Asthma

Asthma is a chronic inflammatory disorder of airways, in which many cells and cellular elements play a role.

→ Risk factors

Specific abnormality underlying asthma is hyper-reactivity of the airways to one / more stimuli.

* There are no. of possible risk/trigger factors:
  - Allergens - pollen, moulds, house dust mite, animals (dander, saliva, urine).
  - Industrial chemicals - isocynate containing paints, aluminium, hair sprays
  - Drugs - aspirin, ibuprofen, β-adrenoceptor blockers
  - Food - (rare) nuts, fish, seafood, dairy products, food coloring
  - Miscellaneous - cold air, exercise, hyperventilation, viral respiratory tract disease, emotions, stress

* Having a blood relative with asthma
* Being overweight
* Smoking, passive smoking.

Chronic Obstructive Pulmonary Disease

COPD is a group of clinicopathologic conditions characterized by airflow limitation due to chronic, partial or complete obstruction to the airflow at any level from trachea to the smallest airway, that is not fully reversible.

→ Symptoms:

COPD symptoms often don't appear until significant lung damage has occured and they usually worsen over time, particularly if smoking exposure continues.

Signs and symptoms of COPD may include:

- Shortness of breath, especially during physical activities
- wheezing
- Chest tightness
- Chronic cough (that may produce sputum that may be clear, white, yellow or greenish
- Frequent respiratory infections

- Lack of energy
- Unintended weight loss (in later stages)
- Swelling in ankles, feet or legs

People with COPD are also likely to experience episodes called exacerbations during which symptoms become worse than usual day to day.

→ Pathophysiology page - 10

→ Treatment

(A.) Bronchodilators:

they usually come in inhalers. They relax muscle around your airway.

(a) Anticholinergics (anti muscarinics)

SAMA (short)	LAMA (long)
Ipratropium Br.	Tiotropium Br
Oxitropium Br.	Glycopyrronium Br
	Glycopyrrolate

(b) β2 Sympathomimetics (agonists)

SABA	LABA
Salbutamol	Salmeterol
Terbutaline	Formeterol

(c) Methyl xanthines

Theophylline
Aminophylline
Doxophylline

(B.) Corticosteroids

this reduce airway inflammation and help prevent exacerbations.

Topical / Inhalation	Systemic / oral / iv
Beclomethasone	Prednisolone
Fluticasone	Hydrocortisone
Budesonide	Methyl prednisolone

(C.) Long acting β_2 - agonist (oral) + inhaled corticosteroid
(LABA + ICS)

- Salmeterol | Fluticsone
- Formeterol | Budesonide

(D.) Mucolytics

mucus regulatory drug

Bromohexine
Acetylcysteine
Carbocisteen

* NON - PHARMACOLOGICAL (GOLD)

1. Smoking cessation
2. Basic information about COPD
3. General approach to therapy
4. Strategies to help minimize dyspnea
5. Advice about, when to seek help
6. Decision making during exacerbation
7. Maintenance of exercise & physical activity
8. Adequate sleep and healthy diet

Surgery — lung volume reduction
bullectomy
lung transplant
bronchoscopic intervention

Management

Diagnosis

Symptoms
Risk factors
Spirometry

Initial assessment

Exacerbation history
Smoking status
Co-morbidities
α_1-antitrypsin

↓

Initial Management

Smoking cessation
Vaccination
Active lifestyle, exerci
Self management
Education
Manage co-morbidities
Risk factor managemer
Written action plan
Inhaler techniques
-Breathlessnes

←

Review

Symptoms
Exacerbation
Smoking status
Exposure to risk factor
Spirometry
Physical activities
Exercises

↓

Treatment

Pharmacological
Non-pharmacological.

3. Endocrine System

DIABETES MELLITUS

Diabetes Mellitus is a clinical syndrome characterized by an increase in plasma blood glucose (hyperglycaemia). It may cause most common type, I and II.

⟶ TYPES

Diabetes mellitus may be classified according to etiology, the most common types being

Type I – • formerly refered to as Insulin - dependent diabetes mellitus.
• it is a disease that causes destruction of insulin producing β-cells, it is either autoimmune T cell mediated destruction [Type IA], or idiopathic [Type IB]
• it generally occurs in young age (below 30).

Type-2 – formerly referred to a non-insulin dependent diabetes mellitus.
- Common above the age of 40y
- It is caused by relative insulin deficiency or insulin resistance.

Other types – Pre diabetes (before stage 2 when sugar level is on border line.

Gastational diabetes (caused during pregnancy and it ends after parturation

Monogenic diabetes syndrome (rare, 4% of population)

Drug induced diabetes (HIV, AIDS, RA, any autoimmune disease, have intake of steroids can cau this type, it affect β-cells or any cell that induce diabetes)

→ Risk factors

Type-1

- Family history : having parents with type 1 diabetes increases risk of person having same type.
- Age : Type 1 usually develops in younger children and adults.
- Genetics : having specific genes may increase risk of type 1 diabetes

Type - II

- doing less or no exercise : no physical activity increases chances of this type.
- Hypertension : one of major risk factor
- Obesity : being overweight, especially having excess weight
- Heart and blood vessel disease and stroke
- Cholesterol : high density lipoprotein (HDL)
- Fats : high levels of triglycerides

→ Oral hyperglycaemic drugs

(a.) Enhance insulin secreation

(1) K_{ATP} channel blockers

* Sulfonylureas
  - Tolbutamide
  - Glipizide
  - Glimepride
  - Glyburide

* Meglitinide (phenylalanine analouges)
  - Repaglinide
  - Nateglinide

(2) * Dipeptidyl peptidase -4 inhibitors
  - Sitagliptin
  - Linagliptin
  - Alogliptin

(3) Glucagone like peptide 1 receptor agonist
  - Liraglutide

(b.) Overcome insulin resistance

(1) Biguanide (AMPK activator)
  - Metformin

(2) Thiazolidinedione
  - Pioglitazone

(C.) Miscellaneous drugs

(1) Sodium - glucose co-transport 2 inhibitor (SGLT-2)

Dapagliflozin
canagliflozin

(2) Dopamine D_2 agonist

Bromocriptine

(3) Amylin analogue

Pramlintide

(D.) Retard carbohydrate absorption

(1) α - glucosidase inhibitors

Acarbose
Voglibose

→ Pathophysiology

Type - 1
(insulin dependent diabetes mellitus)

Autoantigens
· Genetic and Environmental factors

↓

Activation of Th 1 - cells

↓

Release of cytokines
(IL-2 and IFN - γ)

↓

Insulitis
(inflammation of β - cells)

↓

Leading to death of
β - cells

↓

Causes Type 1 Diabetes Mellitus

Type - II
(non- insulin dependent diabetes mellitus)

Genetic & Fav background comorbidities

Environmental factors

Constitutional factors
- Obesity
- Hypertension
- Low physical activity

↘ ↓ ↙

β- cells dysfunction (failure)

↓

Decreased Insulin secreation
+
Insulin resistance

- Impaired glucose utilization
- Receptors defects
- Glucose toxicity of islets

↓

PRE - Diabetes

↓

Hyperglycaemia

↓

Type - 2 DM.

→ Long term complications of Type-1 & II

Complications that can occur over time:

(A.) Microvascular / neuropathic

Retinopathy, cataract
- impaired vision

Nephropathy
- renal failure

Peripheral neuropathy
- sensoryloss
  Motor weakness
  Pain

Autonomic neuropathy
- Postural hypotension
- Gastro intestinal problems
  (gastroparesis, altered bowel habits)

Foot disease
- Ulceration
  Arthropathy

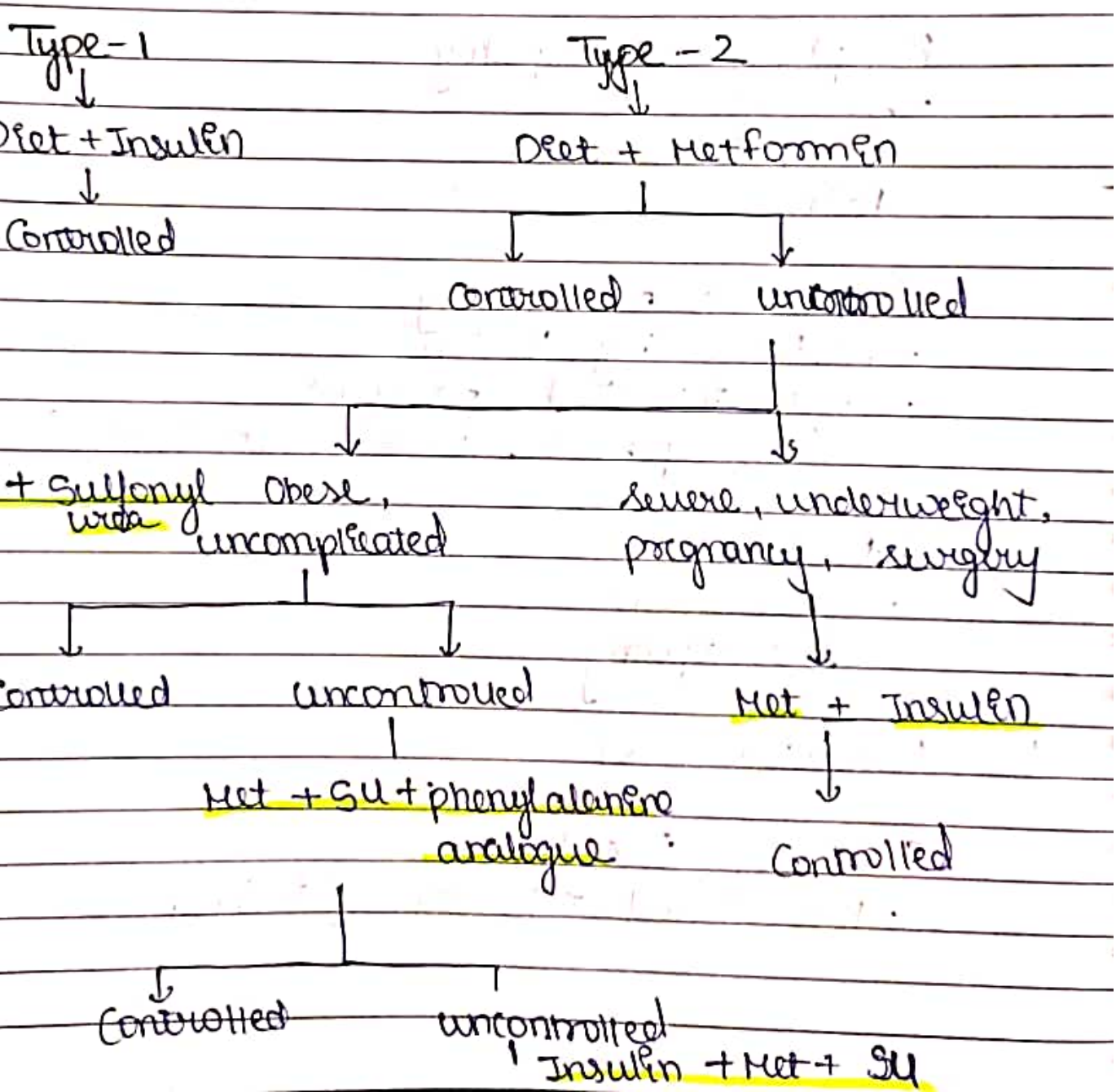
(B.) Macrovascular

Coronary circulation
- Myocardial infarction / ischaemia

Cerebral circulation
- Transient ischaemic attack
- Stroke

Peripheral circulation
- Ischaemia
- claudication

→ Management



→ Insulin types and explained any one—

Insulin is synthesized by β-cells of pancreatic islets from a single chain polypeptide precursor (110 AA) from which 23 AAs are first removed to produce proinsulin. The C peptide with 35 AAs is removed, Remaining with 2 chain polypeptide having 51 AA.
The A-chain has 21 while B-chain has 30 AA.

* Types of insulin preparations and insulin analogues.

Rapid acting

Lispro
Aspart
Glulisine
} Duration - 3-5h
Appearance - clear

Short acting

Regular (Soluble)
} Duration - 6-8h
Appearance - clear

Intermediate acting

Insulin zinc suspension
Isophane insulin
} Duration - 12-20h
Appearance - cloudy

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Long acting

	Duration	
Glargine	24h	Appearance - clear
Determir	20 - 24h	

→ HUMAN INSULIN :

Human insulin are produced by recombinant DNA technology using Escherichia coli or yeast.

They have similar amino acid sequence as insulin.

Eg : Regular human insulin & human NPH insulin

Human insulin is more water soluble as well as hydrophobic than other insulin.
It has a slightly more raped S.C absorption, it has defined peak concentration.

It is modified to produce isophane (NPH) and lente.

E-coli

Produced by – prb (proinsulin recombinant bacterial)

Yeast

pyr (precursor yeast recombinant)

Porcine

emp (enzymatic modification of porcine insulin)

→ PHARMACOLOGY

(A.) Metformin:

It is a drug of class Biguanide used to treat hyperglycemia

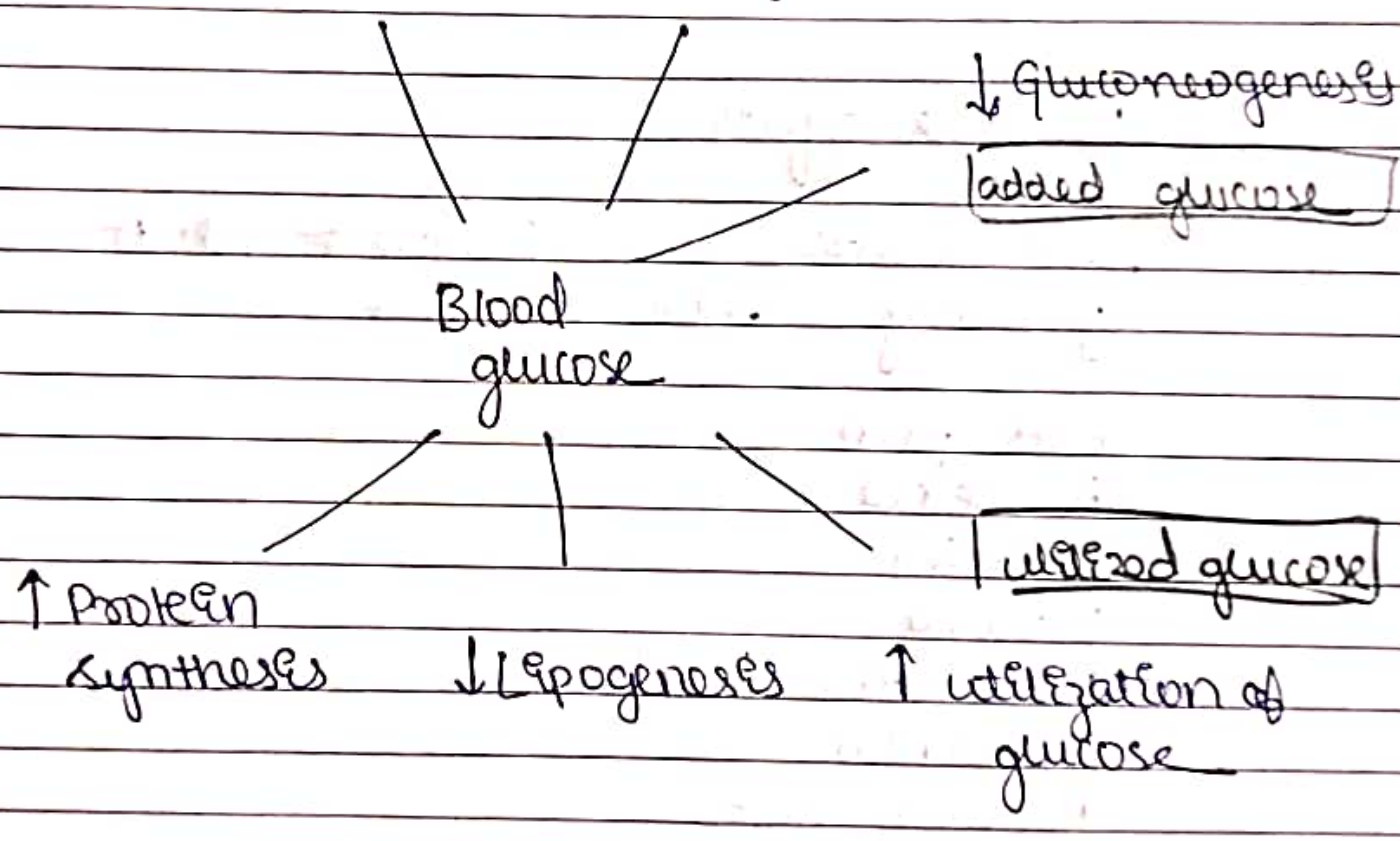
Biguanides do not cause insulin release but presence of insulin is essential for their action.

Mechanism of action:

- Hepatic gluconeogenesis (lyses) – it decreases glucose output from liver this is the major action responsible for lowering glucose from blood.
- Decreased intestinal absorption of glucose – alimentary absorⁿ is also reduced
- Peripheral glucose uptake – enhances insulin-mediated glucose uptake & disposal in skeletal muscle & fat.
  - Glycogen storage in skeletal muscle
  - reduce lipogenesis

Unlike sulfonylurea, metformin does not produce hypoglycemia and do not cause hyperinsulinemia.

- Pharmaco



Therapeutic uses

Metformin is the first choice drug for type 2 DM.

- prevent onset of type 2 DM
- Anti-hyperglycaemic
- weight loss promoting
- Not hypoglycaemic
- prevent macrovascular as well as microvascular complication of Diabetes
- No acceleration of β-cell failure
- Also used in polycystic ovarian disease, as it improves ovulation and fertility.

(B.) Sulfonyl urea

It is divided into two generations. Second are more potent than first.

Drugs – Tolbutamide
Glimepiride
Glepizide

Mechanism of Action

- Stimulates insulin secreation from β-cells
- SU acts on sulfonylurea receptor on the cell membrane, it restrict ATP sensitive K channels.
- The outward flow of K is restricted Intracellular K concentration rises Membrane is depolarized.
- Influx of Ca^{++} by channel is increased and release of Ca^{++} from intracellular stores.
- Ca^{++} ions promote fusion of insulin
- Exocytotic release of insulin.

* Lung volumes - they are :
  - tidal vol - air entering more
  - residual vol - air left after forced expiration
  - inspiratory reserve vol - max air inspiration
  - expiratory reserve vol - max air expiration

* Maximal inspiratory pressure (PI Max) and Maximal expiratory pressure (PE Max) - They are tests that is used to measure strength of respiratory muscle.

* washing asymmetry - it detects hidden diffusion defect. it checks the percentage of haemoglobin that is oxygenated.

SU
↓
Binds to receptor of β-cells
↓
BLOCKS ATP-sensitive K channel
↓
Influx of Ca^{++} ions into β-cells
↓
Depolarization of membrane
↓
Increased release of stored insulin from cells.

Pharmacokinetics

Absorption - orally
Distribution - plasma bound (least with low volume)
Metabolized - liver
Excreated - Kidney (through urine in active/inactive form)

Adverse effects

Incidence of adverse effects are low (3-7%)

Hypoglycaemia - is most common problem it is occasionally severe and rarely fatal.

GI disturbances - Nausea
Vomiting
Diarrhoea

weight gain

Hypersensitivity – rashes, photosensitivity, purpura, agranulocytosis

Safty of SUs during pregnancy is not established. Change liver to insulin is advised.
SUs are secreated in milk.

Therapeutic uses

SUs are useful in patient with type 2 DM.

THYROID DISEASE

The thyroid gland secreats 3 hormones –
thyroxine T_4
triiodothyroxine T_3
Calcitonin

Thyrotoxicosis

Also called hyperthyroidism

It is due to excessive secreation of thyroid hormones.
Two main causes are 'Graves disease' and toxic nodular goiter.

→ Signs and Symptoms

Common : Weight loss
Increased appetite
Heat intolerance, sweating
Palpitations
Tremor
Dyspnoea
Fatigue
Emotional labelity
Moist skin

Less common : Osteoporosis
Diarrhoea
Ankle swelling

Anxiety, psychosis
Muscle weakness
Infertility

Rare : Vomiting
Ageusia
Anosmia
Exacerbation of Asthma

→ Treatment

Non-pharmacological :
Cease smoking
Diet: Control

Self Management

Exercise - physical activity

Keep body hydrated. Avoid caffeinated drinks for anxiety

Pharmacological :

Inhibit hormone synthesis (Thionamides)

Methimazole
Carbimazole

Inhibit Iodine trapping

thiocyanate SCN^-
perchlorate ClO_4^-

Pituitary gland → TSH - thyroid stimulating hormone
Thyroid gland - T_3, T_4

Inhibit hormone release

Iodine
Iodides of Na, K
Organic Iodine

Destroy thyroid tissue

Radioactive Iodine ^{131}I

* MOA of Carbimazole :

Carbimazole bind to thyroid peroxidase enzyme and inhibit synthesis of thyroid hormone.

They inhibit -
Inhibit
(a) Oxidation of Iodine
(b) Iodination of tyrosine
c) Inhibit coupling of iodotyrosine residues to form T_3 & T_4

* Carbimazole converted to thiamazole after absorption.

Adverse effects

Rash
Fever
Agranulocytosis
Leucopenia
Hepatotox (Rare)
Aplastic anemia
Thrombocytopenia
Dermatitis
Skin eruptions

Hypothyroidism

It is a clinical state that results from decreased production of thyroid hormones or very rarely from tissue resistance.

Etiology

- Failure of thyroid gland by autoimmune distruction
- Effects of treament of thyrotoxicosis
- Also be drug induced by agents such as amiodarone, lithium
- Failure of hypothalamus
- Insufficient release of ^pituitary gland hormone by (TSH)

Signs and Symptoms

Skin :- dry skin, cool skin
flaking of skin
Reduced sweating
- puffy face, eye
dry hair
brittle nails

Oral Contraceptives

* Oral contraceptives

- These are hormonal preparation used for reversible suppression of fertility.
- These are hormonal pills that used to prevent concept of fertilization
- These pills contains estrogen or progesteron or both.
- Various types of oral contraceptives are given. Infra.

* Combined pill

- These pill contains both estrogen and progesteron.
- Widely used and most effective
- Mainly used.

Estrogen - Ethinyl, Estradiol
Progesteron - Norgestrel, Levonorgestrel, Desogestrel.

- Can be given as -

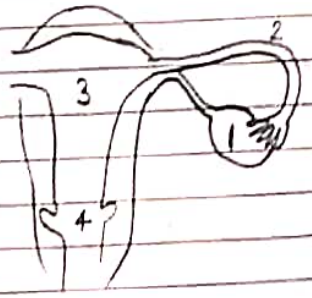
Monophasic	Biphasic	Triphasic
Dose of estrogen and progesterion is fixed through out the treatment	Dose of estrogen kept constant but, progesteron varies are in phase of menstrual cycle.	Dose of estrogen is slightly more & Dose of progesteron ↑ in three successive phase of menstruation

* DOSE :

Estrogen - 20-30 µg
Progestin - 0.1 - 1 mg

* MOA of contraceptives

① Acts on hypothalamic - pituitary axes
↓
Inhibit release of FSH, LH
↓
Inhibit ovulation



2. Contraction of fallopian tube & uterus
↓
interfere with fertilization

3. Makes endometrium less suitable for implantation.

4. Thick, viscid cervical mucus secretion
↓
Prevents sperm penetration (progestins).

* Progestin only (mini pill)

- Mini pill contains very low dose of progestin
- It is taken daily continously without any gap.

* MOA

M.C tends to become irregular and other contribute to the contraceptive action.

Dose: Norgestrel – 75 µg
Norethendron – 350 µg

- Pregnancy should be suspected if Amenorrhoea of more than 2 month occurs.

* Emergency (post coital pill):

- These pills are for use in a women not taking any contraceptive who had a sexual intercourse risking unwanted pregnancy.

- These emergency contraception should be reserved for unexpected or accidental exposure rape, condom rupture only. Bez it has many side effects.

- These will interfer ∑ implantation and also have antiovulatory effect.

Estrogen

Estrogen are naturally occuring sex hormones produced by ovary, adrenal gland and placenta.

Mechanism of Action

Estrogen
↓ ← Enter cell by diffusion
Estrogen receptor < α / β
↓
Release of receptor binding protein
↓
Formation of estrogen receptor
↓
Binds to target gene
↓
Co-regulator < activator / repressor
↓
Biological & Clinical effect.

Pharmacological actions

1. Sex organ - it brings about changes in the female including growth of uterus, fallopian tube, vagina.

2. Secondary sex character - estrogen produced at puberty causes growth of breasts.

pubic and axillary hair appears

behaviour is influenced.

pigmentation of genital region.

3. Metabolic effects - increases bone formation

converts Vit D to D_3 in Kidney

increases HDL levels and decreases LDL levels.

Therapeutic uses

Currently, the two most common uses of estrogens are as contraceptive and for hormone replacement therapy (HRT) in postmenopausal women, but there are some other indication as well.

1. Oral contraceptive – most common use for synthetic estrogen is for contraception.

2. Hormone replacement therapy – short term estrogen therapy is used to relieve menopausal symptoms such as depression, sleeplessness, irritation.

3. Vaginites – estrogen helps prevent drying, thinning and inflammation of vaginal walls.

4. Delayed puberty in girls – pubertal changes are brought about by a cyclic estrogen treatment.

5. Dysmenorrhoea – estrogen therapy is given to prevent painful menstruation.

6. Dysfunctional uterine bleeding – progestin given cyclically in rational & effective therapy. Estrogen have adjuvant value.

Hormone Replacement Therapy

Hormone Replacement Therapy (HRT) is an effective treatment symptoms.

Clinicians used systemic HRT to treat women in menopause, believing that HRT could benefit cardiovascular health prevent osteoporosis and help women live longer and healthier lives.

* Benefits of HRT:

Hormone replacement therapy is most effective treatment for vasomotor and therapy vaginal symptoms associated with menopause.

Estrogen only therapy decreases coronary heart disease and reduces risk of breast cancer in women.

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* Diagnosis

1. Slit lamp exam:

Instrument consist of microscope and high energy beam light

2. Visual acuity tests:

Checks to see if conjunctivitis has affected your vision.

3. Eye culture:

Collect sample of cells, inside of your eyelids with a cotton swab & examined.

* Types of hormone replacement therapy:

ESTROGEN

The body produces three types of estrogen that can be supplemented.

Estrone [E_1], made in our fat tissue, present in body after menopause

Estradiol [E_2], strongest estrogen, present in body before menopause.

Estriol [E_3], weakest estrogen made by ovaries present in body during pregnancy.

Conjugated equine estrogen [CEE] is most commonly prescribed oral estrogen replacement. It includes mainly estrone [E_1] and is derived from pregnant horse urine.

Estradiol [E_2] is also available as oral, tropical and vaginal preparations.

* Starting doses include

Conjugated equine estrogen	0.45 - 0.65 mg OD
Estradiol	10 mcg intravaginally
Micronized 17β-estradiol	0.5 - 1 mg OD

PROGESTERONE

Any women with a uterus taking oral or transdermal estrogen must also take progesterone to prevent endometrial cancer.

Recent advancements to improve absorption & duration of action of progesterone have resulted in development of micronized progesterone [MP].

Commone starting dose:

MP — 100 - 200 mg OD
Medroxyprogesterone acetate 1.5 - 5 mg OD

Osteoporosis

[OESTEOPENIA]

Osteoporosis is reduction in bone density that result when rate of bone resorption (destruction of bone tissue that promotes bone loss) exceeds the rate of bone absorption.

It is due to loss of bone mass or bone mineral density characterized by height reduction, fractures, back/neck pain.

Mostly the bone of spine, hip, lumber and femur.

(a.) Signs and symptoms:

- There typically are no symptoms in early stages of bone loss.
- Back pain, caused by a fracture or collapsed vertebra
- Loss of height over time
- A stooped posture
- Brittle & fragile bone

* Predisposing factor (Risk).

Genetics – Single gene disorder (LRP5)
Inheritance

Endocrine disease – Hypogonadism
Hyperparathyroidism
Hyperthyroidism
Cushings syndrome.

Inflammatory disease – Inflammatory bowel dis
Rheumatoid arthritis

Drugs – Heparin
Alcohol intake
Levothyroxine
Glucocorticoids

GI disease – Malabsorption
Chronic liver disease

Lung disease – COPD
Cystic fibrosis

Miscellaneous – Heavy smoking
HIV infection
Anorexia
Myeloma
Homocystinuria (AA - Methionine lack)

* Pathogenesis

The mechanism competence of the skeleton is maintained by the process of bone remodelling.

In this bone remodelling a quantum of bone is removed by oestoclast followed by formation of new bone by oestoblasts.

= ~~Oesto~~ osteoporosis results from ↑es bone breakdown by oestoclasts

and ↓es bone formation by oestoblast leading to loss of bond mas.

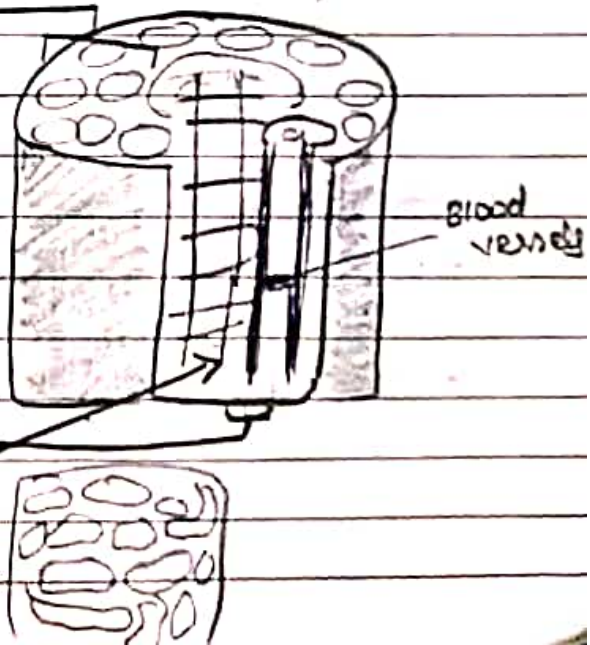
= loss of bone mass leads to loss in density of bone.

(Other pathological causes:)

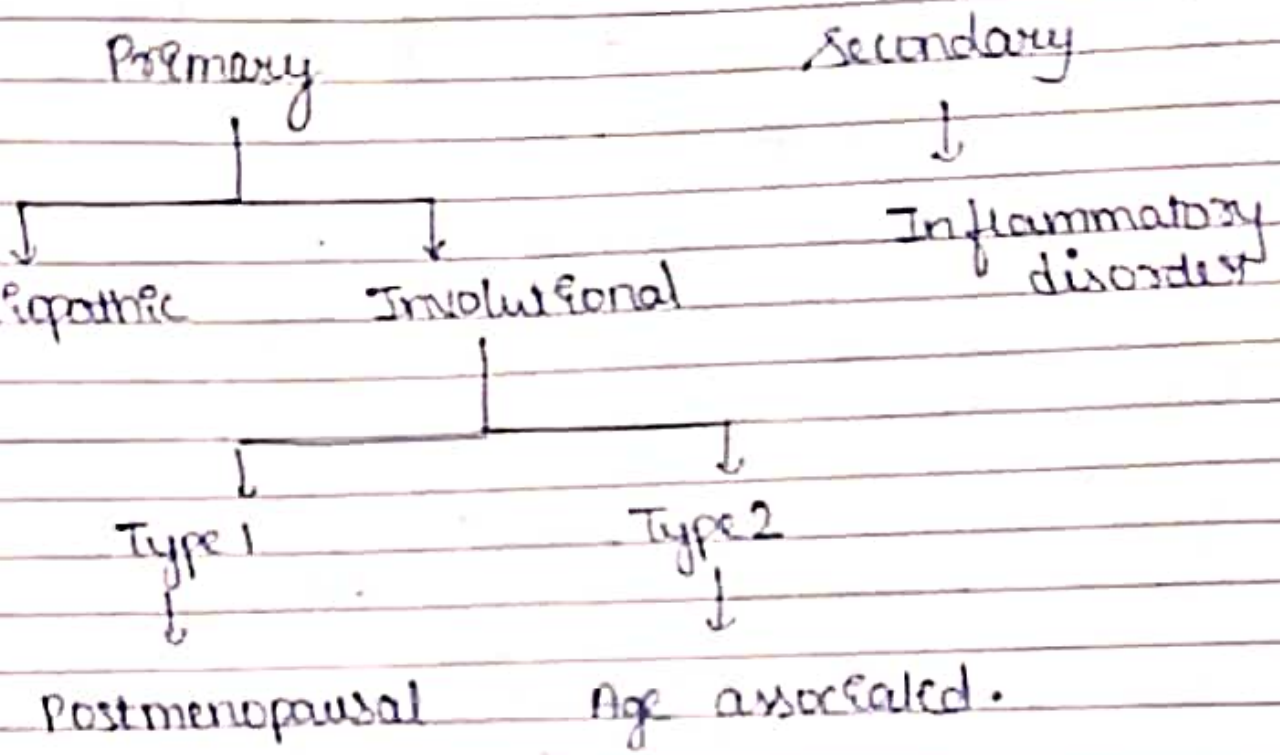
Thinning of cortical bone

Widening of haversian cannals

↓ No. of Trabeculae of in spongy bone



* Types



* Diagnosis

Dual - energy X - Ray absorptiometry
↓
DEXA SCAN

- It tests for bone density
- It shows the patient bone density compared to the normal bone ".
- Result is T- score
  ≤ 2.5
  ↳ is osteoporosis

* Treatment

Non- pharmacological

Exercise - bearing physical activities improves balance and posture. It also can strengthen bone. Reduces chances of fracture.

Good nutrition - eating healthy diets and getting enough calcium & Vit D.

Quit smoking - Smoking cigarettes speeds up bone loss

Limit alcohol - If anyone choose to drink alcohol, do so in moderation.
women - one drink daily
men - two " "

Surgery - surgery with internal fixation is frequently required to realign and stabilise oestoporotic fractures

Vertebroplasty used in treatment of painful vertebral compression fracture.

Dynamics

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Risk factors :

1. Exposure to infected individual.
2. Poor hygienic habits
3. Contact with towels, napkins, pillow cases, handles
4. History of ocular disease (dry eye anatomic surface)
5. History of autoimmune disorder
6. Immunosupression and trauma can weakens immune system.

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4. Cool compress

to relieve discomfort associated with bacterial conjunctivetis.
4 time a day.

Sign and Symptoms

- Pinkness or redness of the eye.
- Thick, sticky discharge from the eye
- Burning, itching, mild pain or discomfort in the eye.
- swollen or reddened eyelids.
- Blurred vision
- Pus (discharge, yellow in color)

Pharmacological

1. Bisphosphonates

first line treatment for osteoporosis
target bone surface
impairs bone resorption.
given for 5y

Alendronate
Ibandronate
Clodronate
Pamidronate
Etidronate
Risedronate

2. Denosumab

Antibody that inhibit bone resorption
Subcutaneous IV every 6 m

3. Calcium and Vit D

4. Parathyroid hormone analogue

Teriparatide.
works by stimulating new bone formation
↑ bone density
SC IV 20 µg OD (2 y)

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Pharmacological

Antibacterial agents

1. Non - steroidal anti - inflammatory (NSAIDs)

   Ibuprofin
   Bromofenac

2. Antibiotics

   Eye drop
   Levofloxacin
   Moxifloxacin

   Ointment
   Erythromycin

3. Artificial tears (for dryness)

   Carboxymethyl cellulose

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Ophthalmology

Glaucoma

Glaucoma is a group of eye conditions that damage optic nerve.
Damage of optic nerve is often related to high pressure in your eye. But glaucoma can happen even with normal eye pressure.

⟶ Types

a. Open angle glaucoma
b. Closed angle glaucoma
c. Congenital glaucoma
d. Secondary glaucoma

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Treatment

Non - pharmacological.

1. Compresses
To relieve the discomfort try applying either a warm or cold compress I a moist wash cloth

2. Avoid contact lenses
removing contact lens and wear glasses

3. Rinse your eye
when you're exposed to allergens your body releases a chemical called histamine, causing redness, tears, etching in eye. So rinse your eye when required

4. Avoid triggers.
Avoid any triggers if possible.

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→ Symptoms

a. Open - angle glaucoma

- No symptoms in early stages
- gradually, patchy blind spots in our side vision (peripheral vision).
- later stage, difficulty seeing things in your central vision

b. Closed - angle glaucoma

- Severe headache
- Severe eye pain
- Nausea or vomiting
- Blurred vision
- Halos / colored ring around lights
- Eye redness

* Pathogenesis

Closed Angle

Anatomic abnormalities

↓

Crowding of ocular structures

↓

Resistance to the flow of aqueous humor (from posterior to anterior chamber)

↓

↑ pressure, lens bows forward

↓

↑ IOP. (Elevated intraocular pressure)

↓

Mechanical damage to nerve axons

Ischemia of nerve axon

Visual field defects

Loss of vision

Photophobia

Adverse effects

Nausea
Vomiting
Breast tenderness
water retention with oedema
weight gain
Increased chances of breast cancer
Increased incidence of liver disease

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Open Angle

Risk factors
- ↑ IOP
- ↑ age
- myopia
- family history

Genetic factors
↑ Susceptibility

Steroid Responsiveness
↑ IOP

Primary open-angle glucoma

↓

Ca^{++} ion influx , ↑ NO in retinal ganglion cells

↓

Retinal ganglion cell injury

↓

Optic nerve damage

↓

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Pathophysiology:

Patient presents with red eye

↓

Mild Pain or no pain, with mild blurring or normal vision

↓

excess of blood in the blood vessels (Hyperaemia)

↓

Continuous Discharge of fluids

↓

Mucopurulent to purulent

↓

Acute bacterial conjunctivitis

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Damage to optic nerve.

Ischemic damage due to blood vessels compression

Apoptoses of retinal ganglion cells.

Progressive visual defects

End stage visual loss

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Pharmacotherapy of Bacterial

Bacterial conjunctivitis :

It is a red eye with a stiky yellow or yellow / green discharge.

Eyelids may be stuck together upon waking. Can affect one or both eyes. usually spread by direct contact only.

Bacteria that can cause conjunctivitis a
- Streptococcus pneumoniae
- Haemophilus influenzae

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Treatment

β - adrenergic blockers : Timolol
Betaxolol

α - adrenergic agonists : Brimonidine

Prostaglandin analogues : Bimatoprost

Carbonic anhydrase : Acetazolamide
inhibitors

Miotics :- Pelocarpine.

(a.) β - adrenergic blocker :

Topical β - adrenergic blockers have been first line treatment drug.

No effects on pupil size, tone of ciliary muscle

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↓

histamine promotes vasodilatation and edema.

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(b.) α - adrenergic agonists

- α 1 constrict ciliary blood vessels — reduce aqueous secretion.
- α 2 in ciliary epithelium — reduce aqueous secretion
- Secondary role — enhancing drainage of aqueous.

(c.) Prostaglandin analogues

It decreases intraocular pressure Decreases ocular irritation and pain.

(d.) Carbonic anhydrase inhibitors

This inhibitors, limits generation of bicarbonate ions (HCO_3^-) from ciliary epithelial cells
Hence reduction of aqueous humour.

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Pathogenesis

Microbes enter the eye on contact with infected objects

↓

Inflammation of eye

↓

Dilatation of blood vessels of eye

↓

Swelling, redness, discharge

→ Allergic:

Allergen enter tear film

↓

Comes in contact with conjuctival mast cells that bear IgE antibodies

↓

Degranulation of mast cells releases histamine

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(e.) Miotics.

In 1970s - were standard antiglaucoma drug.

Last option bez of several drawbacks - myopia, headache, blurred vision.

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Treatment of closed angle glaucoma

a) Eye drops:
eye drops are used to lower eye pressure and can be effective in managing any glaucoma.

b) laser treatment: iridotomy
laser trabeculoplasty. can be used to treat closed angle

c) surgery
in addition to medication and laser, surgery can be successful in treating closed < glaucoma

- Glaucoma drainage device
- Cyclophotocoagulation.
- Trabeculectomy

Pharmacology

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* Classification

(a.) Infectious.

Viral

Bacterial :
hyperacute
acute
chronic

(b.) Non-Infectious

Allergic
Toxins
Chemical
Foreign body
Trauma
Neoplasm

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Conjunctivitis

Conjunctivitis is an inflammation or infection of the transparent membrane (conjunctiva) that lines the eyelid and covers the white part of the eyeball.

Also known as "PINK EYE"

When small blood vessels in the conjunctiva become inflamed, they're more visible. This is what causes the white of eye to appear reddish or pink.

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Treatment for open angle glaucoma.

a) Laser

laser therapy (LIGHT trial) is an appropriate and effective first-line treatment.

works by targeting tissue, that drain fluid inside the eye and improve the drainage outflow, thereby lowering eye pressure.

b) Eye drops

In situations where person does not want to go laser treatment, but rather prefers eye drops.

Most of eye drops lowers eye pressure by either slowing the formation of fluid inside the eye (called aqueous humor) or by promoting better drainage of the fluid.

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Diagnosis

Glaucoma is usually picked up during a routine eye test, often before it causes any symptoms.

Eye pressure test:

It is an eye pressure Test it uses an instrument called a tonometer to measure the pressure inside your eye.

Visual field test:

A visual field test checks for missing areas of vision.

Optic nerve assessment:

The optic nerve, which connects your eye to your brain, can become damaged in glaucoma.

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d) History collection

if patients family has any glaucoma conditions in past

e) Physical examination.

f) Fundus photography

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Management

* Open - angle glaucoma

People with newly diagnosed chronic open angle glaucoma (COAG)

↓

Offer 360° selective laser trabeculop (SLT)

↓

→ Offer a generic prostaglandin analog (PGA)

↓

→ Offer a generic PGA

↓

→ 360° SLT

↓

Continue to monitor intraocular pressure (IOP)

Neuromuscular system
- slow speech
poor memory
muscle pain
depression
weakness

Gastrointestinal
- weight loss
decreased appetite
constipation
ascites

Cardiovascular
- reduced cardiac output
bradycardia
cardiac enlargement

Treatment:
Non-pharmacological
Avoid eating fatty food.

Iodine containing diet is recommended.

Controlled diet with enough intake of sufficient calories.

Exercise and other activities helps reduced cardiovascular symptoms

Keep your body hydrated, drink enough water

Keep your skin & scalp moist and apply

Pharmacological

Treatment for hypothyroidism includes thyroid hormone medicine

- Levothyroxine (Levo-T)

This medication is taken orally.

It helps returns hormone level to a healthy range.

Complimentary prescribed are:-
↳ Iron supplements
multivitamins containing iron
Aluminium hydroxide, found in antacids
Calcium supplements

Pharmacokinetics.

Absorption - oral
Metabolized - not metabolized
Excretion - Kidney (unchanged)
Distribution - negligibly bound to plasma protein.
half life - 6.2 h (plasma)
17.6 h (blood)

Adverse effects

Side effects are frequent, but generally not serious

Abd pain
Anorexia
Bloating
Nausea
Mild diarrhoea
Tiredness
Metallic taste

Major side effects

Lactic acidosis
Renal failure
Vit B_{12} deficiency

2. Respiratory System

[A.] Pulmonary Function Test

Pulmonary function test are used to aid diagnosis, quantify functional impairment and monitor treatment or progression of disease.

Airway narrowing, lung volume and gas exchange capacity are quantified and compared

They are:

* Spirometry – it is one of the most commonly ordered tests of lung function. It measures the lung volume exchange during forced breathing. It detects the obstructive lesions in major airways.

* Diffusion capacity – it estimates the transfer of oxygen in alveolar air to the RBCs. factors that depends are – area of alveolar membrane, thickness of the membrane, Driving pressure, Hb.