General Pathology

Questions

201. Alcohol keratin is composed of
a. Desmin
b. Pre keratin
c. Vimentin
d. Neurofilaments

202. The cell most sensitive to ischemia is
a. Myocardial fibres
b. Glial cells of brain
c. Renal tubular epithelium
d. Cortical neuron

203. Oncocytes are seen in
a. Pituitary
b. Thyroid
c. Pancreas
d. Thymus

204. Coagulation necrosis is seen in all except
a. Myasthenia gravis
b. Burns
c. Tuberculosis
d. Zenker's degeneration

205. One of the following is an apoptosis inhibitor gene (SGPGI 04)
a. P53
b. Bcl 2
c. Rb
d. c myc

206. Exudation of plasma & leukocytes in acute inflammation is from the (PGI 89)
a. Venules
b. Capillaries
c. Arterioles
d. Both b & c

207. Phagocytosis was discovered by (TN-99)
a. Ehrlich
b. Metchnikoff
c. Ruska
d. Pasteur

208. Bradykinin is a clevage product of (TNPSC - 2K)
a Kininogen
b. Pre kallikrien
c. Kallikrein
d. Histamine

209. Pro-inflammatory cytokines are all except
a. IL1
b. IL2
c. IL 6
d. TNFα

210. Granuloma seen in all except (AIIMS 01)
a. TB
b. Yersinia
c. Mycoplasma
d. Leprosy

211. Chemotaxis mediated by (ICS 98)
a. Histamine
b. Leukotriene B4 & C5a
c. Leukotriene C4 & C3a
d. Bradykinin

212. Which leukotriene is adhesion factor for the neutrophil on the cell surface to attach to endothelium (JIPMER 01)
a. B4
b. C4
c. D4
d. E4

213 Keloid scars are made up of (JIPMER 98)
a. Dense collagen
b. Loose fibrous tissue
c. Granulomatous tissue
d. Loose areolar tissue

214. Articular cartilage is made up of (JIPMER 81)
a. Type I Collagen
b. Type II Collagen
c. Type III Collagen
d. Type IV collagen

215. Endothelium, leukocyte interaction during inflammation mediated through (PGI - 03)
a. Selectin
b. Intergrin
c. Defensin
d. Endothelin

216. In acute inflammation pain is mediated by (TN 93)
a. Serotonin
b. Histamine
c. Bradykinin
d. Cytokine

217. The most common glycoprotein in BM is
a. Laminin
b. Fibronectin
c. Type IV collagen
d. Heparan sulfate

218. Pale infarct not seen in (AP-95)
a. Lung
b. Liver
c. Spleen
d. Heart

219. In shock characteristic feature is (PGI- 97)
a. Cardiac failure
b. Poor perfusion of tissue
c. Cyanosis
d. Oedema

220. All are risk factors for DVT except (AI 03)
a. Duration at surgery >30min
b. Obesity
c. Age <40yrs
d. Use of OCPs

221. Hyperviscosity is seen in (PGI 03)
a. Cryoglobulinemia
b. Multiple myeloma
c. MGUS
d. Lymphoma
e. Macro globulinemia

222. Which of the following technique is used in gene therapy (PGI - 01)
a. Electroporation
b. Electro focussing
c. Selectively targeted recombination
d. Intranuclear injection

223. True regarding western blot
a. Gel electrophoresis of protein
b. DNA detection
c. RNA detection
d. Ribosome detection
e. Carbohydrate detection

224. LE cell is a
a. Lymphocyte
b. Neutrophil
c. Basophil
d. Eosinophil
e. Monocytes

225. AL type of amyloid is seen in
a. Medullary carcinoma of thyroid
b. Primary amyloidosis
c. Multiple myeloma
d. Familial amyloidosis

226. Secondary Amyloidosis occurs in (TN - 90)
a. Rheumatoid arthritis
b. Tuberculosis
c. Hodgkins lymphoma
d. All

227. Congo red with amyloid produces (AIIMS - 78)
a. Dark brown
b. Blue colour
c. Brilliant pink
d. Khaki

228. Antitopoisomerase I is a marker of (JIPMER 99)
a. Systemic sclerosis
b. Classic PAN
c. Nephrotic syndrome
d. RA

229. Mast cells release interleukin (AP - 98)
a. 1
b. 2
c. 3
d. 4

230. The HLA class III region genes are important elements in (AI 03)
a. Transplant rejection phenomenon
b. Governing susceptibility to autoimmune disease
c. Immune surveillance
d. Antigen presentation and elimination

231. Most common opportunistic infection in children with neutropenia is
a. Gram negative bacilli
b. Streptococci
c. Staphylococci
d. Pneumococci

232. Pseudomyxoma peritonei is seen with (AI-89)
a. Mucinous cystadenocarcinoma ovary
b. Carcinoid appendix
c. Endometrial carcinoma
d. Ileal carcinoid

233. MHC - II positive cells are all except (PGI - 2000)
a. B cells
b. T cells
c. Macrophages
d. Platelets
e. RBC

234. Anti-RA antibodies belong to (JIPMER 93)
a. IgA
b. IgG
c. IgE
d. IgM

235. Blood borne metastasis is unusual of (AIIMS - 92)
a. Osteosarcoma
b. Medullary carcinoma breast
c. Choriocarcinoma
d. Renal cell carcinoma

236. Chloroma is a tumor of (AP-92)
a. Soft tissue
b. Bone
c. Haemopoetic cell
d. Ovary

237. Marker for ovarian tumours of epithelial origin (JIPMER 95)
a. Fibronectin
b. Acid Phosphatase
c. CA125
d. LDH

238. Carcinoma bronchus is not associated with
a. SIADH
b. Cerebellar degeneration
c. Acanthosis Nigricans
d. Hypocalcaemia

239. Feature common to Ca. pancreas and Ca. lung and Ca. stomach is (TN-99)
a. Thrombophlebitis
b. Migratory thrombophlebitis
c. Ascites
d. DIC

240. Cancer suppressor gene is important in which malignancy (TN-99)
a. Retinoblastoma
b. Malignant Melanoma
c. Liver carcinoma
d. Lung cancer

241. Inherited Cancer Syndrome include all except (Kerala 2001)
a. Retinoblastoma
b. Neuroblastoma
c. Xeroderma pigmentosa
d. Familial polyposis coli

242. Cancer cells derive energy from (AIIMS 2001)
a. Glycolysis
b. Oxidative metabolism
c. Increased mitochondria
d. None

243. Which of the following tumours is completely cured with chemotherapy (PGI 01)
a. Ovarian tumor
b. Choriocarcinoma
c. Ca. Lung
d. Ca. stomach
e. All

244. Which is the guardian gene (JIPMER 03)
a. P53 gene
b. Myc proto oncogene
c. Erb-B_1
d. Cyclin D

245. Transition from G2 to M phase of the cell cycle is controlled by (AIIMS 03)
a. Retinoblastoma gene product
b. p53 protein
c. Cyclin E
d. Cyclin B

246. Kaposi sarcoma occurs with infection due to
a. HHV 8
b. HHV 4
c. HHV 2
d. HHV 1

247. Biphasic pattern on histology is seen in which tumour (MAH 02)
a. Rhabdomyosarcoma
b. Synovial cell sarcoma
c. Osteosarcoma
d. Neurofibroma

248. The correct sequence of cell cycle (AI-03)
a. G0 - G1-S-G2-M
b. G0-G1-G2-S-M
c. G0-M-G2-S-G1
d. G0-G1-S-M-G2

249. Choristoma is
a. Normal tissue at abnormal sites
b. Normal tissue excess at normal site
c. Abnormal tissue at any site
d. None

250. Malignancy with familial inheritance (AI-99)

a. Ca. breast
b. Ca. ovary
c. Ca. testes
d. Ca. pancreas

General Pathology

Answers

201. (b) Pre keratin *(Robbins 7th ed, pg 134; 6th ed pg 28)*
This question tests our by knowledge about various intermediate filaments.
Intermediate filaments → these cellular components produce flexible intracellular scaffold that organises the cytoplasm and resists the forces applied to cells

They or 4 types	**Found in**
(1) Keratin filaments	Epithelial cells
(2) Desmin filaments	Muscle cells
(3) Vimentin filaments	Connective tissue cells
(4) Glial filaments	Astrocytes

Filaments accumulating in cell injury are keratin & Neurofilaments
Mallory body or Alcoholic hyaline composed predominantly of keratin filaments
Thus Pre keratin becomes the answer for this question.

202. (d) Cortical neuron *(Robbins 7th ed pg 139; 6th ed, pg 133)*
Of the various factors which determine development of infarct an important one is vulnerability of a tissue to hypoxia
Necrosis→ undergo irreversible damage when deprived for 3-4min of their Blood supply
Myocardium → fibres die within 20-30min.

203. (b) Thyroid *(Robbins 7th ed, pg 33; 6th ed, pg 27)*
Oncocytomas are benign tumour typified by presence of large mitochondria which gives them characteristic eosinophilic appea - rance
These tumours are found in
(1) Salivary glands
(2) Thyroid
(3) Parathyroid
(4) Kidney

204. (d) Zenker's degeneration *(Robbins 7th ed pg 21)*
Types of necrosis
(a) Coagulative →MC type
In this type typically occurs in solid organs (kidney, heart, adrenals) due to deficient blood supply and anoxia.
Cells retain their outline for a long term.

Brain is an exception that being solid organ still shows liquefactive necrosis

other situation where coagulative necrosis seen → (i) Skin in burns (ii) Coagulative necrosis of liver cells due to virus & toxic chemicals

(b) Liquefactive Necrosis

→ Liquefaction of necrotic cells by lysosomal enzymes by neutrophils or necrotic cells themselves in

(1) Suppuration

(2) Brain infarct

(c) Fat necrosis two types

(a) Enzymatic → Seen in Acute pancreatitis

(b) Non enzymatic→ (Due to trauma)

(a) Breast

(b) Subcutaneous tissue

(c) Abdomen

(d) Fibrinoid necrosis: it is a connective tissue necrosis; Seen in

(a) Malignant hypertension

(b) Auto immune disease - Rheumatic fever
PAN
SLE

205. (b) Bcl 2 *(Robbins 7th ed, pg 39; 6th ed, pg 22)*

Important apoptotic genes are	**Imp pro-apoptotic genes are**
(a) Bcl2	(a) Ba x
(b) Bcl X	(b) Bad

206. (a) Venules (**Robbins 7th ed, pg 513, 56)**

Post capillary venules are important points of interchange between lumen of vessels and surrounding tissue. Both vascular leakage & leucocyte exudation occur preferentially in venules in many types of inflammation

→ leucocyte diapedesis can occur through capillaries in lungs

207. (b) Metchnikoff *(Robbins 7th ed pg 549; 6th ed, pg 530)*

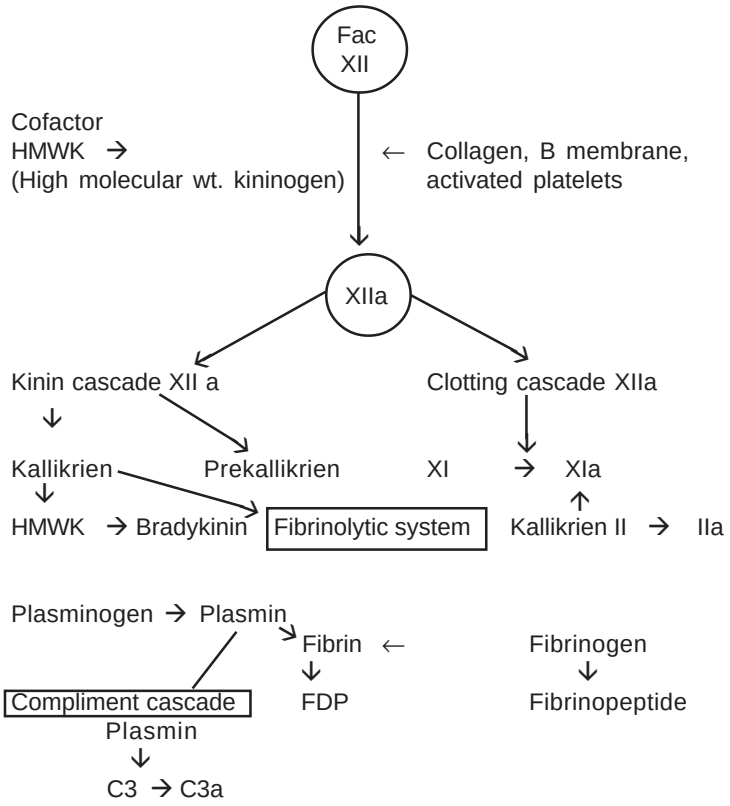
Russian biologist metchnikoff discovered phagocytosis.

Paul Ehrlich → gave humoral theory of inflammation

Ruska→ invented Electron microscope

Pasteur →invented vaccine for small pox
invented pasteurization
gave germ theory for disease

208. (a) Kininogen *(Robbins 7th ed, pg 67 fig 2.15; 6th ed, pg 68 fig 3.17)*



209. (b) IL 2

(Robbins 7th ed pg 75 table (2.6); Robbins 6th ed, pg 78 table 3.7)

Role of mediators in different reactions of inflammation

Vasodilation	-	PGs Nitric oxide Histamine
↑ Permeability	-	Vasoactive amine (5HT) C3a & C5a Bradykinin Leukotrienes C4, D4, E4 PAF Substance P
Chemotaxis, leucocyte recruitment and activation		C5a Leukotrienes By Chemokines

	IL1, TNF,
	Bacterial Products
Fever	IL1, TNF, PGs
Pain	PGs, Bradykinin
Tissue damage	Neutrophil & macrophage
	Lysosomal enzymes
	Oxygen metals
	Nitric oxide

210. (c) Mycoplasma *(Robbins 7th ed, pg 83; 6th ed pg 83, 356 Table 2.7)*
Disease with Granulomatous inflammation:
Tuberculosis
Leprosy
Syphilis
Cat scratch disease
For option (b) 6th ed pg 356
→within submucosal tissue yersinia produce micro abscess rimmed by activated macrophage resembling stellate granuloma of LGV & cat scratch disease.
Thus yersinia also is granulomatous histology

211. (b) Leukotriene B4 & C5a *(Robbins 7th, ed pg 75; 6th ed pg 78)*
→See Q No. 209 table 2.6 Robbins 7th/ e; 3.7 Robbins 6th edition

212. (a) B4 *(Robbins 6th ed, pg 78)*
B4 → highly chemotactic for neutrophil, eosinophil, monocytes
C4 & D4 → Most potent vasoactive & spasmogenic substance known

213. (a) Dense collagen *(Robbins 7th ed pg 115; 6th ed pg 110)*
The accumulation of excessive amount of collagen gives rise to tumourous scar called keloid

214. (b) Type II *(Robbins 7th ed pg 104 table 3.2)*
Type I→ MC, in skin (90%) bone, tendons, most other organs
II→ Cartilage, vitreous humour
III→ Blood vessel, uterus, skin (10%)
IV→ All Basement membrane
V→ 2-5% of interstitial tissue, Blood vessels
VI→ Interstitial tissue
VII→ Dermo epidermal junction
VIII→ Endothelium, Descemets membrane
IX→ Cartilage

215. (a) & (b) Selectin & intergrin *(Robbins 6th ed 57)*
Endothelium leucocyte adhesion molecule

(a) Selectin
→P found on platelets & Endothelium
→E found on Endothelium
above bind to sialyl lewis x receptors of leucocyte
(b) Intergrins → receptor to ICAM & VCAM
for ICAM →CD 11 /CD 18 (intergrins) (LFA -1, MAC I)
for VCAM → αy β1
d α 4β7
(c) Immunoglobulin like molecule
ICAM
VCAM
(d) Mucin like glycoprotein
GlyCAM1, Psyl, ESL1 & CD34

216. (c) Bradykinin
See Q. No. 209

217. (a) Laminin *(Robbins 7th ed, pg 105; 6th ed pg 100)*
- Laminin is most common glycoprotein in BM
- mediates cell attachment to connective tissue
- Involved in angiogenesis
- it binds to intergrins

218. (a) Lung *(Robbins 7th ed pg 138; 6th ed pg 132)*
Red infarcts occur in
(a) Tissue with dual blood supply (in lungs, intestine)
(b) Loose tissue (lungs)
(c) Venous occlusion (in ovarian tissue)
(d) In tissue that was previously congested because of sluggish blood flow.

219. (b) Poor perfusion *(Robbins 7th ed pg 119; 6th ed pg 132)*
Shock constitutes systemic hypoperfusion owing to reduction in cardiac output or in the effective circulating volume. The end results are hypotension followed by impaired tissue perfusion & cellular hypoxia

220. (c) age <40yrs *(Bailey 23rd ed, pg 253)*
High risk →Gen. urological surgery in Pt >40yrs
→extensive pelvic or abdominal surgery
→Major ortho surgery of lower limb
Moderate →Gen. surgery in pt >40yrs of >30min duration; Gen. surgery in Pt <40yrs on OCP
Low risk →Uncomplicated surgery in Pt <40yrs without additional risk

→Minor surgery (<30min) in and who are <40yrs without risk

- ♦ Age < 40 is a favourable factor
- ♦ Use of OCP classifies as moderate risk even when pt. is <40yrs
- ♦ Duration of surgery >30minutes is a major surgery and enhances risk
- ♦ Obesity is an independent risk factor

221. (a),(b),(d),(e) *(William Heamatology 6th ed, pg 1268)*
Hyperviscosity is seen in
Multiple Myeloma
Waldenstorm macroglobulinemia
Cryoglobulinemia
MPS (Myelo proliferative syndrome)

222. (a), (c), (d) *(Harper 15th ed pg 823,824,825)*
Methods to introduce genes into cell for gene therapy
(a) Intranuclear injection
(b) Transfection
(c) Electroporation
(d) Retrovirus
(e) Plasmid lipid complex
(f) Site directed complexes
(g) Adenovirus (for introduction of C.J. gene in respiratory tract cells)

223. (a) Gel electrophoresis of protein *(Harper 16th ed pg 403,407)*
also Southern blot →for DNA
Northern blot →for RNA
Western blot → as in this question for proteins
there is no eastern blot mentioned

224. (b) & (e) *(Robbins 7th ed, pg 230; 6th ed pg 219)*
LE cells is any phagocytic leukocyte (neutrophil or macrophage) that has engulfed denatured nucleus of injured cells

225. (b) & (c) *(Robbins 7th ed pg 260; 6th ed pg 253)*

Generalised amyloidosis	**Disease associated**	**Fibril**
(1)Immunocyte dyscrasia (Primary amyloidosis)	multiple myeloma & other monoclonal B cell proliferation	AL
(2)Reactive systemic amyloidosis	Chronic inflammatory disease	AA
(3)Hemodialysis assoc.	CRF	Aβ2m
Hereditary Familial Mediterranean fever		AA

Familial Amyloidotic neuropathies		ATTR
Systemic Senile		ATTR
Localised Amyloidosis		
Senile cerebral	Alzheimers	Aβ
Endocrine MCT		A cal
Islet of langerhans	Type II DM	AI APP
Isolated atrial amyloidosis		A- ANF

226. (d) All (*Robbins 7th ed, pg 260,261)*
At one time tuberculosis, bronchiectasis, chronic osteomyelitis were most important conditions. But now RA,Ankylosing spondylitis and IBD especially regional enteritis and Ulcerative colitis.
RA is M.C.
Reactive systemic amyloidosis may also occur in association with tumour M.C being RCC & Hodgkin disease.

227. (c) Brilliant Pink *(Robbins 7th ed pg 263; 6th ed pg 251)*
Most widely used stain is congo red
→ Under ordinary light → Pink or red colour to tissue deposit
→ In polarised light → apple green birefringence

228. (a) Systemic sclerosis
(Robbins 7th ed pg 229 table 6.9; 6th ed, pg 218 table 7.8)

Antibodies	**Disease with high concentration with antibodies**
Anti dsDNA	SLE
Anti sm	SLE
Antihistone	Drug induced LE
DNA topoisomeraseI	Diffuse systemic sclerosis
Anticentromere	Limited scleroderma (CREST)
SS - A	
SS - B	Sjogrens Syndrome
Histidyl t-RNA Synthetase	Inflammatory myopathies

229. (a),(c),(d) *(Robbins 7th ed, pg 208; 6th ed, pg 198)*
Most cells are believed to produce a variety of cytokines, including TNF - α, IL -1,IL-3,IL-4,IL-5,IL-6 and GM-CSF as well as chemokines, such as Macrophage inflammatory protein (MIP) -1 α and MIP Iβ

230. (b) Governing susceptibility to auto immune disease
(Ananthanaryanan 6th ed, pg 108; Roilt's Immunology pg 262)
HLA class III molecules are heterogenous. They include complement component linked to the formation of C3 convertase.
Deficiency of these early components of the classical pathway viz.

C1,C2 & C4 is associated with autoimmune diseases like SLE and other collagen vascular disease

HLA class III contains genes for:

(a) Complement components C2 & C4 of classical pathway
(b) Properdin factor B of alternate pathway
(c) Heat shock proteins
(d) Tumor Necrosis Factor α and β

231. (c) Staphylococci (Robbins 7th ed, pg 240 & 6th ed, pg 233)

Please refer to the table given on these pages

Pathogen Type	T-cell defects	B-cell defect	Granulocyte defect	Complement defect
Bacteria	Bacterial sepsis	Haemophilus Streptococci Staphylococci	Staphylococci Pseudomonas	Neisseriae infection
Viruses	CMV,EBV, Severe Varicella etc,	Enteroviral Encephalitis		
Fungi and Parasites	Candida and Pneumocystis carinii	Intestinal giardiasis	Candida Nocardia, Aspergillus	
Special feature	Aggressive disease with opportunistic pathogen failures to clear infection	Recurrent Sinopulmonary Infections, sepsis, Chronic meningitis		

However Harrison 16th edition on pg 353 specifically states that "The most common bacteria are staphylococcus aureus and enteric gram negative bacteria" . This is in reference to abnormal neutrophil function. So you decide for yourself !

232. (a) Mucinous cystadenocarcinoma ovary

(Robbins 7th ed pg 279; 6th ed, pg 269 & 1071)

A condition associated with mucinous ovarian neoplasms in pseudomyxoma peritonei. This disorder combines the presence of an ovarian tumour with extensive mucinous ascites, cystic epithelial implants on the peritoneal surfaces and adhesion. Pseudomyxoma peritonei, if extensive, may result in intestinal obstruction and death. Recent evidence strongly supports an extraovarian (usually appendiceal) primary mucinous tumour in many cases with secondary ovarian and peritoneal spread. It is not related carcinoid of appendix.

233. (d) and (c)

(Robbins 7th ed, pg 204; 6th ed, pg 194; Ananthanarayan 6th ed, pg 122)

"Class I antigens are expressed on all nucleated cells and platelets" They are the principal antigens involved in graft rejection and cell mediated cytolysis. They may function as component of hormone receptors.

HLA-class II antigens are more restricted in distribution being found only on cells of immune system - macrophages, dendritic cells, activated T cells and particularly on B cells.

234. (d) IgM *(Robbins 7th ed, pg 1308; 6th ed pg 1250)*

80% of individuals with RA have auto antibodies to the Fc portion of autologous IgG (rheumatoid factors). These are mostly IgM antibodies, perhaps generated within joints but may be of other classes. These self associated (RA-IgG) to form immune complexes in the sera, synovial fluid and synovial membranes.

235. (b) Medullary carcinoma breast

(Robbins 7th ed pg 1145; 6th ed, pg 1111)

Medullary carcinoma of breast metastasises by lymphatic spread. All the rest of the options spread via vascular invasion.

236. (c) Haemopietic cell

(Robbins 7th ed pg 694; 6th ed pg 678 first line; Harrisons 16th ed, pg 633)

"Rarely patients may present with symptoms of mass lesions located in soft tissues, breast, uterus, cranial or spinal dura, GI, lung, mediastinum, prostate, bone or other organs. The mass lesion represents a tumor of leukemic cells and is called as granulocytic sarcoma or chloroma"

237. (c) CA 125 *(Ref 7th ed, pg 339 table 7.13; 6th ed pg 325 table 8.12)*

Markers	**Associated cancers**
<u>Hormones</u>	
HCG	Trophoblastic, Non-seminomatous testicular tumors
Calcitonin	Medullary carcinoma of thyroid
Catecholamine & metabolites	Pheochromocytoma and related tumours
Ectopic hormones	See following question
<u>Oncofetal antigens</u>	
α - Fetoprotein	Liver cell cancer, Non-seminomatous germ cell tumours of testis
CEA	Carcinomas of the colon, pancreas, lung, stomach and breast
<u>Isoenzymes:</u>	
Prostatic acid phosphatase	Prostate cancer

Neuron specific enolase Small cell cancer of lung, Neuroblastoma

Specific Proteins:

Immunoglobulins, PSA Multiple myeloma and other gammopathies, Prostate cancer

Mucins and other glycoproteins:

CA-125	Ovarian cancer
CA-19-9	Colon cancer, Pancreatic cancer
CA-15-3	Breast cancer

238. (b) Cerebellar degeneration *(Robbins 6th ed pg 746, 321)*

Paraneoplastic syndrome associated with Bronchogenic carcinoma includes

Endocrinopathies

(a) SIADH which leads to hyponatremia
(b) Cushing syndrome due to expression of ACTH
(c) Calcitonin elaboration leads to hypocalcaemia
(d) Gonadotropins lead to Gynaecomastia
(e) Serotonin lead to carcinoid syndrome
(f) Elaboration of parathormone, parathyroid hormone - related peptide, Prostaglandin E and cytokines lead to hypercalcaemia.

Other systemic manifestations are:

(a) Lambert - Eaton myasthenic syndrome
(b) Peripheral neuropathy - predominantly sensory
(c) Dermatologic abnormalities particularly Acanthosis nigricans
(d) Leukaemoid reaction
(e) Hypertrophic pulmonary osteoarthropathy
(f) Horner syndrome
(g) Pain in the distribution of ulnar nerve

Other Paraneoplastic syndromes are:

Clinical syndrome	Major forms of underlying cancer	Causal mechanism
Endocrinopathies:		
Cushing syndrome	Small cell Ca. of lung Pancreatic Ca	ACTH are ACTH like substance Neural tumour
SIADH	Small cell Ca of lung Intracranial neoplasms	ADH or Atrial Natriuretic hormone
Hypercalcaemia	Squamous cell Ca lung Breast carcinoma Renal Carcinoma Adult T cell leukaemia/ lymphoma Ovarian Ca	Parathyroid hormone related peptide, TGF-α, TNF-α, IL-1
Hypoglycaemia	Fibrosarcoma Other mesenchymal sarcoma Hepatocellular Ca	Insulin or insulin like substance

Carcinoid syndrome	Bronchial adenoma (carcinoid) Pancreatic Ca Gastric Ca	Serotonin, bradykinin, ? histamine
Polycythemia	Renal Ca Cerebellar hemangioma Hepatocellular Ca	Erythropoietin
Nerve and muscle syndrome:		
Myasthenia	Bronchogenic Ca	Immunologic
Disorder of the central & peripheral nervous system	Breast Ca	
Dermatologic Disorders:		
Acanthosis nigricans	Gastric Ca Lung Ca Uterine Ca	? Immunologic, ? Secretion of epidermal growth factor
Dermatomyositis	Bronchogenic Ca, Breast Ca	? Immunologic
Osseous, Articular and soft tissue changes:		
Hypertrophic osteo arthropathy & clubbing of fingers	Bronchogenic Ca	Unknown
Vascular & haematologic changes:		
Venous thrombosis (Trousseau phenomenon)	Pancreatic Ca Bronchogenic Ca Other cancers	Tumour products (Mucins that activate clotting)
Non bacterial thrombotic endocarditis	Advanced Cancers	Hyper coagulopathy
Anaemia	Thymic neoplasm	Unknown
Others:		
Nephrotic syndrome	Various Cancers	Tumor antigens, Immune complexes

239. (a), (b),(c) i.e., Thrombophlebitis, Migratory thrombophlebitis, DIC
(Harrison 16th ed, pg 569 table 86.2)
(Robbins 7th ed pg 657 table 13:10)
(Robbins 6th ed pg table (14.10), 640 for d)
For explanation see previous question

240. (a) Retinoblastoma , (b) Malignant melanoma
(Robbins 7th ed, pg 300 table 7.9; 6th ed, pg 287, table 7.7)
Cancer suppressor genes were first studied in Retinoblastoma following which knudson proposed two - hit hypothesis of oncogenesis. Rb gene which is supposed to mediate development

of Retinoblastoma is referred to sometimes as recessive cancer gene.

Now a large no. of Cancer suppressor genes are known as shown in this table.

Subcellular location	Gene	Function	Tumours Associated with mutation	
			Somatic	Inherited
Cell surface	TGF-β receptor	Growth Inhibition	Ca colon	Unknown
	E.cadh-erin	Cell adhesion	Ca stomach, breast	Familial gastric Ca
Under plasma membrane	NF-1	Inhibition of ras Signal trans-duction	Schwannoma	NF-1 and sarcoma
Cytoskeleton	NF-2	Unknown	Schwannomas of meningiomas	NF-II, acoustic aschwannoma + meningiomas
Cytosol	APC	Inhibition of signal transduction	Ca stomach, Colon, Pancreas, Melanoma	FAP coli, colon Ca
Nucleus	Rb	Regulation of cell	Retinoblastoma cycle Osteosarcoma, Ca breast, colon, lung	Retinoblastoma, Osteosarcoma
	p53	Regulation of all cycle & apoptosis in response to DNA damage	Most human Cancers	Li-Fraumeni Syndrome, Multiple carcinoma & sarcoma
	WT-1	Nuclear transcription	Wilm's Tumor	Wilm's Tumor
	P-16 (INK4A)	Regulation of cell cycle by Inhibiting cyclin dependent kinases	Pancreatic, Esophageal Ca	Malignant Melanoma
	BRCA-1	DNA repair		Ca breast & ovary
	BRCA-2	DNA repair		Ca male & female breast

241. (c) Xeroderma pigmentosa

(Robbins 7th ed pg 307, 323 & 1245; 6th ed, pg 275 table (8.6))

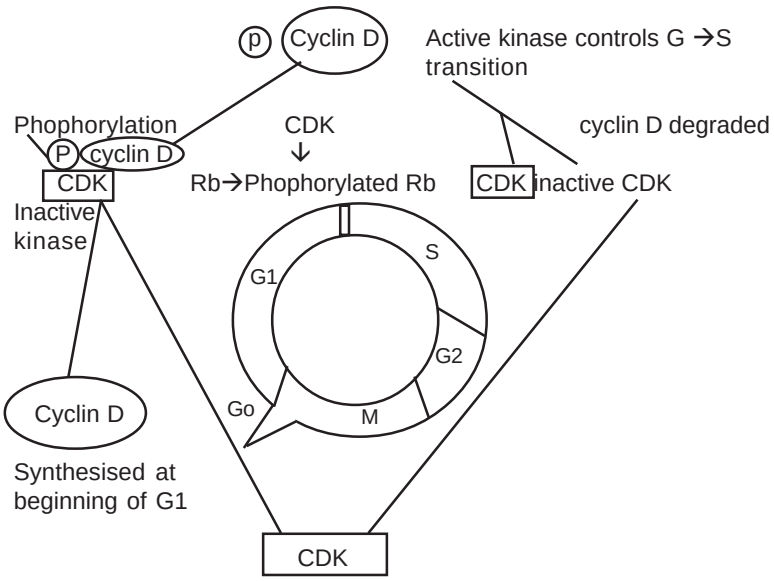
Inherited Cancer syndromes (Autosomal Dominant):
Familial Retinoblastoma
Familial Adenomatous polyps of colon
MEN Syndrome
NF type 1 & 2
Von hippel - Lindau syndrome
Familial Cancers:
Breast Ca
Ovarian Ca
Colon Ca other than Familial Adenomatous polyps
Autosomal Recessive syndromes:
Xeroderma Pigmentosa
Ataxia - telangiectasia
Bloom syndrome
Fanconi anaemia

242. (a) Glycolysis *(Harper 16th ed pg 136 & 25th edition pg 190)*
In fast growing cancer cells glycolysis precedes at a much faster rate than is required by citric acid cycle.Thus,more pyruvate is produced than can be metabolised. This, inturn leads to excessive lactate which favours a relative acid local environment in the tumour, a situation that may have implication for certain type of cancer therapy.

243. (b) Chorio Carcinoma. (e) All *(CMDT 2004 pg 1598)*
Chemotherapy is curative in:
- ♦ Choriocarcinoma
- ♦ Hodgkins Lymphoma
- ♦ Diffuse large cells and high grade lymphoma
- ♦ Ca of testis
- ♦ Some cases of acute leukaemia
- ♦ Embryonal rhabdomyosarcoma

244. (a) p53 gene *(Robbins 7th ed, pg 303; 6th ed, pg 290)*
A little of 50% human tumors contain mutation in p53 gene

245. (d) Cyclin B *(Robbins 7th ed, pg 290; 6th ed, pg 283)*
The orderly progression of cells through various phases of cell cycle is orchestrated by cyclins and dependent kinase (CDK) and their inhibitor.....CDK drive the cell cycle by phosphorylating critical target proteins that are required for progression of cells to next phase of cell cycle. CDKs are activated by Phophorylation after binding to cyclins.



Cyclin B/CDK_1	Regulates the transition from G2 to M phase
Cyclin D/CD_4 Cyclin D/CDK_6 Cyclin E/CDK_2	} Regulate transition from G1-S
Cyclin A/CDK_2 and Cyclin B/CDK_1	} Active in S phase

246. (a) HHV 8 *(Robbins 7th ed, pg 257; 6th ed, pg 248,249)*
Patients with AIDS have a high incidence of certain tumors especially kaposi sarcoma, Non hodgkins lymphoma & Ca cervix in women. The basis is multifactorial: profound defect in T-cell immunity, dysregulated B cell & monocyte function and multiple infection with known and unknown (e.g HHV 8, EBV, HPV)
Ananthanarayan 6th edition pg 448 stages: In 1994DNA sequences presumed to represent a new herpes virus were identified from tissues of Kaposi sarcoma from AIDS patient. This has been named HHV 8. This has subsequently been identified in Kaposi sarcoma in patients not infected with HIV. It has therefore been referred to as KSHV (Kaposi sarcoma associated virus) but etiological relationship is set to be proved.

247. (b) Synovial cell sarcoma *(Robbins 7th ed, pg 1303)*
See morphology block

The histologic hall mark of biphasic synovial sarcoma is dual line of differentiation of tumour cells (i.e. epithelial like spindle like)
Synovial sarcoma may some times be monophasic having spindle cell or rarely epithelial cells only

248. (a) G0-G1-S-G2-M *(Robbins 7th ed, pg 290; 6th ed pg 283)*
Ref to Q. 245 for explanation

249. (a) Normal tissue at abnormal site (*Robbins 7th ed, pg 272)*
An ectopic rest of normal tissue is sometimes called choristoma en rest of adrenal cell under kidney capsule

250. (a) Ca Breast
(b) Ca Ovary
(Robbins 7th ed pg 287 table 7.6; 6th ed, pg 275 table 8.6)
For explanation ref to Q.241.