DNB CET REVIEW
FOR PRIMARY AND POST DIPLOMA

SECOND EDITION

Dr VAIBHAV BHARAT
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(PG Resident)

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(PG Resident)

KALAM BOOKS
PREFACE

The idea of this book was born because of the lack of any comprehensive book on Primary DNB CET. This is a recall based book and we have not cooked up anything other than some of the missing options. We thank all our readers for the response to our previous edition and to bring forward the mistakes.

When one of my friend asked me if I remember all the things I wrote in this book, I politely replied “No, but I had to when I appeared for the exam”.

The question bank is naturally splitted into paper wise and further into subject wise pattern for the sake of your convenience. Appendixes and charts for the most productive and most repeated topics has been created especially for the last moment studies in addition to saving printing cost of the book.

We have also obtained few data regarding pass percentage of different specialties in DNB final exams by virtue of “Right to information act 2005” and we hope this will be helpful to you in selecting a branch after your selection.

We heartily invite any suggestions, corrections or future papers recalls on mail id dnbctreview@gmail.com

Thanks
Dr Vaibhav Bharat
Dr Ishad Aggarwal
Foreword

This book has been written as a course material for DNB primary (Board speciality) and secondary (Post diploma) entrance examinations conducted by the National board of Examinations, New Delhi. The aim has been to provide students with recently asked multiple choice questions, with additional material to help them to tackle potential new questions.

The references of the text book have been taken from the recent editions of standard national and international medical publications and authors. The material is formatted to be student friendly. The “Appendix” which has been prepared with a great deal of effort by the authors will make things simple for the aspirants to memorize the essentials with least effort.

The DNB exams being conducted by a centralised institution namely the NBE, has proved to be a fair organization giving credit to merit alone. Deserving candidates therefore have better chances of selection. For all these reasons, followed by strict training regulations of NBE, DNB degrees are acquiring greater credibility and international recognition. The need for well written books for the same is being increasingly felt. This book is an attempt in the direction to fulfil these needs.

I will advise all the aspirants to go through the text, especially the charts and not just memorize the answers, as it will help you to compete better for not only this exam but the state entrance, all India entrances also.

The RTI information included regarding the pass percentage will also clear your myths and doubts, increase your confidence and help you to select a stream.

Lastly, I heartily congratulate the authors for their excellent piece of work which will surely help the upcoming aspirants.

Dr. A. K. Sahay
MCh, DNB Plastic surgery
Department of Plastic Surgery
Durgapur steel plant hospital
Durgapur
DEDICATION AND ACKNOWLEDGEMENT

We take this opportunity to dedicate this work and to thank people who directly contributed to this book or indirectly inspire us.

- Almighty
- Our Parents and Grandparents
- Dr Rama Gopal (Our Publisher)
- Dr Harshvardhan Goel
- Ishan Agarwal
- Dr Gaurav Bharat (RDGMC)
- Dr Saurabh Bharat (MPCD, NYFA)
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- Dr Naval Asija (SRMC, NIHFW)
- Dr Parimal Tara (SRMC)
- Dr Pradeep Kumar (SRMC)
- Dr Vivek Dudeja (SRMC, NSCBMC Jabalpur)
- Dr Shweta Raut (GMC Bhopal)
- Dr Triloki Nath Soni (RDGMC)
- Dr Savyasanchi Tiwari (RDGMC)
- Dr Jorawar singh Bhatia (RDGMC)
- Dr Vipan Bhandari (SRMC)
- Dr Prashant Bharadwaj (SRMC)
- Dr Varun Gupta (SRMC, ASCOMS)
- Dr Md Faizer Nazer (SRMC, RPGMC Tanda)
- Dr Abhay Kumar Singh (SRMC, Safdarjung)
- Dr Nirupam Sharan (SRMC, Patel Chest)
- Dr Raghu Prakash (SRMC, CNMC Kolkata)
- Dr Mankeshwar (SRMC)
- Dr Harsha Guduru (SRMC)
- Dr Shailendra Rana (MPCD, AIIMS)
- Dr Brajesh Kumar (SRMC, AMC)
- Dr Deepak Ranjan (SRMC, AMC)
- Dr Diwakar Kumar (SRMC, AMC)
- Dr Saumin Dhaneria (RDGMC Ujjain)
- Dr Ripon Chaudhary (SRMC, MMIMSR)
- Dr Md Sadat Wasim (DSP)
- Dr Ambarish Maiti
  (St. Petersburg State Medical Academy)

- Dr A K Singh
  (Director & HOD General surgery DSP Hospital Durgapur)
- Dr N Kulshreshta
  (Jt Director, General surgery DSP hospital Durgapur)
- Dr A K Sahay (MCh DNB Plastic Surgery DSP hospital Durgapur)
- Department of General surgery, DSP Hospital Durgapur
- Prof. G. Chatterjee (HOD, Dept of Dermatology IPGMER)
- Dr Arghya Prasun Ghosh (Assoc. Prof IPGMER)
- Dr Abhishek De (Asst. Prof IPGMER)
- Dr Sumit Sen (Assoc. Prof IPGMER)
- Department of Dermatology IPGMER
- Dr Brig. Ashok Hooda (AMC)
- Dr Trupti Surana (IPGMER)
- Dr S. Biswas (IPGMER)
- Dr Rakesh Patel (IPGMER)
- Dr Keshab Sil (IPGMER)
- Dr Nandini (IPGMER)
- Dr Nidhi Chaudhary (IPGMER)
- Dr Sampriti Sendur (IPGMER)
- Dr Neha Sori (IPGMER)
- Dr Rakibul Hasan (IPGMER)
- Dr Shatabdi (IPGMER)
- Dr Kunal Mahajan (PGI Rohtak)
- Dr Vivek Sharma (IPGMER)
- Dr Tony (IPGMER)
- Dr Atanu Chakraborty (IPGMER)
- Dr Arindam (IPGMER)
- Dr Shagun Gupta (IPGMER)
- Dr Bhavika Sen (IPGMER)
- Dr Kanishk Sinha (KMC Katihar)
- Dr Shahbaaz (KMC Katihar)
- Dr Sachin Goel (Safdarjung hospital)
- Dr Kumar Abhishek (Sikkim Manipal)
- Dr Romit Saxena (MAMC)
- Dr Mayank Porwal (GRMC Gwalior)
- Dr Vikram Singh Rathore (GRMC)
- Dr Aprajita Pandey (GRMC)
- Dr Madhusudan Ponnesswamy (SGPGI)
- Dr Sushma kataria (RML)
- Dr Darshan (RML)
- Dr Alwin Antony (CMC Kochi)
Ref. No.RTI/PIO-341/2011-13355
To
Dr Vaibhav khare

Dated: July 25 2011

Subject: Information under RTI act 2005

Ref: Your application dated 20/06/2011 received in PIO dated 27/06/2011

Sir,

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This is with reference to your application seeking information under RTI act 2005

Your sincerely

Dinesh chand
Deputy Director & PIO
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A= Appeared, P= Passed, % = Pass percentage

NOTE: Blank cells represent non conduction of examination in that session or no candidate applied
In December 2010 session practical results of some centres for psychiatry and surgical oncology was not declared till the date of reply of this RTI
1. Parathyroid gland develops from?
   (A) Brachial cyst  (B) Brachial cleft
   (C) Brachial pouch  (D) Brachial arch

2. Which is not a type of epiphysis?
   (A) Traction  (B) Atavistic
   (C) Friction  (D) Pressure

3. Which of the following is a Flexor of Abdomen?
   (A) Psoas  (B) Piriformis
   (C) Pectoralis major  (D) External oblique

4. Clavipectoral fascia is not pierced by?
   (A) Medial pectoral nerve  (B) Lateral pectoral nerve
   (C) Cephalic vein  (D) Thoracoacromian artery

5. Boundaries of foramen of Winslow are all EXCEPT:
   (A) Caudate lobe of liver  (B) Inferior vena cava
   (C) Free border of lesser omentum  (D) 4th part of Duodenum

6. During ejaculation, sperms are released from?
   (A) Epididymis  (B) Vas deferens
   (C) Rete testes  (D) Seminal vesicle

7. Lower 1/5th of vagina is formed by?
   (A) Urogenital sinus  (B) Paramesonephric duct
   (C) Mesonephric duct  (D) Mullerian duct

8. True regarding Prostate gland is?
   (A) Only glandular tissue
   (B) Glandular tissue covered with transitional epithelium
   (C) Glandular tissue and fibromuscular stroma
   (D) Entire gland is composed of collagen

9. Uterine artery is a branch of?
   (A) External iliac artery  (B) Internal iliac artery
   (C) Aorta  (D) Inferior vesical artery

10. White fibrocartilage is present in all EXCEPT:
    (A) Acetabular labrum  (B) Intervertebral disc
     (C) Meniscus  (D) Pinna

11. What is denonvillers fascia?
    (A) Between prostate and rectum
    (B) Between rectum and sacral wall
    (C) Between rectal ampulla and perineal body
    (D) Between rectum and urinary bladder
12. Skin over the angle of mandible is supplied by?
   (A) Trigeminal nerve  
   (B) Posterior rami of C2, C3, C4  
   (C) Cervical spinal nerve (C2-C3)  
   (D) Cervical branch of mandibular nerve

13. Appendix of testis is derived from?
   (A) Paramesonephric duct  
   (B) Mesonephric duct  
   (C) Hind gut  
   (D) Cloaca

14. Infection of CNS spreads in inner ear through?
   (A) Cochlear Aqueduct  
   (B) Endolymphatic sac  
   (C) Vestibular Aqueduct  
   (D) Hyrtle’s fissure

15. Klumpke’s paralysis involves?
   (A) C5 C6  
   (B) C6 C7  
   (C) C7 T1  
   (D) C8 T1

16. Craniosacral outflow is mediated by?
   (A) Sympathetic postganglionic fibers  
   (B) Sympathetic preganglionic fibers  
   (C) Parasympathetic postganglionic fibers  
   (D) Parasympathetic preganglionic fibers

17. Chromatin condensation occurs in?
   (A) Prophase  
   (B) Metaphase  
   (C) Anaphase  
   (D) Telophase

18. Superficial palmar arch is at the level of?
   (A) Proximal border of extended thumb  
   (B) Distal border of extended thumb  
   (C) Proximal transverse palm crease  
   (D) Distal transverse palm crease

19. Peripheral and central chemoreceptors respond to?
   (A) Increased arterial pH  
   (B) Increased arterial Oxygen  
   (C) Increased arterial CO₂  
   (D) Decreased arterial CO₂

20. True about oligodendrocytes is?
   (A) Forms Myelin sheath  
   (B) Forms blood brain barrier  
   (C) Secretes CSF  
   (D) All of the above

21. Predominant site of erythropoiesis during 6th month of gestation is?
   (A) Yolk sac  
   (B) Liver  
   (C) Bone marrow  
   (D) Thymus

22. During under water diving the main danger is due to?
   (A) Oxygen and nitrogen  
   (B) CO₂ and nitrogen  
   (C) Oxygen only  
   (D) Nitrogen only
(23) Maximum filling of ventricles is seen in?
(A) Protodiastole  (B) Isovolumetric relaxation
(C) Ventricular phase of diastole  (D) Atrial contraction

(24) Serotonin secreting cell in brain is?
(A) Raphe nucleus  (B) Enterochromaffin cells
(C) Magnan cells  (D) Betz cells

(25) True about thymus gland is?
(A) Size increases after puberty  (B) Red and white cell mass
(C) Consists of Hassalls corpuscles  (D) Forms immunoglobulins

(26) Gammaglobulins are formed by?
(A) B cells  (B) T cells
(C) Plasma cells  (D) Liver

(27) Androgen binding protein is secreted by?
(A) Pituitary  (B) Liver
(C) Sertoli cells  (D) Leydig cells

(28) MIS is secreted by?
(A) Sertoli cell  (B) Leidig cell
(C) Supporting cells  (D) Semeniferous tubules

(29) Meileu interior means?
(A) ECF  (B) ICF
(C) Plasma  (D) Lymph

(30) Which of the following decreases appetite?
(A) Orexins  (B) Neuropeptide Y
(C) Ghrelin  (D) Leptin

(31) Orexins are implicated in all EXCEPT:
(A) Wakefulness  (B) Sexual behaviour
(C) Appetite  (D) Alzheimer’s disease

(32) What happens when carotid sinus is pressed?
(A) Heart rate decreases, peripheral resistance increases
(B) Heart rate and peripheral resistance decreases
(C) Peripheral resistance and contractility decreases
(D) Peripheral resistance and contractility increases

(33) Time duration required to generate an action potential is?
(A) Threshold  (B) Rheobase
(C) Chronaxie  (D) Refractory period

(34) Satiety center is located at?
(A) Ventromedial nucleus  (B) Lateral hypothalamus
(C) Supra median nucleus  (D) Preoptic nucleus
(35) **Potassium in which compartment is responsible for cardiac and neural function?**

(A) Intracellular  
(B) Extracellular  
(C) Intravascular  
(D) Extravascular

(36) **All are correct about stomach EXCEPT:**

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

(37) **Which of the following has highest pH?**

(A) Bile  
(B) Pancreatic juice  
(C) Saliva  
(D) Gastric juice

(38) **Band not covered by actin filament is?**

(A) H band  
(B) I band  
(C) M band  
(D) Z band

(39) **Free fatty acid is transported in blood by?**

(A) Albumin  
(B) Globulin  
(C) Fibrinogen  
(D) Carnitine

(40) **Iodine uptake into thyroid gland is an example of?**

(A) Primary active transport  
(B) Secondary active transport  
(C) Facilitated diffusion  
(D) Endocytosis

---

**BIOCHEMISTRY**

(41) **Alpha oxidation of fatty acid takes place in?**

(A) ER  
(B) Golgi apparatus  
(C) Mitochondria  
(D) Peroxisomes

(42) **Amino acid used in Carnitine synthesis is?**

(A) Alanine  
(B) Lysine  
(C) Arginine  
(D) Tyrosine

(43) **Chylomicrons core is formed by?**

(A) Triglyceride  
(B) Triglyceride and Cholesterol  
(C) Triglyceride, Cholesterol and Phospholipids  
(D) Free fatty acids

(44) **Vitamin A is present in all EXCEPT:**

(A) Sunflower seeds  
(B) Egg  
(C) Milk  
(D) Tomato
(45) Which hormone synthesized from Tyrosine?
(A) Calcitriol  (B) Calcitonin  
(C) Thyroxin (D) Cortisol

(46) Activator of enzyme sulfite oxidase is?
(A) Copper  (B) Zinc  
(C) Molybdenum (D) Iron

(47) Cofactor involve in sulphur containing amino acid metabolism is?
(A) Folic acid (B) Biotin  
(C) Vitamin B 1 (D) Vitamin B 12

(48) Glycogen phosphorylase coenzyme associated is?
(A) Thiamine pyrophosphate (B) Tetrahydrofolate  
(C) Flavin mononuleotide (D) Pyridoxal phosphate

(49) Enzyme that is responsible for unwinding of DNA is?
(A) Ligase (B) DNA primase  
(C) Helicase (D) DNA polymerase

(50) DNA replication occurs in which phase of cell cycle?
(A) G1 phase (B) S phase  
(C) G2 phase (D) M phase

(51) Inheritance of Familial Hypercholesterolemia is?
(A) Autosomal dominant (B) Autosomal recessive  
(C) X Linked dominant (D) X Linked recessive

(52) PFK is allosterically activated by?
(A) Fructose 1, 6 bisphosphate (B) Fructose 2, 6 bisphosphate  
(C) Phosphoenolpyruvate (D) Pyruvate

(53) Which one of the following enzymes is obtained from Thermophilus aquaticus bacterium which is heat stable and used in PCR at high temperature?
(A) DNA polymerase III (B) Endonuclease  
(C) Taq polymerase (D) DNA gyrase

(MICROBIOLOGY)

(54) Which of the following gives positive test with both weil felix and OX 19?
(A) Spotted fever (B) Scrub typhus  
(C) Epidemic typhus (D) None of the above

(55) Number of nucleus present in mature cyst of E. Histolytica is?
(A) 1 (B) 2  
(C) 4 (D) 8
(56) Gonorrhea can be identified by?
(A) Growth on MacConkey medium  (B) Growth at 37° C
(C) By the fermentation of glucose  (D) Growth in 45%/60% bile

(57) All are capsulated EXCEPT:
(A) Klebsiella pneumonia  (B) Hemophilus influenzae
(C) Bacillus anthracis  (D) Escherichia coli

(58) Food poisoning in canned food is caused by?
(A) Staphylococcus  (B) Salmonella
(C) Clostridium botulinum  (D) Bacillus cereus

(59) Food poisoning that does not presents within 6 hours is due to?
(A) Staphylococcus  (B) Salmonella
(C) Clostridium botulinum  (D) Bacillus cereus

(60) Which of the following is a strict aerobic bacterium?
(A) Staphylococcus aureus  (B) E. Coli
(C) Streptococcus pneumoniae  (D) Pseudomonas

(61) Atypical pneumonia is caused by all EXCEPT:
(A) Klebsiella  (B) Adeno virus
(C) Chlamydia  (D) Hemophilus

(62) Antigenic variation is seen in all EXCEPT:
(A) Influenza type A  (B) Influenza type B
(C) Influenza type C  (D) None of the above

(63) Which of the following acts as intermediate host of malaria parasite?
(A) Culex  (B) Female anopheles
(C) Thromboculid mite  (D) Human

(64) Schizonts are not seen in peripheral smear in which type of malaria?
(A) P. Vivax  (B) P. Falciparum
(C) P. Ovale  (D) P. Malariae

(65) Most common cause of dysentery in adults is?
(A) Cryptoparvum  (B) Giardia
(C) Strongoloides  (D) Entamoeba histolytica

(66) Cause of Meliodosis is?
(A) Burkholderia mallei  (B) Burkholderia pseudomallei
(C) Burkholderia cepacia  (D) None

(67) Localized myogenic infection is caused by which bacteria?
(A) Staphyloccocus  (B) Clostridium
(C) Cornybacterium  (D) Streptococcal

(68) Which of the following parasite causes myocarditis?
(A) Trichenella spiralis  (B) Trichuris trichura
(C) Ancyclostoma duodenale  (D) Tienea solium
(69) Which of the following parasite can enters through intact skin?
(A) Giardia  (B) Whip worm
(C) Strongyloids  (D) Trichinella

(70) Chagas disease is caused by?
(A) Trypanosoma cruzi  (B) Trypanosoma gambiense
(C) Trypanosoma brucei  (D) Leishmania donovani

(71) All are dimorphic fungi EXCEPT:
(A) Histoplasma  (B) Paracoccidiodes
(C) Cryptococcus  (D) Blastomyces

(72) Most common cause of bacillary angiomatosis is?
(A) B. quintana  (B) B. bacilliformis
(C) B. hensae  (D) B. elizabethi

(73) Metachromatic granules are seen in?
(A) Corynbacterium  (B) E.coli
(C) Yersinia  (D) Pseudomonas

(74) Skin test useful in Hydatid disease is?
(A) Casoni’s test  (B) Schick test
(C) Patch test  (D) Dick’s test

(75) Acute Meningoencephalitis is caused by?
(A) Acantamoeba  (B) Nageleria
(C) Meningococcus  (D) Balmuthia

(76) Most common cause of pyomyositis is?
(A) Streptococcus pyogenes  (B) Pseudomonas
(C) Staphylococcus Aureus  (D) E. Coli

(77) Innate immunity is stimulated by which part of bacteria?
(A) Carbohydrate sequence in cell wall  (B) Flagella
(C) Bacterial cell membrane  (D) Nucleus

(78) Continuous cell line for viruses not present for?
(A) Vero  (B) Hep2
(C) WT-38  (D) Hela

(79) Annexin V is associated with?
(A) Necrosis  (B) Apoptosis
(C) Atherosclerosis  (D) Inflammation
(80) Which of the following has a direct role in apoptosis?
(A) Nitric oxide  (B) Adenylcyclase
(C) cAMP  (D) Cytochrome C

(81) Fixative used for bone histopathology is?
(A) 10% formalin  (B) Normal saline
(C) Rectified spirit  (D) Nothing

(82) Paraneoplastic syndrome not seen in renal cell cancer is?
(A) Acanthosis nigricans  (B) Amyloidosis
(C) Polycythemia  (D) SLE

(83) Which of the following is a chemotactic factor?
(A) Prostaglandins  (B) Prostacyclins
(C) Thromboxane  (D) Leukotrienes

(84) All are markers of Mantle cell lymphoma EXCEPT:
(A) CD 5  (B) CD 19
(C) CD 20  (D) CD 23

(85) Adding glucose to stored blood causes?
(A) Prevent hemolysis  (B) Gives nutrition to cells
(C) Increase acidosis of blood  (D) Prevent Hyperkalemia

(86) Gastric carcinoma is associated with all EXCEPT:
(A) Inactivation of p53  (B) Over expression of C-erb
(C) Over expression of C-met  (D) Activation of RAS

(87) Complete wound strength is gained by?
(A) Never regained  (B) 1 month
(C) 6 months  (D) 1 year

(88) Chromosomal translocation seen in CML is?
(A) 2:8  (B) 8:14
(C) 9:22  (D) 15:17

(89) Increased PT and Normal PTT are found in?
(A) Von Willibrand’s disease  (B) Factor 7 deficiency
(C) Factor 8 deficiency  (D) Thrombin deficiency

(90) Which of the following is a chemokines?
(A) Leukotriene A4  (B) IL-8
(C) C5  (D) C3

(91) Asteroid bodies are seen in?
(A) Sarcoidosis  (B) Syphilis
(C) Chromoblastomycosis  (D) Sporotrichosis

(92) Karyotype in Klinefelter’s syndrome is?
(A) 47 XXY  (B) 45XO
(C) 46XXX  (D) 45XXX
(93) **Most common cause of hereditary spherocytosis?**  
(A) Spectrin  (B) Glycophorin  
(C) Ankyrin  (D) Band 4

---

**PHARMACOLOGY**

(94) **Microtubule formation is inhibited by?**  
(A) Paclitaxel  (B) Vincristine  
(C) Etoposide  (D) Irinotecan

(95) **Atropine is most sensitive to?**  
(A) Mucous and pharyngeal secretions  (B) Heart  
(C) Pupil  (D) GI tract motility

(96) **Which is not an effect of atropine?**  
(A) Rise of body temperature  (B) Decreased salivary secretion  
(C) Bradycardia  (D) Increased A-V conduction

(97) **In diabetes mellitus with increased HbA1C, drug that is not used in treatment is?**  
(A) Sulfonylureas  (B) Acarbose  
(C) Biguinides  (D) Thiazolidinediones

(98) **HbA1C is decreased most by?**  
(A) Biguanides  (B) Sulfonylureas  
(C) Thiazolidinediones  (D) Acarbose

(99) **Drug used in post prandial sugar control is?**  
(A) Alfa glucosidase  (B) Biguinides  
(C) Sulfonylurea  (D) Repaglinide

(100) **Drug that can cause hypertrophic pyloric stenosis is?**  
(A) Tertacyclin  (B) Erythromycin  
(C) Ampicillin  (D) Rifampicin

(101) **Drug that should not be given with Apomorphine is?**  
(A) Dopamine agonist  (B) Spironolactone  
(C) Ondansetron  (D) Aspirin

(102) **In Anaphylactic shock epinephrine given by which route?**  
(A) Intravenous route  (B) Oral  
(C) Subcutaneous  (D) Intramusular

(103) **Which of the following is an example of endogenous/Physiological Antagonism?**  
(A) Heparin-Protamine  (B) Prostacycline-Thromboxone  
(C) Adrenaline-Phenoxybenzamine  (D) Physiostigmine-Acetylcholine
(104) In treatment of shock Dobutamine is preferred over Dopamine because?
(A) It causes less arrhythmia  
(B) It causes less renal vasodilatation  
(C) It causes less coronary vasoconstriction  
(D) All the above

(105) Long acting insulin is?
(A) Insulin glargine  
(B) Insulin Lispro  
(C) Insulin aspart  
(D) Insulin glulisine

(106) Pseudolymphoma is caused by?
(A) Thiazides  
(B) Penicillin  
(C) Dapsone  
(D) Sulfonamides

(107) Side effects of isoniazid are all EXCEPT:
(A) Hepatitis  
(B) Optic neuritis  
(C) Peripheral Neuropathy  
(D) Thrombocytopenia

(108) Drug that inhibits cell wall synthesis is?
(A) Tetracyclins  
(B) Penicillins  
(C) Aminoglycosides  
(D) Chloramphenicol

(109) Which of the following Tetracycline can be used in renal failure without dose adjustment?
(A) Oxytetracyclin  
(B) Doxycyclin  
(C) Demiclocyclin  
(D) Tetracycline

(110) Which is not an alkaloid?
(A) Morphine  
(B) Neostigmine  
(C) Emetine  
(D) Atropine

(111) The effect of morphine which has least tolerance is?
(A) Analgesia  
(B) Respiratory depression  
(C) Constipation  
(D) Bradycardia

(112) Which of the following is a bacteriostatic Antitubercular drug?
(A) Streptomycin  
(B) Ethambutol  
(C) Isoniazid  
(D) Rifampicin

(113) Drug used in prophylaxis of meningococcal meningitis is?
(A) Ciprofloxacin  
(B) Rifampicin  
(C) Penicillin  
(A) Gentamycin

(114) All are true about rifampicin EXCEPT:
(A) Microsomal enzyme inducer  
(B) Used in treatment of meningococcal meningitis  
(C) May cause OCP failure  
(D) Bactericidal in nature

(115) Imatinib primarily acts on?
(A) BCR-ABL  
(B) Tyrosine kinase  
(C) PGDFR  
(D) None
(116) Drugs used in the treatment of obesity is/are?
(A) Orlistat  (B) Sibutramine  
(C) Rimonabant  (D) All of the above

(117) Rifampicin acts by?
(A) DNA dependent RNA polymerase  (B) RNA dependent DNA polymerase  
(C) Mycolic acid inhibition  (D) Mycolic acid incorporation defects

(118) All are true about estrogen EXCEPT:
(A) Causes cholestasis  (B) Used in treatment of gynecomastia  
(C) Used in HRT  (D) Increased risk of breast cancer

(119) Most hepatotoxic is?
(A) Olgendralone  (B) Methandrostenolone  
(C) Stanozolol  (D) Nandrolone

(120) Fastest acting antithyroid drug is?
(A) Potassium iodide  (B) Propylthiouracil  
(C) Carbimazole  (D) Cholestyramine

(121) All of the following are topical steroids EXCEPT:
(A) Hydrocortisone valerate  (B) Fluticasone propionate  
(C) Triamcinolone  (D) Prednisolone

(122) Cidofovir can be used for?
(A) Respiratory papillomatosis  (B) Herpes simplex  
(C) Herpes zoster  (D) All of the above

(123) Thanatology deals with?
(A) Death  (B) Snakes  
(C) Poison  (D) Fingerprints

(124) Fish tailing of margins in stab wound is seen with?
(A) Single edged knife  (B) Double edged knife  
(C) Bayonet  (D) None

(125) Sin of Gomorrah is also known as?
(A) Anal coitus  (B) Oral coitus  
(C) Lesbianism  (D) Bestiality
(1) ANSWER: (C) Brachial pouch
REF: Langman’s medical embryology 9th edition page 384-385
See APPENDIX-6 for “BRACHIAL ARCHES”
“Inferior parathyroid develops from 3rd brachial pouch while superior parathyroid develops from 4th brachial pouch”

(2) ANSWER: (C) Friction
REF: Human osteology by BDC 1st edition page 5-6

<table>
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<tr>
<th>Epiphysis</th>
<th>Definition/ Function</th>
<th>Example</th>
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<td>Pressure epiphyses</td>
<td>Takes part in transmission of weight</td>
<td>Head of femur, condyles of tibia, lower end of radius</td>
</tr>
<tr>
<td>Traction epiphyses</td>
<td>Provide attachment to more than 1 tendon</td>
<td>Trochanter of femur, tubercles of humerus, mastoid process.</td>
</tr>
<tr>
<td>Atavistic epiphyses</td>
<td>Phylogenetically an independent bone which in man becomes fused to another bone</td>
<td>Coracoid process of scapula, posterior tubercle of talus, ostrigonum.</td>
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<tr>
<td>Aberrant epiphyses</td>
<td>Not always present</td>
<td>Head of metacarpal, base of other metacarpal</td>
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(3) ANSWER: (D) External oblique
REF: BDC 4th edition vol-2 page 201

**ACTIONS OF THE MUSCLES OF ANTERIOR ABDOMINAL WALL:**

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<thead>
<tr>
<th>Support of viscera</th>
<th>Due to the tone of the oblique muscles especially the internal oblique</th>
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<tr>
<td>Expulsive acts</td>
<td>Obliques assisted by the transversus</td>
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<tr>
<td>Forceful expiratory acts</td>
<td>External oblique</td>
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<tr>
<td>Flexion of trunk</td>
<td>Rectus abdominis</td>
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<tr>
<td>Lateral flexion of trunk</td>
<td>Oblique muscles</td>
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<tr>
<td>Rotation of trunk</td>
<td>Combined action of external oblique with opposite internal oblique</td>
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**ACTIONS OF MUSCLES OF POSTERIOR ABDOMINAL WALL:**

<table>
<thead>
<tr>
<th>Psoas major</th>
<th>With iliacus it acts as a powerful flexor of the hip joint as in raising the trunk from recumbent to sitting posture</th>
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<tbody>
<tr>
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<td>Helps in maintaining the stability of hip joint</td>
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<tr>
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<td>Lateral flexion of trunk, Medial rotation of hip</td>
</tr>
<tr>
<td>Psoas minor</td>
<td>Weak flexor of trunk</td>
</tr>
<tr>
<td>Iliacus</td>
<td>With psoas it flexes the hip joint</td>
</tr>
<tr>
<td>Quadratus lumborum</td>
<td>Fixes the last rib during inspiration so that contraction of the diaphragm takes more effectively</td>
</tr>
<tr>
<td></td>
<td>When pelvis is fixed it causes lateral flexion of vertebral column</td>
</tr>
<tr>
<td></td>
<td>Extend the lumbar vertebral column</td>
</tr>
</tbody>
</table>
(4) **ANSWER: (A) Medial pectoral nerve**

REF: BDC 4th edition vol 1 page 46

**CLAVIPECTORAL FASCIA:**
- Extent: from clavicle above to axillary fascia below.
- Upper part splits to enclose subclavius muscle while lower part splits to enclose pectoralis minor muscle.
- It helps to pull up axillary fascia.
- Upper thickened part is called the costocoracoidal ligament.

**Structures piercing clavipectoral fascia:**
- Lateral pectoral nerve
- Cephalic vein
- Thoracoacromial vessels
- Lymphatics from the breast and pectoral region

(5) **ANSWER: (D) 4th part of Duodenum**

REF: Gray’s anatomy 39th edition, page 3136

Repeat from June 2009 and June 2011

In human anatomy, the omental foramen (Epiploic foramen, foramen of Winslow, or uncommonly aditus) is the passage of communication, or foramen, between the greater sac (general cavity of the abdomen), and the lesser sac.

**Borders of Epiploic foramen:**
It has the following borders:
- **Anterior:** the free border of the lesser omentum, known as the hepatoduodenal ligament. This has two layers and within these layers are the common bile duct, hepatic artery, and hepatic portal vein.
- **Posterior:** the peritoneum covering the inferior vena cava
- **Superior:** the peritoneum covering the caudate lobe of the liver
- **Inferior:** the peritoneum covering the commencement of the duodenum and the hepatic artery, the latter passing forward below the foramen before ascending between the two layers of the lesser omentum.
- **Left lateral:** Gastrosplenic ligament and Splenorenal ligament.

(6) **ANSWER: (A) Epididymis**


Repeat from December 2010

Sperms are formed in seminiferous tubules and stored in epididymis till ejaculation. The sequence of sperm movement from within the testis to urethra is as follows

Seminiferous tubules ——— Straight tubule ——— Rete testis ——— Efferent tubule ——— Epididymis ——— Vas deferens ——— Ejaculatory ducts ——— Urethra
(7) ANSWER: (A) Urogenital sinus
REF: Gray’s anatomy 39th ed p. 1385, Blueprints Obstetrics and Gynecology by Tamara L. Callahan, Aaron B. Caughey Page 140, Pediatric and adolescent gynecology by Marc R. Laufer, Donald Peter Goldstein Page 337
See APPENDIX-5 for “Adult derivatives and vestigial remains of embryonic urogenital structures”

Repeated from June 2011
- The sinovaginal bulb originates from urogenital sinus.
- Distally sinovaginal bulb and proximally overgrowth of Mullerian duct at Mullerian tubercle results in formation of vaginal plate which on canalization forms upper and lower vagina respectively.
- The first and second portions of Mullerian duct forms the fimbriae and the fallopian tubes while distal segment forms the uterus and upper vagina.
- Distal most portion of sinovaginal bulb forms the Hymen.

(8) ANSWER: (C) Glandular tissue and fibromuscular stroma
Indirect repeat from December 2010
The anterior part of the prostate is composed mainly of fibromuscular stroma, which is continuous with detrusor fibers. Toward the apex of the gland, this fibromuscular tissue blends with striated muscle from the levator.
Puboprostatic ligaments also blend with this area.

(9) **ANSWER: (B) Internal iliac artery**

REF: BDC 4th edition page 387

**BRANCHES OF THE INTERNAL ILIAC ARTERY:**

- **branches of anterior division:** (6 in males, 7 in females)
  1. Superior vesical
  2. Obturator
  3. Middle rectal
  4. Inferior vesical (in males)
  5. Vaginal artery (in females)
  6. Inferior gluteal
  7. Internal pudendal
  8. Uterine artery (in females)
- **Branches of posterior division:**
  1. Iliolumbar
  2. Two lateral sacral
  3. Superior gluteal artery

**BRANCHES OF ABDOMINAL AORTA:**

- **Ventral branches:**
  1. Celiac trunk
  2. Superior mesenteric artery
  3. Inferior mesenteric artery
- **Lateral branches:**
  1. Inferior phrenic artery
  2. Middle suprarenal artery
  3. Renal arteries
  4. Testicular or ovarian arteries.
- **Dorsal arteries:**
  1. Lumbar arteries – 4 pairs.
  2. Median sacral artery (unpaired)

(10) **ANSWER: (D) Pinna**


See APPENDIX-2 for “Types of cartilage”

“Pinna or auricle is formed of elastic cartilage not fibrocartilage”

(11) **ANSWER: (A) Between prostate and rectum**

REF: BDC 4th edition vo. l 2 page 380

**SUPPORTS OF RECTUM:**

1. **Rectovesical fascia of Denonviller’s:** Extends from rectum behind to the seminal vesicles and prostate in front.
2. **Fascia of waldeyer:** Attaches lower part of rectal ampulla to sacrum, it is the condensation of pelvic fascia and contains superior rectal vessels and lymphatics
3. Pelvic floor and levator ani
4. Lateral ligament of rectum
5. Pelvic peritoneum.

(12) **ANSWER: (C) Cervical spinal nerve (C2-C3)**
 **REF: Gray’s anatomy 39th edition page 515**

The *Great auricular nerve* forms part of the cervical plexus and is derived from the anterior primary rami of the *second and third cervical spinal nerves*. It passes up from the neck, lying on sternocleidomastoid, towards the angle of the jaw, and supplies much of the lower part of the auricle of the ear, and skin overlying the parotid gland.

![Diagram of the head and neck](image)

(13) **ANSWER: (A) Paramesonephric duct**
 **REF: Langman’s medical embryology 9th ed p. 340–341**

See APPENDIX-5 for “ADULT DERIVATIVES AND VESTIGIAL REMAINS OF EMBRYONIC UROGENITAL STRUCTURES”

(14) **ANSWER: (A) Cochlear aqueduct**
 **REF: Dhingra 4th edition page 10**

Indirect repeat from December 2010

*Cochlear aqueduct*: connects scala tympani to CSF
Endolymphatic sac: Endolymphatic duct is formed by the union of two ducts, one each from the saccule and the utricle. It passes through the vestibular aqueduct. Its terminal part is dilated to form endolymphatic sac which lies between the two layers of dura on the posterior surface of the petrous bone. Endolymphatic sac is surgically important. It is exposed for drainage or shunt operation in Meniere’s disease.

Hyrtle’s fissure: Hyrtle’s fissure also called tympanomeningeal hiatus; it is an embryonic remnant that connects CSF space to middle ear just anterior and inferior to the round window. It runs parallel to cochlear aqueduct. It can be the source of congenital CSF otorrhoea or meningitis from middle ear infections. Normally it gets obliterated.

(15) ANSWER: (D) C8 T1
REF: BDC 4th edition vol 1 Page 53
KLUMPKE’S PARALYSIS:
Site of injury: lower trunk of brachial plexus
Cause of injury: undue abduction of the arms
Nerve roots involved: mainly T1 partly C8
Muscles paralyzed:
• Intrinsic muscles of the hand (T1)
• Ulnar flexors of wrist and fingers (C8)
Disability:
1. Claw hand
2. Horner’s syndrome
3. Vasomotor changes
4. Trophic changes
5. Cutaneous anesthesia on the ulnar border of hand and forearm

(16) ANSWER: (D) Parasympathetic preganglionic fibers
The **parasympathetic nervous system** (PSNS) is one of the two main divisions of the autonomic nervous system (ANS). The ANS is responsible for regulation of internal organs and glands, which occurs unconsciously.

**Functions:**
To be specific, the parasympathetic system is responsible for stimulation of “rest-and-digest” activities that occur when the body is at rest, including sexual arousal, salivation, lacrimation (tears), urination, digestion, and defecation. Its action is described as being complementary to that of one of the other main branches of the ANS, the sympathetic nervous system, which is responsible for stimulating activities associated with the fight-or-flight response.

A useful acronym to summarize the functions of the parasympathetic nervous system is **SLUDG** *(salivation, lacrimation, urination, digestion, and gastric emptying)*

**Physical location:**
Parasympathetic nerve fibers arise from the central nervous system with the **S2, S3, and S4** spinal nerves and from the third, seventh, ninth, and tenth cranial nerves. Because of its location, the parasympathetic system is commonly referred to as having **“craniosacral outflow”**, which stands in contrast to the sympathetic nervous system, which is said to have “thoracolumbar outflow”.

The parasympathetic nerves that arise from the S2, S3, and S4 spinal nerves are commonly referred to as the **pelvic splanchnic nerves** or the “nervi erigentes”.

**Pelvic Splanchnic Control:**
The pelvic splanchnic nerves, S2-4, work in tandem to innervate the pelvic viscera. Unlike in the cranium, where one PSN was in charge of one particular tissue or region, for the most part the **pelvic splanchnics each contribute fibers to pelvic viscera by first travelling to one or more plexuses before being dispersed to the target tissue**. These plexuses are composed of mixed autonomic nerve fibers (PSN and SN) and include the vesical, prostatic, rectal, uterovaginal, and inferior hypogastric plexus. The **preganglionic neurons** in these neurons do not synapse in named ganglion as in the cranium but rather in the walls of the tissues or organs that they innervate.

(17) **ANSWER:** (A) Prophase
**REF:** Langman’s Medical Embryology 9th edition page 5
“Throughout prophase the chromosomes continue to condense, shorten, and thicken, but only at prometaphase do the chromatids become distinguishable”

(18) ANSWER: (B) Distal border of extended thumb

The superficial palmar arch is an anastomosis fed mainly by the ulnar artery. About a third of the superficial palmar arches are formed by the ulnar alone; a further third are completed by the superficial palmar branch of the radial artery; and a third by the arteria radialis indicis, a branch of arteria princeps pollicis or the median artery.

If one were to fully extend the thumb, the superficial palmar arch would lie approximately at the level of a line drawn from the distal border of the thumb across the palm. The superficial palmar arch is more distal than the deep palmar arch.

(19) ANSWER: (C) Increased arterial CO2
REF: Ganong’s 22nd ed chapter 36, Guyton and Hall 11th edition page 211 & 518

Chemoreceptors act principally to detect variation of the oxygen concentration in the arterial blood, whilst also monitoring arterial carbon dioxide and pH.
The chemoreceptors are chemosensitive cells sensitive to oxygen lack, carbon dioxide excess, and hydrogen ion excess. They are located in several small chemoreceptor organs about 2 millimeters in size (two carotid bodies, one of which lies in the bifurcation of each common carotid artery, and usually one to three aortic bodies adjacent to the aorta). Carotid bodies and aortic bodies detect changes primarily in oxygen. They also sense increases in CO2 partial pressure and decreases in arterial pH, but to a lesser degree than for O2.

(20) ANSWER: (A) Forms Myelin sheath
“The myelin sheath is produced by oligodendrocytes in the central nervous system and by Schwann cells in the peripheral nervous system”
• Oligodendrocytes myelinate CNS axons and are most commonly seen as intrafascicular cells in myelinated tracts.
• Oligodendrocytes may enclose up to 50 axons in separate myelin sheaths: the largest calibre axons are usually ensheathed on a 1:1 basis.

(21) ANSWER: (B) Liver
REF: Guyton, 6th edition, page 420
Production of Red Blood Cells: In the early weeks of embryonic life, primitive, nucleated red blood cells are produced in the yolk sac. During the middle trimester of gestation, the liver is the main organ for production of red blood cells, but reasonable numbers are also produced in the spleen and lymph nodes. Then, during the last month or so of gestation and after birth, red blood cells are produced exclusively in the bone marrow.

(22) ANSWER: (A) Oxygen and nitrogen
REF: Ganong’s 22nd ed chapter 37
Indirect repeat from December 2010
POTENTIAL PROBLEMS ASSOCIATED WITH EXPOSURE TO INCREASED BAROMETRIC PRESSURE UNDER WATER:

Oxygen toxicity
Lung damage, Convulsions
Nitrogen narcosis
Euphoria, Impaired performance
High-pressure nervous syndrome
Tremors, Somnolence
Decompression sickness
Pain, Paralyses
Air embolism
Sudden death

(23) ANSWER: (C) Ventricular phase of diastole
Since the ventricles are 80% full by the first quarter of diastole, this is referred to as rapid ventricular filling.

<table>
<thead>
<tr>
<th>Valvular events</th>
<th>Cardiac events</th>
<th>ECG</th>
<th>JVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening of A-V valve</td>
<td>End of isovolumetric relaxation phase</td>
<td>End of T wave</td>
<td>V-Y descent</td>
</tr>
<tr>
<td>Closure of A-V valve</td>
<td>End of diastole or beginning of isovolumetric contraction</td>
<td>Later half of ‘r’ wave</td>
<td>End of ‘X’ descent</td>
</tr>
<tr>
<td>Opening of semilunar valve</td>
<td>End of isovolumetric contraction</td>
<td>S-T segment</td>
<td>Peak of ‘c’ wave</td>
</tr>
<tr>
<td>Closure of semilunar valve</td>
<td>Beginning of isovolumetric relaxation, beginning of diastole</td>
<td>Later half of ‘t’ wave</td>
<td></td>
</tr>
</tbody>
</table>

(24) **ANSWER: (A) Raphe nucleus**

REF: Molecular and cellular signalling By Martin Beckerman page 292

“Serotonin producing cells in CNS are organised in clusters called the raphe nucleus”

Serotonin (5-hydroxytryptamine; 5-HT) is present in highest concentration in blood platelets and in the gastrointestinal tract, where it is found in the enterochromaffin cells and the myenteric plexus. Lesser amounts are found in the brain and in the retina.

(25) **ANSWER: (C) Consists of Hassalls corpuscles**


- Size of thymus is maximum at puberty and decreases afterwards
- Red pulp and white pulp is a part of spleen not thymus
- Thymus plays a role in T cell maturation not immunoglobulins formation

**THYMUS:**

The thymus is composed of two identical lobes and is located anatomically in the anterior superior mediastinum, in front of the heart and behind the sternum.

The thymus is largest and most active during the neonatal and pre-adolescent periods. By the early teens, the thymus begins to atrophy and thymic stroma is replaced by adipose (fat) tissue. Nevertheless, residual T lymphopoiesis continues throughout adult life. The thymus reaches maximum weight (20 to 37 grams) by the time of puberty.

<table>
<thead>
<tr>
<th>Age</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>birth</td>
<td>about 15 grams;</td>
</tr>
<tr>
<td>puberty</td>
<td>about 35 grams</td>
</tr>
<tr>
<td>twenty-five years</td>
<td>25 grams</td>
</tr>
<tr>
<td>sixty years</td>
<td>less than 15 grams</td>
</tr>
<tr>
<td>seventy years</td>
<td>as low as 5 grams</td>
</tr>
</tbody>
</table>
Histology:
Histologically, the thymus can be divided into a central medulla and a peripheral cortex which is surrounded by an outer capsule.

Cortex:
The cortical portion is mainly composed of lymphoid cells, supported by a network of finely-branched epithelial reticular cells. The cortex is the location of the earliest events in thymocyte development, where T cell receptor gene rearrangement and positive selection takes place.

Medulla:
In the medullary portion, the reticulum is coarser than in the cortex, the lymphoid cells are relatively fewer in number, and there are found peculiar nest-like bodies, the concentric corpuscles of Hassall. These concentric corpuscles are composed of a central mass, consisting of one or more granular cells, and of a capsule formed of epithelioid cells

26) ANSWER: (C) Plasma cells
REF: Vasudevan Biochemistry page 307, Ganong's 22nd ed chapter 27
On the basis of electrophoresis Globulins are classified as:
- Alpha 1 and 2 globulins: e.g. – Glycoprotein’s and hormone binding globulins
- Beta globulins:
- Gamma globulins: Immunoglobulin’s are a type of gamma globulins and they are produced by plasma cells

27) ANSWER: (C) Sertoli cells
REF: Ganong’s 22nd ed chapter 23
The Sertoli cells secrete:
- Androgen-binding protein (ABP)
- Inhibin
- MIS
- They contain aromatase (CYP19), the enzyme responsible for conversion of androgens to estrogens, and they can produce estrogens
Also know: The leydig cells secrete testosterone

28) ANSWER: (A) Sertoli cell
REF: Ganong’s 22nd ed chapter 23
See previous question for explanation

29) ANSWER: (A) ECF
REF: Textbook of biochemistry by G. P. Talwar and I. M. Srivastava page 276
“La fixit de milieu interior”, was coined by Claude Bernard and it means that in spite of ever changing external environment the internal environment remains same.
In our body cells are bathed in the interstitial fluid which constitutes the internal milieu. Since interstitial fluid is part of ECF, best possible answer is ECF

30) ANSWER: (D) Leptin
REF: Harrison’s 18th ed chapter 77
FACTORS AFFECTING APPETITE

<table>
<thead>
<tr>
<th>INCREASE</th>
<th>DECREASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Neuropeptide –Y</td>
<td>• alpha-MSH (melanin stimulating hormone)</td>
</tr>
<tr>
<td>• MCH (melanin concentrating hormone)</td>
<td>• CART (cocaine and amphetamine related transcript)</td>
</tr>
<tr>
<td>• AGRP (Agouti related peptide)</td>
<td>• GLP-1 (Glucagon related peptide)</td>
</tr>
<tr>
<td>• Orexin</td>
<td>• Serotonin</td>
</tr>
<tr>
<td>• Endocannabinoid</td>
<td>• Glucose</td>
</tr>
<tr>
<td>• CCK</td>
<td>• Ketones</td>
</tr>
<tr>
<td>• Ghrelin</td>
<td>• Leptin</td>
</tr>
<tr>
<td>• Gut peptides</td>
<td>• Insulin</td>
</tr>
<tr>
<td>• PYY</td>
<td>• Cortisol</td>
</tr>
<tr>
<td>• Vagal stimulation</td>
<td></td>
</tr>
</tbody>
</table>

(31) ANSWER: (B) Sexual behaviour

REF: The orexin/hypocretin system: physiology and pathophysiology by Seiji Nishino, Takeshi Sakurai Page 107

OREXINS:
- **Orexins**, also called *hypocretins*, are the common names given to a pair of excitatory neuropeptide hormones
- Orexin-A/hypocretin-1 is 33 amino acid residues long and has two intrachain disulfide bonds, while Orexin-B/hypocretin-2 is a linear 28 amino acid residue peptide
- Secreted in the lateral and posterior hypothalamus
- The orexin peptides bind to the two G-protein coupled orexin receptors, **OX<sub>1</sub>** and **OX<sub>2</sub>**, with Orexin-A binding to both **OX<sub>1</sub>** and **OX<sub>2</sub>** with approximately equal affinity while Orexin-B binds mainly to **OX<sub>2</sub>** and is 5 times less potent at **OX<sub>1</sub>**

Functions of orexins:
- Orexin seems to promote **wakefulness**
- A link between orexin and Alzheimer’s disease has been recently suggested. The enigmatic protein amyloid beta builds up over time in the brain and is correlated with Alzheimer’s disease. The recent research shows that amyloid beta expression rises during the day and falls during the night, and that this is controlled by orexin.
- Orexin **increases the craving for food**
- Orexin-A (OXA) has been recently demonstrated to have direct effect on a part of the lipid metabolism. OXA stimulates glucose uptake in 3T3-L1 adipocytes and that increased energy uptake is stored as lipids (triacylglycerol). OXA thus increases lipogenesis. It also inhibits lipolysis and stimulates the secretion of adiponectin

(32) ANSWER: (B) Heart rate and peripheral resistance decreases

REF: Ganong’s 22nd ed chapter 31, Basic and Bedside Electrocardiography by Romulo F. Baltazar Page 195

CAROTID SINUS PRESSURE:
- The most commonly used and most effective vagal maneuver in terminating **SVT** is **carotid sinus** pressure.
- It should be performed under cardiac monitoring in recumbent position.
• With neck hyperextended the common carotids is identified by its pulsations and followed distally as close to the mandible as possible, usually to the **angle of mandible**, where the common carotid bifurcates. It is the bifurcation where carotid sinus is located.

• Carotid sinus pressure will produce increased baroreceptor discharge inhibits the tonic discharge of the vasoconstrictor nerves and excites the vagal innervation of the heart, producing **vasodilation**, **venodilation**, **a drop in blood pressure**, **bradycardia**, and **a decrease in cardiac output**

• Carotid pressure is applied gently and constantly using both middle and index finger.

• The maneuver can be repeated several times until the response is elicited but should **never exceeds more than 5 seconds**

• It is contraindicated in carotid stenosis

---

**Answer: (B) Rheobase**

**Ref:** Electrotherapy Simplified by Nanda page 276

The terms “chronaxie” and “rheobase” were coined in 1909 by the French physiologist Louis Lapicque

**Chronaxie** (or **chronaxy**) is the minimum time over which an electric current, double the strength of the rheobase, needs to be applied, in order to stimulate a muscle fiber or nerve cell.

**Rheobase** is the minimal current amplitude of indefinite duration (practically, a few hundred milliseconds) that results in the depolarization threshold of the cell membranes being reached (i.e. an action potential or the contraction of a muscle).

In the case of a nerve or single muscle cell, rheobase is half the current that needs to be applied for the duration of chronaxie to result in an action potential or muscle twitch. This can be understood better by looking at a strength duration relationship

---

**Answer: (A) Ventromedial nucleus**

**Ref:** Ganong’s 22nd ed chapter 14

See APPENDIX- 12 for “THE HYPOTHALAMIC NUCLEI”

Hypothalamic regulation of the appetite for food depends primarily on the interaction of two areas: a lateral **feeding center** in the bed nucleus of the medial forebrain bundle at its junction with the pallidohypothalamic fibers and medial **satiety center** in the ventromedial nucleus
(35) **ANSWER:** (A) **Intracellular**

**REF:** Guyton’s physiology 11th edition page 46 & 358, Ganong’s 22nd edition chapter 1

“Potassium cations are important in neuron (brain and nerve) function, and in influencing osmotic balance between cells and the interstitial fluid”

- Potassium is the most abundant cation in intracellular fluid.
- Phosphate is the most abundant intracellular anion.
- Sodium is the most abundant extracellular cation.
- Chloride is the most abundant extracellular anion.

(36) **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”

**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
(37) **ANSWER: (B) Pancreatic juice**

REF: Ganong’s 22nd ed chapter 26

<table>
<thead>
<tr>
<th>Secretion</th>
<th>Mean pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saliva</td>
<td>5.5</td>
</tr>
<tr>
<td>Gastric</td>
<td>1.0</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>8.0</td>
</tr>
<tr>
<td>Bile</td>
<td>7.0</td>
</tr>
</tbody>
</table>

(38) **ANSWER: (A) H band**

REF: Ganong’s 22nd ed

- The area between two adjacent Z lines is called a **sarcomere**

- The thick filaments, which are about twice the diameter of the thin filaments, are made up of myosin; the thin filaments are made up of actin, tropomyosin and troponin
- The thick filaments are lined up to form the **A bands**, whereas the array of thin filaments forms the less dense **I bands**
- The lighter **H bands** in the center of the A bands are the regions where, when the muscle is relaxed, the thin filaments do not overlap the thick filaments
- The **Z lines** transect the fibrils and connect to the thin filaments. If a transverse section through the A band is examined under the electron microscope, each thick filament is seen to be surrounded by six thin filaments in a regular hexagonal pattern.
(39) ANSWER: (A) Albumin
REF: Guyton’s physiology 11th edition page 841
In blood free fatty acids are transported in combination with albumin however they are transported across mitochondrial membrane by carnitine system.

(40) ANSWER: (B) Secondary active transport
REF: Ganong’s 22nd ed chapter 18
The thyroid cell membranes facing the capillaries contain a symporter, or iodide pump, that transports Na⁺ and I⁻ into the cells against the electrochemical gradient for I⁻. This Na⁺/I⁻ symporter (NIS) is capable of producing intracellular I⁻ concentrations that are 20–40 times as great as the concentration in plasma. The process involved is secondary active transport

(41) ANSWER: (D) Peroxisomes
Although mitochondrial β oxidation (oxidation of the fatty acyl group at the C-3, or β position—hence the name β oxidation) is by far the most important catabolic fate for fatty acids, two less common pathways of fatty acid catabolism are α and ω oxidation.

Alpha oxidation (α-oxidation) is a process by which certain fatty acids are broken down by removal of a single carbon from the carboxyl end. In humans, alpha-oxidation is used in peroxisomes to break down dietary phytanic acid, which cannot undergo beta-oxidation.

<table>
<thead>
<tr>
<th>Fatty acid oxidation</th>
<th>Major site</th>
</tr>
</thead>
<tbody>
<tr>
<td>β oxidation</td>
<td>Mitochondria in animal cell, Peroxisomes in plant cell</td>
</tr>
<tr>
<td>ω oxidation</td>
<td>Endoplasmic reticulum</td>
</tr>
<tr>
<td>α oxidation</td>
<td>Peroxisomes</td>
</tr>
</tbody>
</table>

(42) ANSWER: (B) Lysine
Carnitine is a quaternary ammonium compound biosynthesized from the amino acids lysine and methionine

(43) ANSWER: (B) Triglyceride and Cholesterol
MOLECULAR STRUCTURE OF CHYLOMICRONS:
The surface is a layer of phospholipids, with head groups facing the aqueous phase. Triacylglycerols sequestered in the interior make up more than 80% of the mass. Several apolipoproteins that protrude from the surface (B-48, C-III, C-II) act as signals in the uptake and metabolism of chylomicron contents. The diameter of chylomicrons ranges from about 100 to 500 nm.

**Core**: it’s made up of neutral lipids like triacylglycerols and cholesterol/cholesterol esters

**Shell**: composed of apolipoproteins, phospholipids

**Important points**:
- Maximum content of triglycerides: chylomicrons
- Maximum content of exogenous triglycerides: chylomicrons
- Maximum content of endogenous triglycerides: VLDL
- Maximum cholesterol: LDL

(44) **ANSWER: (A) Sunflower seeds**

REF: Harrison’s 18th ed chapter 74

“Sunflower seeds are unusually rich in B complex vitamins. Sunflower seeds also contain a potent antioxidant team of selenium and vitamin E and are significantly deficient in vitamin A”

There are two sources of dietary vitamin A. The first, or active form (retinol), is immediately available to the body and can be obtained from animal products such as milk, egg yolk, fish and liver. The second, or precursor form (beta-carotene), can be obtained from fruit and vegetables, and is converted to the active form in the body.

“Tomato is a good source of vitamins especially vitamin A and vitamin C”

(45) **ANSWER: (C) Thyroxin**

REF: Vasudevan biochemistry page 205, Lehninger Principles of Biochemistry 4th edition page 678, 859

**Important specialized products from tyrosine**:
- Thyroxine
- Melanin
- Catecholamines (dopamine, norepinephrine and epinephrine)

(46) **ANSWER: (C) Molybdenum**

ENZYME COFACTOR
Ketoglutrate dehydrogenase TPP
Carbonic anhydrase, lactate/alcohol dehydrogenase Zn
Xanthine oxidase, sulphite oxidase Mo
Amine oxidase, superoxide dismutase, cytochrome oxidase, tyrosinase Cu
Kinase, peptidase Mg
Hydrolase, decarboxylase, transferase Mn
Hb and cytochrome Fe
carboxylation biotin
transamination Pyridoxine

(47) ANSWER: (D) Vitamin B 12
REF: Lehninger Principles of Biochemistry 4th edition page 674
Sulphur containing amino acids are methionine, cystine and cysteine
Vitamin B 12 is a co-factor for methionine synthetase; it helps to convert homocysteine to methionine

(48) ANSWER: (D) Pyridoxal phosphate
REF: Lehninger Principles of Biochemistry 4th edition page 562
Pyridoxal phosphate is an essential cofactor in the glycogen phosphorylase reaction; its phosphate group acts as a general acid catalyst, promoting attack by Pi on the glycosidic bond. (This is an unusual role for this cofactor; its more typical role is as a cofactor in amino acid metabolism)
Flavin mononucleotide (FMN), or riboflavin-5’-phosphate, is a biomolecule produced from riboflavin (vitamin B2) by the enzyme riboflavin kinase and functions as prosthetic group of various oxidoreductases including NADH dehydrogenase as well as cofactor in biological blue-light photo receptors

(49) ANSWER: (C) Helicase
REF: Lippincott’s biochemistry 3rd Ed page: 400
PROTEINS INVOLVED IN REPLICATION:

<table>
<thead>
<tr>
<th>PROTEIN</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Helicase</td>
<td>Causes unwinding of DNA using ATP</td>
</tr>
<tr>
<td>SSB (Single strand binding protein)</td>
<td>Keeps two strands separate, prevents renneiling of two strands</td>
</tr>
<tr>
<td>Topoisomerase I and II</td>
<td>They have both nuclease (strand cutting) and ligase activity, hence they relieve torsional strain that results from helicase induced unwinding</td>
</tr>
<tr>
<td>DNA primase</td>
<td>Initiates synthesis of RNA primer</td>
</tr>
<tr>
<td>DNA polymerase</td>
<td>Deoxyribonucleotide polymerisation</td>
</tr>
<tr>
<td>DNA ligase /nick sealing enzyme</td>
<td>Seals the single stranded nick between nascent chains and okazaki fragments</td>
</tr>
</tbody>
</table>
(50) ANSWER: (B) S phase
REF: Ganong’s physiology 22nd ed chapter 1
“S or synthetic phase of cell cycle is the one in which replication of DNA OCCURS.

(51) ANSWER: (A) Autosomal dominant
In familial hypercholesterolemia, a human genetic disorder, blood levels of cholesterol are extremely high and severe atherosclerosis develops in childhood. These individuals have a defective LDL receptor and lack receptor-mediated uptake of cholesterol carried by LDL. Consequently, cholesterol is not cleared from the blood; it accumulates and contributes to the formation of atherosclerotic plaques.

Familial hypercholesterolemia is a genetic disorder caused by a defect on chromosome 19. The condition is typically passed down through families in an autosomal dominant manner. The most common genetic defects in FH are LDLR mutations (prevalence 1 in 500, depending on the population), ApoB mutations (prevalence 1 in 1000), PCSK9 mutations (less than 1 in 2500) and LDLRAP1

(52) ANSWER: (B) Fructose 2, 6 bisphosphate
REF: Lehninger Principles of Biochemistry 4th edition page 527
In the second of the two priming reactions of glycolysis, phosphofructokinase-1 (PFK-1) catalyzes the transfer of a phosphoryl group from ATP to fructose 6-phosphate to yield fructose 1, 6 bisphosphate:

In some organisms, fructose 2, 6-bisphosphate (not to be confused with the PFK-1 reaction product, fructose 1, 6- bisphosphate) is a potent allosteric activator of PFK-1.

(53) ANSWER: (C) Taq polymerase
REF: Lehninger Principles of Biochemistry 4th edition page 321
PCR uses a heat-stable DNA polymerase, such as the Taq polymerase (derived from a bacterium that lives at 90 °C), which remains active after every heating step and does not have to be replenished.

(54) ANSWER: (A) Spotted fever
- The Weil-Felix test is an agglutination test for the diagnosis of rickettsial infections
- The basis of the test is the presence of antigenic cross-reactivity between Rickettsia spp. and certain serotypes of non-motile Proteus spp
• Typhus group rickettsiae (Rickettsia prowazekii, R. typhi) react with P. vulgaris OX19,
• Scrub typhus (Orientia tsutsugamushi) reacts with P. mirabilis OXK.
• The spotted fever group rickettsiae (R. rickettsii, R. africae, R. japonica, etc.) react with P. vulgaris OX2 and OX19, to varying degrees, depending on the species

(55) ANSWER: (C) 4
REF: Paniker 6th ed p. 27

There are a lot of differences between E. histolytica and Entamoeba coli however we should remember a few important ones.
• It is important to distinguish the E. histolytica cyst from the cysts of nonpathogenic intestinal protozoa such as Entamoeba coli by its appearance.
• E. histolytica cysts have a maximum of four nuclei, while the commensal Entamoeba coli cyst has up to 8 nuclei.
• Additionally, in E. histolytica, the endosome is centrally located in the nucleus, while it is usually off-center in Entamoeba coli.
• Finally, chromatoidal bodies in E. histolytica cysts are rounded, while they are jagged in Entamoeba coli.

(56) ANSWER: (C) By the fermentation of glucose
REF: Anantnarayan 8th ed 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.

Other features:
• Gonococcus possess capsule while meningococcus doesn’t.
• Gonococcus is lens shaped while meningococcus is kidney shaped
• Gonococcus has plasmid , meningococcus rarely has

(57) ANSWER: (D) Escherichia coli
REF: Anantnarayan 8th ed various chapters, Jawetz 24th edition Chapter 16
(Note: Although K strain of E. coli is capsulated it is the best possible answer amongst the options provided)

E coli are classified by more than 150 different heat-stable somatic O (lipopolysaccharide) antigens, more than 100 heat-labile K (capsular) antigens, and more than 50 H (flagellar) antigens.

**K antigens** are external to O antigens on some but not all Enterobacteriaceae. Some are polysaccharides, including the K antigens of E. coli; others are proteins.

A common mnemonic used to remember some encapsulated pathogens is:

**“Even Some Super Killers Have Pretty Nice big Cars”**

Escherichia coli (K strain), Streptococcus pneumoniae, Salmonella, Klebsiella pneumoniae, Haemophilus influenzae, Pseudomonas aeruginosa, Neisseria meningitidis, Bacillus anthracis and Cryptococcus neoformans.

(58) **ANSWER: (C) Clostridium botulinum**

REF: Harrison’s 18th ed chapter 141

See APPENDIX-22 for “ACUTE INFECTIOUS DIARRHEA”

The temperatures obtained in open kettle canning are not high enough to destroy all spoilage and food poisoning organisms that may be in the food.

**Food-borne botulism** is caused by consumption of foods contaminated with botulinum toxin, which is a metabolic waste produced under anaerobic condition by the bacterium Clostridium botulinum. Most botulism cases are sporadic; outbreaks are typically small, involving two or three cases. The wide variation in reported rates of botulism by region and continent probably reflects differences both in true incidence and in diagnostic and reporting capacity. Worldwide, the highest incidence rate is reported from the Republic of Georgia and Armenia in the southern Caucasus region, where illness is associated with risky home-canning practices.
(59) ANSWER: (B) Salmonella  
REF: Harrison's 18th ed chapter 141  
See APPENDIX-22 for “ACUTE INFECTIOUS DIARRHEA”  
“Salmonella typically presents after 12 hours of infection”

(60) ANSWER: (D) Pseudomonas  
REF: Anantnarayan 8th ed various chapters  
Aerobic and anaerobic bacteria can be identified by growing them in liquid culture:

<table>
<thead>
<tr>
<th>Obligate aerobe</th>
<th>Require oxygen and will not grow under anaerobic conditions (ie, in the absence of O₂)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obligate anaerobe</td>
<td>Do not use oxygen for growth and metabolism but obtain their energy from fermentation reactions</td>
</tr>
<tr>
<td>Facultative anaerobe</td>
<td>Bacteria that can grow either oxidatively, using oxygen as a terminal electron acceptor, or anaerobically, using fermentation</td>
</tr>
<tr>
<td>Microaerophile</td>
<td>Requires environments containing lower levels of oxygen than are present in the atmosphere</td>
</tr>
</tbody>
</table>

Aerobic bacteria:  
- Bacillus anthracis  
- Bordetella pertussis  
- Brucella  
- Klebsiella  
- Listeria monocytogenes  
- Mycobacterium  
- Nocardia  
- Niesseria  
- Pseudomonas  
- Proteus  
- Pasteurella  
- Vibrio  
- Yersinia

(61) ANSWER: (C) Chlamydia  
REF: Robbin's 7th ed page 747  
Community acquired acute pneumonia:  
Streptococcus pneumonia, Haemophilus influenza, Moraxella catarrhalis, Staphylococcus aureus, Legionella, Enterobacteriacea (E.Coli, klebsiella), Pseudomonas  
Community acquired atypical pneumonia:  
Mycoplasma pneumoniea, chlymadia, coxiella, viruses (RSV, parainfluenza, influenza, adenovirus, SARS)

(62) ANSWER: (C) Influenza type C  
REF: Park 18th ed Page 144
INFLUENZA VIRUS:
- Family orthomyxoviridae
- Incubation period: 18-72 hours
- Types: A, B, C (No cross immunity and antigenically distinct)
- Both A and B have surface antigens, H (hemagglutinin), N (neuraminidase)
- **Major antigenic variations occur in A, some in B while C seems to be antigenically stable.**
- Rimantidine and Amantadine are used as prophylaxis for influenza A

**Antigenic variations:**
1. Antigenic shift: sudden change, due to genetic reassortment, causes major pandemics
2. Antigenic drift: A gradual change, due to point mutations, causes epidemics.

(63) **ANSWER: (D) Human**

REF: Jawett’s 24th edition Section VI. Parasitology Chapter 46, Paniker various chapters
Repeat from December 2009, June 2009

**Man is the definitive host in most parasitic infections except in:**
- Echinococcus granulosus
- Plasmodium
- Taenia Solium (man is both definitive and intermediate host)
- Toxoplasma gondii
- Sarcocystis lindemani

In other parasitic infections man acts as definitive host

**Two intermediate hosts are seen in:**
- Lung fluke (paragonimus)
- Chinese tape worm (chlonorchis)
- Fish tape worm (diphyllobothrum latum)
- Metagonimus westermanni

(64) **ANSWER: (B) P. Falciparum**

REF: Jawett’s 24th edition table 46–2

**SOME CHARACTERISTIC FEATURES OF THE MALARIA PARASITES OF HUMANS (ROMANOWSKY-STAINED PREPARATIONS):**

<table>
<thead>
<tr>
<th></th>
<th><em>P. vivax</em> (Benign Tertian Malaria)</th>
<th><em>P. malariae</em> (Quartan Malaria)</th>
<th><em>P. falciparum</em> (Malignant Tertian Malaria)</th>
<th><em>P. ovale</em> (Ovale Malaria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of usual maximum parasitemia</td>
<td>Large rings (1/3–1/2 red cell)</td>
<td>Large rings (1/3 red cell diameter).</td>
<td>Small rings (1/5 red cell diameter). Often two</td>
<td>Large rings (1/3 red cell diameter).</td>
</tr>
<tr>
<td>Ring stage trophozoites</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**P. ovale**

*P falciparum* (Malignant Tertian Malaria)
December 2011 Paper | 57

### Pigment in developing trophozoites
- Older trophozoites: Usually one chromatin granule; ring delicate. Usually one chromatin granule; ring delicate.

### Mature Schizonts (segmenters)
- More than 12 merozoites (14–24).
- Fewer than 12 large merozoites (6–12).
- Often in rosette.

### Gametocytes
- Round or oval.
- Crescentic.

### Distribution in peripheral blood
- All forms.
- Only rings and crescents (gametocytes). ¹

¹Ordinarily, only ring stages or gametocytes are seen in peripheral blood infected with *P falciparum*; post-ring stages make red cells sticky, and they tend to be retained in deep capillary beds except in overwhelming, usually fatal infections.

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(65) **ANSWER: (D) Entamoeba**

**REF: Various**

This was a difficult one and quite a nagging question, because no book gives a perfect answer. However after consulting various websites, CMDT, Harrison I reached the conclusion that most common cause of dysentery is bacterial > parasitic.

Robbin’s states that shighella is the prominent cause of dysentery in countries with poor hygiene, like India etc, while amongst parasites, it states that entameoba histolytica is major cause of dysentery.

(66) **ANSWER: (B) Burkholderia pseudomallei**

**REF: Ananthnarayan 8th edition Page 314**

“Melidiosis is caused by burkholderia pseudomallei”

**BURKHOLDERIA PSEUDOMELLEI**
- Causes melidiosis
- Motile
- May cause hemoptysis, hence resembles TB
- Regional lymphadenitis, nodular lymphangitis, localized skin infection.
- Latency and reactivation might occur
- Mode of spread: skin abrasion or inhalation
- Safety pin appearance
- Treatment: ceftazidime or carbpenams

**BURKOLDERIA MALLEI**
- Causes glanders
- Non motile
- Bipolar staining
- Agent of biological warfare, category b
- Induces strauss reaction
- Mallein test used for diagnosis
- Treatment is same as melidiosis

(67) ANSWER: (A) Staphylococcus
REF: Harrison’s 18th ed chapter 125

Important causes of muscle infections:
- Most common cause of pyomyositis: staphylococcus aureus
- Gas gangrene: Cl. Perferingens
- Severe muscle pain: pleurodynia due to coxsackie B, trichinosis
- Rhabdomyolysis: clostridium, streptococcal

(68) ANSWER: (A) Trichenella spiralis
REF: Myocarditis: from bench to bedside by Leslie T. Cooper Page 445 table 19-2
Repeat from June 2010

Parasitic infections associated with eosinophilic myocarditis:

Protozoa:
1. Trypanosoma cruzi (Chagas disease)
2. Toxoplasma gondii (Toxoplasmosis)

Metazoa:
1. Trichenella spiralis
2. Toxocara canis
3. Echinococcus granulosus (Hydatid cyst)
4. Schistosomiasis

(69) ANSWER: (C) Strongyloids
REF: Jawetz, Melnick, & Adelberg’s Medical Microbiology, 24th edition chapter 46 Table 46–4
Indirect repeat in December 2010, See APPENDIX-74 for “DISEASES DUE TO HELMINTHS”

Skin penetration is seen in:
1. Hookworms; Ancylostoma duodenale, Necator americanus
2. Strongyloides
3. Schistosoma (Cercariae larvae)

(70) ANSWER: (A) Trypanosoma cruzi

Although the genus Trypanosoma contains many species of protozoans, only T. cruzi, T. brucei gambiense, and T. brucei rhodesiense cause disease in humans. T. cruzi is the etiologic agent of Chagas' disease in the Americas; T. b. gambiense and T. b. rhodesiense cause African trypanosomiasis

<table>
<thead>
<tr>
<th>Trypanosome</th>
<th>Vector</th>
<th>Disease</th>
<th>Infective stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. brucei</td>
<td>Tse tse fly</td>
<td>African trypanosomiasis</td>
<td>Metacyclic trypomastigote</td>
</tr>
<tr>
<td></td>
<td></td>
<td>sleeping sickness</td>
<td></td>
</tr>
<tr>
<td>T. cruzi</td>
<td>Reduvid bug</td>
<td>Chaga's disease (south american trypanosomiasis)</td>
<td>Metacyclic trypomastigote</td>
</tr>
</tbody>
</table>
(71) ANSWER: (C) Cryptococcus  
REF: Chakraborty 2nd edition page 211  
Repeat in December 2010  
“Dimorphic fungi are fungi which can exist as mold/hyphal/filamentous form or as yeast”

<table>
<thead>
<tr>
<th>Yeast and yeast like</th>
<th>Moulds</th>
<th>Dimorphic fungus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cryptococcus</td>
<td>Dermatophyes</td>
<td>Histoplasma capsulatum</td>
</tr>
<tr>
<td>Candida</td>
<td>Aspergillus</td>
<td>Blastomyces sermatidis</td>
</tr>
<tr>
<td></td>
<td>Zygomycoses</td>
<td>Paracoccidioides brasiliense</td>
</tr>
<tr>
<td></td>
<td>Penicillium</td>
<td>Penicillium marneffei</td>
</tr>
<tr>
<td></td>
<td>Malassezia furfur</td>
<td>Sporothrix schenkii</td>
</tr>
<tr>
<td></td>
<td>Madurella mycosis</td>
<td>Coccidioides immitis</td>
</tr>
<tr>
<td></td>
<td>Philosphora</td>
<td>Candida albicans</td>
</tr>
<tr>
<td></td>
<td>Pseudoallescheria</td>
<td></td>
</tr>
</tbody>
</table>

Mnemonic for dimorphic fungi; **HB PSC** (use **HB** pencil in **PSC** exam)

(72) ANSWER: (C) B. henslae  
REF: Harrison’s 18th ed table 160-1  
Bartonella species are fastidious, facultative intracellular, slow-growing, gram-negative bacteria that cause a broad spectrum of diseases in humans

<table>
<thead>
<tr>
<th>Bartonella Species</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. henselae</td>
<td>Cat-scratch disease, bacillary angiomatosis, bacillary peliosis, bacteremia, endocarditis</td>
</tr>
<tr>
<td>B. quintana</td>
<td>Trench fever, chronic bacteremia, bacillary angiomatosis, endocarditis</td>
</tr>
<tr>
<td>B. bacilliformis</td>
<td>Bartonellosis (Carrion’s disease)</td>
</tr>
</tbody>
</table>

(73) ANSWER: (a) Corynebacterium  
REF: Ananthnarayan 8th edition page 238  
Metachromatic granules are inclusion bodies in bacterial cells that alter the colour of particular stains. Methylene blue stains metachromatic granules pink; not blue. In corynebacteria, the metachromatic granules are composed of polyphosphates and act as energy storage sites.  
**Features of Corynebacterium:**
- Gram-positive, catalase positive, non-spore-forming, non-motile, rod-shaped bacteria that is straight or slightly curved.
- The bacteria group together in a characteristic way, which has been described as the form of a “V”, “palisades”, or “Chinese letters”
- They may also appear elliptical. They are aerobic or facultatively anaerobic, chemoorganotrophs, with a 51–65% genomic G:C content
- Granules known as babes ernst or volutin granules composed of polymetaphosphate are seen
- Selective media: cysteine-tellurite blood agar or tinsdale medium rapid growth: loeffer’s serum slope

(74) ANSWER: (A) Casoni’s test  
REF: Paniker’s 6th ed p. 149
The **Casoni test** is a skin test used in the diagnosis of hydatid disease. The test involves the intradermal injection of 0.5 ml of sterilised fluid from hydatid cysts. A wheal response occurring at the injection site within 20 minutes is considered positive. The test is positive in about 90% of cases of hydatid disease affecting the liver, but positive in less than 50% of patients with hydatid disease elsewhere in the body; false positive results are also common. Consequently, serological tests are now generally used.

Dick’s test: Scarlet fever
Schick’s test: Diphtheria

(75) **ANSWER: (B) Nageleria**

**REF:** Jawetz, Melnick, & Adelberg’s Medical Microbiology, 24th edition Chapter 46

Free-living amebas of the genera *Naegleria*, *Acanthamoeba*, and *Balamuthia* live in brackish or freshwater habitats around the world (including lakes, tap water, swimming pools, and air conditioning and heating ducts) and are accidental and opportunistic agents of disease.

Chronic granulomatous disease from *Acanthamoeba* and *Balamuthia* may infect both immunocompetent and immunosuppressed humans and animals. Infection of the CNS from the skin lesion may occur weeks or months later. It is termed granulomatous amebic encephalitis to distinguish it from the explosive, rapid brain infection from *Naegleria* (**primary amebic meningoencephalitis**).

*Naegleria* (the “brain-eating ameba”) is the causative agent of primary amebic meningoencephalitis (PAM).

- *Naegleria* prefers warm freshwater, and most cases occur in otherwise healthy children, who usually have swum in lakes or swimming pools during the previous 2 weeks. *Naegleria* enters the central nervous system via water inhaled or splashed into the nose.
- The earliest manifestations are anosmia (usually perceived as alterations in taste), headache, fever, photophobia, nausea, and vomiting. Cranial nerve palsies, especially of the third, fourth, and sixth nerves, are documented and rapid progression of disease, with seizures, coma, and death within 7–10 days of the onset of symptoms, are common.
- Pathologic examination reveals hemorrhagic necrosis of brain tissue (often most prominent in the olfactory bulbs), evidence of increased intracranial pressure, scant purulent material that may contain a few amebas, and marked leptomenigitis.
- The diagnosis of PAM is based on the finding of motile *Naegleria* trophozoites in wet mounts of freshly obtained cerebrospinal fluid (CSF).
- Unfortunately, the prognosis for PAM is dismal. The few survivors who have been reported were treated with high-dose amphotericin B and rifampin in combination heading.

**Acanthamoeba Infections**

- Acanthamoeba species are free-living amebas that cause two major clinical syndromes: granulomatous amebic encephalitis and keratitis.
- Granulomatousamebicencephalitisoccursindebilitated,chronicallyill,andimmunosuppressed individuals.
- Granulomatous amebic encephalitis tends to present as a space-occupying lesion in the brain. Common symptoms include altered mental status, stiff neck, and headache along with focal findings including hemiparesis, ataxia, and cranial nerve palsies. Seizures and coma often precede death.
- Diagnosis is usually made by detection of Acanthamoeba trophozoites or cysts in biopsy specimens.
• There have been case reports of survivors treated with multidrug combinations that included pentamidine, sulfadiazine, flucytosine, rifampin, and fluconazole.
• Acanthamoeba keratitis is associated with corneal injuries complicated by exposure to water or soil and with the wearing of contact lenses. In contact lens–associated infection, extended wear, breaches in hygiene and disinfection procedures, swimming with contact lenses in place, and the use of homemade saline solutions contaminated with Acanthamoeba are important risk factors.
• Current therapy involves topical administration of a cationic antiseptic agent such as a biguanide or chlorhexidine, with or without a diamidine agent.

(76) **ANSWER: (C) Staphylococcus Aureus**


Pyomyositis, also known as tropical pyomyositis or myositis tropicans, is a bacterial infection of the skeletal muscles which results in a pus-filled abscess

Pyomyositis is most often caused by the bacterium *Staphylococcus aureus*. The infection can affect any skeletal muscle, but most often infects the large muscle groups such as the quadriceps or gluteal muscles.

(77) **ANSWER: (A) Carbohydrate sequence in cell wall**


**Innate immunity** does not have the specificity of adaptive immunity, but is capable of distinguishing self from nonself.

Bacterial lipopolysaccharides (LPS) are the major outer surface membrane components present in almost all Gram-negative bacteria and act as extremely strong stimulators of innate or natural immunity in diverse eukaryotic species ranging from insects to humans.

LPS consist of a poly- or oligosaccharide region that is anchored in the outer bacterial membrane by a specific carbohydrate lipid moiety termed lipid A. The lipid A component is the primary immunostimulatory centre of LPS.

(78) **ANSWER: (C) WT-38**

REF: Anathnarayan 8th ed page 441

**SOME CELL CULTURE LINES:**

<table>
<thead>
<tr>
<th>Primary cell culture</th>
<th>Diploid cell culture</th>
<th>Continuous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal cells freshly taken from body and cultured</td>
<td>WT-38</td>
<td>HeLa</td>
</tr>
<tr>
<td>Capable of limited growth and can’t be maintained in serial culture</td>
<td>HL-8</td>
<td>Hep-2</td>
</tr>
<tr>
<td>Rhesus monkey kidney culture</td>
<td>cells of single type and they retain their original diploid chromosome number and karyotype</td>
<td>KB</td>
</tr>
<tr>
<td>Human amnion cell culture</td>
<td></td>
<td>McCoy</td>
</tr>
<tr>
<td>Chick embryo fibroblast culture</td>
<td></td>
<td>Detroit-6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vero</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Derived from cancer cells</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Continuous indefinite serial culture possible</td>
</tr>
</tbody>
</table>
PATHOLOGY

(79) ANSWER: (B) Apoptosis
Indirect repeat from June 2008, June 2009 & June 2010
See APPENDIX-27 for “APOPTOSIS VS NECROSIS”

Annexin A5 (or annexin V) is a cellular protein in the annexin group. The function of the protein is unknown; however, annexin A5 has been proposed to play a role in the inhibition of blood coagulation by competing for phosphatidylinerine binding sites with prothrombin and also to inhibit the activity of phospholipase A1.

Antibodies directed against annexin A5 are found in patients with a disease called the antiphospholipid syndrome (APS), a thrombophilic disease associated with auto antibodies against phospholipid compounds.

Annexin A5 is used as a probe in the annexin A5 affinity assay to detect cells that have expressed phosphatidylinerine on the cell surface, a feature found in apoptosis as well as other forms of cell death.

(80) ANSWER: (D) Cytochrome C
See APPENDIX-27 for “APOPTOSIS VS NECROSIS”

“Cytochrome c is released by the mitochondria in response to pro-apoptotic stimuli and forms apoptosomes.”

INTRINSIC PATHWAY:
As its name suggests, the intrinsic pathway is initiated from within the cell. This is usually in response to cellular signals resulting from DNA damage, a defective cell cycle, detachment from the extracellular matrix, hypoxia, loss of cell survival factors, or other types of severe cell stress. This pathway involves the release of pro-apoptotic proteins that activate caspase enzymes from the mitochondria. This process ultimately triggers apoptosis.
Cytochrome c binds the adaptor apoptotic protease activating factor-1, forming a large multiprotein structure known as the apoptosome. Assembly of the apoptosome is highly regulated and may be driven by nucleotide exchange factors and/or ATPase-activating proteins. The primary function of the apoptosome seems to be multimerization and allosteric regulation of the catalytic activity of caspase 9. Initiator caspase 9 is recruited into the apoptosome and activated from within the adaptor protein complex, which in turn activates the downstream effector caspases 3, 6, and/or 7.

**EXTRINSIC PATHWAY:**

The extrinsic pathway begins outside the cell through the activation of specific pro-apoptotic receptors on the cell surface. These are activated by specific molecules known as pro-apoptotic ligands. These ligands include Apo2L/TRAIL and CD95L/FasL and bind their cognate receptors DR4/DR5 and CD95/Fas, respectively. Unlike the intrinsic pathway, the extrinsic pathway triggers apoptosis independently of the p53 protein.

**ANSWER:** (A) 10% formaldehyde


“10% natural buffered formalin (NBF) is the most commonly used fixative for bone specimens”

Primary fixatives of bone tissue:
1. 10% formalin
2. Glutaraldehyde
3. Paraformaldehyde
4. Alcohol based solutions

**Secondary fixatives of bone tissue:**
1. Zenker’s solution (mercuric chloride, potassium dichromate, sodium sulfate, water, and acetic acid)
2. Bouin’s solution (picric acid, acetic acid and formaldehyde)

(82) ANSWER: (D) SLE
REF: Harrison’s 18th ed chapter 100

A spectrum of paraneoplastic syndromes has been associated with these malignancies, including erythrocytosis, hypercalcemia, nonmetastatic hepatic dysfunction (Stauffer syndrome), and acquired dysfibrinogenemia. Erythrocytosis is noted at presentation in only about 3% of patients. Anemia, a sign of advanced disease, is more common.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1475999/ says

The paraneoplastic syndromes associated with renal cell carcinoma range from those manifesting in constitutional symptoms (i.e. fever, cachexia, and weight loss) to those that result in specific metabolic and biochemical abnormalities (i.e. hypercalcemia, nonmetastatic hepatic dysfunction, amyloidosis, hyperglycemia, Hypertension, Cushing’s syndrome, acanthosis nigricans).

(83) ANSWER: (D) Leukotrienes
REF: Robbin’s 7th ed page 56

“Endogenous chemo attractants are C5a, leukotriene B4, IL-8”

See APPENDIX-19 for “CYTOKINES

(84) ANSWER: (D) CD23
REF: Robbin’s 7th ed page 682

**MANTLE CELL LYMPHOMA:**
- The tumor closely resembles normal mantle zone B cell that surrounds germinal centers.
- Have a characteristic chromosomal translocation, t(11;14), between the immunoglobulin heavy chain gene on chromosome 14 and the *bcl-1* gene on chromosome 11
- The most common presentation of mantle cell lymphoma is with palpable lymphadenopathy
- **Immunophenotype:**
  1. CD 19, CD 20 positive
  2. CD 5 positive and CD 23 negative
  3. Characteristically positive for cyclin D1 protein

(85) ANSWER: (A) Prevent hemolysis

**PACKED RED CELLS VERSUS RED CELLS IN ADDITIVE SOLUTIONS:**
After whole blood is collected into CPD or CPDA-1, the red cells may be concentrated by centrifugation and removal of most of the plasma (“packed RBCs”). Approximately 20% of the anticoagulant-containing plasma must be left with the red cells to provide metabolic substrate for
the red cells during storage. Another approach to red cell preservation involves virtually complete removal of the anticoagulated plasma from the red cells (“dry pack”), followed by resuspension of the red cells in 100 ml of an additive solution. Such additive solutions contain saline, adenine, and glucose, with or without mannitol to decrease hemolysis. The duration of red cell storage in additive solutions is extended to 42 days. Red cells in additive solution are now the most common preparations available for transfusion. Red cells collected in any of the anticoagulants and preservatives must be stored at 1° to 6°C to maintain optimum function.

Note: • A number of chemical agents—dihydroxyacetone, pyruvate, phosphoenolpyruvate, and inosine—are capable of maintaining near-normal red cell 2,3-DPG content during storage or of replenishing 2,3-DPG after storage. Although none of these chemicals are likely to be used in transfusion because of their side effects
• Glycerol is gradually added to the red cells as a cryoprotectant for frozen RBC to a final concentration of 40% (weight/volume).

(86) ANSWER: (B) Over expression of C-erb

MOLECULAR STOMACH CARCINOGENESIS:
• p53 mutation commonly participate in early steps of stomach carcinogenesis.
• Amplification and overexpression of c-met and cyclin E genes are frequently associated with advanced stages
• Reduced expression of p27 participates in both development and progression of gastric carcinoma.
• K-ras mutation, HER-2/c-erb/B2 amplification and APC mutation preferentially occurs in well differentiated type.
• Inactivation of cadherin and catenins and amplification of K-sam and c-met are frequently associated with poorly differentiated with or scirrhous type carcinoma.

(87) ANSWER: (A) Never regained
REF: Robbin’s 7th ed p. 113
“Most wounds do not regain the full tensile strength of unwounded skin after healing is complete”

WOUND STRENGTH:
• Carefully sutured wound have tensile strength of 70% of unwounded skin largely because of placement of sutures
• When sutures are removed, usually at the end of first week the wound strength is approximately 10%
• Then there is rapid gain of strength for next 4 weeks
• Reaches a plateau of wound strength of about 70-80% of unwounded skin by 3rd month and persists at this level for life.

(88) ANSWER: (C) 9:22
REF: Robbin’s 7th ed page 696
See APPENDIX-29 for “TRANSLOCATION”
“In more than 90% of CML cases, karyotyping reveals the so called Philadelphia chromosome, which is created by reciprocal (9,22) (q34,q11) translocation”
(89) **ANSWER: (B) Factor 7 deficiency**


See APPENDIX-50 for “BLEEDING/COAGULATION DISORDERS” and APPROACH TO A PATIENT WITH BLEEDING DISORDER

“The prothrombin time (PT) is prolonged in factor VII (FVII) deficiency and the international normalized ratio (INR) is elevated. The activated partial thromboplastin time (aPTT) is within the reference range in isolated factor VII deficiency”

(90) **ANSWER: (B) IL-8**

REF: Robbin’s 7th edition page 72

See APPENDIX-19 for “CYTOKINES”

Their name is derived from their ability to induce directed chemotaxis in nearby responsive cells; they are chemotactic cytokines

- **C-X-C OR ALPHA CHEMOKINE**
  - a) CXC chemokines specifically induce the migration of neutrophils
  - b) Secreted by activated macrophage, endothelial cell and other cell
  - c) Includes IL-8

- **C-C OR BETA CHEMOKINE:**
  - a) induce chemotaxis of monocytes, eosinophils, lymphocytes but not neutrophils
  - b) Includes
    - I. Monocyte chemoattractant protein (MCP-1)
    - II. Eotaxin
    - III. Macrophage inflammatory protein (MIP)
    - IV. RANTES

- **C-CHEMOKINE OR GAMMA CHEMOKINES**
  - a) Relatively specific for lymphocyte
  - b) Include lymphotactin

- **CX3C CHEMOKINE**
  - a) Two forms
    - I. Cell surface bound
    - II. Soluble
      - b) Include fractalkine

(91) **ANSWER: (A) Sarcoidosis**

REF: Robbins’ 7th edition, page 734, 8th ed p. 738

Repeat from June 2010

“Asteroid bodies are seen in Sarcoidosis”

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

An asteroid body is a histopathologic finding, seen in granulomatous diseases such as Sarcoidosis and foreign body giant cell reactions, other granulomatous conditions and tuberculosis.

(92) **ANSWER: (A) 47XXY**
REF: Harrison’s 18th ed table 349-2

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Common Chromosomal Complement</th>
<th>Gonad</th>
<th>External</th>
<th>Internal</th>
<th>Breast Development</th>
</tr>
</thead>
<tbody>
<tr>
<td>Klinefelter’s syndrome</td>
<td>47,XXY or 46,XY/47,XXY</td>
<td>Hyalinized testes</td>
<td>Male</td>
<td>Male</td>
<td>Gynecomastia</td>
</tr>
</tbody>
</table>

**Clinical Features**

Small testes, azoospermia, decreased facial and axillary hair, decreased libido, tall stature and increased leg length, decreased penile length, increased risk of breast tumors, thromboembolic disease, learning difficulties, obesity, diabetes mellitus, varicose veins

93) **ANSWER: (C) Ankyrin**


Indirect repeat from December 2010

Most common cause of hereditary spherocytosis is **ankyrin gene mutation**

**Pathophysiology of hereditary spherocytosis:**

Hereditary spherocytosis is an *autosomal dominant* or recessive trait, most commonly (though not exclusively) found in Northern European and Japanese families, although an estimated 25% of cases are due to spontaneous mutations. A patient has a 50% chance of passing the mutation onto his/her offspring. Hereditary spherocytosis is caused by a variety of molecular defects in the genes that code for Spectrin (alpha and beta), Ankyrin, band-3 protein, protein 4.2 and other erythrocyte membrane proteins:

These proteins are necessary to maintain the normal shape of an erythrocyte, which is a biconcave disk.

**INHERITED DISEASES OF THE RED CELL MEMBRANE-CYTOSKELETON:**

<table>
<thead>
<tr>
<th>Gene</th>
<th>Chromosomal Location</th>
<th>Protein Produced</th>
<th>Disease(s) with Certain Mutations (Inheritance)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPTA1</td>
<td>1q22-q23</td>
<td>α-Spectrin</td>
<td>HS (recessive) HE (dominant)</td>
<td>Rare. Mutations of this gene account for about 65% of HE. More severe forms may be due to coexistence of an otherwise silent mutant allele.</td>
</tr>
<tr>
<td>SPTB</td>
<td>14q23-q24.1</td>
<td>β-Spectrin</td>
<td>HS (dominant) HE (dominant)</td>
<td>Rare. Mutations of this gene account for ~30% of HE, including some severe forms. May account for majority of HS.</td>
</tr>
<tr>
<td>ANK1</td>
<td>8p11.2</td>
<td>Ankyrin Band 3 (anion channel)</td>
<td>HS (dominant) HE (dominant)</td>
<td>Mutations of this gene may account for ~25% of HS.</td>
</tr>
<tr>
<td>SLC4A1</td>
<td>17q21</td>
<td></td>
<td>Southeast Asian ovalocytosis (dominant) HE (dominant)</td>
<td>Polymorphic mutation (deletion of 9 amino acids); clinically asymptomatic; protective against Plasmodium falciparum.</td>
</tr>
<tr>
<td>EPB41</td>
<td>1p33-p34.2</td>
<td>Band 4.1</td>
<td></td>
<td>Mutations of this gene account for about 5% of HE, mostly with prominent morphology but no</td>
</tr>
<tr>
<td>EPB42</td>
<td>15q15-q21</td>
<td>Band 4.2</td>
<td>HS (recessive)</td>
<td>hemolysis in heterozygotes; severe hemolysis in homozygotes. Mutations of this gene account for about 3% of HS. Very rare; associated with total loss of all Rh antigens.</td>
</tr>
<tr>
<td>-------</td>
<td>-----------</td>
<td>----------</td>
<td>-----------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>RHAG</td>
<td>6p21.1-p11</td>
<td>Rhesus antigen</td>
<td>Chronic nonspherocytic hemolytic anemia</td>
<td>Note: HS, hereditary spherocytosis; HE, hereditary elliptocytosis.</td>
</tr>
</tbody>
</table>

**PHARMACOLOGY**

(94) **ANSWER: (B) Vincristine**  
REF: KDT 5th edition page 774  
See APPENDIX-37 for “CLASSIFICATION OF CANCER CHEMOTHERAPY AGENTS”

(95) **ANSWER: (A) Mucous and pharyngeal secretions**  
REF: KDT 5th edition page 95  
**Sensitivity of atropine to various organ systems:**  
Saliva, sweat, bronchial secretions > eyes, bronchial muscles, heart > smooth muscle of intestine and bladder > gastric glands and smooth muscle  
This probably is due to:  
• Other factors that influence the cholinergic tone of various organs.  
• Variations in synaptic gaps  
**Some important points about atropine**  
• Natural alkaloid, anticholinergic drug  
• Used as a mydriatic in form of (1%) ointment in children < 5yrs of age.  
• Effects in belladonna poisoning or dhatura toxicity are due to atropine.  
• Atropine is specific anti-dote for anti CHE and early mushroom poisoning.  
• May be used in counteracting bradycardia and partial heart blocks in patients with increased vagal discharge, like, MI, digitalis toxicity.  
• Contraindications of atropine:  
  • Absolute c/I in patients with narrow angle glaucoma.  
  • Caution is advocated in elderly males with prostate hypertrophy.

(96) **ANSWER: (C) Bradycardia**  
REF: KDT 4th Ed p. 94
EFFECTS OF ATROPINE:

<table>
<thead>
<tr>
<th></th>
<th>CNS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Stimulates medullary, vasomotor and respiratory center</td>
</tr>
<tr>
<td></td>
<td>• Depresses vestibular excitation, hence anti motion sickness</td>
</tr>
<tr>
<td></td>
<td>• Suppresses cholinergic activity in basal ganglia, hence decreases tremor.</td>
</tr>
<tr>
<td></td>
<td>• High doses may cause cortical excitation, restlessness, disorientation and hallucinations</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>CVS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td>• Abbreviates A-V refractory period</td>
</tr>
<tr>
<td></td>
<td>• Facilitates A-V conduction</td>
</tr>
<tr>
<td></td>
<td>• No consistent effect on BP</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>EYE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Mydriasis</td>
</tr>
<tr>
<td></td>
<td>• Abolition of light reflexes</td>
</tr>
<tr>
<td></td>
<td>• Cycloplegia</td>
</tr>
<tr>
<td></td>
<td>• Rise of IOT</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Smooth muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• All visceral smooth muscles are relaxed</td>
</tr>
<tr>
<td></td>
<td>• Constipation</td>
</tr>
<tr>
<td></td>
<td>• Bronchodilation</td>
</tr>
<tr>
<td></td>
<td>• Urinary retention</td>
</tr>
<tr>
<td></td>
<td>• Urinary bladder and ureter relaxation</td>
</tr>
<tr>
<td></td>
<td>• Effect on uterus is minimal</td>
</tr>
<tr>
<td></td>
<td>• Effect of biliary tract is less marked</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Glands</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Decrease sweat, salivary, tracheobronchial and lachrymal secretions</td>
</tr>
<tr>
<td></td>
<td>• Decreases secretion of acid and pepsin</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Body temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• rise</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Local anesthetic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• effect present</td>
</tr>
</tbody>
</table>

(97) **ANSWER: (C) Biguanides**

REF: Harrison's 18th ed chapter 344 table 344-11, KDT 5th edition page 249

"Glucosidase inhibitors are not as potent as other drugs in reducing HBA1C levels"

For better understanding of the topic see next question

(98) **ANSWER: (B) Sulfonylureas**

REF: Harrison's 18th ed chapter 344, table 344–11, KDT 5th edition page 249

According to Harrison:

Insulin secretagogues, biguanides, GLP-1 receptor agonists, and thiazolidinediones improve glycemic control to a similar degree (1–2% reduction in A1C) and are more effective than - glucosidase inhibitors and DPP-IV inhibitors.

According to the table 344–11

<table>
<thead>
<tr>
<th>Drug</th>
<th>% reduction in HBA1C levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphonylureas</td>
<td>1–2</td>
</tr>
<tr>
<td>Biguanides</td>
<td>1–2</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td>0.5–1.4</td>
</tr>
<tr>
<td>Glucosidase inhibitor</td>
<td>0.5–0.8</td>
</tr>
<tr>
<td>DPP-1V inhibitor</td>
<td>0.5–0.8</td>
</tr>
</tbody>
</table>

KDT states that:

Sulphonylureas have a more reducing effect on HBA1C than biguanides.
Hence the **drug with most reduction in HBA1C should be sulphonylureas.**

A few important points about newer anti diabetic drugs are as follows:

**Dipeptidyl peptidase IV inhibitors:**
- **MOA:** Prolong endogenous GLP-1 action
- **EXAMPLES:** Saxagliptin, Sitagliptin, Vildagliptin
- **Reduce dose with renal disease**

**GLP-1 receptor agonists**
- **MOA:** Increases Insulin, decreases glucagon, slow gastric emptying and satiety
- **EXAMPLES:** Exenatide, liraglutide
- **Causes Weight loss, do not cause hypoglycaemia**
- **Reduce dose with Renal disease, agents that also slow GI motility**

**Amylin agonists**
- **MOA:** Slow gastric emptying, glucagon
- **EXAMPLES:** Pramlintide

(99) **ANSWER:** (D) Repaglinide

**REF:** KDT 4th edition page 249

Metaglinide analogues like repaglinide and netaglinide acts on ATP sensitive K channels and hence causes insulin secretion by causing depolarization. They can be used to limit post prandial hyperglycemia without producing late phase hypoglycemia.

(100) **ANSWER:** (B) Erythromycin

**REF:** Schwartz 8th ed p. 1486

**SIDE EFFECTS OF ERYTHROMYCIN:**
- Administration of erythromycin in early infancy has been linked to subsequent development of hypertrophic pyloric stenosis.
- Erythromycin estolate has been associated with reversible hepatotoxicity in pregnant women in the form of elevated serum glutamic-oxaloacetic transaminase and is not recommended during pregnancy.
- It may also alter the effectiveness of combined oral contraceptive pills because of its effect on the gut flora.
- More serious side-effects include arrhythmia with prolonged QTc intervals including Torsades-de-Pointe and reversible deafness.
- Allergic reactions range from urticaria to anaphylaxis. Cholestasis, Stevens–Johnson syndrome, and toxic epidermal necrolysis are some other rare side-effects that may occur.

Erythromycin and related antibiotics act as non-peptide motilin agonists, and are sometimes used for their ability to stimulate gastrointestinal motility.

Intravenous erythromycin may also be used in endoscopy as an adjunct to clear gastric contents.

(101) **ANSWER:** (C) Ondansetron

**REF:** Goodman Gillman 11th ed p. 536

Based upon reports of profound hypotension and loss of consciousness when apomorphine was introduced with ondansetron. Due to the risk of dangerously low blood pressure (hypotension) and loss of consciousness, ondansetron and apomorphine should not be combined.
Ondansetron: (serotonin 5-HT3 receptor antagonist)
- Primary drugs used to treat and prevent chemotherapy-induced nausea and vomiting
- Ondansetron is also effective in controlling post-operative nausea and vomiting (PONV) and post-radiation nausea and vomiting, and is a possible therapy for nausea and vomiting due to acute or chronic medical illness or acute gastroenteritis.
- A 2006 double-blind, randomized controlled trial indicated that ondansetron may have value in the treatment of schizophrenia, as an adjunct to haloperidol.
- Ondansetron lowers the cravings for alcohol, especially in early-onset alcoholics.
- Ondansetron blocks the 5-HT3 receptor in the enteric nervous system, and thereby reduces colonic contractions, sensory perception, and motility. A large number of drugs in this category, 5-HT3 antagonist, have been shown to have this effect, which positively impacts irritable bowel syndrome with diarrhea (IBS-D)
- Ondansetron was found to be as effective as pethidine (meperidine, Demerol) when given as a single IV dose before anesthesia.

(102) ANSWER: (D) Intramuscular
REF: KDT 5th edition page 116

USES OF ADRENALINE

<table>
<thead>
<tr>
<th>USE</th>
<th>DOSE</th>
<th>ROUTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaphylactic shock</td>
<td>0.5 mg</td>
<td>intramuscular</td>
</tr>
<tr>
<td>Local anesthetics</td>
<td>1:100000 or 1:200000</td>
<td></td>
</tr>
<tr>
<td>Control of bleeding</td>
<td>1 in 10000</td>
<td></td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td></td>
<td>intravenous</td>
</tr>
</tbody>
</table>

(103) ANSWER: (B) Prostacycline-Thrombaxone
REF: KDT 5th edition page 49

ANTAGONISM:

<table>
<thead>
<tr>
<th>Physical</th>
<th>Based upon physical properties of the drugs</th>
<th>Charcoal adsorbs alkaloids and can prevent their absorption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical</td>
<td>Two drugs react and form an inactive product</td>
<td>KmnO4 oxidises alkaloids tannins + alkaloids chelating agents nitrites form methemoglobinemia</td>
</tr>
<tr>
<td>Physiological/functional</td>
<td>The drugs react with different receptors and exert different effects</td>
<td>Histamine and adrenaline on bronchial muscles and BP hydrochlorothiazide and triamterene on urinary K excretion</td>
</tr>
<tr>
<td>Receptor</td>
<td>Antagonist interferes with binding of agonist on receptor</td>
<td></td>
</tr>
</tbody>
</table>

One needs to know the basic concept of antagonism and the receptors drugs act on to deal with this question
- For physiological antagonism both drugs must act on different receptors
- Protamine binds to heparin and deactivates it, hence not physiological antagonism
- Adrenaline and phenoxybenzamine act on same receptors but are antagonists, hence not physiological.
- Acetylcholine and physostigmine also are antagonists on same receptors.

TXA2 is generated by blood platelets, while PGI2 is produced by vascular endothelium. TXA2 is a potent vasoconstrictor. It also initiates the release reaction, followed by platelet aggregation. PGI2 is a vasodilator, especially potent in coronary circulation. It also inhibits platelet aggregation by virtue of stimulation of platelet adenyl cyclase. A balance between formation and release of PGI2, TXA2 and/or cyclic endoperoxides in circulation is of utmost importance for the control of intra-arterial thrombi formation and possibly plays a role in the pathogenesis of atherosclerosis.

Both thromboxane and prostacyclins act on different receptors, but their actions are exact opposite, hence they are physiological antagonists.

(104) ANSWER: (D) All of the above

REF: KDT 5th ed p. 470

<table>
<thead>
<tr>
<th>Dopamine</th>
<th>Acts on D1, D2, alpha and beta1 receptors</th>
<th>Low doses: renal vasodilation (D1 action)</th>
<th>Moderate doses: Positive ionotropism (beta1 action)</th>
<th>High doses: Vasoconstriction (alpha1 action)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>Relatively beta1 specific agonist</td>
<td></td>
<td>Ionotropi i.e increases force of cardiac contraction without causing vasoconstriction, increasing afterload, heart rate or BP.</td>
<td></td>
</tr>
</tbody>
</table>

Therefore since dobutamine is beta1 specific, doesn’t cause vasoconstriction, it is less arrhythmogenic, and doesn’t affect renal vasculature it is preferred in cardiogenic shock, however it is less suitable for shocks in which vasoconstriction is required.

(105) ANSWER: (A) Insulin glargine

REF: Pharmacology ReCap 2.0 for Bachelor of Dentistry Students by Dr. J. G. Buch page 304, Harrison’s 18th ed chapter 344

INSULINS:

<table>
<thead>
<tr>
<th>Highly purified mono-component insulin</th>
<th>Porcine Actrapid-Regular</th>
<th>Short acting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Porcine Monotard-Lente</td>
<td></td>
<td>Intermediate acting</td>
</tr>
<tr>
<td>Porcine Insulatard-NPH</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Porcine Mixtard</td>
<td></td>
<td>30% regular, 70% Isophane</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Human insulin</th>
<th>Human Actrapid-Regular</th>
<th>Short acting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human Monotard-Lente</td>
<td></td>
<td>Intermediate acting</td>
</tr>
<tr>
<td>Human Insulatard-NPH</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Human Mixtard</td>
<td></td>
<td>30% regular, 70% Isophane</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Insulin analogues</th>
<th>Insulin Lispro</th>
<th>Ultra short acting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin aspart</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin glulisine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Insulin glargine</th>
<th>Long acting</th>
</tr>
</thead>
</table>

(106) ANSWER: (B) Penicillin

REF: Rooks 7th ed p. 57.53
Drugs Reported to Cause Pseudo lymphomas of the Skin:
- Anticonvulsants
- Antipsychotics: chlorpromazine, thioridazine
- Antihypertensives: captopril, atenolol, verapamil, diltiazem, moduretic, beta blockers, hydrochlorothiazide
- Cytotoxics: cyclosporine, methotrexate
- Antirheumatics: gold, salicylates, phenacetin, D-penicillamine, allopurinol
- Antibiotics: penicillin, nitrofurantoin
- Antidepressants: fluoxetine, doxepin, desipramine, amitriptyline hydrochloride, lithium
- Anxiolytics: alprazolam, clonazepam, lorazepam
- Antihistamines: diphenhydramine, cimetidine, ranitidine
- Antiarrhythmics: mexiletine chloride

Other causes
- Viral infections – HIV, molluscum, herpes zoster, parapox and pox.
- Infections – syphilis and Borrelia.
- Parasites – scabies, leeches and coral.
- Antigen injections – de-sensitisation procedures and vaccinations.
- Metals – tattoos, earrings.
- Treatment for haematological disease.
- Ethnic scarification/female genital mutilation.

(107) ANSWER: (D) Thrombocytopenia


CHARACTERISTICS OF ANTITUBERCULOUS DRUGS:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Most Common Side Effects</th>
<th>Tests for Side Effects</th>
<th>Drug Interactions</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoniazid</td>
<td>Peripheral neuropathy (due to pyridoxine deficiency), hepatitis, rash, Optic neuritis, Seizures, Psychosis.</td>
<td>AST and ALT. neurologic examination.</td>
<td>Phenytoin (synergistic); disulfiram.</td>
<td>Bactericidal to both extracellular and intracellular organisms. Pyridoxine, 10 mg orally daily as prophylaxis for neuritis; 50–100 mg orally daily as treatment.</td>
</tr>
<tr>
<td>Rifampin</td>
<td>Hepatitis, fever, rash, flu-like illness, gastrointestinal upset, bleeding problems, renal failure.</td>
<td>CBC, platelets, AST and ALT.</td>
<td>Rifampin inhibits the effect of oral contraceptives, quinidine, corticosteroids, warfarin, methadone, digoxin, oral hypoglycemics; aminosalicylic acid may interfere with absorption of rifampin. Significant interactions</td>
<td>Bactericidal to all populations of organisms. Colors urine and other body secretions orange. Discoloring of contact lenses.</td>
</tr>
<tr>
<td>Drug</td>
<td>Effect</td>
<td>Side Effect</td>
<td>Mechanism of Action</td>
<td></td>
</tr>
<tr>
<td>--------------------</td>
<td>---------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Pyrazinamide</td>
<td>Hyperuricemia, hepatotoxicity, rash,</td>
<td>Uric acid, AST, ALT.</td>
<td>with protease inhibitors and nonnucleoside reverse transcriptase inhibitors.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>gastrointestinal upset, joint aches.</td>
<td></td>
<td>Bactericidal to intracellular organisms.</td>
<td></td>
</tr>
<tr>
<td>Ethambutol</td>
<td>Optic neuritis (reversible with discontinuance of drug; rare at 15 mg/kg); rash.</td>
<td>Red-green color discrimination and visual acuity (difficult to test in children under 3 years of age).</td>
<td>Rare.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bacteriostatic to both intracellular and extracellular organisms. Use with caution in renal disease or when ophthalmologic testing is not feasible.</td>
<td></td>
</tr>
<tr>
<td>Streptomycin</td>
<td>Eighth nerve damage (ototoxicity), nephrotoxicity.</td>
<td>Vestibular function (audiograms); BUN and creatinine.</td>
<td>Neuromuscular blocking agents may be potentiated and cause prolonged paralysis.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bactericidal to extracellular organisms. Use with caution in older patients or those with renal disease.</td>
<td></td>
</tr>
</tbody>
</table>

AST, aspartate aminotransferase; ALT, alanine aminotransferase; CBC, complete blood count; BUN, blood urea nitrogen

(108) **ANSWER: (B) Penicillin**

REF: KDT 6th edition page 688

**MECHANISMS OF ACTIONS OF ANTIBIOTICS:**

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell wall synthesis inhibitors</td>
<td>Beta lactams, cycloserine, fosfomycin, bacitracin, vancomycin</td>
</tr>
<tr>
<td>Protein synthesis inhibitors</td>
<td>Aminoglycosides</td>
</tr>
<tr>
<td>- freeze initiation</td>
<td>Tetracyclines, puromycins, chloramphenicol</td>
</tr>
<tr>
<td>- inhibit elongation</td>
<td>Clindamycin, erythromycin</td>
</tr>
<tr>
<td>- inhibit translocation</td>
<td></td>
</tr>
<tr>
<td>Misreading of RNA code</td>
<td>Aminoglycosides</td>
</tr>
<tr>
<td>Inhibit DNA gyrase</td>
<td>Nalidixic acid and nitrofurantoin</td>
</tr>
<tr>
<td>Drugs binding to ribosomes</td>
<td>30 S – Tetracycline, streptomycin</td>
</tr>
<tr>
<td></td>
<td>50 S – chloramphenicol, clindamycin, erythromycin</td>
</tr>
<tr>
<td></td>
<td>Both 30 S and 50 S – Aminoglycosides except streptomycin</td>
</tr>
</tbody>
</table>

(109) **ANSWER: (B) Doxycycline**

REF: KDT 6th edition page 673

**Drugs that don’t require dose alteration in renal failure:**

- Doxycycline
- Chloramphenicol
- Clindamycin
- Rifampicin
- Rifapentine
- Rifabutin

**Drugs that are contraindicated in renal failure**
- Nitrofurantoin
- Nalidixic acid
- Sulphonamides
- Tetracycline except Doxycycline
- Methenamine
- Voriconazole
- cidofovir

**Answer:** (B) Neostigmine  
**Ref:** KDT 6th edition various chapters

**Some alkaloids used as drugs are:**
- Local anesthetic and stimulant cocaine
- The psychedelic psilocin
- The stimulant caffeine; nicotine
- The analgesic morphine
- The antibacterial berberine
- The anticancer compound vincristine
- The antihypertension agent reserpine
- The cholinomimetic galatamine
- The spasmyloytic agent atropine
- The vasodilator vincamine
- The anti-arrhythmia compound quinidine
- The anti-asthma therapeutic ephedrine
- The antimalarial drug quinine
- The emetic emetine

**Answer:** (C) Constipation  
**Ref:** KDT 6th edition page 457  
Tolerance is exhibited to all actions except miosis and constipation

**Answer:** (B) Ethambutol  
**Ref:** KDT 6th edition page 703  
**Bacteriostatic anti tubercular drugs are:**
- Thiactazone
- Ethionamide
- Ethambutol
- PAS
- Cycloserine
(113) ANSWER: (B) Rifampicin
REF: Park 20th edition page 151
Repeat from December 2009
“Rifampicin is the drug of choice for chemoprophylaxis of meningococcal meningitis”
Current recommendations regarding chemoprophylaxis of close contacts are early institution of Rifampicin 600 mg twice a day for 2 days for adults, or minocycline 100 mg every 12 hours.

(114) ANSWER: (B) Used in treatment of meningococcal meningitis
REF: KDT 5th edition page 701
• Rifampicin is used in prophylaxis of meningococcal meningitis not treatment
• Rifampicin is a microsomal enzyme inducer, hence it raises metabolism of OCP and hence may cause it failure

(115) ANSWER: (B) Tyrosine kinase
Imatinib is the drug of choice for chronic phase of CML
MOA:
Imatinib inhibits protein tyrosine kinase activities of ABL and its activated derivatives v-ABL, BCR-ABL, and EVT6-ABL, the platelet-derived growth factor receptor (PDGFR), and KIT receptor tyrosine kinases.
Adverse effects:
Imatinib is administered orally. The main side effects are fluid retention, nausea, muscle cramps, diarrhea, and skin rashes. The management of these side effects is usually supportive. Myelosuppression is the most common hematologic side effect. Myelosuppression, while rare, may require holding drug and/or growth factor support. Doses <300 mg/d seem ineffective and may lead to development of resistance.

(116) ANSWER: (D) All above
REF: Harrison’s 18th edition chapter 75

**DRUGS USED IN TREATMENT OF OBESITY:**

<table>
<thead>
<tr>
<th>Drug Type</th>
<th>Drug Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serotonin and norepinephrine reuptake inhibitor</td>
<td>Sibutramine</td>
</tr>
<tr>
<td>Synthetic hydrogenated derivative of a naturally occurring lipase inhibitor</td>
<td>Orlistat</td>
</tr>
<tr>
<td>Selective cannabinoid CB1 receptor antagonist</td>
<td>Rimonabant</td>
</tr>
</tbody>
</table>

(117) ANSWER: (A) DNA dependent RNA polymerase
REF: Goodman & Gillman 2008 edition page 786
Rifampin forms a stable complex with DNA dependent RNA polymerase, leading to suppression of initiation of chain formation (but not chain elongation) in RNA synthesis. High concentrations of rifampin can inhibit RNA synthesis in mammalian mitochondria, viral DNA–dependent RNA polymerases, and reverse transcriptases. Rifampin is bactericidal for both intracellular and extracellular microorganisms.

(118) ANSWER: (B) Used in treatment of gynecomastia
Estrogen receptor antagonists are used in the treatment of gynecomastia not estrogen

Adverse effects of Estrogen:
- Suppression of libido and gynecomastia
- Fusion of epiphyses
- Endometrial carcinoma
- Increased incidence of breast cancer
- Increased incidence of cholestasis and gall stones
- Increased chances of thromboembolism

(119) ANSWER: (C) Stanozolol

This question is based upon anabolic steroids
- Of the anabolic steroid the 17 alpha substituted ones are most hepatotoxic.
- 17 alpha anabolic steroids: Oxandrolone, stanozolol, oxymetholone
- Oxandrolone: risk of hepatic toxicity is much lower than other 17 alpha anabolic steroids

(120) ANSWER: (A) Potassium iodide

“The fastest acting anti-thyroid agent is iodine itself, reducing thyroid hormone synthesis within three days through a presumed autoregulatory mechanism”

ANTITHYROID COMPOUNDS

<table>
<thead>
<tr>
<th>Process Affected</th>
<th>Examples of Inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active transport of iodide Complex anions</td>
<td>Perchlorate, F-fluoborate, Pertechnetate, Thiocyanate</td>
</tr>
<tr>
<td>Iodination of thyroglobulin Thionamides</td>
<td>Propylthiouracil, Methimazole, Carbimazole Thiocyanate; iodide, Aniline derivatives; sulfonamides</td>
</tr>
<tr>
<td>Coupling reaction</td>
<td>Thionamides; sulfonamides</td>
</tr>
<tr>
<td>Hormone release</td>
<td>Lithium salts, iodide, Lodothyrosine</td>
</tr>
<tr>
<td>Iodothyronine deiodination</td>
<td>Nitrothyroxines</td>
</tr>
<tr>
<td>Peripheral deiodination</td>
<td>Thiouracil derivatives; Amiodarone Oral cholecystographic agents</td>
</tr>
<tr>
<td>Hormone action</td>
<td>Thyroxine analogs; Amiodarone</td>
</tr>
<tr>
<td>Hormone excretion inactivation</td>
<td>Inducers of hepatic drug-metabolizing enzymes: phenobarbital, rifampin, carbamazepine, phenytoin</td>
</tr>
<tr>
<td>Binding in gut</td>
<td>Cholestyramine</td>
</tr>
</tbody>
</table>

(121) ANSWER: (D) Prednisolone

TOPICAL STEROIDS:

Group I
- Very potent: up to 600 times stronger than hydrocortisone
- Clobetasol propionate 0.05% (Dermovate)
• Betamethasone dipropionate 0.25% (Diprolene)
• Halobetasol propionate 0.05% (Ultravate, Halox)
• Diflurorasone diacetate 0.05% (Psorcon)

Group II
• Fluocinonide 0.05% (Lidex)
• Halcinonide 0.05% (Halog)
• Amcinonide 0.05% (Cyclocort)
• Desoximetasone 0.25% (Topicort)

Group III
• Triamcinolone acetonide 0.5% (Kenalog, Aristocort cream)
• Mometasone furoate 0.1% (Elocon ointment)
• Fluticasone propionate 0.005% (Cutivate)
• Betamethasone dipropionate 0.05% (Diprosone)

Group IV
• Fluocinolone acetonide 0.01-0.2% (Synalar, Synemol, Fluonid)
• Hydrocortisone valerate 0.2% (Westcort)
• Hydrocortisone butyrate 0.1% (Locoid)
• Flurandrenolide 0.05% (Cordran)
• Triamcinolone acetonide 0.1% (Kenalog, Aristocort A ointment)
• Mometasone furoate 0.1% (Elocon cream, lotion)

Group V
• Triamcinolone acetonide 0.1% (Kenalog, Aristocort, kenacort-a vail, cream, lotion)
• Fluticasone propionate 0.05% (Cutivate cream)
• Desonide 0.05% (Tridesilon, DesOwen ointment)
• Fluocinolone acetonide 0.025% (Synalar, Synemol cream)
• Hydrocortisone valerate 0.2% (Westcort cream)

Group VI
• Alclometasone dipropionate 0.05% (Aclovate cream, ointment)
• Triamcinolone acetonide 0.025% (Aristocort A cream, Kenalog lotion)
• Fluocinolone acetonide 0.01% (Capex shampoo, Dermasmooth)
• Desonide 0.05% (DesOwen cream, lotion)

Group VII
• The weakest class of topical steroids. Has poor lipid permeability, and can not penetrate mucous membranes well.
• Hydrocortisone 2.5% (Hytone cream, lotion, ointment.
• Hydrocortisone 1% (Many overthecounter brands)

(122) ANSWER: (D) All of the above

REF: Harrison 18th edition chapter 178

USES OF CIDOFOVIR:
1. Cidofovir demonstrated a statistically significant effect in delaying the progression of CMV retinitis lesions in newly diagnosed patients, as well as in previously treated patients who had failed other therapies.
2. Cidofovir has shown efficacy as an anti-HSV, anti-VZV and as an anti-CMV
3. Cidofovir has also shown efficacy in the treatment of acyclovir resistant herpes
4. Cidofovir has also been investigated as a treatment for progressive multifocal leukoencephalopathy, but as of 2005 studies are inconclusive.
5. Cidofovir might have anti-smallpox efficacy and might be used on a limited basis in the event of a bioterror incident involving smallpox cases. In fact, there is a very high chance for Cidofovir to work against smallpox. A cidofovir derivative with much higher activity against smallpox that can be taken orally has been developed.
6. Cidofovir shows anti-BK virus activity in a subgroup of transplant patients
7. Cidofovir is being investigated as a complementary intralesional therapy against papillomatosis caused by HPV.
8. It has been suggested as an antitumor agent, due to its suppression of FGF2.

(123) ANSWER: (A) Death
The term Thanatology is of Greek origin; Thanatos meaning death and logus meaning the science. In other words it means the subject which deals with scientific study of death, types of death, the various events or changes that occur in the cadaver and their medicolegal significance.

(124) ANSWER: (A) Single edged knife
REF: Oxford Handbook of Forensic Medicine by Jonathan P. Wyatt, Tim Squires, Guy Norfolk Page 135
Types of stab wound on the basis of weapon used:
- Double sharp-edged blades can cause linear wounds with two ‘pointed’ or V-shaped ends.
- Single sharp-edged blades can result in one pointed end and one “fishtail” or ‘squared off’ wound, the latter reflecting localized blunt trauma from the blunt edge, with localised tearing and bruising of skin.
- A bayonet is a knife-like attachment put on the end of a gun for melee combat.

(125) ANSWER: (B) Oral coitus
REF: Textbook Of Forensic Medicine And Toxicology: Principles And Practice 4th edition by Krishan vij, Forensic Medicine and Toxicology by R.N.Karmakar Page 455
BUCCAL COITUS (ORAL COITUS OR SIN OF GOMORRAH)
This offence is mentioned in the Bible. It used to be practised in the town of Gomorrah and hence the name, Sin of Gomorrah, is attributed to this practice. Oral coitus may be practised by both sexes

(126) ANSWER: (C) Arsenic
Typically, after acute arsenic and thallium poisoning the nails show multiple bands of transverse leukonychia distributed along the whole width of the nail plate (Mee’s lines).
## APPENDIX – 1

### Cranial Nerve Motor Nuclei

<table>
<thead>
<tr>
<th>Nucleus</th>
<th>Position</th>
<th>Cranial Nerve</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Somatic motor – voluntary</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oculomotor</td>
<td>Upper midbrain</td>
<td>Oculomotor III</td>
<td>Eyeball movements: extrinsic ocular muscles</td>
</tr>
<tr>
<td>Trochlear</td>
<td>Lower midbrain</td>
<td>Trochlear IV</td>
<td>Eyeball movements: extrinsic ocular muscles</td>
</tr>
<tr>
<td>Abducens</td>
<td>Pons</td>
<td>Abducens VI</td>
<td>Eyeball movements: extrinsic ocular muscles</td>
</tr>
<tr>
<td>Hypoglossal</td>
<td>Medulla</td>
<td>Hypoglossal XII</td>
<td>Tongue muscles and movements</td>
</tr>
<tr>
<td><strong>Branchiomotor (special visceral motor) – voluntary</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trigeminal</td>
<td>Pons</td>
<td>Mandibular Vc (1st)</td>
<td>Chewing, tensor tympani</td>
</tr>
<tr>
<td>Facial</td>
<td>Pons</td>
<td>Facial VII (2nd)</td>
<td>Facial expression, Buccinator, stapedius</td>
</tr>
<tr>
<td>Nucleus ambiguus</td>
<td>Medulla</td>
<td>Glossopharyngeal IX (3rd) Vagus X, various branches (4th) Vagus (X), recurrent laryngeal (sixth)</td>
<td>Muscles of swallowing and phonation</td>
</tr>
<tr>
<td>Cervical accessory nucleus</td>
<td>Upper cervical spinal cord</td>
<td>Spinal accessory XI</td>
<td>Sternocleidomastoid, trapezius</td>
</tr>
<tr>
<td><strong>Parasympathetic (general visceral motor) – involuntary</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Edinger–Westphal</td>
<td>Midbrain</td>
<td>Oculomotor III</td>
<td>Ciliary muscle: lens accommodation, iris muscle: pupilloconstriction</td>
</tr>
<tr>
<td>Salivatory: superior</td>
<td>Pons</td>
<td>Facial VII</td>
<td>Secretomotor: lacrimal, nasal, palate, submandibular, sublingual glands</td>
</tr>
<tr>
<td>Salivatory: inferior</td>
<td>Upper medulla</td>
<td>Glossopharyngeal IX</td>
<td>Secretomotor: parotid gland</td>
</tr>
<tr>
<td>Dorsal motor of vagus</td>
<td>Medulla</td>
<td>Vagus X</td>
<td>Heart, foregut and midgut derivatives</td>
</tr>
</tbody>
</table>

Cranial nerve with branchial arch in brackets
## Types of Cartilages

<table>
<thead>
<tr>
<th></th>
<th>Hyaline Cartilage</th>
<th>Elastic Cartilage</th>
<th>Fibrocartilage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Composition</strong></td>
<td>Type II collagen, Chondroitin sulfate</td>
<td>Type II collagen, Elastin fibers</td>
<td>Type I collagen</td>
</tr>
<tr>
<td><strong>Perichondrium</strong></td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Example</strong></td>
<td>Articular cartilage, Arytenoid cartilage, Thyroid cartilage, Cricoid cartilage, Epiphyseal growth plate, Nasal septum, Costal cartilage of ribs</td>
<td>Auricle, Auditory tube, Epiglottis, Cuneiform cartilage of larynx, Epiglottis, External auditory canal</td>
<td>Articular discs, Intervertebral discs, Menisci, Pubic symphysis, Glenoid labrum, Acetabular labrum</td>
</tr>
</tbody>
</table>
## Gait Abnormalities

<table>
<thead>
<tr>
<th>Gait</th>
<th>Also Known as</th>
<th>Description</th>
<th>Found In</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemiparetic gait</td>
<td>Circumduction gait, Pyramidal gait</td>
<td>Mild cases: only indication of paresis may be that the ball/heel of the shoe may be worn more on the affected side. Severe cases: knee extended, ankle plantar flexed. Patient therefore circumducts the affected leg to ambulate</td>
<td>Hemiplegia (upper motor neuron disease)</td>
</tr>
<tr>
<td>Ataxic gait</td>
<td>Broad based gait</td>
<td>patient spreads legs apart to widen the base to compensate for the imbalance while standing or walking</td>
<td>Cerebellar sign, alcoholism</td>
</tr>
<tr>
<td>Lurching gait</td>
<td>Jack knife gait, Gluteus Maximus Lurch</td>
<td>The trunk lurches back on the stance phase side hyperextending</td>
<td>Gluteus Maximus weakness</td>
</tr>
<tr>
<td></td>
<td>Trendelenburg's gait, Gluteus medius lurch</td>
<td>The trunk shifts over the side of the weak muscle in stance phase to minimise the fall of the swing phase side of the pelvis.</td>
<td>Hip dislocation Coxa vara Short limb Unilateral superior gluteal nerve injury</td>
</tr>
<tr>
<td>Waddling gait</td>
<td>Myopathic gait</td>
<td>The patient uses circumduction to compensate for gluteal weakness</td>
<td>Muscular dystrophy Bilateral superior gluteal nerve injury</td>
</tr>
<tr>
<td>Shuffling gait</td>
<td>Festinating gait</td>
<td>Short steps, appears to shuffle his or her legs rather than put them forward. steps and pace may vary with a tendency for the patient to accelerate</td>
<td>Parkinson's disease (extrapyramidal)</td>
</tr>
<tr>
<td>Stepping gait</td>
<td>high-stepping, slapping</td>
<td>high steps as if climbing stairs while walking on a level surface, trying to avoid injury to the feet (from dragging them) by stepping high</td>
<td>peripheral neuropathies (bilateral foot drop)</td>
</tr>
<tr>
<td>Spastic gait</td>
<td>Scissoring gait</td>
<td>legs are held in adduction at the hip and the thighs rub against each other as the patient walks</td>
<td>Cerebral diplegia (cerebral palsy)</td>
</tr>
<tr>
<td>Charlie Chaplin gait</td>
<td>Out toe gait</td>
<td>‘spread-eagle’ or frog leg position</td>
<td>Tibial torsion</td>
</tr>
<tr>
<td>Antalgic gait</td>
<td></td>
<td>Patient favours the affected painful extremity and walks, putting weight on the normal leg</td>
<td>Limb pain</td>
</tr>
</tbody>
</table>
## Lymphatic Drainage of Perineal Structures:

<table>
<thead>
<tr>
<th>Lymph Node Group</th>
<th>Lymph Nodes Drained</th>
<th>Structures Drained In Males</th>
<th>Structures Drained In Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumbar/Aortic</td>
<td>Common iliac nodes</td>
<td>Testis, Testicular vessels, Epididymis</td>
<td>Ovaries, Ovarian vessels, fundus &amp; upper part of body of uterus, uterine tubes (except isthmus &amp; intrauterine parts)</td>
</tr>
<tr>
<td>Inferior mesenteric</td>
<td>Pararectal nodes</td>
<td>Sigmoid colon, Descending colon, Superior most rectum</td>
<td>Sigmoid colon, Descending colon, Superior most rectum</td>
</tr>
<tr>
<td>Common iliac</td>
<td>External &amp; Internal iliac nodes</td>
<td>Base of bladder, Inferior part of pelvic ureters, Prostate, Prostatic urethra, Membranous urethra, Seminal vesicles, Cavernous bodies, Anal canal (above pectinate line), inferior rectum</td>
<td>Base of bladder, Inferior part of pelvic ureters, Anal canal (above pectinate line), inferior rectum, cervix, middle &amp; lower 1/3 vagina</td>
</tr>
<tr>
<td>Internal iliac node</td>
<td>Sacral nodes</td>
<td>Base of bladder, Inferior part of pelvic ureters, Prostate, Prostatic urethra, Membranous urethra, Seminal vesicles, Cavernous bodies, Anal canal (above pectinate line), inferior rectum</td>
<td>Base of bladder, Inferior part of pelvic ureters, Anal canal (above pectinate line), inferior rectum, cervix, middle &amp; lower 1/3 vagina</td>
</tr>
<tr>
<td>External iliac nodes</td>
<td>Deep inguinal nodes</td>
<td>Superior aspect of bladder, Superior part of pelvic ureters, spongy urethra (minor)</td>
<td>Superior aspect of bladder, Superior part of pelvic ureters, upper 1/3 vagina, cervix, lower body of uterus</td>
</tr>
<tr>
<td>Superficial inguinal nodes</td>
<td></td>
<td>Lower limbs, Anterior abdominal wall inferior to umbilicus, Skin of perineum, Gluteal region, Anal canal (below pectinate line), Prepuce of penis, Scrotum</td>
<td>Lower limbs, Anterior abdominal wall inferior to umbilicus, Skin of perineum, Gluteal region, Anal canal (below pectinate line), vulva, Prepuce of clitoris, vaginal ostium (below hymen)</td>
</tr>
<tr>
<td>Deep inguinal nodes</td>
<td>Superficial inguinal nodes</td>
<td>Glans of penis, Spongy urethra (major)</td>
<td>Glans of clitoris</td>
</tr>
<tr>
<td>Sacral nodes</td>
<td>Inferior rectum</td>
<td>Inferior rectum, inferior vagina</td>
<td></td>
</tr>
<tr>
<td>Pararectal nodes</td>
<td>Superior rectum</td>
<td>Superior rectum</td>
<td></td>
</tr>
</tbody>
</table>
## APPENDIX - 5

### Adult Derivatives and Vestigial Remains of Embryonic Urogenital Structures

<table>
<thead>
<tr>
<th>Embryonic Structure</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gonadal Ridge</strong></td>
<td><strong>Testis</strong></td>
<td><strong>Ovary</strong></td>
</tr>
<tr>
<td><strong>Cortex</strong></td>
<td><strong>Seminiferous tubules</strong></td>
<td><strong>Ovarian follicles</strong></td>
</tr>
<tr>
<td><strong>Medulla</strong></td>
<td><strong>Rete testis</strong></td>
<td><strong>Medulla</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Rete ovarii</strong></td>
</tr>
<tr>
<td><strong>Gubernaculum</strong></td>
<td><strong>Gubernaculum testis</strong></td>
<td><strong>Ovarian ligament</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Round ligament of uterus</strong></td>
</tr>
<tr>
<td><strong>Mesonephric tubules</strong></td>
<td><strong>Ductus efferentes</strong></td>
<td><strong>Epoophoron</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Paradidymis</strong></td>
<td><strong>Paroophoron</strong></td>
</tr>
<tr>
<td><strong>Mesonephric duct</strong></td>
<td><strong>Appendix of epididymis</strong></td>
<td><strong>Appendix vesiculosa</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Ductus epididymidis</strong></td>
<td><strong>Duct of epoophoron</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Ductus deferens</strong></td>
<td><strong>Duct of Gartner</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Ureter, pelvis, calices, and collecting tubules</strong></td>
<td><strong>Ureter, pelvis, calices, and collecting tubules</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Ejaculatory duct and seminal vesicle</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Paramesonephric duct</strong></td>
<td><strong>Appendix of testis</strong></td>
<td><strong>Hydatid (of Morgagni)</strong></td>
</tr>
<tr>
<td>(Mullerian duct)</td>
<td></td>
<td><strong>Uterine tube</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Uterus</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Upper Vagina</strong></td>
</tr>
<tr>
<td><strong>Urogenital sinus</strong></td>
<td><strong>Urinary bladder</strong></td>
<td><strong>Urinary bladder</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Urethra (except glandular portion)</strong></td>
<td><strong>Urethra</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Prostatic utricle</strong></td>
<td><strong>Lower Vagina (from sinovaginal bulb)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Prostate gland</strong></td>
<td><strong>Urethral and paraurethral glands</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Bulbourethral glands</strong></td>
<td><strong>Greater vestibular glands</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Vestibule of vagina</strong></td>
</tr>
<tr>
<td><strong>Müllerian tubercle</strong></td>
<td><strong>Seminal colliculus</strong></td>
<td><strong>Hymen</strong></td>
</tr>
<tr>
<td><strong>Genital tubercle</strong></td>
<td><strong>Penis</strong></td>
<td><strong>Clitoris</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Glans penis</strong></td>
<td><strong>Glans clitoridis</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Corpora cavernosa penis</strong></td>
<td><strong>Corpora cavernosa clitoridis</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Corpus spongiosum</strong></td>
<td><strong>Bulb of the vestibule</strong></td>
</tr>
<tr>
<td><strong>Urogenital folds</strong></td>
<td><strong>Ventral aspect of penis</strong></td>
<td><strong>Labia minora</strong></td>
</tr>
<tr>
<td><strong>Labioscrotal swellings</strong></td>
<td><strong>Scrotum</strong></td>
<td><strong>Labia majora</strong></td>
</tr>
</tbody>
</table>
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.

<table>
<thead>
<tr>
<th>Pharyngeal Arch</th>
<th>Muscular Contributions</th>
<th>Skeletal Contributions</th>
<th>Nerve</th>
<th>Artery</th>
<th>Corresponding Pouch Structures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st (mandibular arch)</td>
<td>Muscles of mastication, Anterior belly of the digastric, Mylohyoid, Tensor tympani, Tensor veli palatini</td>
<td>Maxilla, mandible (only as a model for mandible not actual formation of mandible), Incus and Malleus, Meckel's cartilage, Ant. ligament of malleus, Sphenomandibular ligament</td>
<td>Trigeminal nerve (V2 and V3)</td>
<td>Maxillary artery, external carotid artery</td>
<td>Eustachian tube, middle ear, mastoid antrum, and inner layer of the tympanic membrane.</td>
</tr>
<tr>
<td>2nd (hyoid arch)</td>
<td>Muscles of facial expression, Buccinator, Platysma, Stapedius, Stylohyoid, Posterior belly of the digastric</td>
<td>Stapes, Styloid process, hyoid (lesser horn and upper part of body), Reichert's cartilage, Stylohyoid ligament</td>
<td>Facial nerve (VII)</td>
<td>Stapedial Artery</td>
<td>middle ear, palatine tonsils</td>
</tr>
<tr>
<td>3rd</td>
<td>Stylopharyngeus</td>
<td>Hyoid (greater horn and lower part of body), thymus</td>
<td>Glossopharyngeal nerve (IX)</td>
<td>Common carotid/Internal carotid</td>
<td>Inferior parathyroid, Thymus</td>
</tr>
<tr>
<td>4th</td>
<td>Cricothyroid muscle, all intrinsic muscles of soft palate including levator veli palatini</td>
<td>Thyroid cartilage, epiglottic cartilage</td>
<td>Vagus nerve (X) Superior laryngeal nerve</td>
<td>Right 4th aortic arch: subclavian artery Left 4th aortic arch: aortic arch</td>
<td>Superior parathyroid, ultimobranchial body (which forms the Para follicular C-Cells of thyroid gland)</td>
</tr>
<tr>
<td>6th</td>
<td>All intrinsic muscles of larynx except the cricothyroid muscle</td>
<td>Cricoid cartilage, arytenoid cartilages, corniculate cartilage</td>
<td>Vagus nerve (X) Recurrent laryngeal nerve</td>
<td>Right 6th aortic arch: pulmonary artery Left 6th aortic arch: Pulmonary artery and ductus arteriosus</td>
<td>Rudimentary structure, becomes part of the fourth pouch contributing to thyroid C-cells.</td>
</tr>
</tbody>
</table>
Pattern of the branchial arches
I-IV Branchial arches
1-4 Branchial pouches (inside)
Pharyngeal grooves (outside)
a Tuberculum laterale
b Tuberculum impar
c Foramen cecum
d Ductus thyreoglossus
e Sinus cervicalis
**APPENDIX - 7**

**Brachial Plexus Lesions-chart 1**

<table>
<thead>
<tr>
<th>Nerve (Segment)</th>
<th>Motor Deficit(s)</th>
<th>Sensory Deficits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long Thoracic (C 5, 6, 7)</td>
<td>Winged Scapula- Serratus Anterior</td>
<td>None</td>
</tr>
<tr>
<td>Suprascapular (C 5, 6)</td>
<td>Hard to start shoulder abduction - Supraspinatus</td>
<td>None</td>
</tr>
<tr>
<td>Axillary (C 5, 6)</td>
<td>Difficult abducting arm to horizontal - Deltoid</td>
<td>Lateral side of arm below point of shoulder</td>
</tr>
<tr>
<td>Musculocutaneous C 5, 6, (7)</td>
<td>Very weak flexion of elbow joint- Biceps &amp; Brachialis</td>
<td>Lateral forearm</td>
</tr>
<tr>
<td>Radial (C 5 - T1)</td>
<td>Drop Wrist - Extensor carpi radialis longus &amp; brevis, Ext. carpi ulnaris</td>
<td>Posterior lateral &amp;arm; dorsum of hand</td>
</tr>
<tr>
<td>Median (C 5 - T1) at Elbow</td>
<td>Pronation of radioulnar joints-Pronator teres &amp; quadratus</td>
<td>Radial portion of palm; palmar surface &amp; tips of radial 3½ digits</td>
</tr>
<tr>
<td>Median (C 5 - T1) at Wrist</td>
<td>Weakened opposition of thumb - thenar muscles</td>
<td>Palmar surface &amp; tips of radial 3½ digits</td>
</tr>
<tr>
<td>Ulnar (C 8, T1) at Elbow</td>
<td>&quot;Clawing&quot; of fingers 3 &amp; 4- M.P. joints hyper extended; P.I.P. Flexed - Interossei &amp; Lumbricalis</td>
<td>Ulnar and dorsal aspect of palm and of ulnar 1½ digits</td>
</tr>
<tr>
<td>Ulnar (C 8, T1) at Wrist</td>
<td>&quot;Clawing&quot; of fingers 3 &amp; 4- M.P. joints hyper extended; P.I.P. Flexed - Interossei &amp; Lumbricalis</td>
<td>Ulnar and dorsal aspect of palm and of ulnar 1½ digits</td>
</tr>
</tbody>
</table>
## Upper and Lower Root Lesions - chart 2

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Motor Deficits</th>
<th>Sensory Deficits</th>
<th>Nerves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erb’s Palsy</td>
<td>Loss of abduction, flexion and rotation at shoulder; Weak shoulder extension - deltoid, rotator cuff</td>
<td>Posterior and lateral aspect of arm - axillary n.</td>
<td>Axillary, Suprascapular, Upper and Lower subscapular</td>
</tr>
<tr>
<td>Klumke’s Palsy</td>
<td>Loss of opposition of thumb - Thenar muscles</td>
<td>Ulnar side of forearm, hand &amp; ulnar 1½ &amp; digits - ulnar and medial antebrachial cutaneous</td>
<td>Thenar branch of Median nerve</td>
</tr>
<tr>
<td></td>
<td>Loss of adduction of thumb - Adductor pollicis</td>
<td></td>
<td>Ulnar nerve</td>
</tr>
<tr>
<td></td>
<td>Loss of following finger movements: abduction and adduction of M.P. joints; flexion at M.P. &amp; extension of I.P. joints, Lumbricals &amp; interossei</td>
<td></td>
<td>Deep branch of Ulnar &amp; Median</td>
</tr>
</tbody>
</table>
Dermatomal Distribution:
Dermatomal distribution is important to determine the site of lesion and is repeatedly required to solve case scenario type questions other than the direct questions. A few things that might help you in remembering this chart are:

- Divide body into 5 segments
  1. Head/face (supplied by cranial nerve 5)
  2. Neck and upper limb (supplied by C)
  3. Thorax and abdomen (supplied by T)
  4. Lower limb (supplied by L)
  5. Perineum (supplied by S)
- C1 does not take part in dermatomal distribution as it is busy with the spinal accessory nerve (11th cranial nerve).
- First spinal nerve of each (cervical, thoracic, lumbar and sacral) actually goes to the superior/previous body component i.e.
  - C2 (in place of C1) goes to head in place supplying cervical region.
  - T1 goes to upper limb in place of supplying thorax.
  - L1 goes to the abdomen (above the inguinal ligament and small part below it).
  - S1 goes to lower limb instead of supplying perineum.
- Now you know the upper limit and lower limit of each segment. Fill up the rest.
Vertebral Levels (Mnemonic: All Bifurcations (BI4-cation) are at The Level of 4)

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td>Hyoid bone; Bifurcation of common carotid artery.</td>
</tr>
<tr>
<td>C5</td>
<td>Thyroid cartilage</td>
</tr>
</tbody>
</table>
| C6    | Cricoid cartilage  
|       | Trachea begins  
|       | Oesophagus begins  
|       | Pharynx and larynx ends |
| C7    | Thoracic duct reaches its greatest height; Isthmus of thyroid gland |
| T1    | Sternoclavicular joint; Highest point of apex of lung. |
| T2    | Sternal notch  
|       | Jugular notch |
| T4    | Sternal angle (of Louis)  
|       | Junction of superior and inferior mediastinum  
|       | Ascending aorta ends  
|       | Arch of aorta begins and ends.  
|       | Bifurcation of trachea |
| T8    | IVC hiatus |
| T9    | Xiphisternal joint |
| T10   | Oesophageal hiatus |
| T12   | Aortic hiatus  
|       | Thoracic duct through diaphragm  
|       | Azygos vein through diaphragm |
| L1    | End of spinal cord in adults  
|       | Transpyloric plane  
|       | Pylorus of stomach  
|       | Superior mesenteric artery origin  
|       | Hilum of kidneys (renal artery–left is above and right is below)  
|       | Celiac artery originates just above and renal arteries originate just below this line. |
| L2    | Thoracic duct begins; Azygos and hemiazygos begin |
| L3    | Umbilicus  
|       | End of spinal cord in newborns  
|       | Inferior mesenteric artery |
| L4    | Iliac crest  
|       | Aorta bifurcates into common iliac arteries.  
|       | Inferior vena cava formed from common iliac veins. |
| S1    | Sacral promontory |
| S2    | Posterior superior iliac spine  
|       | End of dural sac (Dura, arachnoid, subarachnoid space, CSF)  
|       | Middle of sacroiliac joint |
| S3    | Posterior inferior iliac spine  
|       | End of sigmoid colon  
|       | Rectum begins (important landmark in surgery of recto sigmoid carcinoma). |
## Common Types of Covering Epithelia in Human Body

<table>
<thead>
<tr>
<th>Type</th>
<th>Cell Form</th>
<th>Examples of distribution</th>
<th>Main Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple</td>
<td>Squamous</td>
<td>Lining of vessels (endothelium). Serous lining of cavities; pericardium, pleura, peritoneum (mesothelium).</td>
<td>Facilitates the movement of the viscera (mesothelium), active transport by pinocytosis (mesothelium and endothelium), and secretion of biologically active molecules (mesothelium).</td>
</tr>
<tr>
<td></td>
<td>Cuboidal</td>
<td>Covering the ovary, thyroid.</td>
<td>Covering, secretion.</td>
</tr>
<tr>
<td></td>
<td>Columnar</td>
<td>Lining of intestine, gallbladder.</td>
<td>Protection, lubrication, absorption, secretion.</td>
</tr>
<tr>
<td>Pseudod-stratified</td>
<td>Some columnar and some cuboidal</td>
<td>Lining of trachea, bronchi, nasal cavity.</td>
<td>Protection, secretion; cilia-mediated transport of particles trapped in mucus.</td>
</tr>
<tr>
<td>Stratified</td>
<td>Surface layer squamous keratinized (dry)</td>
<td>Epidermis.</td>
<td>Protection; prevents water loss.</td>
</tr>
<tr>
<td></td>
<td>Surface layer squamous nonkeratinized (moist)</td>
<td>Mouth, esophagus, larynx, vagina, anal canal.</td>
<td>Protection, secretion; prevents water loss.</td>
</tr>
<tr>
<td></td>
<td>Cuboidal</td>
<td>Sweat glands, developing ovarian follicles.</td>
<td>Protection, secretion.</td>
</tr>
<tr>
<td></td>
<td>Transitional: domelike to flattened, depending on the functional state of the organ</td>
<td>Bladder, ureters, renal calyces.</td>
<td>Protection, distensibility.</td>
</tr>
<tr>
<td></td>
<td>Columnar</td>
<td>Conjunctiva.</td>
<td>Protection.</td>
</tr>
</tbody>
</table>
**Sleep Physiology**

<table>
<thead>
<tr>
<th></th>
<th>Awake</th>
<th>Nrem Sleep</th>
<th>Rem Sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AKA</strong></td>
<td></td>
<td>Slow wave sleep cycle</td>
<td>Paradoxical sleep (EEG pattern similar to awake)</td>
</tr>
<tr>
<td><strong>EEG</strong></td>
<td><strong>Beta Wave</strong>: Day to day wakefulness, highest in frequency and lowest in amplitude, and also more desynchronous than other waves. Best recorded at parietal and occipital region. <strong>Alpha Wave</strong>: During periods of relaxation, while still awake (eyes closed), slower, increase in amplitude and become more synchronous. Best recorded at parietal and frontal region.</td>
<td><strong>Stage 1 Nrem</strong> → <strong>theta Waves</strong>: The first stage of sleep is characterized by theta waves, which are even slower in frequency and greater in amplitude than alpha waves. <strong>Stage 2 Nrem</strong> → (1 &amp; 2 are light sleep stages) <strong>Theta Waves</strong>: continues along with two unusual wave phenomena <strong>K Complexes</strong>: sudden increase in wave amplitude. <strong>Sleep Spindles</strong>: sudden increase in wave frequency. <strong>Stage 3 Nrem</strong> → <strong>delta Wave</strong>: slowest and highest amplitude brain waves (&lt;50% delta waves) <strong>Stage 4 Nrem</strong> → &gt; 50% delta waves (stage 3&amp;4=deep sleep, slow-wave sleep)</td>
<td><strong>Stage 5 Sleep</strong> → Rapid low voltage EEG similar to awake (combination of alpha, beta, and desynchronous waves)</td>
</tr>
<tr>
<td><strong>PGO (Ponto-geniculo-occipital) waves</strong></td>
<td>Present (Corresponds to eye movement of both awake and sleep)</td>
<td>Absent</td>
<td>Present (most prominent just before REM sleep, First sign of REM sleep)</td>
</tr>
<tr>
<td><strong>EMG</strong></td>
<td></td>
<td></td>
<td>Loss of muscle tone (REM atonia) - non reciprocal flaccid paralysis of both flexors and extensors.</td>
</tr>
<tr>
<td><strong>EOG</strong></td>
<td>Rapid eye movement</td>
<td>Non rapid eye movement</td>
<td>Rapid eye movement (slower compared to those of the awake state)</td>
</tr>
<tr>
<td><strong>THERMO-REGULATION</strong></td>
<td>Regulated at normal set point</td>
<td>Regulated at lower set point, shivering and sweating thresholds decreases</td>
<td>Not well regulated, no response to hot and cold, shivering or sweating thresholds disturbed</td>
</tr>
<tr>
<td><strong>RS</strong></td>
<td>Regular breathing</td>
<td>Regular breathing- exclusively under chemical/mechanical control, ( \downarrow )PO2, ( \uparrow )PCO2, 15% fall in minutes volume, ( \downarrow )hypoxic ventilator response</td>
<td>Irregular breathing- due to lack of chemical control and depends on only higher cortical drive, ( \downarrow \downarrow ) hypoxic ventilator response</td>
</tr>
<tr>
<td><strong>URINE</strong></td>
<td>Concentrated urine</td>
<td>( \uparrow )concentration, ( \downarrow )Quantity</td>
<td>( \uparrow \uparrow )concentration, ( \downarrow \downarrow )Quantity</td>
</tr>
</tbody>
</table>
CVS

<table>
<thead>
<tr>
<th>CVS</th>
<th>↓ BP, ↓ CO, cerebral blood flow ↑ a little</th>
<th>Tonic Rem BP &amp; CO same as NREM Cerebral blood flow ↑ by more than 50%</th>
<th>Phasic Rem BP &amp; CO- variable, may increase up to 30% above normal, Cerebral blood flow -200% above normal</th>
</tr>
</thead>
</table>

ANS

<table>
<thead>
<tr>
<th>ANS</th>
<th>Sympathetic (++) Parasympathetic (++)</th>
<th>Sympathetic same as awake (++) Parasympathetic slightly increased (+++)</th>
<th>Tonic Rem Sympathetic decrease (+) Parasympathetic same as NREM (+++)</th>
<th>Phasic Rem Sympathetic &gt; parasympathetic (both increases)</th>
</tr>
</thead>
</table>

GENITAL

<table>
<thead>
<tr>
<th>GENITAL</th>
<th>Parasympathetic predominance causes penile erection, clitoris erection and vaginal engorgement.</th>
</tr>
</thead>
</table>

DREAMING

<table>
<thead>
<tr>
<th>DREAMING</th>
<th>Day dreaming Hypnogogic hallucination</th>
<th>Dreams cannot be remembered</th>
<th>Dreams can be remembered</th>
</tr>
</thead>
</table>

DISORDERS

<table>
<thead>
<tr>
<th>DISORDERS</th>
<th>Narcolepsy: Excessive daytime sleepiness is often associated with: Cataplexy (sudden loss of muscle tone and paralysis of voluntary muscles), Hypnagogic hallucinations (pre-sleep dreams) &amp; Automatic behaviours (doing something &quot;automatically&quot; and not remembering afterwards)</th>
<th>Somnambulism: sleep walking Somniloquy: sleep walking Pavor nocturnus: Sleep terrors/night terrors Enuresis: bed wetting Bruxism: tooth grinding Restless legs syndrome</th>
<th>REM sleep behaviour disorder (RBD): Muscle paralysis is incomplete or absent. RBD is characterized by the acting out of dreams that are vivid, intense, and violent. Dream-enacting behaviours include talking, yelling, punching, kicking, sitting etc. Nightmares: frightening dreams that occur during REM sleep, associated with tachycardia, tachypnoea, profuse sweating, and arousal Catathrenia: breath holding and expiratory groaning during sleep</th>
</tr>
</thead>
</table>

Also Know:

In a normal night’s sleep, a sleeper begins in stage 1, moves down through the stages, to stage 4, then back up through the stages, with the exception that stage 1 is replaced by REM, then the sleeper goes back down through the stages again. One cycle, from stage 1 to REM takes approximately 90 minutes. This cycle is repeated throughout the night, with the length of REM periods increasing, and the length of delta sleep decreasing, until during the last few cycles there is no delta sleep at all.

The sequence of EEG waves can be memorised as “Gay BAT Dance”. From Gamma, beta, alpha, theta to delta frequency decreases and amplitude increases. (Gay as in happy)

Polysomnography (PSG) is a multi-parametric test used in the study of sleep and as a diagnostic tool in sleep medicine. The PSG monitors many body functions including brain (EEG), eye movements (EOG), muscle activity or skeletal muscle activation (EMG) and heart rhythm (ECG) during sleep.
The Hypothalamic Nuclei:

<table>
<thead>
<tr>
<th>Region</th>
<th>Area</th>
<th>Nucleus</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Medial</td>
<td>Preoptic nucleus</td>
<td>Thermo-regulation/Heat loss centre</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Supraoptic nucleus (SO)</td>
<td>vasopressin (ADH) release</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paraventricular nucleus (PV)</td>
<td>CRH release</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Oxytocin and to a lesser extent antidiuretic hormone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anterior hypothalamic nucleus (AH)</td>
<td>Thermo-regulation/Heat loss centre (set point comparison)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Heat loss if core temp &gt; set point</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Suprachiasmatic nucleus</td>
<td>Circadian rhythms</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Part of supraoptic nucleus (SO)</td>
<td>vasopressin release</td>
</tr>
<tr>
<td>Tuberal</td>
<td>Medial</td>
<td>Dorso-medial hypothalamic nucleus (DM)</td>
<td>Blood Pressure Heart Rate GI stimulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventromedial nucleus (VM)</td>
<td>satiety centre (controls eating)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lesion causes voracious appetite</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arcuate nucleus (AR)/Infundibular nucleus/Periventricular nucleus</td>
<td>Endocrine function (releasing hormones) - controls Adenohypophysis</td>
</tr>
<tr>
<td></td>
<td>Lateral</td>
<td>Lateral hypothalamic area</td>
<td>Feeding centre (thirst and hunger)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- lesion causes anorexia</td>
</tr>
<tr>
<td>Posterior</td>
<td>Medial</td>
<td>Mammillary nuclei (part of mammillary bodies) (MB)</td>
<td>memory feeding reflex</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Posterior nucleus (PN)</td>
<td>Increase blood pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pupillary dilation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thermoregulation (generates shivering, if core temp &lt; set point)</td>
</tr>
</tbody>
</table>

Note: Paraventricular nucleus is *not* to be confused with periventricular nucleus.
## APPENDIX - 13

### Mechanoreceptors

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Ending</th>
<th>Site</th>
<th>Fibre</th>
<th>Type</th>
<th>Sensation</th>
<th>Figure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pacinian corpuscle</td>
<td>Encapsulated (onion like)</td>
<td>Dermis, deep fibrous tissue</td>
<td>Aβ fibers</td>
<td>Rapid adapting</td>
<td>Pressure, Vibration (200-300 Hz), touch</td>
<td><img src="image1.png" alt="Pacinian corpuscle" /></td>
</tr>
<tr>
<td>Meissners corpuscle</td>
<td>Encapsulated</td>
<td>Non hairy superficial skin (lip, nipple, prepuce, fingertip)</td>
<td>Aβ fibers</td>
<td>Rapid adapting</td>
<td>Pressure, Vibration (50 Hz), touch</td>
<td><img src="image2.png" alt="Meissners corpuscle" /></td>
</tr>
<tr>
<td>Ruffini’s ending</td>
<td>Spray ending</td>
<td>Dermis, deep fibrous tissue</td>
<td>Aβ fibers</td>
<td>Slow adapting</td>
<td>Heat, Pressure, touch</td>
<td><img src="image3.png" alt="Ruffini’s ending" /></td>
</tr>
<tr>
<td>Merrel’s disc</td>
<td>Expanded</td>
<td>Hairy Superficial skin</td>
<td>Aβ fibers</td>
<td>Slow adapting</td>
<td>Tactile localization, continuous touch</td>
<td><img src="image4.png" alt="Merrel’s disc" /></td>
</tr>
<tr>
<td>Krause’s end bulb</td>
<td>Encapsulated</td>
<td>Skin</td>
<td>Aδ and C-fibers</td>
<td>Slow adapting</td>
<td>Cold</td>
<td><img src="image5.png" alt="Krause’s end bulb" /></td>
</tr>
<tr>
<td>Free nerve ending</td>
<td>Uncapsulated</td>
<td>Skin, muscle, viscera</td>
<td>Aδ (Fast-pricking pain) C-fiber (slow-aching pain)</td>
<td>Non adapting</td>
<td>Nociception-pain</td>
<td><img src="image6.png" alt="Free nerve ending" /></td>
</tr>
<tr>
<td>Golgi tendon organ</td>
<td>Muscle ending</td>
<td>Joint space, muscle</td>
<td>Aα fiber</td>
<td>Slow adapting</td>
<td>Proprioception, position sense</td>
<td><img src="image7.png" alt="Golgi tendon organ" /></td>
</tr>
<tr>
<td>Muscle spindle</td>
<td>Muscle ending</td>
<td>Muscle</td>
<td>Aα fiber</td>
<td>Slow adapting</td>
<td>Stretch</td>
<td><img src="image8.png" alt="Muscle spindle" /></td>
</tr>
</tbody>
</table>

- **Aβ** fibers: Rapid adapting (phasic)
- **Aδ** and **C**-fibers: Slow adapting (tonic)
- **Aα** fiber: Slow adapting (tonic)
## Renal Physiology:

<table>
<thead>
<tr>
<th>Substance</th>
<th>Description</th>
<th>Proximal Tubule</th>
<th>Loop of Henle</th>
<th>Distal Tubule</th>
<th>Collecting Duct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>If glucose is not reabsorbed by the kidney, it appears in the urine, in a condition known as glucosuria. This is associated with diabetes mellitus.</td>
<td>Reabsorption (almost 100%) via sodium-glucose transport proteins (apical) and GLUT (basolateral).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oligopeptides, proteins, and amino acids</td>
<td>All are reabsorbed nearly completely.</td>
<td></td>
<td></td>
<td></td>
<td>Reabsorption in medullary collecting ducts</td>
</tr>
<tr>
<td>Urea</td>
<td>Regulation of osmolality. Variates with ADH</td>
<td>Reabsorption (50%) via passive transport</td>
<td>Secretion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium</td>
<td>Uses Na-H antiport, Na-glucose symport, sodium ion channels (minor)</td>
<td>Reabsorption (65%, isosmotic)</td>
<td>Reabsorption (5%, sodium-chloride symporter)</td>
<td>Reabsorption (5%, principal cells), stimulated by aldosterone via ENaC</td>
<td></td>
</tr>
<tr>
<td>Chloride</td>
<td>Usually follows sodium. Active (transcellular) and passive (paracellular)</td>
<td>Reabsorption (thin ascending, thick ascending, Na-K-2Cl symporter)</td>
<td>Reabsorption (sodium-chloride symporter)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water</td>
<td>Uses aquaporin water channels. See also diuretic.</td>
<td>Absorbed osmotically along with solutes</td>
<td>Reabsorption (descending)</td>
<td>Reabsorption (regulated by ADH, via arginine vasopressin receptor 2)</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Helps maintain acid-base balance.</td>
<td>Reabsorption (80–90%)</td>
<td>Reabsorption (thick ascending)</td>
<td>Reabsorption (intercalated cells, via band 3 and pendrin)</td>
<td></td>
</tr>
<tr>
<td>Protons</td>
<td>Uses vacuolar H+ATPase</td>
<td></td>
<td></td>
<td>Secretion (intercalated cells)</td>
<td></td>
</tr>
<tr>
<td>Substance</td>
<td>Function</td>
<td>Site of Reabsorption</td>
<td>Mode of Reabsorption</td>
<td>Mode of Secretion</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td>----------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>------------------</td>
<td></td>
</tr>
<tr>
<td>Potassium</td>
<td>Varies upon dietary needs.</td>
<td>Reabsorption (65%)</td>
<td>reabsorption (20%, thick ascending, Na-K-2Cl symporter)</td>
<td>secretion (common, via Na+/K+ – ATPase, increased by aldosterone), or reabsorption (rare, hydrogen potassium ATPase)</td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>Uses calcium ATPase, sodium-calcium exchanger</td>
<td>Reabsorption</td>
<td>reabsorption (thick ascending) via passive transport</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnesium</td>
<td>Calcium and magnesium compete, and an excess of one can lead to excretion of the other.</td>
<td>Reabsorption</td>
<td>reabsorption (thick ascending)</td>
<td>Reabsorption</td>
<td></td>
</tr>
<tr>
<td>Phosphate</td>
<td>Excreted as titratable acid.</td>
<td>Reabsorption (85%) via sodium/phosphate cotransporter. Inhibited by parathyroid hormone.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carboxylate</td>
<td></td>
<td>Reabsorption (100%) via carboxylate transporters.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# APPENDIX - 15

## Tracts of Spinal Cord:

### Ascending tracts of Spinal cord:

<table>
<thead>
<tr>
<th>Situation</th>
<th>Tract</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior white funiculus</td>
<td>Anterior spinothalamic tract</td>
<td>Crude touch sensation</td>
</tr>
<tr>
<td>Lateral white funiculus</td>
<td>Lateral spinothalamic tract</td>
<td>Pain &amp; temperature sensation</td>
</tr>
<tr>
<td></td>
<td>Ventral spino cerebellar tract</td>
<td>Subconscious kinesthetic sensations</td>
</tr>
<tr>
<td></td>
<td>Dorsal spino cerebellar tract</td>
<td>Subconscious kinesthetic sensations</td>
</tr>
<tr>
<td></td>
<td>Spinoctetal tract</td>
<td>Concerned with spinovisual reflex</td>
</tr>
<tr>
<td></td>
<td>Fasiculus dorsolateralis</td>
<td>Pain &amp; temperature sensations</td>
</tr>
<tr>
<td></td>
<td>Spinoreticular tract</td>
<td>Consciousness &amp; awareness</td>
</tr>
<tr>
<td></td>
<td>Spinooolivary tract</td>
<td>Proprioception</td>
</tr>
<tr>
<td></td>
<td>Spinovestibular tract</td>
<td>Proprioception</td>
</tr>
<tr>
<td>Posterior white funiculus</td>
<td>Fasciculus gracilis</td>
<td>Tactile sensation</td>
</tr>
<tr>
<td></td>
<td>Fasciculus cuneatus</td>
<td>Tactile localization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tactile discrimination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vibratory sensation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Conscious kinesthetic sensation stereognosis</td>
</tr>
</tbody>
</table>

### Descending tracts of Spinal cord:

<table>
<thead>
<tr>
<th>Situation</th>
<th>Tract</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyramidal tracts</td>
<td>Anterior corticospinal tract</td>
<td>Control voluntary movements</td>
</tr>
<tr>
<td></td>
<td>Lateral corticospinal tract</td>
<td>Forms upper motor neurons</td>
</tr>
<tr>
<td>Extra Pyramidal tracts</td>
<td>Medial longitudinal fasciculus</td>
<td>Coordination of reflex ocular movement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Integration of movements of eyes &amp; neck</td>
</tr>
<tr>
<td></td>
<td>Anterior vestibulospinal tract</td>
<td>Maintenance of muscle tone &amp; posture</td>
</tr>
<tr>
<td></td>
<td>Lateral vestibulospinal tract</td>
<td>Maintenance of position of head &amp; body during acceleration</td>
</tr>
<tr>
<td></td>
<td>Reticulospinal tract</td>
<td>Coordination of voluntary &amp; reflex movements. Control of muscle tone.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control of respiration &amp; blood vessels.</td>
</tr>
<tr>
<td></td>
<td>Tectospinal tract</td>
<td>Control of movement of head in response to visual &amp; auditory impulses.</td>
</tr>
<tr>
<td></td>
<td>Rubrospinal tract</td>
<td>Facilitatory influence on flexor muscle tone.</td>
</tr>
<tr>
<td></td>
<td>Olivospinal tract</td>
<td>Control of movements due to proprioception.</td>
</tr>
</tbody>
</table>
## Types of Hypoxia

<table>
<thead>
<tr>
<th>Type of hypoxia</th>
<th>Pathophysiology</th>
<th>Arterial PO2</th>
<th>O2 content of arterial blood</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemic Hypoxia</td>
<td>Due to reduced O2 carrying capacity</td>
<td>Normal</td>
<td>Reduced</td>
<td>Anemia (Reduced RBC, Reduced Hb) CO poisoning</td>
</tr>
<tr>
<td>Hypoxic Hypoxia</td>
<td>Inadequate gas exchange</td>
<td>Reduced</td>
<td>Reduced</td>
<td>High altitude Respiratory disease</td>
</tr>
<tr>
<td>Histotoxic Hypoxia</td>
<td>Inability of cells to utilize available O₂</td>
<td>Normal</td>
<td>Normal</td>
<td>Cyanide poisoning</td>
</tr>
<tr>
<td>Stagnant Hypoxia</td>
<td>due to slow circulation</td>
<td>Normal</td>
<td>Normal</td>
<td>Circulatory Shock Congestive heart failure</td>
</tr>
</tbody>
</table>
## Lysosomal Storage Disorders

<table>
<thead>
<tr>
<th>(1) Lipid Storage Disorder (Sphingolipidoses)</th>
<th>Disease</th>
<th>Deficiency</th>
<th>Inheritance</th>
</tr>
</thead>
<tbody>
<tr>
<td>GM1 gangliosidoses</td>
<td>GM1 gangliosidoses</td>
<td>Beta galactosidase</td>
<td>AR</td>
</tr>
<tr>
<td>GM2 gangliosidoses</td>
<td>Tay-sach disease</td>
<td>Beta hexosaminidase –A &amp; B</td>
<td>AR</td>
</tr>
<tr>
<td>Sandhoff disease</td>
<td>Sandhoff disease</td>
<td>Beta hexosaminidase –A &amp; B</td>
<td>AR</td>
</tr>
<tr>
<td>Gaucher disease (most common LSD)</td>
<td>Glucocerebrosidase</td>
<td>AR</td>
<td></td>
</tr>
<tr>
<td>Niemann-Pick disease</td>
<td>Type-A&amp;B</td>
<td>Sphingomyelinase</td>
<td>AR</td>
</tr>
<tr>
<td>Farber’s disease</td>
<td>Ceramidase</td>
<td>AR</td>
<td></td>
</tr>
<tr>
<td>Fabry disease</td>
<td>Galectosylceramidase</td>
<td>AR</td>
<td></td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS I- Hurler syndrome</td>
<td>α-L-iduronidase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS II- Hunter syndrome</td>
<td>Iduronate sulfatase</td>
<td>XR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS III</td>
<td>Sanfilippo syndrome A</td>
<td>Heparan sulfamidase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS III</td>
<td>Sanfilippo syndrome B</td>
<td>N-acetylglucosaminidase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS III</td>
<td>Sanfilippo syndrome C</td>
<td>Acetyl-CoA:alpha-glucosaminide acetyltransferase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS III</td>
<td>Sanfilippo syndrome D</td>
<td>N-acetylglucosamine 6-sulfatase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS IV</td>
<td>Morquio syndrome A</td>
<td>Galactose-6-sulfate sulfatase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS IV</td>
<td>Morquio syndrome B</td>
<td>Beta-galactosidase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS VI- Maroteaux-Lamy syndrome</td>
<td>N-acetylglucosamine-4-sulfatase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS VII- Sly syndrome</td>
<td>β-glucuronidase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>MPS IX- Natowicz syndrome</td>
<td>Hyaluronidase</td>
<td>AR</td>
</tr>
<tr>
<td>(3) Glycoproteinosis</td>
<td>Mucolipidosis</td>
<td>Sialidosis (mucolipidosis I)</td>
<td>sialidase</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>Mucolipidosis II</td>
<td>N-acetylglucosamine-1-phosphotransferase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>Mucolipidosis III (pseudo-Hurler polydystrophy)</td>
<td>N-acetylglucosamine-1-phosphotransferase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>Fucosidosis</td>
<td>alpha-1-fucosidase</td>
<td>AR</td>
</tr>
<tr>
<td>Fabry disease</td>
<td>Mannosidosis</td>
<td>Alpha &amp; Beta-mannosidosis</td>
<td>AR</td>
</tr>
</tbody>
</table>

**NOTE:** All except Fabry’s and Hunter’s disease (x linked recessive) are autosomal recessive.
## Glycogen Storage Disorders:

<table>
<thead>
<tr>
<th>Number</th>
<th>Enzyme deficiency</th>
<th>Eponym</th>
<th>Hypoglycemia</th>
<th>Hepatomegaly</th>
<th>Hyperlipidemia</th>
<th>Muscle symptoms</th>
<th>Development/prognosis</th>
<th>Other symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSD type I</td>
<td>glucose-6-phosphatase</td>
<td>von Gierke's disease</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
<td>Growth failure</td>
<td>Lactic acidosis, hyperuricemia</td>
</tr>
<tr>
<td>GSD type II</td>
<td>acid maltase</td>
<td>Pompe's disease</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Muscle weakness</td>
<td>Death by age ~2 years (infantile variant)</td>
<td>Heart failure</td>
</tr>
<tr>
<td>GSD type III</td>
<td>glycogen debrancher</td>
<td>Cori's disease or Forbes' disease</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Myopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSD type IV</td>
<td>glycogen branching enzyme</td>
<td>Andersen disease</td>
<td>No</td>
<td>Yes, also cirrhosis</td>
<td>No</td>
<td>None</td>
<td>Failure to thrive, death at age ~5 years</td>
<td></td>
</tr>
<tr>
<td>GSD type V</td>
<td>muscle glycogen phosphorylase</td>
<td>McArdle disease</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Exercise-induced cramps, Rhabdomyolysis</td>
<td>Renal failure by myoglobinuria</td>
<td></td>
</tr>
<tr>
<td>GSD type VI</td>
<td>liver glycogen phosphorylase</td>
<td>Hers' disease</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSD type VII</td>
<td>muscle phosphofructokinase</td>
<td>Tarui's disease</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Exercise-induced muscle cramps and weakness, growth retardation</td>
<td>Haemolytic anaemia</td>
<td></td>
</tr>
<tr>
<td>GSD type IX</td>
<td>phosphorylase kinase, PHKA 2</td>
<td>–</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>None</td>
<td>Delayed motor development, Growth retardation</td>
<td></td>
</tr>
<tr>
<td>GSD type XI</td>
<td>glucose transporter, GLUT 2</td>
<td>Fanconi-Bickel syndrome</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSD type XII</td>
<td>Aldolase A</td>
<td>Red cell aldolase deficiency</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Exercise intolerance, cramps</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------</td>
<td>------------</td>
<td>------------------------------</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>-----------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSD type XIII</td>
<td>$\beta$-enolase</td>
<td>$-$</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Exercise intolerance, cramps</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Increasing intensity of myalgias over decades</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum CK: Episodic elevations; Reduced with rest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSD type 0</td>
<td>glycogen synthase</td>
<td>$-$</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Occasional muscle cramping</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Cytokines:

<table>
<thead>
<tr>
<th>Name</th>
<th>Major Cellular Source</th>
<th>Selected Biologic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>IFN-α, β</td>
<td>Macrophages (IFNα) fibroblasts (IFNβ)</td>
<td>Antiviral</td>
</tr>
<tr>
<td>IFN-γ</td>
<td>CD4+ T cells, NK cells</td>
<td>Activates macrophages, TH1 differentiation</td>
</tr>
<tr>
<td>TNF-α</td>
<td>Macrophages, T cells</td>
<td>Cell activation, Fever, cachexia, antitumor</td>
</tr>
<tr>
<td>TNF-α, LT (lymphotoxin)</td>
<td>T cells</td>
<td>Activates PMNs</td>
</tr>
<tr>
<td>IL-1</td>
<td>Macrophages</td>
<td>Cell activation, Fever (Pro inflammatory)</td>
</tr>
<tr>
<td>IL-2</td>
<td>T cells</td>
<td>T cell growth and activation</td>
</tr>
<tr>
<td>IL-3</td>
<td>T cells</td>
<td>Hematopoiesis</td>
</tr>
<tr>
<td>IL-4</td>
<td>T cells, mast cells</td>
<td>B cell proliferation and switching to IgE, TH2 differentiation</td>
</tr>
<tr>
<td>IL-5</td>
<td>T cells</td>
<td>Differentiation of eosinophil, activates B cells</td>
</tr>
<tr>
<td>IL-7</td>
<td>Bone marrow stroma cells</td>
<td>T cell progenitor differentiation</td>
</tr>
<tr>
<td>IL-8</td>
<td>Macrophages, T cells</td>
<td>Chemotactic for neutrophils</td>
</tr>
<tr>
<td>IL-10</td>
<td>Macrophages, T cells</td>
<td>Inhibits activated macrophages and dendritic cells, Anti-inflammatory</td>
</tr>
<tr>
<td>IL-12</td>
<td>Macrophages</td>
<td>Differentiation of T cells, activation of NK cells</td>
</tr>
<tr>
<td>GM-CSF</td>
<td>T cells, macrophages, monocytes</td>
<td>Differentiation of myeloid progenitor cells</td>
</tr>
<tr>
<td>M-CSF</td>
<td>Macrophages, monocytes, fibroblasts</td>
<td>Differentiation of monocytes and macrophages</td>
</tr>
<tr>
<td>G-CSF</td>
<td>Fibroblasts, monocytes, macrophages</td>
<td>Stimulates neutrophil production in bone marrow</td>
</tr>
</tbody>
</table>
## Immunoglobulins

<table>
<thead>
<tr>
<th></th>
<th>IgG</th>
<th>IgA</th>
<th>IgM</th>
<th>IgD</th>
<th>IgE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heavy chain</strong></td>
<td>γ</td>
<td>α</td>
<td>μ</td>
<td>δ</td>
<td>ε</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td>75–85 (max)</td>
<td>7–15</td>
<td>5–10</td>
<td>0.3</td>
<td>0.019 (min)</td>
</tr>
<tr>
<td><strong>Carbohydrate %</strong></td>
<td>4</td>
<td>10</td>
<td>10.7</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td><strong>Serum half-life (days)</strong></td>
<td>21 (max)</td>
<td>6</td>
<td>5</td>
<td>3</td>
<td>2 (min)</td>
</tr>
<tr>
<td><strong>Concentration</strong></td>
<td>12 mg/ml</td>
<td>2 mg/ml</td>
<td>1.2 mg/ml</td>
<td>0.03 mg/ml</td>
<td>0.00004 mg/ml</td>
</tr>
<tr>
<td><strong>Molecular form</strong></td>
<td>monomer</td>
<td>Serum IgA = monomer</td>
<td>Secretory IgA = Dimer</td>
<td>Pentamer</td>
<td>Monomer</td>
</tr>
<tr>
<td><strong>Molecular weight</strong></td>
<td>150,000 (lightest)</td>
<td>Serum IgA = 160,000</td>
<td>Secretory IgA = 400,000</td>
<td>950,000 (heaviest)</td>
<td>175,000</td>
</tr>
<tr>
<td><strong>Subclasses</strong></td>
<td>IgG1 &gt; IgG2 &gt; IgG3 &gt; IgG4</td>
<td>IgA1 = circulatory, IgA2 = secretory</td>
<td>M1, M2</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td><strong>Compliment activation</strong></td>
<td>Classical ++</td>
<td>Alternate (only IgA1 fixes complemet)</td>
<td>Classical +++</td>
<td>Alternate</td>
<td>Alternate or None</td>
</tr>
<tr>
<td><strong>Sedimentation coefficient</strong></td>
<td>7S</td>
<td>Monomer = 7S</td>
<td>7S</td>
<td>7S</td>
<td>8S</td>
</tr>
<tr>
<td><strong>Placental crossing</strong></td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>Presence in milk</strong></td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Compartment</strong></td>
<td>Equal in intravascular (45%) and extravascular (55%) compartment</td>
<td>Mostly extravascular (Minimum intravascular distribution)</td>
<td>mostly intravascular</td>
<td>Mostly extravascular</td>
<td></td>
</tr>
<tr>
<td><strong>Heat stability</strong></td>
<td>Stable</td>
<td>Stable</td>
<td>Stable</td>
<td>Stable</td>
<td>Heat labile</td>
</tr>
<tr>
<td><strong>Also know</strong></td>
<td>Produces secondary response</td>
<td>First to appear in fetus (20 wk)</td>
<td>Produces primary response</td>
<td>Homocytotropic</td>
<td></td>
</tr>
</tbody>
</table>
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