Dt:
Pg:

1. Iron Deficiency Anaemia - Anaemia is a deficiency of RBC which affects the oxygen carrying ability of blood causing unusual tiredness and other symptoms. The deficiency occurs either through the reduced production of red blood cell or an increased loss of red blood cell.

The RBC is manufactured in Bone Marrow and has a life time of approx 4 months. To produce RBC, the body needs iron, vit. B_{12} and folic acid. If there is a lack of one or more of these ingredients anaemia will develop.

[Causes]

1. In Children, Iron deficiency is caused by low iron diet. In adults, it is due to with vegetarians because the main general dietary source of Iron is red meat.

2. Babies can develop, iron deficiency. If they are born prematurely, as their iron storage is incomplete until the final stage of pregnancy.

3. Reduced absorption of Iron from the intestine is due to small intestinal disease such as gluten intolerance and Crohn's disease (digerade the lining of small intestine)

Dt.:
Ps.:
Delta

4. Ulcer in the stomach and duodenum can also lead to cause of iron deficiency Anaemia.

Pathophysiology

1. Iron Balance is achieved by regulation of Iron absorption in the proximal small intestine either diminished absorption of dietary Iron or excessive loss of Body iron can cause Iron deficiency.

2. Pernecious Anaemia or vitamin B_{12} Anaemia

- Pernecious Anaemia referred to a type of auto immune Anaemia.
Intrinsic factor is necessary for vitamin B_{12} absorption so, impaired absorption of vit. B_{12} can result in to B_{12} deficient Anaemia.
A pernecious Anaemia is a deficiency of Blood cells but many other cells in the body needs vit. B_{12} including nerve cell. Hence, deficiency may affect the brain functions. Generally pernecious Anaemia is a disease in which the red blood cells are abnormaly formed due to its inabilit to absorb vitamin B_{12}. Pernecious Anaemia also refered to a disorder of Atrophied fruital cells leading to absent of intrensic factor which resulting in an ability to absorb vitamin B_{12}.

DL:
Pg:

Pathophysiology

Food vit. B_{12}

↓ Digestion

Food vit. B_{12}

↓ Release

vit. B_{12} + Intrensic factor ⊕ ← Small Intestine

↓

B_{12} - Intrensic factor complex

⊖ ↓ enter

Intestine

⊖ ↓ release

B_{12} ← absorb

↓

Urine pass

Causes

- When Prietal cells shrinked in size. they produce less intrensic factor
- Various pathological conditions that affect the first part of ~~intrin~~ intestine that is illeum from where the B_{12} is absorbed, can cause anaemia.

Symptoms

- Vit. B_{12} deficiency affects three systems of the Body.

1. Haemapoitic system
2. GIT system

Dt.:
Pg.:

3. Nervous System

• Haemapoetic system

Symptoms: Fatigue, Diginess, Ringing in ears, yellowish skin, Heart rate increase, Chest pain.

• GIT system

Symptoms: Loss of apetite, Diarrhea, abdominal cramps, weight loss and red colored tongue.

• Nervous System

Symptoms: Numbness, Tingling and Burning of arms, legs, hand and feet, Muscle weakness, Loss of Balance during walking, change in reflexes, irritation, mental confusion and depression.

☆ Pathophysiology

Cobalamene is an organometallic substance which contain corrin ring, a centrally located cobalt atom and various axial legands. The Basic structure of vitamin B_{12} is synthesized by microorganism but most animals are capable of converting vitamin B_{12} in to the two co-enzymes

Dt. Pg.

Vitamin B_{12}

adenoryl cobalamine → L-methylmalonic acid → Succinyl coenzyme A

methyl cobalamine → (Methyl transferase) → homocystine → Methionine

If deficiency of cobalamine takes place then

Cobalamine ⊖ → ⊖ Thymidine synthetase enzyme function → megaloblastic changes in dividing → ⊖ DNA Synthesis

Cobalamene in diet → release → parietal cells → Intrensic factor + cobalamene → IF-CBC complex → ⊕

Dt.:
Pg.:

Transcobalamine
TC – II
↓
IF – CBC – TC – II ← Complex formation
↓
CBC – TC – II
↓ TC – II → remove out
endocytosis
CBC or B12
↓
absob

3. Follic acid Deficiency

This deficiency is a low level of B- complex vitamins results in anaemia of RBC which are large in size but few in numbers.

Uses of follic acid

1. RBC maturation
2. cellular and growth repair
3. DNA – RNA Syhthesis

☆ Manufacture by small intestine and store in liver for 6 months.

Sources: green leafy vegetables, mushrooms, apple, Rajma and soya.

my notes

Causes:

It occurs from a diet lacking in foods with high follic acid content
Sometime body does not absorb food containing follic acid.
Age, Alcolism, Birth control pills, anti convulsion therapy; Sulpha antibiotics, illness, Smoking and stress.

Symptoms: Fatigue, Pale colour skin, Anorexia, faster heart beat, red coloured tongue, weakness and weight loss.

Pathophysiology:
Structure of
Follic acid → Pteren ring + P-amino benzoic acid with glutamic acid (PABA)

Human Body does not form follic acid because PABA is deficient in them.

External source: In food follic acid present in form of Polyglutamate + Monoglutamate.

Absorption → Small intestine

Polyglutamates —(small intestine enzyme)→ monoglutamates —(absorb in)→ jejunum

Pg: Delta

Folate present in blood is in form of 5-methyl tetrahydro follic acid

Tetrahydro follic acid

| 5-methyl TFFA

Cell enter

| demethyl (which remove 5-methyl)

THFA

|

active form follic acid.

4. Sickle Cell Anaemia

Sickle cell Anaemia is a inherited blood disorder, the normal RBCs are shaped like disc which give the blood them flexibility to transport through even the smallest blood vessel. In sickle cell Anaemia, the RBCs have an abnormal bow shaped resembling to sickle. Due to this shape, the RBCs become sticky and rigid in nature and become prone getting trapped in small vessels which blocks blood from reaching different part of the body.

Symptoms: Intense pain, severe anaemia episodes of intense pain, organ damage, early death, vunerability of infection, fatigue, Shortness of Breath, pale coloured skin and finger nails, frequent infection, eye infection including Blindness and cardiovascular stroke

5. Thalessemia :

The Thalessemia are an inherited group of disorder in which mutation in genes expressing alpha-globulin and β-globulin result in impaired Haemoglobin synthesis and microcytic anaemia of varying severely. The Thalessemia divided in to α and β type according to which globulin genes are defective.

Thalessemia
↓ in a disorder gene
mutation gene or DNA
↓
α and β globulin syntheses ↓↓
↓
α-β globulin defective
→ ⊖ ⊖ Hb synthesis ↓↓
→ ⊕ ⊕ microcytic anaemia ↑↑

Symptoms: Fatigue, Weakness, pale or yellowish colour skin, facial bone deformities, slow growth, abdominal swelling, dark colour urine.

Thalassemia

α- Thalassemia	β- Thalassemia
→ α- globulin chain ↓↓	→ β-globulin chain ↓↓
→ β- globulin Chain formation in excess	→ α- globulin chain formation in excess

Dt.:
Pg.:

Delta

α-globulin + β-globulin

↓

expression

↓

goes to bone marrow

↓

Blood formation

Disease in Thalassemia = Reduction in Haemoglobin Synthesis
Erythropoesis
Periferal haemolysis

Medium = Deferasirox (↓↓ formation of iron in body and prevent Thalassemia.

6. Haemophilia:

It is a genetic disorder which affects the mechanism of blood clotting. Depending upon the degree of the disorder, there will be a excess bleeding after surgery, Dental procedure and on injury.

Haemophilia are of two types

Haemophilia

↓

Haemophilia A | Haemophilia B | Haemophilia C

↓ | ↓ | ↓

Classical Haemophilia | Christmas Disease | By factor XI coagulation ↓↓

↓ | ↓

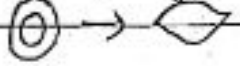
By factor VIII, coagulation ↓↓ | By factor IX coagulation ↓↓

Dt.
Pg.

Haemolytic Anaemia (Hereditary Acquired Anaemia)

This is the disorder in which the RBC's are destroyed prematurely. The cells are broken down at the faster rate than the bone marrow can produce new cells.

Haemolytic anaemia is generally of two types:

Haemolytic Anaemia
- Acquired
- Hereditary
  - Spherocytosis 
  - Elliptocytosis 

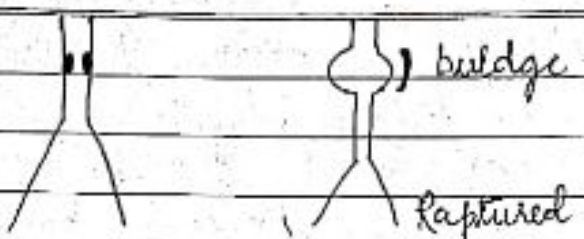
Acquired: Excessive blood loss due to injury leads to this. May also occur due to Hb deficiency.

Causes: Medication which produces antibodies (for which RBC is antigen) leads to RBC destroyment.

Medication ⟶ Antibodies + RBC (Antigen)
↓
RBC destroyed

Sometimes spleen gets destroyed.

Aneurysms: This disease leads to the weakness of artery wall leads to the buldge of artery wall at a particular site.



Hypersplenism: Leads to spleen enlargement due to which more and more RBCs raptured in to it.

Symptoms: Shortness of breathe, increase in heart rate, fatigue, pale appearance, jaundice, enlargement of spleen, pain in upper abdominal.

DISORDERS RELATED TO NERVOUS SYSTEM.

Dt.:
Pg: 2/12

Epilepsy: Epilepsy is a group of neurological disorders characterised by epilectic seizures (fit). It is a neurological disorder mask by sudden recurrent episodes of sensory disturbance, loss of conciousness or convulsion which is associated with abnormal electrical activity in the brain.

Seizures: Seizures are sudden distruption of the brain, normal electrical activity which is associated by altered consciousness and other neurological and behavirioal manifestations.

There are five types of Seizures -

1. Generalised apileptic seizures
2. Myclonic seizures
3. Visual Seizures
4. Generalised tonic - clonic seizures (grand-mal seizures)
5. Absence seizures (Petit - mal seizures)

1. Generalised apileptic seizures: From few minutes to hour → throughout the brain have abnormal electrical activity which leads to jerk in any part of body.

2. Myclonic seizures: Involuntary spasms in tongue, and muscles for few minutes (least)
   - leads to faint due to pain occurrent.

3. Visual seizures: Mainly occur in pediatric case (14 yrs)
   - Brain part which control visual activity, hearing activity (sensation) get affected.

Symptoms of fainting fatigue

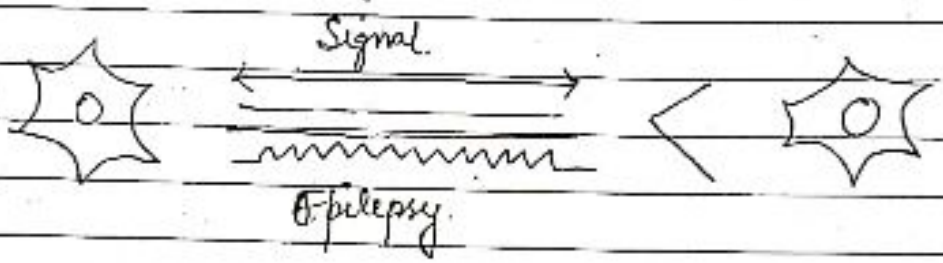
Symptoms

DL:
Pg.:

Delta

→ Treatment occur upto 3 years regularly in children but in aged person upto 5 years.

4. Generalised tonic - chlonic Seizures: duration - 2 to 5 m
Seizures → person loudly. (Loud cry)
ery → loss of consciousness
→ Fall on the ground.



Symptoms: Skin colour converts in to bluish colour person may bite his tongue, loss of bowel and bladder control, trouble of breathing.

5. Absense Seizures: Generally in baby (4 years) and upto adolescence.
Duration - upto 3 - 5 seconds.

CAUSES OF EPILEPSY

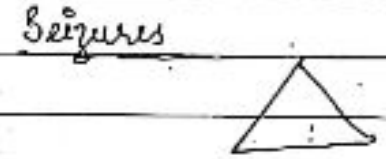
1. Trauma of birth
2. Insufficient oxygen supply at birth
3. Head injury
4. Heavy bleeding
5. Incompatibility between mother blood and new born baby.
6. Alcoholism
7. Inflammation of membrane covering the brain and spinal cord.

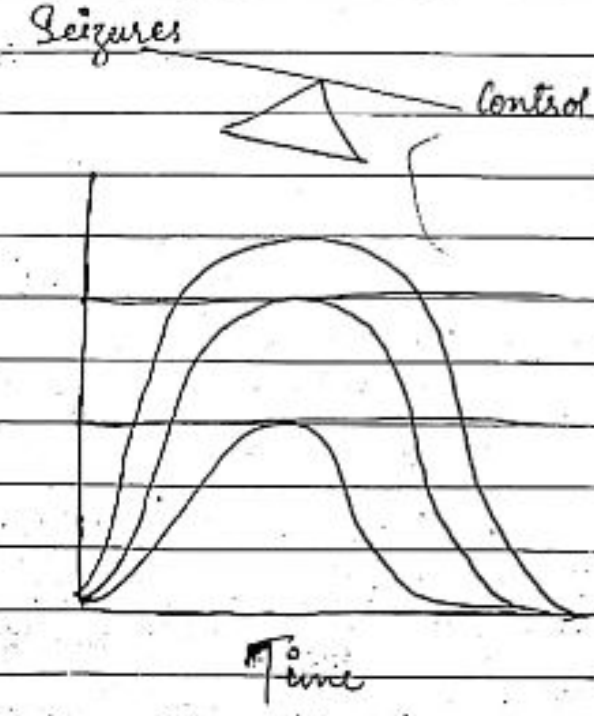
8 Phenylketonuria
9. Lead and mercury poisoning, co poisoning.
10. Genetic factor.

PATHOPHYSIOLOGY

Gene
↓ code
Protein subunits
⊕ ⊕
↓
Voltage gated and ligand gated ion channels. generates

[Na^+ and K^+ – Inhibitory
Ca^{+2} and Cl^- - Excitatory]





Dt.:
Pg.:

Delta

PARKINSON DISEASE

Parkinson Disease is a movement disorder mark by tremors, rigidity, bradykinesia (slowing of movement) and positive postural instability. It occurs when cells in one of the movement control center of the brain, bigin to die for unknown reason.

Symptoms :
- Tremors in hand, arms, legs, jaw and face
- Rigidity or stiffness of the limbs and trun
- Posture instability
- Coordination tremor
- Change in speaking
- writing ability (↓↓)
- sleep disturbance
- Depression

Causes :
- Environmental factor
- Ageing
- Genetic factor
- Oxidation stress
- Cerebrovascular disease and some medication like anticycotic drugs.

Pathophysiology :

Disease → starts degeneration → in substance nigra of mid brain

Nigrostriatial track

Dil:
Pg:

In Normal case

Human Body contain Dopamine
(+)(+)
stimulate motor neurons
Start coordination, organisation and maintain muscle tone.

In Disease Case

Dopamine ↓↓
(-)(-)
motor neurons ↓↓
(-)(-)
Lack of coordination, organisation and maintanence of muscle tone.

Oxidative Stress

oxidase,
MAO (Monoamino^disease)

MAO-A - MAO-B
↓
responsible for Parkinson

MAOB + Aldehyde dehydrogenase Enzymes
↓
increase free hydroxyl free radical.
↓
Then hydroxyl free radical + ferrous ion
(-)(-)
↓

decrease lipid membrane and DNA

↓ ⊕⊕

Then start neural degeneration (cells degrade)

Ageing

Ageing

↓

starts defects in mitochondrial electron transport chain

↓ ⊕

then degeneration in cells starts.

Cerebrovascular Disease

Produces by metabolism of antipsycotic Drug. ← MPTP (neurotoxin)

(N-methyl 4-Phenyl tetrahydroxy pyridine

↓ ⊕⊕

Starts degeneration.

PSYCHIATRIC

PSYCHOTIC DISORDER:

Depression:

It is the mental health disorder. It is also known as mood disorder characterised by persisten low mood and a feeling of sadness and loss interest. It is a parmanent problem and the average length of depressive episode is a 6 to 8 months.

Types of Depression

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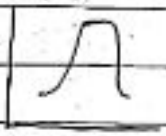
Date: Page: Delhi

There are five types of Depression

1. Major Depression – It goes to suicide thinking.
2. Dysthymic Depression – 
   - First increase
   - then stable
   - then gets decrease.
3. Bipolar Disorder (Mania) → Excitation, Sudden
4. Postnatal Depression - (After having Baby)
   - depression during birth of baby and pregnancy due to lack of sleep.
5. Seasonal Affective disorders (SAD)

Bipolar → Excitatory depression
Bipolar → sudden depression.

☆ Seasonal Affective disorders

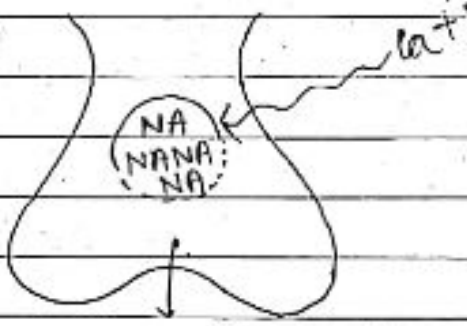
Light → Eye → hit retina
↓ ⊕⊕
Stimulate brain
↓
mood enhance

Symptoms :
- excessive sleep
- withdrawal from people.

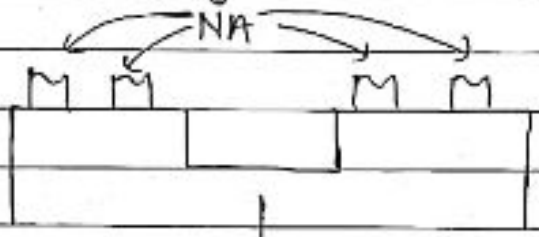
Dt.:
Pg.:

Delta

Pathophysiology:



NA (Non adrenaline)



activity starts

mood change (enhance)

- In case of depression, effective loss, formation not happen, no mood change, no activity starts.

MANIA (Bipolar Disease)

Mania is an abnormally elevated mental state typically characterised by feeling of euphoria, lack of inhibitions, raising thoughts, and diminished need for sleep, talkativeness, risk taking, and irritability. In extreme case, mania can induce hallucinations and other psychotic symptoms. Mania may be accompanied by the

over activity of non-adergenic transmission that is opposite to Depression.

Causes: 1. Abuse of Drug (specially coccain) of stimulant drug.
Ex; coccain and Amphitamines (taken by sports).

2. Glucocorticoid → by acute stress and chronic stress.

Symptoms:
- Abnormally elevated mood.
- Decreased need for sleep.
- Inflated self esteemed.
- talkativeness
- Raising thoughts
- Distructibility
- Increase in goal directed activity.

Pathophysiology: Scientist give two types of theories.

In Normal case.
Oligodendrocytes (non-nerve cells)
↓
Mylein sheath formation
↓
then it wraping and insulating exon
+
conduct nerve impulse into brain.

In case of Bipolar disorder
Oligodendrocytes
↓
Mylein Sheath get degrade
↓
then loss of insulating exon

Dt.:
Pg.:

Delta

2. Brain → Central power → cells related to emotions
↓
Damage

SCHIZOPHRENIA DISORDER:

It is a psychotic disorder marked by severely impaired thinking emotions and behaviors. Schizophrenia patients are unable to filter sensory stimuli and may have enhanced perception of sounds, colour and other feature of their environment.

Schizophrenia (Three phases)

- Acute Phase
  - Contact reality (contains true, wrong & effective symptoms)
- Stabilization Phase
  - Symptoms controls
    - relaps ↑↑
- Maintenance Phase.
  - Symptomes ge stabelised (mai -ained) + Medication (conti

Types of Schizophrenia Disorder.

1. Paranoid
2. Disorganized
3. Catatonic
4. Undifferentiated
5. Residual.

Symptoms.

DL:
PG:

Delta

1. Paranoid - Dilusions, excess of hearing voice (hallucination), Abnormal Emotions, Excessive Jealousy, suicidal and violent behaviours.

2. Disorganised - Disorganised speech & thinking, Patient behave like a kid.

3. Catatonic - Disturbance of movements, Rigidity and agitation.

4. Undifferentiated - +ve symptoms
                    -ve symptoms:

Patient have one out of two symptoms.

5. Residual - +ve symptoms
              -ve symptoms

Patient have both symptoms.

Pathophysiology

Genetic Changes
↓
Biochemical Abnormalities like DA & SHT ⟷ neurological abnormalities develop
↓
cause rhizophrenia.

Environmental factor
→ Infection
→ Injury to females during pregnancy
→ Social issue
→ Life stress

Dt.:
Pg.:

Delta

ALZHEIMER'S DISEASE (AD)

Alzheimer's Disease is the most common form of dementia. It is a neurologic disease characterised by loss of mental ability which serve enough to interfare with normal activities of daily life and this is lasting at last for 6 months.

Mental function (↓↓)

↓

Slight memory loss

↓

Ability loss to maintain, to execute, or to plan daily works

↓

loss of judgement powes (↓↓)

↓

communication skills decrase (↓↓)

Causes: 1. Genetic
2. Nerve cell destroy responsible for memory, then memory gets destroyed
3. Effect cerebral cortex responsible for judgement
4. Brain get shrink at last stage and death occur.

Pathophysiology:

Brain → (Abnormal proteins) neurofibillary tangles
Brain → amyloid plagues
] Tau Proteins

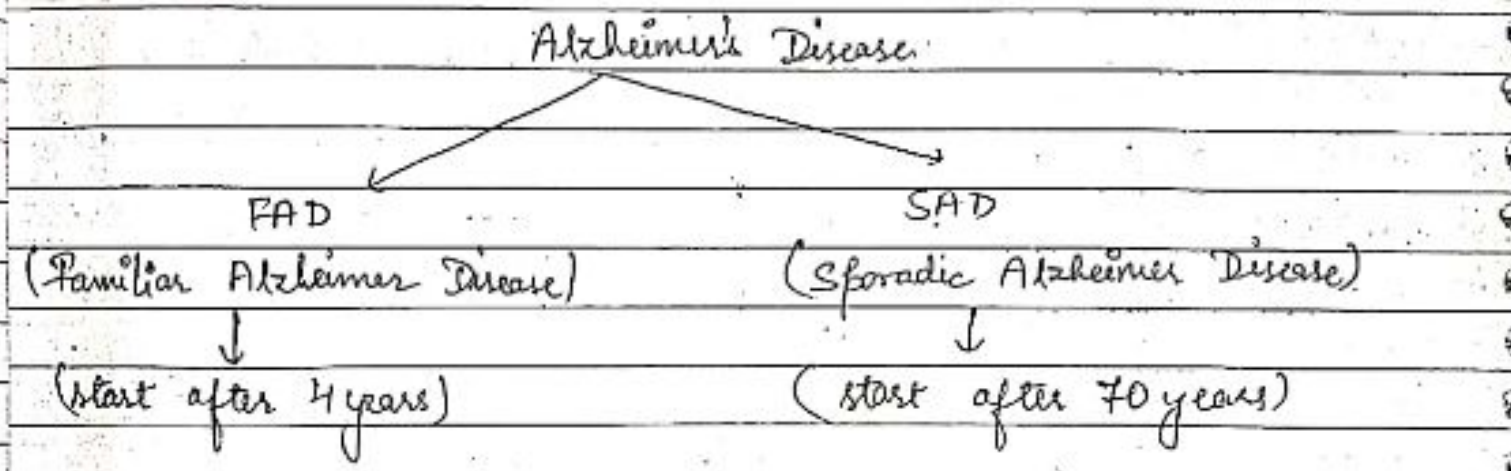
Dt.:
Pg.:

In case of disease

⇒ chemically changes in protein occur

neurons death get increased

Types of Alzheimer's disease:



Symptoms:

1. Symptoms are begin with memory lapse to progress to more disturbing loss.
2. Inability to perform routine task
3. Loss of judgement
4. personality and behaviour changes.
5. Person may suffer from confusion or agitation in the evening.
6. In the final stage, people may have severe problems with eating, communicating and controlling their bladder and bowel functions.

STROKE:

Stroke is a stage in which the blood flow to brain stops and due to this, the brain cell begin to

Dt.:
Ps.:
Delta

die. They are three kinds of stroke. The most common is called Ischemic stroke. which is caused by a blood clot that blocks the blood vessel in the brain. The other one is called hemorrhagic stroke. It is due to blood vessel breaks and bleeds in to the brain. The other type is known as Transient Ischemic attack or stroke in which mini stroke are observed.

blood supply stops
↓
cause brain cell get lack of oxygen(↓↓)
↓ ⊕ ⊕
then cell death increase (↑↑)
↓
affected brain part get death
↓
affected brain part causes memory loss, movement loss, hearing loss.

Factors in which affected brain depends. (Now it comes that Brain is affected)

1. Stroke Power -
2. Small stroke
   ↳ Temporary loss of movement legs, arms and face.
3. Large stroke -
   ↳ half body paralysis

Dt.:
Pg.:

Delta

→ Full body paralysis
→ Permanent loss of inability of speaking
→ Death.

Symptoms:
1. Trouble with speaking and understanding.
2. Paralysis and numbness of face, arms or legs.
3. Trouble with seeing in one or both eyes.
4. Headache and trouble with walking.
5. Sudden numbness and weakness of face, arm or legs.
6. Sudden confusion and sudden trouble in walking.
7. Sudden loss of balance or coordination.
8. Sudden severe headache with unknown region.

Pathophysiology: There are 3.

1. Arteries Rupture
↓ (+)(+)
Leakage of blood and formation of clot (↑↑)

2. Narrowing of Arteries
↓ (−)(−)
Blood supply (↓↓)
↓ (−)(−)
then oxygen supply (↓↓)
↓ (+)(+)
Brain cell death (↑↑)

Dt.:
Pg.:
Delta

3. Hemorrahagic Stroke

↓ Due to high BP

Pressure gets increase

↓

chances of Arteries rupture gets increased

↓

Blood comes from Arteries

↓ (+) (+)

leak blood get increase (↑↑)

↓

Brain cells get death.

PEPTIC ULCERS:

A peptic ulcer is an open sore in the upper digestive track.

Peptic ulcers

- Gastric ulcer → Present in lining of the stomach.
- Deudenal ulcer → Present in upper part of small intestine

Helicobacter pylori → Bacteria (spiral shape) which is present in acidic part of stomach. It causes degradation of lining of mucosa.

NSAIDS → Non-steroidal anti-inflammatory drugs.

Symptoms

- Abdominal pain
- Vomiting of blood
- Melina (condition in which a foul smell comes from faeces)
- Weight loss
- Heart Burn
- GERD (Gastro esophagial Reflux Disease)
  - food gets in the mouth in upper side. It occurs when secreation in excess occur.
- Epigastric pain during the meal.

Dt.:
Pg.:

Delta

Pathophysiology:

* Helicobacter pylori
↓
causes mucosa formation (↓↓)
↓
then tissues open

* Helicobacter pylori
↓ ⊕⊕
causes gastric secreation (↑↑)
↓
increase in parital cells
↓
gastric secreation bind with open tissues
↓
cause peptic ulcer

* Cyclooxygenase enzyme
↓ ↓
Cox-I Cox-II
↓
Prostaglandins (20-carbon skeleton causes inflammation)

Delta

Prostaglandins

(+) (+)

↓ inflammation (↑↑)

↓ mucosa formation

NSAID: inhibit prostaglandin but mucosa formation get decrease.

DISEASES RELATED TO CARDIOVASCULAR SYSTEM.....

HYPERTENSION:

Hypertension is the high blood pressure and blood pressure is a force by which the blood put agenst the wall of arteries as it flows through them. and Arteries are blood vessels that cassies oxygenated blood from heart to body tissues.

Hypertension

(+) (+)

↓ incrase heart work load

↓ (+) (+)

heart muscles get thick and weak

↓

Finally heart get weak

↓

Further Disease.

Types of Hypertension:

Dt.:
Pg.:

Delta

Hypertension

- Primary hypertension
  - without a proven cause.
- Secondary hypertension
  - It occurs due to medications.

Cushing's Syndrome: Red patches comes on the face.

Cushing's Syndrome + Tumor of pituitary and adrenal gland

↓

hormones of adrinal gland secreate in excess (aldosterone, cortisol, adrenalin)

↓

blood volume ↑↑

↓

Hypertension

Pathophysiology: 3 Theories

1. Kidney failure

⊖⊖

↓

Na+ exertion

↓

Atrial natriuretic factor (↓↓)

↓

(↓↓) TPRT (total peripheral resistance)

↓

Hypertension (↑↑)

Renin Angio' System (RAS System)

⇒ Angiotensinogen
↓ By Renin
Angiotensin I (decapeptide Protein)
↓ ACE Enzyme (Angiotensin converting enzyme)
Angiotensin II (octapeptide protein)
↓ Aminopeptidase
Angiotensin III (heptapeptide)
↓ Angiotensinogenesis
Inactive fragments.

Blood → BP (↑↑) ⊕⊕ ⊖⊖ ↓ Renin
Vasoconstriction + Na+ & H_2O retention + Aldosterone secretion ↑↑
Aldosteron (↑↑)

3. Sympathetic Nervous System (Theory)

B_2 ⊕⊕ → Stress
↓
Stimulate Sympathetic Nervous system
↓ Hormone release.
act on B_2 ⊕
↓
BP ↑↑.

B
B_1 B_2 B_2
B_1 (Heart muscles and Bronchus)
B_2 (Adipose Tissue)
Juxta glomerular cells

Pre hypertension = 129/89
Stage (I) hypertension = 140-159 / 90-99
Stage (II) hypertension = 160-179 / 100-109.

Dt.:
Pg.:
Delta

CHF (CONGESTIVE HEART FAILURE):

In Heart failure, the heart lose the ability to pump in a blood to the tissues which lead to lose blood supply to the organs and tissues resulting in dificient supply of oxygen and nutrients to function properly. In congestive

Inability of heart to supply enough blood.

↓ ⊖⊖

Blood supply ↓↓

↓

tissue and organ Death

↓

Tiredness + arrythemia ← causes Pulmonary edema (Swelling)

Heart failure has two types

1. Left Sided heart failure
2. Right Sided heart failure.

Left ventricle abnormalities (In case of Left sided heart fa

↓ ⊖⊖

Blood supply inhibit

↓

Shortness of breath and coughing

↓

fluid store in lungs

↓ If pulmonary edema then

Blood comes in coughing.

Delta

(In case of Right sided heart failure)

Right ventricles abnormalities

↓

Blood supply to lungs get decrease

↓

Blood get collect to Right side

↓

edema (feet and legs & thighs)

Causes Of CHF :
- Coronary heart disease
- Silent heart attack
- Cardiomyopathy
- Hypertension
- Heart Valve disease
- Congental heart disease
- Alcolism and smoking
- Drug abuse.

Symptoms of CHF :
- Shortness of Breath
- Frequent coughing specially when lying down.
- Swollen feet ankles and legs.
- Abdominal swelling and vomiting.
- fatigue
- Dizziness, fainting
- Sudden death

Pathophysiology: Divided is to two parts

Systolic Dysfunction Diastolic Dysfunction.

Dt.:
Pg.:

Systolic Difunction:

Ventricle contraction ability ↓↓

⊖⊖

Stroke volume – Systemic blood need ↓↓

⊕⊕

heart work load ↑↑

heart failure

It is due to Stress

Diastolic Disfunction

⊖⊖

Ventricle compliance ↓↓

⊕⊕

Venous pressure ↑↑ to maintain ventricular filling

⊕⊕

Work load ↑↑ of heart

CHF.

ISCHEMIC HEART DISEASE:

It is imbalance between myocardial oxygen supply and demand which resulting in myocardial hypoxia (lack of O_2) and accumulation of waste metabolites. Ischemia is an insufficient supply of blood to an organ due to a blocked artery.

IHD causes due to:

IHD

- Silent
- Symptomatic
  - Chest pain
    - angina pectoris

Classification of Ischemia Heart Failure.

IHF

- Infarct effect
  - myocardial infraction
- Non-infarct effect
  1. Chronic ischamic heat disease.
  2. Sudden cardiac death
  3. Angina pectoris
     - Stable Angina
     - Unstable Angina
     - Angina in Brain
     - Transient Ischemic Attack (TIA)

Causes of IHF:

1. Blockage of an Artery
2. formation of Blood clots
3. Silent Ischemia is caused by Emotional or mental stress or exertion without any symptoms.
4. Heavy Exercise
5. (TIA) is caused by Blood clot due to the Blockage of cerebral artery.

Dt.:
Pg.:

Delta

Factors which effect:

1. Hereditary
2. Sex (Male or Female)
3. Age.
4. Smoking
5. High Cholesterol
6. High B.P.
7. Lack of physical activity
8. Diabetes Mallitus
9. Obesity
10. Stress and anger.

ANGINA PECTORIS:

It is an oppressive heavy crushing pain or a constrictive feeling in the centre of the chest behind the sternum or on a left side of the chest. The pain may radiate to one or both arms generally more to the left arm. It can be experienced in the throat, jaw and a stomach and more rarely between the shoulders.

Angina

- Classical Angina
  - atherosclerosis
  - Platelet aggregation
- Variant or Prizmetal's angina
  - Artery spasm
  - magnesium Overload conc. ↓↓.
  - calcium conc. ↑↑.
- Unstable angina
  - Spasm

DL:
Pg.:

Causes: more Physical Exercise ⇈ Psycological stress, extreme cold, heavy meal specially at night time, coronary erythrosclorosis, anaemia, narrowing of the coronary artery, severe aortic stenosis, hypertrophy, Hyperthyredism

Hypoglycemia: blood glucose level ↓↓ ← energy provide

⊖⊖

energy

⊕⊕

O_2 demand

Angina

Hyperthyredism: in CVS (cardio vascular system)

thyroid effect on CVS.

- Palpitation
- excessive intolerance
- edema
- O_2 demand ⇈

⊕⊕

Heart work load to pump blood

Heart muscle weak

⊕⊕

Angina and any disease of heart.

Dt.:
Pg.:

T_3 ⟶ catabolic effect

↓

Protein used as source of energy

↓ prolonged time

-ve Nitrogen Balance

↓

Tissue waste

↓ ⊕⊕

O_2 demand ↑↑

↓

for Body growth

↓

Angina

Pathophysiology :

Narrowing of arteries

↓ ⊖⊖

Blood flow ↓↓

↓ ⊕⊕

O_2 demand ↑↑

↓

Anaerobic metabolism

↓ ⊕⊕

Lactic acid deposition

↓

Myocardial nerve fibre irritation

↓

Pain radiation ⟶ Angina

Lactate ← absence of O_2

Scanned with CamScanner

MYOCARDIAL INFRACTION (MI):

Myocardial Infaction also known as Heart attack. The Myocardial Infaction focus on the heart and the changes is nacrosis (cell death) of myocardial tissue. The word infaction means the clotting of artery.

blood supply inhibit in 1 part of heart

↓ ⊖⊖

O_2 ↓↓

↓

cell or heart tissue damage or death

no blood flow or little blood flow.

↓

cardiac activity does not complete

↓ ⊕⊕

Chances of Infaction

Subendo cardial infaction : Sub type of M.I.

Blood flow ↓↓ in subendo cardiac infaction

↓

Infaction.

Pathophysiology:

Stagnant blood

↓ Or cavity ⊕

Muscle fibre

Dt.:
Pg.:

↓

Hb bluish colour

↓

heart infarcted area colour changes

⊖

Blood vessels weak

↓

Blood goes to adjoining tissue

↓

edema cause.

Symptoms :
- Chest pain
- Sensation, tightness, pressure or squeezing
- Pain radiates to left arm
- Pain also radiates to the lower jaw, neck, Right arm, back and epigastrium.
- Heart Burn
- Shortness of Breadth
- Pulmonary edema
- nausea, vomiting and palpitation
- Loss of consciousness
- Sudden death

~~ARTHEROSCHEROSIS~~ : ATHEROSCLERO

Atherosclerosis is a specific type of arteriosclerosis. It is a disease in which plaque builts up inside the arteries. Plaque is made up of fat, ~~cholesterol~~ cholesterol, calcium and other substances in blood.

Dt:
Pg:

Delta

The plaque hardens the narrow arteries. This limit the flow of oxygen rich blood to the organs and other parts of the body. Although, Atherosclerosis is considered a heart problem but it can affect arteries anywhere in the body.

Types:

Symptomatic

Heart arteries	Arteries supplying the brain	Arteries in the arm and leg.
Obstruction in coronary arteries	obstruction in the carotid artery.	Obstruction of blood in the arteries in arms and legs
heart attack	Stroke	leg pain during walking
Chest pain	sudden numbness	Paralysis -
	weakness	
	dizziness	

Causes: 1. Damage or injury to the inner layer of an artery
2. High Blood pressure
3. High Cholesterol
4. Smoking
5. Some disease like diabetes.

Pathophysiology

LDL → Non-enzymatically Oxidized → Ox-LDL (low Density Lipids)

↓ release

bioactive phospholipids

↓ ⊕ ⊕

Dl.:
Pg.:

endothelial cells

↓ ⊕⊕

Pro-inflammatory + pro coagulant — other initiation on step of Artheroscelosis

↓

leukocytes formation ↑↑

↓

Adhere to ~~intima~~ edema.

ARTERIOSCLEROSIS:

It is the defect occuring in the artery walls. It refers to hardening of the normally flexible valves due to the loss of elasticity of the arterial structure. On young stage, the arteries are flexible due to the presence of protein known as elastin as age increase, there is a loss of elastin which cause thickning of the arterial valves.

Ca^{+2} + Plaque deposit

↓ ⊕⊕

arteries wall thick

↓ ⊖⊖

elastin protein

↓

Walls become brittle and hard.

Heart work load ↑↑

↓ (+)(+)

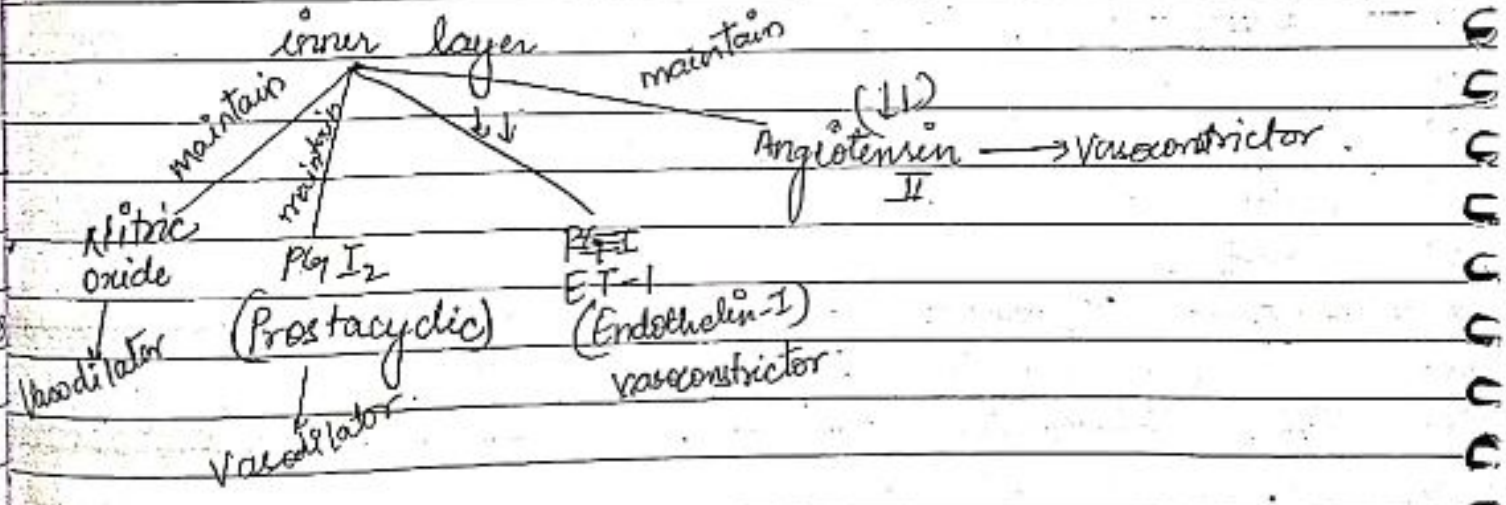
MI.

Cause and Symptoms → Same as Atherosclerosis.

Pathophysiology →

1. Endothelial Disfunction
2. Adhesion of blood cells
3. Immigration of Adhered WBC.

1. Endothelial Disfunction



High BP, Diabetics, Milletus, smoking.

↓

endothelial dysfunction

↓↓ NO ↓↓ PGI2 (+)(+) ET-1 ↑↑ Ang-II

↓

Improper heart work.

Dl.:
Pg.:

Delta

2. Adhesion of blood cells.

Normal artery

↓ Endothelial dysfunction

→ NO ↓↓.

↓ ⊕ ⊕

ET-1 ↑↑

↓

P-selection & E-selection release

(category of cell adhesion molecules) (glycoprotein

VCAM-1 release

(Vascular cell Adhesion molecule)

ICAM-1 release.

(Intracellular cell adhesion molecule)

↓

leucocytes & Thromocytes & fibrin release

↓

Blood clot

3. Immigration of adhesed WBC.

In case of macrophage.

↓

IL-8 (Interleukin)

↓

cellular necrosis + fibroblast + cellular codesion

IL-8 + MCP-1 (CCL-2) ← Chemokine Ligand-2

↳ Macrophage chemoattract Protein-1.

↓

Diapedesis

DISEASES RELATED TO LUNGS....

Asthma :

Asthma is charactised by hyper-responsiveness of tracho-bronchal smooth muscles to a variety of stimuli which resulting in narrowing of air-tubes and often accompanied by increased secreation, mucosal edema and mucous plugging. Asthma is also an inflamma-tory disease.

Asthma

- extrensic
  - episodic
  - status asthmaticus (bd)
  - attack repeat again & again.
- intrensic
  - long lastic
  - status asthmaticus (↑↑)

* allergens
  ↓
  attack on mast cells + inflammatory cells.
  (present in lungs)

Etiology (symptoms & causes)

Dt.:
Pg.:

Delta

↓

lungs → histamine, TNFα, Protease enzyme

lungs → Cell membranes activate ⊕ → Phospholipids formation ⊕⊕ → PG, LT, PAFs stimulate.

lungs → Genes get activate → Protein synthesis → IL, TNFα produce.

IL, TNFα produce → Smooth muscle constrict → mucous get release → air passage constrict → Asthma trigger occur.

Symptoms:
- Shortness of breath (dyspnea)
- Wheezing
- constriction in chest
- tachypnea (rapid breath)
- tachycardia (rapid beating) heart rate
- Inflammation over the chest
- During severe attack person turn blue due to lack of oxygen.
- loss of consciousness
- respiratory arrest & death.

Causes:
- allergens
- Medication like aspirin and beta blockers.
- air pollution like ozone, nitrogendioxide and sulphur dioxide.
- Industrial compounds & chemicals like monoamino chlorine di and triamino chlorine

- Early childhood
- Virde respiratory infection
- heavy exercise
- emotional stress.

Pathophysiology : → Bronchio constriction
→ Inflammation (Inflammed airway)

smoke dust fetters (+)(+) → Inflammed airway
↓ (+)(+)
excess mucous secreate
↓ (−)(−)
Breathing decrese

antigen + Inflammed airway
↓ (+)
antigen - antibody reaction
↓ (+)(+)
Inflammation
↓
Contraction
↓
Branchio spasm
↓
asthma attack.

Dt:
Pg:
Delta

When happens in antigen - antibody reaction.

allergen inhaled

↓

airway pass
ingest

↓

antigen Presenting cells APC + dendrites

TH_1 (cell mediated Immunity activate)

TH_2 (humoral immune response activate)

allergen + antibodies } for 2nd time.

recognise

↓ (+) (+)

HSR

↓ (+) (+)

IL, Histbrachio spasm.

↓

airway constrict

↓

asthma attact.

CHRONIC OBSTRUCTIVE LUNG DISEASE

It is a progressive disease with emphysema (alveolar dist and bronchial fibrosis in variable proportion. The expiratory airflow limitation does not fluctuate but the

Dt.:
Pg.: Delta

are exacerbations precipitated by respiratory infections & pollutants. It is clearly related to smoking and generally starts after the age of 40.

Symptoms:
- Cough with and without mucous
- fatigue
- respiratory infections
- dyspnea
- Wheezing Wheezing.

1-(naphthyl-3-oxy)-(dimethyl propyl amine)-propanol

Dt.:
Pg.:

Delta

16/04/18

RENAL DISEASE.....

Acute Renal failure: Acute kidney failure occurs when illness, infection or injury damage the kidney. Temporary - the kidneys donot remove fluid and waste from the body and maintain the proper level of certain kidney regulated chemicals in the blood stream.

- # heart disease
- # liver damage
- # hemorrahage
- # Seiver burns
- # Cancer

Protein urea
Stenosis
Blockage of vessels
Reflux

Symptoms:

1. Decrease in RBC production
2. Bad Breadth or Bad taste
3. Bone and joints problem

Sunlight on the skin → 7 - dehydrocholesterol → UVB under skin → vit. D_3.

Calcifediol → hydroxilation by kidney → Calcitrion C1,25 dihydroxycholecalciferol or vit D

DL:
Pg.: Delta

1. Edema
2. Puffiness around the eyes
3. Swelling in arms and feet
4. foamy and bloody urine -
5. headache
6. hypertension
7. Fatigue
8. Itching (phosphorus accumulate in body)
9. lower back pain
10. Nocea (By urea retension)

Pathophysiology :

Renal Blood flow ↓↓
↓ ⊖⊖
Glomelcur fitration Rate
} 3 Mech.

Mech.

1. Pre- renal failure
tubular and Glomerular functions changes comes
↓
Renal perfusion → GFR ↓↓.
changes comes

2. Intrensic Renal failure

Delta

Glomerular and tubular dysfunctions due to excess increase in renal efferent-vasoconstrictor.

↓

filtration rate ↓↓.

3° Post-obstructive renal failure:

Tubular Pressure ↑↑

+

Blood filtration ↓↓.

↓

Pressure Gradient get equilize

↓

depressed Glob. F.R.

In case of Ischemia

Renal blood flow ↓↓.

↓

Ischemia or cell death.

↓

O2 free radical + enzyme release

↓

cell injury

↓

Chronic renal failure.

Dt.:
Pg.:

Delta

CHRONIC RENAL FAILURE: Permanent loss of renal function

- Severe illness
- increase creatinine in blood serum.
- end of ARF.

Chronic renal failure is a slowly progressive loss of renal function over a period of month or years and defined as abnormally low glomerular filteration rate which is usually determined indirectly by the creatinine level in blood serum. Chronic renal failure can lead to severe illness and require regular renal replacement therapy which is also known as end stage renal failure disease.

Causes: 1. Diabetes
2. Hypertension
3. Nephropathy
4. glomerulonephritis

Pathophysiology: Nephrons ↓ GFR

* In case of Renal injury ↓ Nephrons

Dt.:
Pg.:

Delta

Kidney ability ↓↓

GFR maintain ↓↓

- hyperfiltration
- Compensatory hypertrophy (cell size ↑↑)

Increase level of Creatinine & urea. (double)↑

* Renal injury + disease + Medication

Growth factor ↑↑ ↑↑

Cytokinins ↑↑ ↑↑

Vasoactive molecule ↑↑ ↑↑

RAS ↑↑ ↑↑

hypertension

↑↑ ↑↑

- Glomerular hypertension
- hypertension
- hyperfiltesation

↓

Glomelural hypertropy (cell size ↑↑)

↓

Sclerosis

↓

health remaining nephron activity ↑↑

JOINTS RELATED DISEASES....

DL:
Pg:

Delta

RHEMATOID ARTHRITIS:

It is a chronic inflammatory auto-immune disorder that cause the immune system to attack the joints. It is the disabling and painful inflammatory condition which can lead to substantial loss of mavility due to pain and joints distruction.
Rhemaloid Arthritis is a systemic disease which often affecting extra articular tissue throughout the body.

Inflammation of joints
↓
Lead to other joints
↓
disease progress
↓
Inflammation (↑↑)
↓
Erosion & distruction
↓
Joints deformities and movement (↓↓)

Deformities:
- Ulnar deviation
- Ulnar drift
- Boutonniere deformity
- Swan neck deformity (fingers tip bend towards palm)
- Z- Thumb deformity.

Dt.:
Pg.:
Delta

Smoking + Infection + any kind of trauma.

Enter in body

Immunity ↑↑↑

Synovial hypertrophy

Chronic inflammation in joints

⊕ ⊕

Extraarticular damage

Symptoms: Two Types

1. Systemetic 2. Extra-articular. 3- Dermatologic

Systemetic: Starting with fever than malaise, (arteralgia) pain in joints.

Weakness before progressing to joint inflammation and swelling. Persistant symmetric polyarthritis of hand and feet. Articular deformities extra-articular involvement, difficulty in performing activities of daily life.

Extra-articular symptoms: GIT bleeding which is due to the side effect of non-steroidal anti inflammatory drugs which is used for analgesic, splenomegaly

DL:
PS:

Delta

(enlargement of spleen), leukopenia (Reduction in WBC), lymphatic infilteration

Dermatological symptoms: Subcutaneous eruption.

Pathophysiology:

Infection + genetic factor + smoke or any other antigen.

Reaction

B-Cells + T-cells

⊕⊕ auto antibodies.

Rheumatoid factor, anticyclic citrullinated peptied antibody produce

Inflammation produce

Molecular and structural damage of bones and joints

CD4 cells ↑↑

inflammation ⊕⊕

→ Interleukins

Tumor necrosis factor ← TNFα, IL-1, cytokine.

Act on

Synovium

Synovial infiltration

⊕⊕

Inflammation

Destruction of Synovium, cartilage and bone.

Proliferation

degeneration of synovial membrane.

Dt.:
Pg.:

Delta

GOUT:

It is a metabolic disorder characterised by hyperuricemia. Uric acid which is a product of purine metabolism and has a low solubility especialy at low PH. When blood level are high it precipitates and deposit in joints kidney and subcutaneous tissue. The deposition increase in size and burst through the skin to form sinus discharging of chaky white material.

Monosodium urate crystals deposit + articular cartilage of joints and tendons

⊕⊕

Inflammation

⊕⊕

Size

Burst

white chalk material skin

chronic gout.

Symptoms: Pain, swelling, redness, hyperthermia of particular area, stiffness in joints, low fever may also be present. Skin become tender and sore it is slightly touched.

Normal Range: 1-4 mg/dl

greater than 1-4 mg/dl (chronic gout).

Dt.:
Pg.:

Delta

1-3 mg/dl (Acute gout)
below 4 mg/dl (No gout)

2° hyperuricemia or 2° Gout

Thiazide, furosemides
Levodopa, clofibrates

⊖⊖

Uric acid secreation

⊕⊕

Blood uric aced

Gout

Acute
- Sudden onset of severe pain
- suffer small joints

Treatment may be occur.

Chronic
- Pain, stiffness (long term)
- Skin of pinna, eyelids, nose, kidney.
- Deformities occur.

Treatment may not be occur.

Acute: Colchicine

Chronic: Allopurinol, probenced.

Dt.:
Pg.:

Pathophysiology :

Uric acid ↑↑

↓

Tissue and blood Na.

↓

Uric acid + Na

↓ ⊕⊕

Monosodium urate (uric acid)

↓ ⊕⊕

Deposit on skin and joints

↓

PG, IL, TNF-α ↑↑

↓

Inflammation occur

↓ ⊖⊖

Granulocyte ↓↓

↓ ⊕⊕

Free radicals

↓ ⊕⊕

Proteolytic enzymes

↓

Lysosomal enzyme

↓

Joints destruction

Dt.:
Pg.:

Gout has four stages
- Asymptomatic
- Acute
- Intercritical
- Chronic

OSTEOPOROSIS:

It is defined as disease characterised by low bone mass and microarchitectural detonation of bone tissue which leading to enhanced bone fragility and a constiguent increase in fracture risk.

Osteoporosis

Primary	Secondary
not related to any disease ageing Hormone dysfunction Decrease level in estrogen due to during menopause of female.	related to other disease. chronic illness Trauma. diabetes

Causes: Ageing
Malnutrition
reduced level of estrogen

Dt.:
Pg.:

Delta

reduced estrogen ⊕⊕ → by osteoblast + osteoclast

reduced estrogen ⊖⊖ → bone production ↓↓ ⊖⊖ ← by osteoblast + osteoclast

Pathophysiology:

matrix degrading enzyme ⟷ matrix synthetic enzyme (Normal case)

In case of disease

matrix degrading enzyme ⟷ matrix synthetic enzyme.

Osteoporosis ⊕⊕ → degrading enzyme ↑↑ ⊕⊕ → degradation of matrix ↑↑. ⊖⊖ → Proteoglycans + collagens ↓↓ ⊕⊕ ← Chondrocyte proliferation → Chondrocytes

If it occurs for long time then it decrease Chondrocytes proliferation ultimately Chondrocyte decrease & cause Osteoporosis.

Inflammatory Bowel Diseases

Dt.: 28/08/18 Tuesday
Pg.:

JAUNDICE:

It is a yellowish discolouration of the skin and eyes caused by abnormaly bigh level of pigment bilirubin in blood stream.
Normal bilirubin - 2.5 - 3 mg/dl.

Old or damaged RBC
↓ spleen
↓ remove
Haemaglobin break down
↓
(H) Bilirubin (dark greenish yellow pigment)
⊕⊕
blood circulation
↓
liver
↓
intestine excrete out as component of bile

skin yellowish colour or jaundice ↑
Bilirubin deposit in tissue ↑
In blood cells ← ⊕⊕

Causes:
1. Any inflammation cause jaundice
2. Damage to the liver due to inflammation
3. Any impairement in liver.
4. Liver Serosis
5. The blockage of bile ducts either by gall stone or tumour.
6. Over production of bilirubin due to excessive breakdown of RBC.

How bone reabsorb calcium?

Dt.:
Pg.:

Delta

2nd theory

Post. menopausal

↓

bone reabsorption

Cytokinins, IL-1, IL-6, TNFx.

↓

Bone reabsorb.

Senile. (Poor mental ability)

↓ release Renal & hydroxylase enzyme ⊖⊖

$1,25(OH)_2D$ ↓↓

$1,25(OH)_2D$ ↓↓

↓

absorption of Ca^{+2} in blood ↓↓

↓

calcium level decrease in plasma.

↓

release PTH (Parathormone)

↓

⊕ bone reabsorb calcium

Dt.:
Pg.:

Delta

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

(COPD, CORD, COAD, CAL)

It is a progressive disease with emphysema (alveoli destruction) and bronchial fibrosis in variable proportions. The expiratory air flow limitations does not fluctuate but there are exacerbations precipitated by respiratory infection and pollutants. It is clearly related to smoking and generally starts after the age of 40

Symptoms: Cough with and without mucous, fatigue, respiratory infections, dyspnea and wheezing

Causes: Chronic smoking, α_1-anti trypsin deficiency, Idiopathic cond^n in which disease comes sporadically with unknown reason, prolonged exposure to dusty environment specially in coal mining

α_1-antitrypsin → liver release → lungs protect

☆ SERPINA = 1 gene form α-1 antitrypsin. It is mutated → α_1-1 antitrypsin formation stop → lung damage.

☆ α_1-1-antitrypsin ⊖⊖ → neutrophile elastase (4 enzyme)

Irritants like smoke, dust etc. release →

Dt.:
Pg.:

Delta

- neutrophil
- CD8 (receptor of lymphocytes)
- B-cells
- Macrophages
- TNF-α (Tumor Necrosis factor)
- Interferon-γ (IFN-γ)

(Belong to cytokines category)

- Matrix Metallo proteinase (MMP6)
- C-reactive protein (CRP)
- Interleukins
- fibrinogen

Interleukins

↓ (++)

Inflammation process

↓ (++)

Tissue damage

↓

Airflow limitation

Ist case:

If inflammation occurs

↓ then

Narrowing of airway

↓

due to this, parenchymal destruction

↓ (--)

loss of tissue lung elasticity

↓

Emphysema (alveolar wall destruction)

↓

lung capacity (↓)

Dt.:
Pg.:

Delta

Third case:

Inflammation

↓

narrowing of airway (hypersecretion of mucus)

↓

[Peribronchial fibrosis]

↑ mucus production (↑↑es)

↓

lung capacity (↓↓es)

Third case:

Inflammation

↓

Airway tissue scar formation.

If patient inhale smoke and dust

↓

Epithelial cells

↓ TGFβ

fibroblast formation

↓ (+)(↑)

cell division (↑↑es)

Smoke and dust

↓

epithelial cells

↓

CXCL8, CXCL9, CXCL10, CXCL11

↓ (+)(↑)

T helper cells

(+)(↑) → Macrophages (+) CXCL8 & CXCL9 Neutrophils

(+)(↑) → Tc cells (+)(↑) → emphysema

INFECTIOUS DISEASE

DL: Pg: ......Delta

LEPROSY...... (Henson's Disease)

causing agent: Mycobacterium Leprase.

It is a chronic granulomatous infection caused by micobacterium Laprae which primarily affects skin, mucous membrane and nerves. Lapracy also known as Hanson's disease which is an infectious disease caused by DNA plasmid carried by Hanson baccillus also known as microbacterium Laprae which is a vector.

Causes:

1. Drinking of contaminated water.
2. Person who lives in andemic areas drinking contaminated water insufficient diet, other diseases like HIV, thyroid, and ge
3. Genetic variation

Classification:

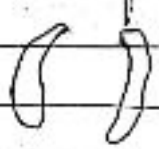
- → Lepromatous (Pale colour patches)
- → Boderline
- → Boderline Lepromatous
- → Intermediate (discoveration of skin (white)
- → Boderline Tubercloid
- → Tubercloid. (Avidamatous ~~locater~~ maculy (one) in body.)

Dt.:
Pg.:

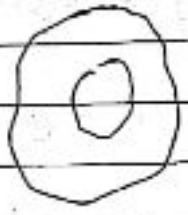
Delta

Types of Hypomalous macules

arciform



annular



→ no hair
→ anaesthetic
→ dry ends scaly

Pathophysiology:

Case-1 M. leprae + 4 α-chain of laminin-2 of Schwann cell

⊕⊕

↓

multiplication of M. leprae ↑↑

⊕⊕

↓

cell mediated immunity ↑↑

↓

Chronic inflammation ↑↑

⊕⊕

↓

Swelling perineurium

↓

Ischemia & fibrosis

&

axonal death.

Case II

M. Leprae

↓ due to their genes

Metabolism in host

↓ remain

Mycobacterium cell wall

↓ ⊕⊕

(Immunoglobulin) IgM stimulate

↓ ⊕⊕

CH-I Stimulated

↓

Lepra reaction

Strong CMI + Weak humoral response

↓

Mild form disease + less bacterial content

X-CMI + Strong humoral response

↓

(Lepromatous Leprosy) LL - lesion spread Skin

Case III

Toll like receptor

TLR_1 & TLR_2.

Drug → Dapsone [DDS]. used in leprosy.

DL:
Pg.:

M. Lapra act on TLR, & TLR2 (Schwann cells)

↓ ⊕⊕

Nerve damage (↑↑)

↓

disease (↑↑)

MENINGITIS :

° Inflammation of meninges

↓ ⊗⊗

Brain cells

↓ ⊗⊗

Regenerate

↓

death

due to
→ Bacteria
→ virus
→ fungi
→ Bleeding into meninges
→ Cancer disease
→ Chemotherapy.

Meningitis is a serious inflammation disease of meninges which is thin membranious covering a brain and spinal cord.

Meningitis

↓ Bacterial

↓ Non-Bacterial

DL:
PG:

Delta

Bacterial

→ Haemophillus influenzae B.
→ Neseria meningitidis
→ Streptococcus pneumoniae.
→ Listeria monocytogenes - Child.

N- Bacterial

→ disease like cancer, AIDS.
→ Drug induced

Causes:

1. Age, habits, living environment, health states, chronic alcoholism, upper respiratory tract infection, patients suffering from AIDS, Tuberculosis and skull fracture.

Symptoms: headache, fever, vomiting, irritation, severe fatigue - confusion, mental retard, coma.

Pathophysiology:

Organism
↓ Nasopharynges
Nasopharyngeal colonies
↓
Invade
↓ (+)(+)
Blood enter
↓
injury of endothelial cells
↓
Meningeal invade.
↓
subarachnoid space inflammation
↓ (+)(+)

Intracranial pressure (+)(+) → cerebral infarction

cerebral vasculitis (inf. of blood vessel wall)

vasogenic edema

BBB (Blood Brain permeability - Brain Barrier)

Intracranial pressure (+)(+) → cytotoxic edema

DOT: Direct observation Treatment

(Cerebro spinal fluid)

CSF outflow resistance

↓

hydrocephalus (fluid retension in brain cavities)

↓

interstial edema (edema within the

↓ (+)(+)

intracranial pressure

↓ ⊖⊖

cerebral blood flow

Tuberculosis

Mycobacterium Tuberculosis
- small rod shaped like bacteria
- acid fast bacteria
- grow in dry state

TB is an infectious disease caused by micobacterium tuberculosis which commonly affect lungs, CNS, lymphatic system, circulatory system, genito urinary system, bone & lungs.

- M. Tuberculosis
- M. bovis
- M. africanum
- M. Microti (cattles & animals)
- Non-Tuberculous Mycobacter

Dl:
Pg:

Delta

Causes: Intake of contaminated food & water

Symptoms: 1. Prolonged cough more than 3 weeks duration
2. Chest pain
3. hemoptysis (blood in cough)
4 Fever, chills
5. Night sweating
6. Loss of apetite
7. Weight loss & letharginess

Pathophysiology:

Bacteria
enter
Pulmonary alveoli
infect
Pulmonary macrophages
replication
(nodule formation) Ghon focus + Dendritic cells infect
Transportation of Bacteria
in lymph node
blood stream
various tissues organ

Dt.:
Pg.:

Delta

TB cells ⊕⊕ → immune system

⊕⊕

T- lymphocytes

release

cytokine like factor IF-Y.

⊕⊕

Activate macrophages

kill bacteria.

Drugs in t.B : (1) Isoniazide
(2) Ethambutol
(3) Streptomycin
(4) Pyrazinamide

} 1st line drugs

TYPHOID :

Infecting agent : Salmonella Typhi
S- Typhimurium

Typhoid fever is a systemic infection caused by Salmonella Typhi which usualy occur through ingestion of contaminated food and water.

Causes: Ingestion of contaminated food & water

Symptomes: loss of apetite
Vomiting
Naus@
headache
constipation
High fever (103° to 10
Diarrohea
confusion
abdominal pain

Dl.:
Pg.:

Delta

Pathophysiology :

Bacteria
↓ enter
intestine (1-3 weeks stay)
↓ invade
intestinal wall
↓ enter
blood stream → Diarrhea, Haemorrhage
↓
WBC carry → Cholecystitis (inflammation in gall bladder)
↓ in
enter gall bladder, liver, spleen and bone marrow
↓
replicate in these organs
↓
gas in GIT tract → pus in faeces.
sometimes blood stream & Kidney

Dt.:
Pg.:
Delta

Urinary Tract Infection

UTI: UTI is an infection of the urinary tract infection which is anycohere from the kidney to the ureter, Bladder and urethra.

causes: 1. Sickle cell anaemia patient
2. highly active sex worker
3 More in females than males (size of urethra small & close to anus)

Symptoms: 1. Urtheritis (Burning sensation thoough out urethra by micturition)

2. Cystitis (pain in mid line suprapubic region) - connection b/w the bladder & skin which is used to drain the urine

3 Haematouria (blood in urine).

4. Cloudy & foul smell urine
5. fever upto more than 3 days
6. Nausea & vomiting
7. frequently urine

DL:
Pg:

Delta

Pathophysiology:

E. coli
↓ enter
bladder mucosa
↓ invade & enter
Periurethral vaginal introitus
↓
multiply
↓ during intercourse
bacteria migrate
↓
further invade
↓
cyst formation

Fav. PH of E. coli → 5-5

Medicine - NFT - 20 mg [lower the PH of urine to 3]

Dt.:
Pg.:

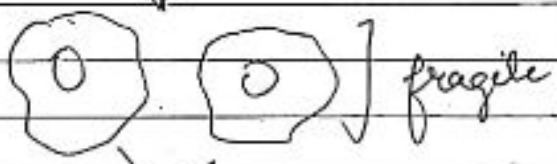
Delta

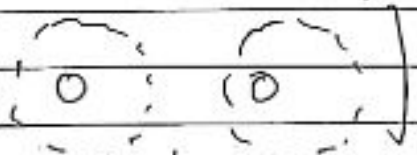
Symptoms: 1. The skin and white of the eyes appear yellow colour.
2. The colour of urine become dark yellow
3. Itching
4. light colour stool
5. loss of apetite
6. Nausea, vomiting, fever.
7. Blockage of bile duct may cause abdominal pain and fever.

Pathophysiology: After 120 days

⊕⊕

RBC breakdown (HT)





enter in RAS

membrane rupture

RBC's Breakdown

cellular content like Hb release in blood

Cholestyramine → improve symptoms.

Dt.:
Pg.:
Delta

Hb free form → phagocytosis

macrophages ingest

Hb Breakdown

Heme → Heme oxygenase cause oxidation → oxidn → biliverdin (green colour pigment)

Co+fe

Cytosolic enzyme biliverdin reductase → redn → Bilirubin (greenish yellow)

Jaundice cause.

Globin → Protein break down → amino acids → No Jaundice occ

Alcoholic Liver Diseases Cirrhosis:

deposition of fats and scars

Protein form → blood clot

Liver

- Germ & Bacterea remove
- remove or neutralize poisons
- Immunogents produce
- Produce bile to absorb fat & fat soluble vita

Disulfuram → Alcohol से जुड़ा हुआ Medicine

Date: Page: Delhi

It is a condition in which the liver does not function properly due to long term damage. The Damage is characterised by replacement of normal liver tissue by scar tissue.

Causes: 1. Excess consuption of ethanol.

2. Chronic Hepatitis C disease HCV

↓

Causes inflammation in liver cells

↓

cirrohsis

3. Hepatitis B and D

HBV → & inflammation → Liver cells injury

HDV → ↓ infect already suffering HBV.

4. Auto immune Hepatitis

Pathophysiology:

Alcohol

↓ metabolism alcohol dehydrogenase

Acetaldehyde

↓ Acetaldehyde dehydrogenase

acetic acid

$CO_2 + H_2O$

NADH Production increase ↑↑↑

Dt.:
Pg.:

Delta

Due to ↑↑ of NADH prodⁿ

⇒ NADH : NAD⁺ changes

NADH ↑↑

⊕⊕ oxidⁿ

fatty acid

NAD⁺ ↓↓

oxdⁿ ⊖⊖

fatty acid ↓↓

⊕⊕

fatty acid level ↑↑ in blood.

signal prodⁿ

glycerol

Triglyceride (↑↑)

Accumulation in liver.

fatty liver

liver cirrhosis

2nd case

After liver cirrhosis

⊕⊕

TNF-α, IL6 & IL8

⊕⊕

liver apoptosis & necrosis (↑↑)

Dt.:
Pg.:

If again alcohol consumption

gut → LPS endotoxin → Portal circulation → Kupffer's cells → progoxytosis of endotoxin → TNF-α → cell death.

Liver apoptosis & necrosis

* HEPATITIS :

It is a disease that affects liver causing it to swell and become inflammed.

Types (May be)
- Mild
- Chronic
- .....

- A
- B
- C
- D
- E

Dt.:
Pg.:

Delta

Virus

↓

enter in body

enter through portal circulation

↓

liver

↓

Swelling + inflammation

↓

Cirrhosis

bcz of
- liver damage
- scar form
- dysfunction

↓

Cancer

Hepatitis - A

- Least threatening
- Symptoms shows in 30 days
- Chances in baby

It is acctually beginning hepatitis that is caused by picrono virus and Generally spread by contaminated food and water.

Symptoms: Nausea, Dark urine, fatigue, vomiting, Abdominal pain.

Treatment: 1. Hepatitis A Vaccine

1 dose → 2 dose

6 months

prevent for 20 years.

2. Gamma Globulin injection.

Dt.:
Pg.:

Delta

Hepatitis-B

- cell damage
- cell death
- cirrohsis

Virus: HbsAg [Hepatitis-B surface Antigen]
Test: Anti HbsAg.

Types: 1. Replicative
⇒ Chronic phase
⇒ virus continuously replicate in liver
} 3 drugs combination (FDA)

2. Non-replicative
⇒ minimal
⇒ low liver injury cause

2. Virus: Cytomegalovirus

Symptoms: Jaundice, discouration of skin, light colour stool formation, Arthritis

Drugs - Adefovir
- Lamivudine
- Interferon-α

Hepatitis-C

- not spread by sexual contact
- spread by saliva

Delta

- shows in AIDS patients due to lack of immunity
- by needles
- by razors

Symptoms: NO jaundice, nausea, vomiting, stool formation, lack of apetite

Diagnosis: antiHCV
- PEG or Pegylation
↓
Cholestrol like substance
+
interferon-α.
↓
inject in patients ∫effect for 3-weeks.

Hep. D

NABQI - N- acetyl, p- benzoquinoneimine.

* Gluconuration saturate
⊖⊖
↓
hepatic glutathione
+++
↓
cirrhosis & hepatotoxity. NABQI metabolite
↓
necrosis ← liver protein + renal protein → degrade

Dt.:
Pg.:

Delta

CANCER :

It is a class of disease in which a group of cell display uncontrolled, invasion and sometimes metastases (Spread by lymph or blood)

Stem cell
↓
new cell formation
↓
cell multiplication
↓
cell differentiation
↓
cell death

Cancer cell.

Cancer Cycle

G_1 Phase (Nucleic acid Synthesis
↓
S phase → DNA Synthesis
↓
G_2 Phase → Post-DNA Synthesis
↓
M Phase (Mitosis)
↓
G_1 / G_2 → G_0 — Non-proliferating

Dt.:
Pg.:

Delta

☆ Cell cycle specific Anti cancer drug

1) anti metabolites
   - 6- mercaptopurines

2) Plant alkaloids
   - ~~Vic~~ Vincristine
   - Vinblastine

3) Bleomycin (antibiotic)

☆ Cell cycle Non- specific
- cell multiplication G_0 phase
- insulating agent
  - Nitrogen mustard
  - Carmustine
  - Lomustine
- Antibiotics
  - Doxrubicin
  - Donrubin

Types of Tumour

Benign Tumour
↳ Suffix → "oma"
Ex:

Dt.:
Pg.:

Delta

fibrosis tissue

↓

fibroma.

= Cartilaginous tissue benign tumour

↓

Chondroma.

Types of Benign Tumour

1. Adenoma
2. Papilloma
3. cystadenoma

* Adenoma — gland produce

↓

epithelial cells

↓

gland like tumour

* papilloma - like cell formation in neck or eyes.

Malignant tumor

- progressive
- infiltration (goes from one part to another)
- invade
- don't develop capsule.

Dt.:
Pg.:

Delta

Cancer arise from mesenchymal tissue ues etc.
↓
Sarcoma.

cancer arise from epithelial tissue
↓
carcinoma.

Maligant tumor follow 3 mech. to spread
⇒ Seedling with in body cavity
⇒ through lymphatic System
⇒ hematologic system.

. Seedling with in body cavity :
Ex: Carcinoma of colon
↓
Penetrate through the gut wall
} through Peritoneal cavity.

Ex: Carcinoma of lungs
↓
spread in pleural cavity

Through lymphatic System

Delta

Ex: Lung Carcinoma

↓

respiratory passage

↓

bronchial lymph nodes

↓

tracheo – bronchial

↓

hilar nodes (roots of the lungs)

Haematologic Spread

Spread through blood. then to liver.

Chemical carcinogens

Aquired mutation

↓

Proto oncogenes and oncogenes altered

↓

cell proliferation dedifferentiation

Inherited mutation

↓

tumor suppressor gene (T.S.)

↓

Decreased apoptosis

Development of Tumour

↓

Delta

Production of metalloproteinases etc.

↓

Invasion of nearby tissue by tumour cells

↓

Angiogenesis

↓

Metastasis

↓

Development of 2° tumour

(*)

26/08/19 — P' cology

Pharmacokinetics : A D M E

↓

in, through, out of the Body.

Dosage form → Solution form → Free drug

Site of action

Storage site for → compare/found

Acidic drug → Penicillin

Specialised Transport

excrete out → Bile, kidney, sweat, saliva, faeces

(A)

Absorption — Biological Membrane. (two layers)

Phospholipids, Cholesterol

thickness = 100 A°

(1) Drug.

Acidic – anionic

Basic – Cationic