ARISE MEDICAL ACADEMY

BUILDING BETTER DOCTORS

ENDOCRINOLOGY

WORKBOOK

By

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RISE WITH ARISE

ARISE MEDICAL ACADEMY

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Chapter 1
Pathogenesis of Diabetes mellitus

→ 1
1.5
2
MODY
BRONZE

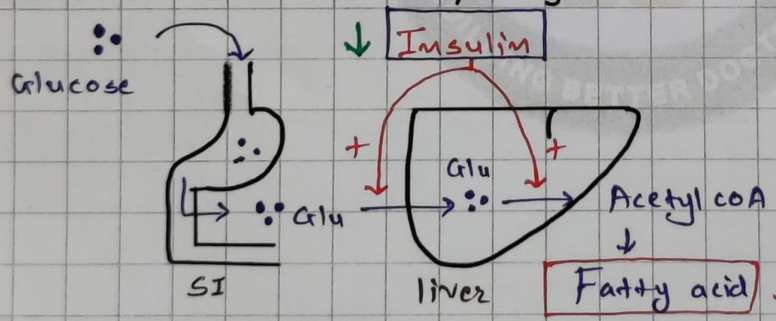
PATHOGENESIS OF TYPE 1 DM

→ < 25 yr age
→ Autoimmune DM
(Antibody mediated distruction of β cell of Pancreas)

→ Trigger
- Cox - Sackie inf.
- Rubella
- Viral infection

→ HLA DR 3 / DR 4 ⊕
↓
Starts wrong antibody synthesis
[anti - islet cell - Ab
anti - GAD - 65 Ab]
↓
distruction of β-cell [≈ 80%]
↓
↓ Insulin → ↑ Blood sugar → DM

Relation of Insulin with body weight



Occur at Age- < 25 yr
B- cell mass at the time of diagnosis- 80-100%. β-cell are distructed
Concordance in identical twins - 30-70%.
body wt → wt. loss

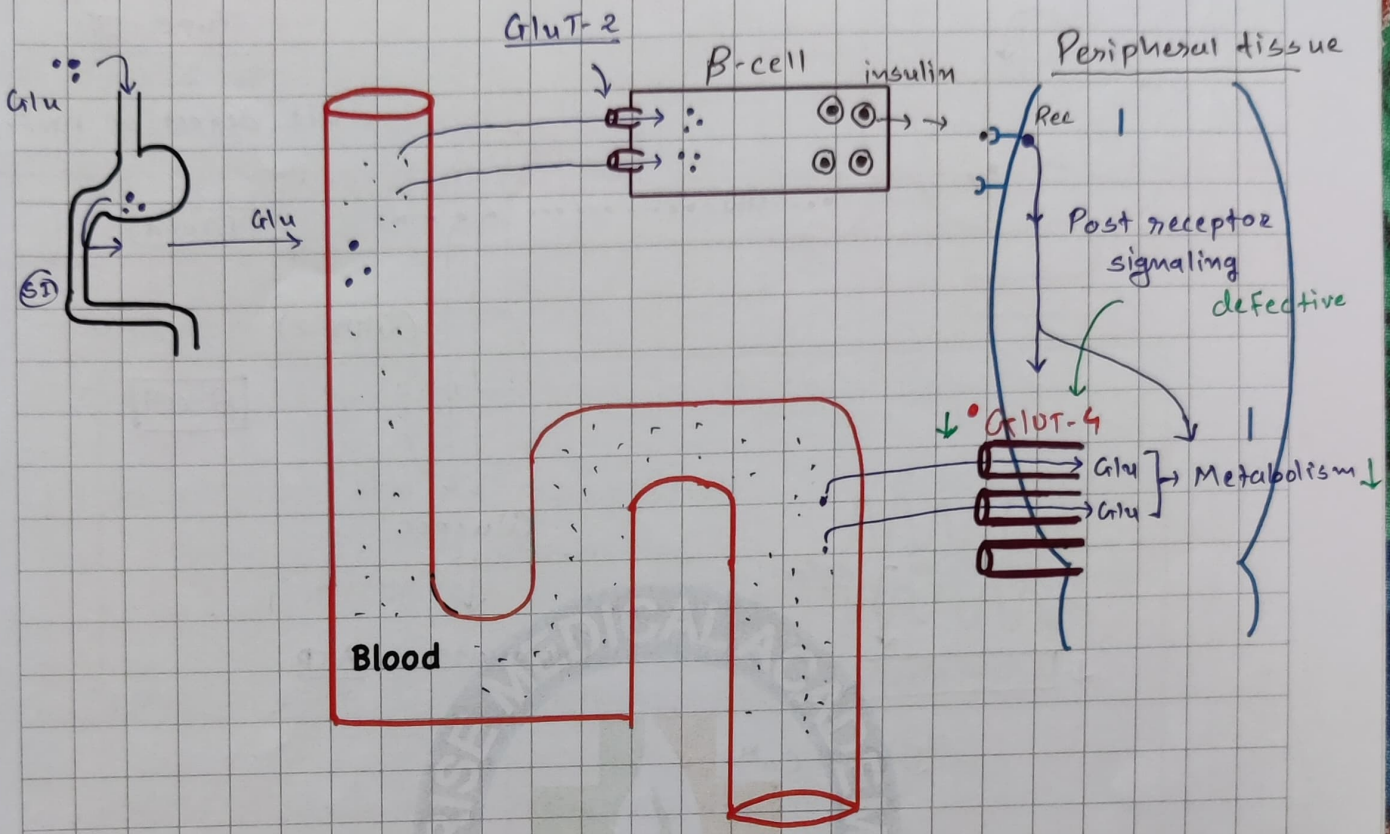
PATHOGENESIS OF TYPE 1.5 DM

Hidden LADA -LATENT AUTOIMMUNE DIABETES OF ADULTS > 25 yr age
↓
Less severe form of Type 1.

Post receptor signal → open GLUT-4 & activate Metabolism of Glu
→ so amount of Glu in Blood start ↓

PATHOGENESIS OF TYPE 2 DM

→ Defect in Post-receptor signal
MC → > 45 yr age



Type your te

Occur at Age- >45 yr
B- cell mass at the time of diagnosis- 50 % of B cell are exhausted
Concordance in identical twins - 70-90%.
Body wt - wt gain

PATHOGENESIS OF Maturity onset of diabetes (MODY)

↓

Defect of HNF-1α (Endoplasmic Reticulum)

↓

↓↓ Conv from ER

↓

↓ insulin release → ↑ Blood sugar → DM

Types of MODY :- → 6 type

↓

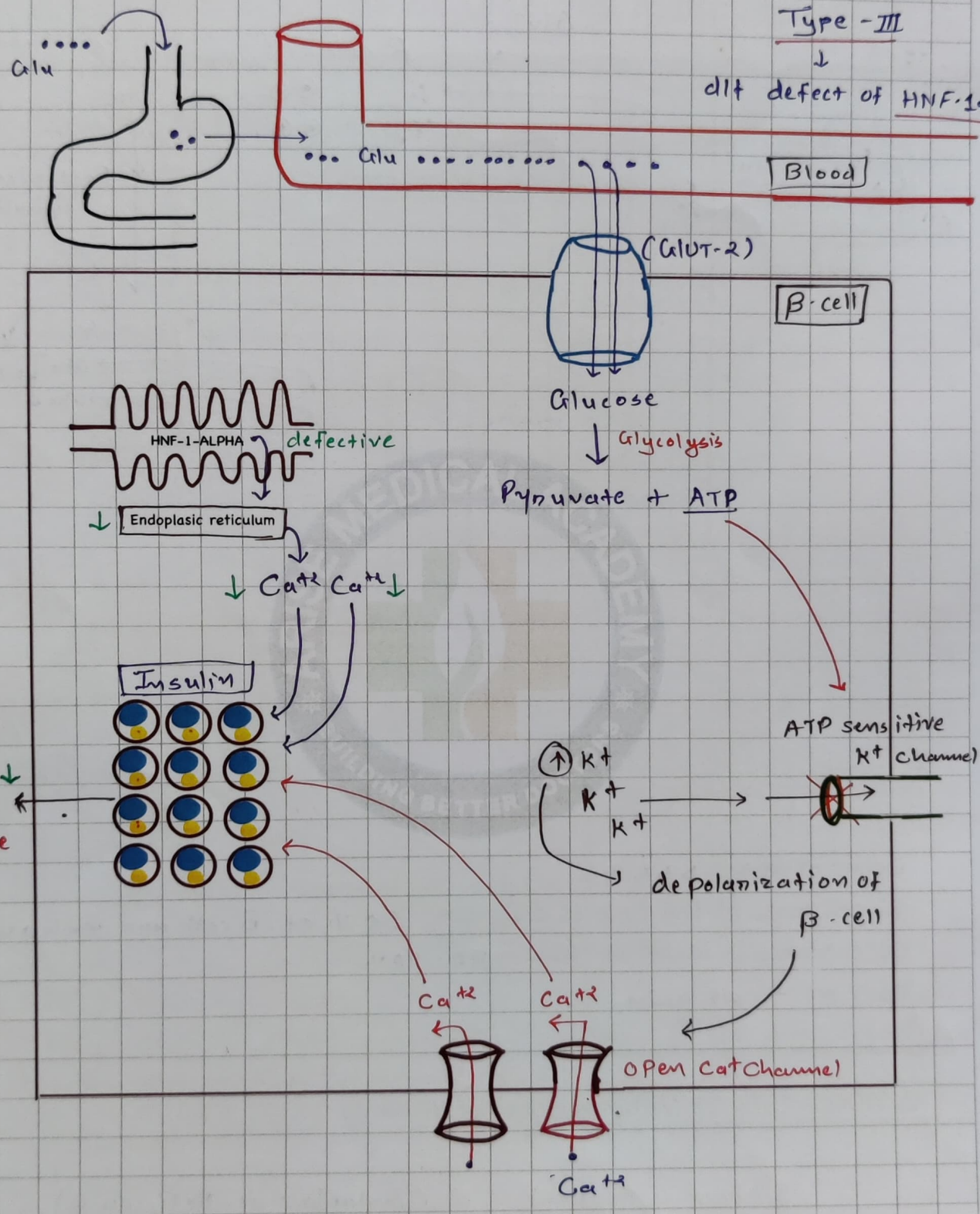
MC type of MODY

↓

Type - III

↓

dlt defect of HNF-1α



Inheritance pattern - Autosomal dominant

So → Can involve 3 Continous generation

MCQ [Grand Father → Father → Son]

Types. Of MODY → 6 Type → Mc Type. III

Age of onset - > 25 yr

B-cell mass- Normal

Rx- K^+ channel Blocker → Sulfonylurea → eg Glimpride

BRONZE DIABETES

Caused by - Iron deposition in β. cell (Pancreas)

↓

distruction of β-cell

↓

↓ Insulin

↓

DM

Iron deposition - skin → Pigmentation
- liver → Cirrhosis
- pancreas → DM

So manifest as → DM + Cirrhosis (Ch. liver ds)

Summary

Type Of DM	Pathogenesis
1	Rapid autoimmune distruction of β-cell
1.5	Slow " " " "
2	Post receptor signal defect
Mody	Mc Type - III → dft defect of HNF - 1α
Bronze	- dft Iron deposition

- Overall M/C type of DM - Type. II
- Overall most rare type of DM- MoDy

Also know

Gene associated with insulin production - CTL4A

chromosome for MODY type 3- chromosome 12

Chapter 2
Diabetes Presentation , diagnosis and Complications

C/f → ↑ apetite → Polyphagia
↑ water intake → Polydipsea
↑ Urination → Poly urea

Diagnosis

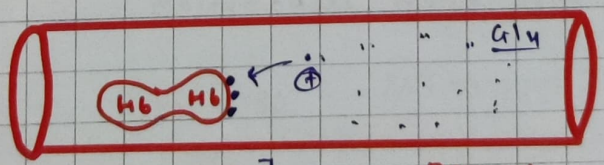
Pre-diabetes

	Normal	Impaired glucose tolerance	Diabete
Fasting	Upto 99	100 - 125	>126 mg/dl
Post prandial (oral glu. Tolerance test)	Upto 139	140 - 199	≥ 200
HbA1C	Upto 5.6%	5.7 - 6.4 %	≥ 6.5%
Random			≥ 200 mg/dl + C/F of DM

Post prandial means - oral glucose (75 gm) → check blood sugar after (2) hr

Best test for DM → HBA1C

HbA1C

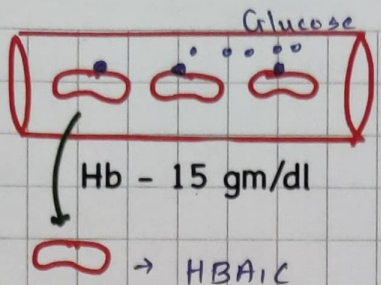


[RBC (Hb) + Glucose] → Glycated Hb [HBA1C]

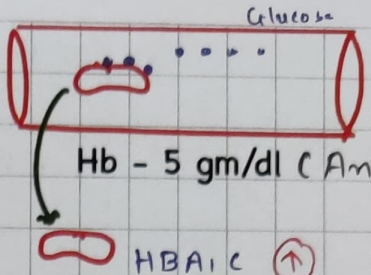
Blood.

★ life span of RBC → 90 - 120 days
• So RBC HBA1C Reflects → Blood sugar level of 90 - 120 days

Sugar level
95 mg/dl

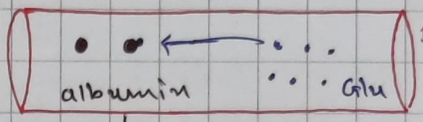


Sugar level
95 mg/dl



False High HBA1C

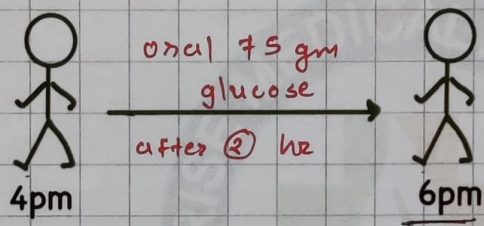
Glycated albumin (s. Fructosamine) -



Shows average blood sugar of ↴

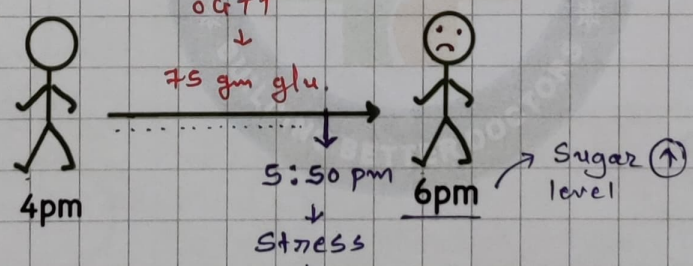
last 2 w - 3 wks.

Oral glucose tolerance test (OGTT)



Extra point

Healthy

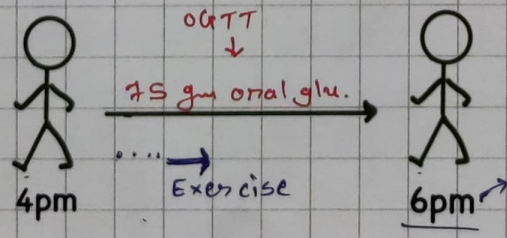


So

False ⊕ OGTT

- Stress
- Infection
- Malnourished

DM



So

False ⊕ ve OGTT

- Exercise in between test

→ d/t gluco neo genesis *

Q. **Mechanism for Fasting hyperglycemia**

healthy — Meal 8 Pm → Fasting sugar at 8am → Blood Sugar Normal

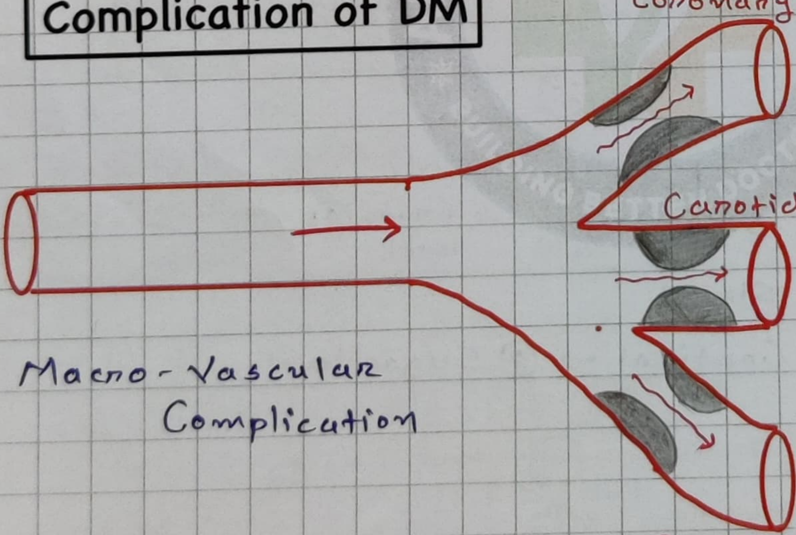
DM — 7pm → Fasting sugar at 8am → ↑↑

Q. **Mechanism for Post prandial hyperglycemia.**

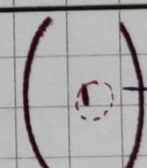
healthy — Meal — after 2 hr → Bld. sugar - (N)

DM — Meal → Bld. sugar - (↑)
↓
d/t (↓) Glucose utilization *

Complication of DM

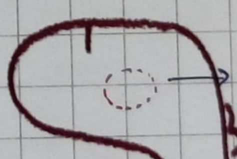


Macro-Vascular Complication



→ IHD / CAD

Myocardium

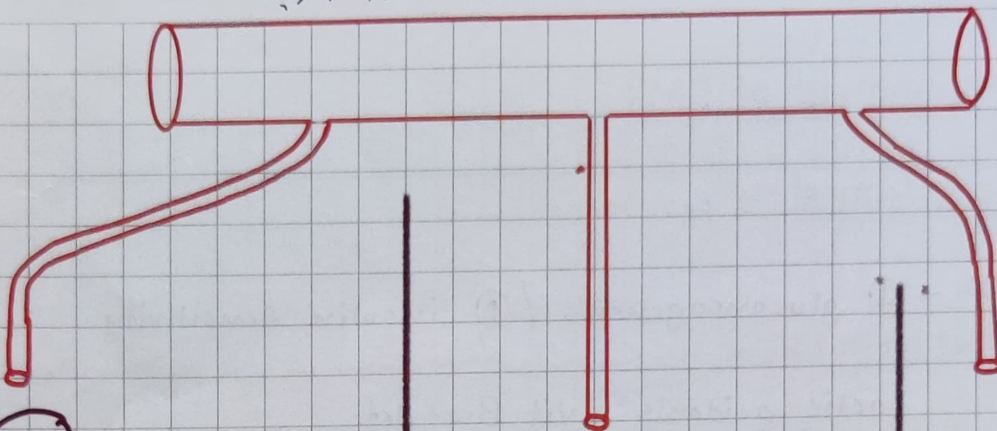


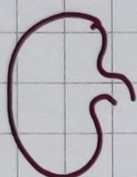
→ ischemic stroke (CVA)

Brain

→ Gangren
↓
(Texas Classification)

→ Micronvascular Complication





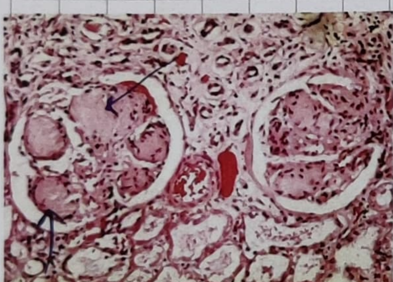
DM-Nephropathy

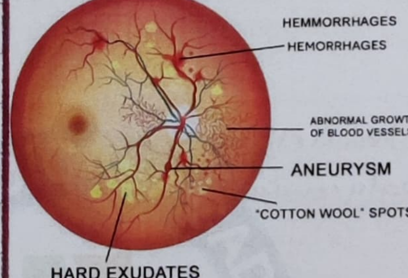
DM Retinopathy

DM-Neuropathy

DM-Neuropathy

Rx
- Gabapentin
- Pregabalin
- Fluoxetin





↳ loss of Sensation (or) tingling (Burning) sensation (Palm & sole)

Earliest change
- ↑ GFR
- Microalbuminuria

Most specific change
↓
Nodular glomerulosclerosis
or
Kimmelstain-wilson bodies

Earliest change
↳ Microaneurysm

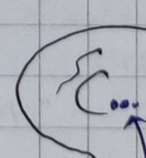
Most specific change
↳ Neovascularization

So retinal examination in type 1 DM after (5) yr and type 2 DM immediatly

(4) Non-vascular Complication of DM

↑ Snow Flake cataract (d/t deposition of Sorbital)

↑ Mucormycosis



Malignant otitis Externa
↓
d/t Pseudomonas

Managment of DM

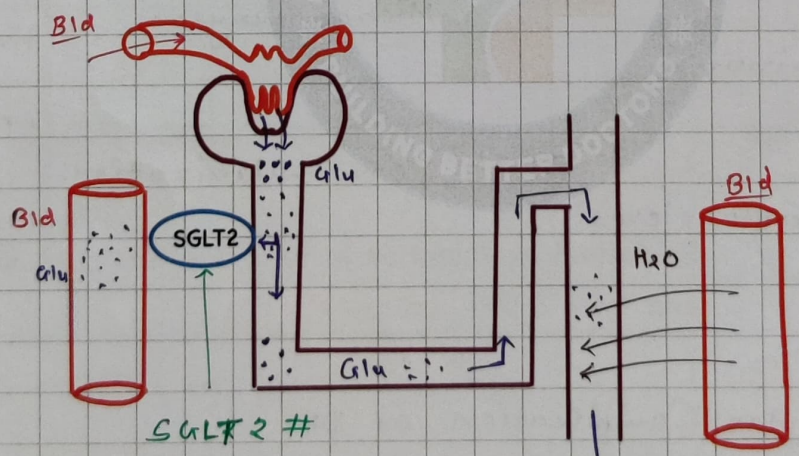
1- BIGUANIDES → Eg. Metformin

MOA → ↓ gluconeogenesis / ↑ insulin sensitivity
Effect on Hb1aC → Reduction → Maximum is with Metformin
S/E - Lactic acidosis , Vit B12 def.
C/I - if GFR < 30 ml/min

2 - sulfonylurea - 1st generation - Tolbutamide
2nd generation - Glimepride / glyburide

MOA → K^+ channel blocker → ↑ insulin Release
S/E → Hypoglycemia (-ide)
MOST POTENT - Glyburide

3- SGLT -2 inhibitors



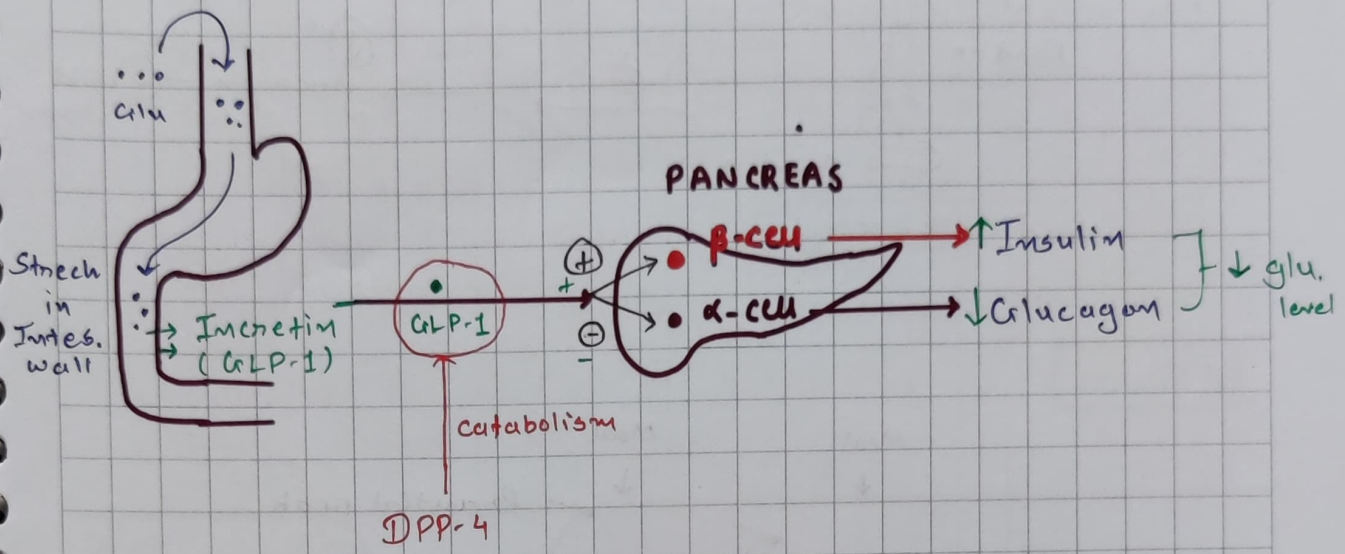
Eg- Gliflozin
S/E - UTI
Preferred in - DM + Heart Failure

loss of Glu. + H_2O in urin

4 thiazolidinediones → Eg. Pioglitazone

MOA → ↑ activity of PPAR → ↑ insulin sensitivity
S/E → ↑ risk of - CAD
- Bladder cancer

5. Incretins



GLP(glucagon like peptide -1) analogue - Exenatide, Linaglutide

MOA - ↑ β-cell activity (Pancreas) → ↑ Insulin

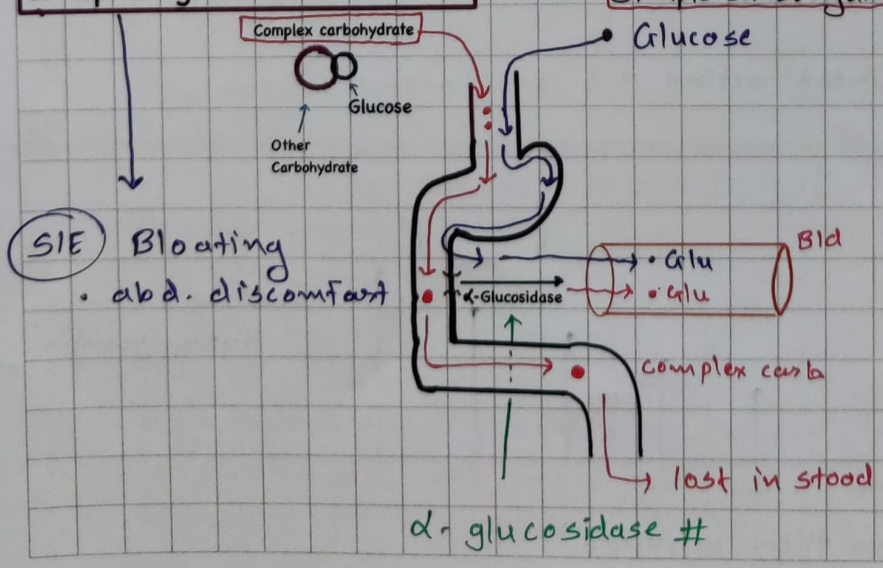
S/E → Pancreatitis, ↑ risk of Medullary cancer, Hypoglycemia

DPP-4 INHIBITOR- Teneli-gliptin

Most renal safe DPP-4 #- Linagliptin

6. Alpha- glucosidase inhibitor

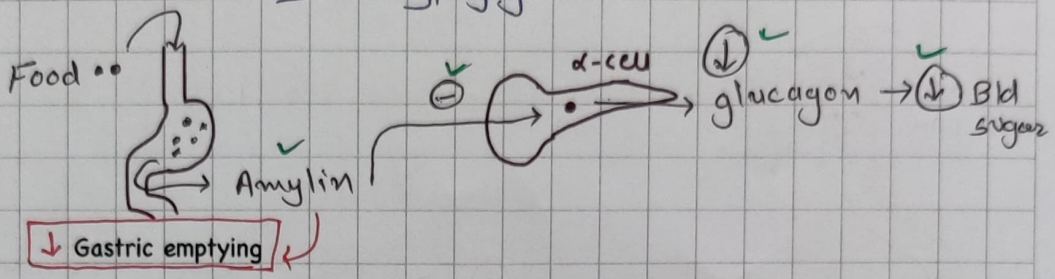
→ # absorption of complex carb :- Voglibose, Acarbose



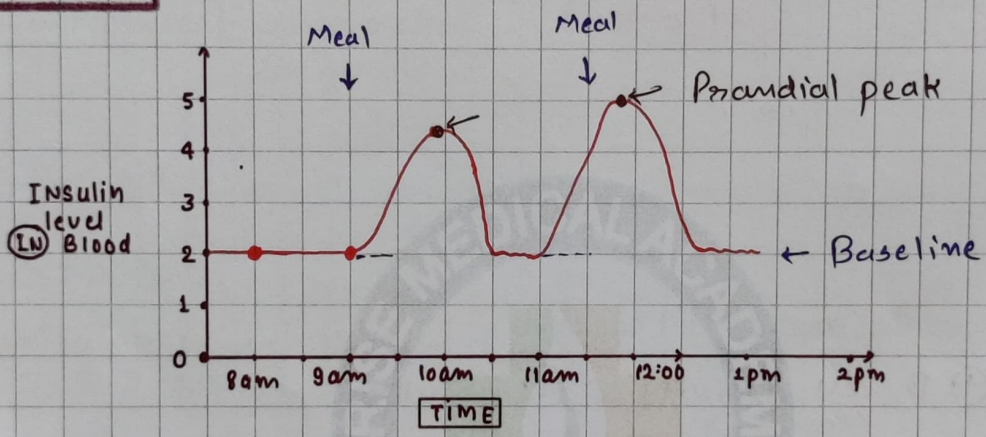
α-glucosidase #

7. Amylin analogue

→ Eg : Pramlintide
→ S/E : Hypoglycemia



8.insulin

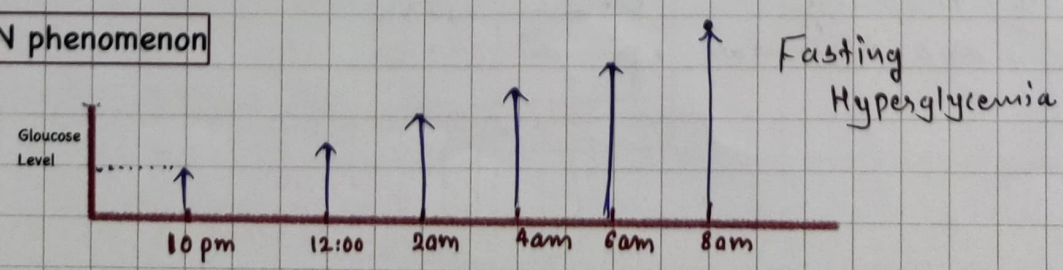


Basal insulin → Long acting
- → NPH → t½ = 10-12 hr
- → glargine → t½ ≈ 24 hr
- → degludec → t½ ≈ 48 hr

Prandial insulin → Short acting → Lispro, aspart, Regular

Complication of insulin therapy

DAWN phenomenon

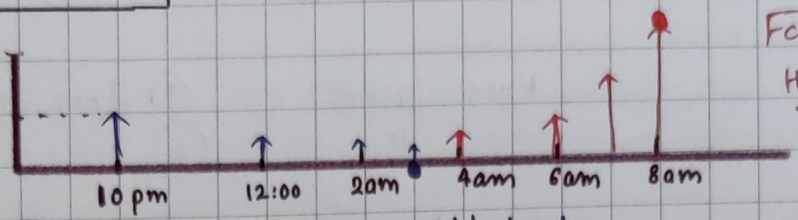


(less insulin then required)

Rx - ↑ dose of night insulin

Somogyi phenomenon

Ix : Mid night sugar level



[taking more insulin then Required]

Midnight Hypoglycemia

↓

Trigger cortisol release ↑

Rx - decrease the dose of night insulin

So in

Type 1 DM → Exogenous Insulin

Type 1.5 DM → Exogenous Insulin

Type 2 - HBA1c level
- 6.5 - 9 % → stand Metformin
- 9 - 10 % → Two drugs
- > 10 % → Two drugs + insulin

MODY → Sulfonyurea (kt channel Blocker)

Summary

*Hb1ac shows average of - 3-4 ~~weeks~~ Months

2. Anemia leads to fasle High HB1ac

3. FALSE + OGTT -- Stress

4. FALSE - ve OGTT - Exercise

5. Fasting hyperglycemia in DM is due to gluconeogenesis

6. anti - diabetic to avoid

If -- DM + HF → Thiazolidine diones

-- DM + medullary ca of thyroid → GLP-1 analoges

-- DM + uti → SGLT2 #

-- DM + MI → Thiazolidinediones

7. Maximum Hb1AC reduction - Metformin
8. Longest acting insulin - degludec
9. Dawn phenomenon - fasting Hyperglycemia. Rx- ↑ dose of night insulin
10. Somogyi phenomenon - fasting .....!!..... Rx- ↓ " " " "

m. Dep

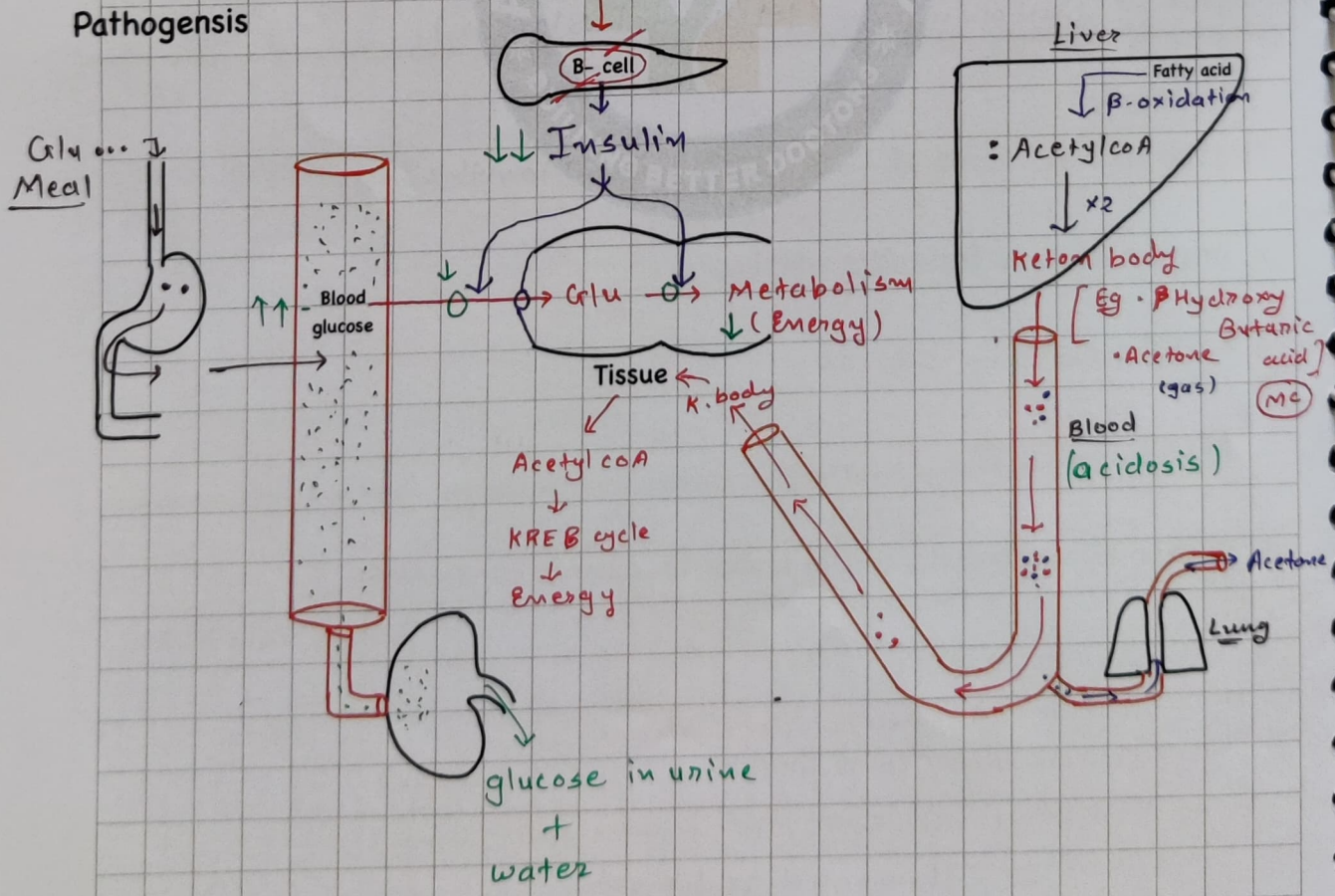
Chapter 3
Diabetic keto acidosis (DKA) & Hyperglycemic Hyperosmolar State (HHS)

DIABETIC KETO ACIDOSIS

Type I DM

Rx : Exo. Insulin (defaulter)

Pathogensis



C/f- Nausea, Vomiting, abd. pain

- dehydration
- Fruity smell of breath d/t acetone

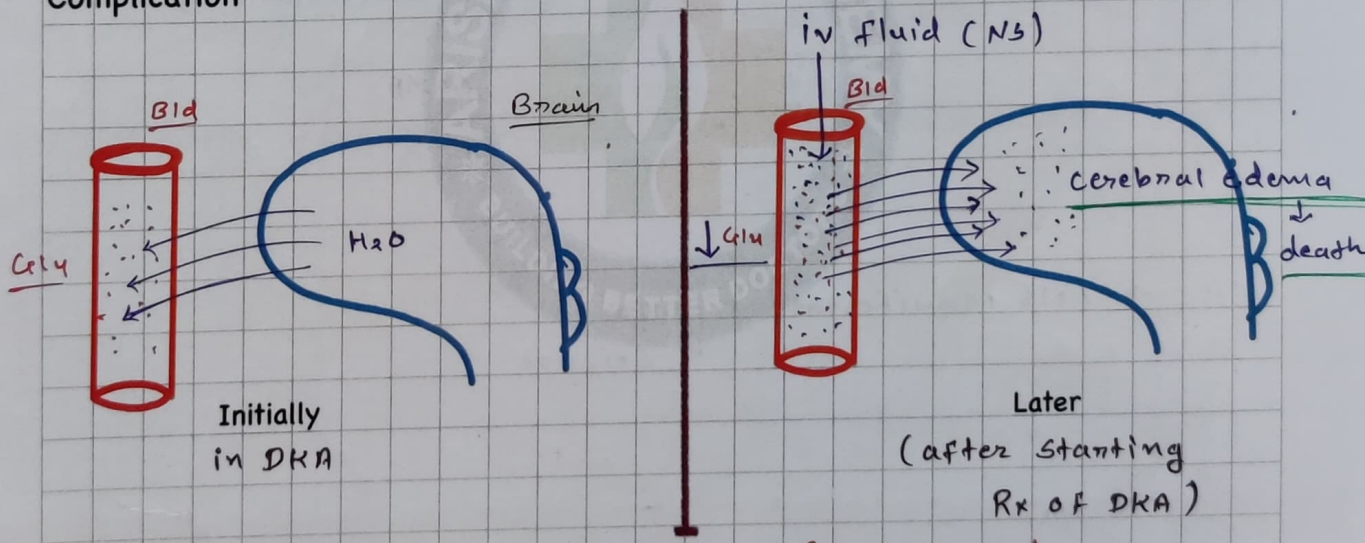
Investigation - blood glucose level- ↑↑↑
- urine keton +++
- ABG - pH- Met. acidosis → Kussmaul breathing
- S. Na+ - ↓↓

Rx- 1st - iv fluid (NS 0.9%)

- inj. Insulin (Regular)

- K+ Supplement to prevent Hypokalemia

Complication



(A) Mcc of death in pt. of DKA = Cerebral edema

Hyperglycemic hyperosmolor state (HHS) / HHC

Coma

Commonly seen with - Type (II) DM

Age - old age

Pathogenesis

- old age lady
- Type II DM

↓

Rx defaulter

↓

↓ thirst

↓

↓ water intake

Infection

↓

Stress

↓

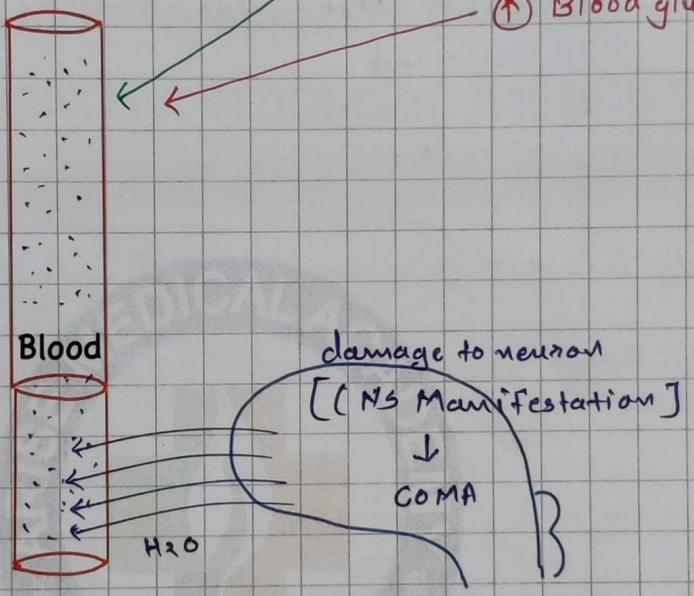
↑ Cortisol

↓

↑ Blood glucose

↑Glu

↑↑↑↑ Blood osmolarity



C/f → CNS Manifestation → Coma
→ Seizure

Treatment → IV Fluid (slowley)
→ Inj. Insulin
→ K+ Supplement
→ Bad prognosis

Summary

1. DKA commonly seen with type I DM and trigger is Rx defaulter / MI, Infection,

2. M/C keton body synthesized in liver is - β Hydroxy butyric acid

3. Keton body having fruity smell- Acetone

4. Keton body can be detected by - Rothera test / Nitroprussside test

5. Main stay of rx in DKA- IV Fluid

6. Regular insulin used in DKA managment .

7. MCC of death in DKA is Cerebral edema

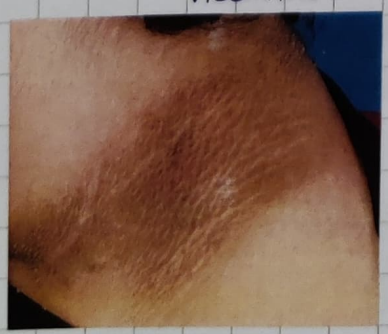
8. HHS seen with type II DM in old age group .

9. C/f of HHS - commonly - CNS t manifestation .

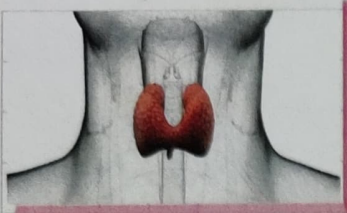
10 . prognosis of HHS - Bad

DM

Acanthosis Nigrican

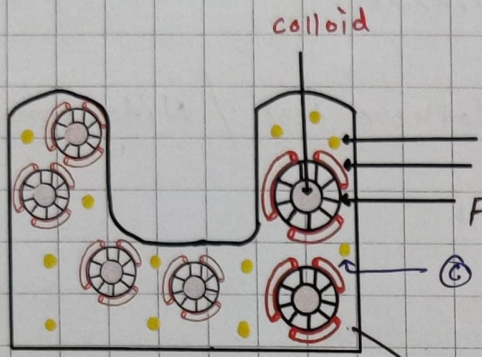


Necrobiosis lipidoica



Chapter 4
Thyroid Physiology

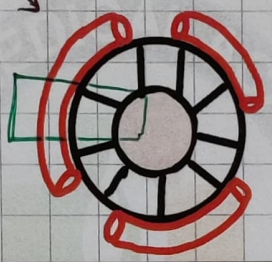
Thyroid gland



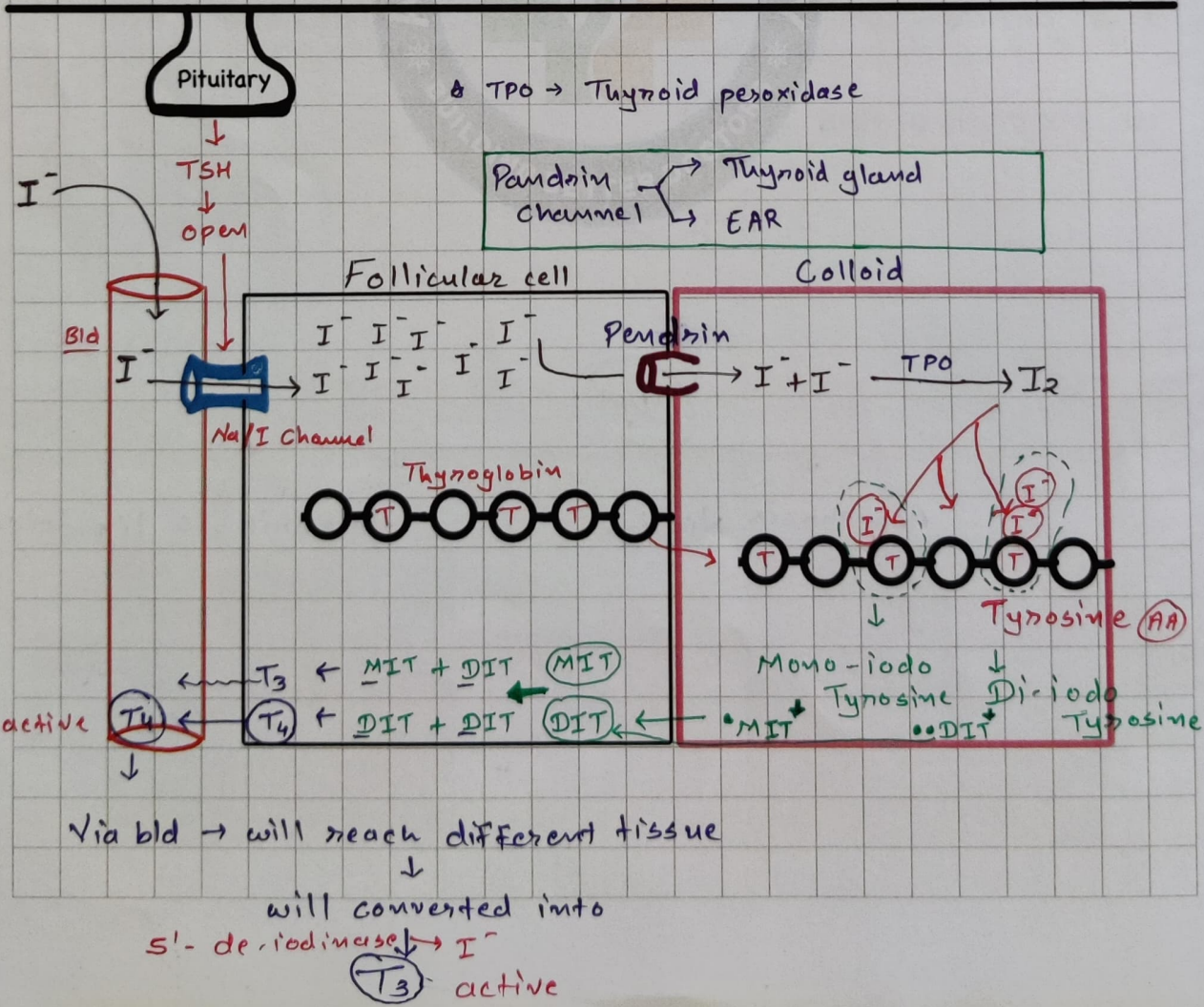
Cancer of parafollicular c cell is called ↓

Medullary Ca of thyroid

(C) cell → Calcitonin



← Thyroid Follicles



* TPO → Thyroid peroxidase

Pendrin Channel → Thyroid gland, EAR

Via bld → will reach different tissue

↓

will converted into

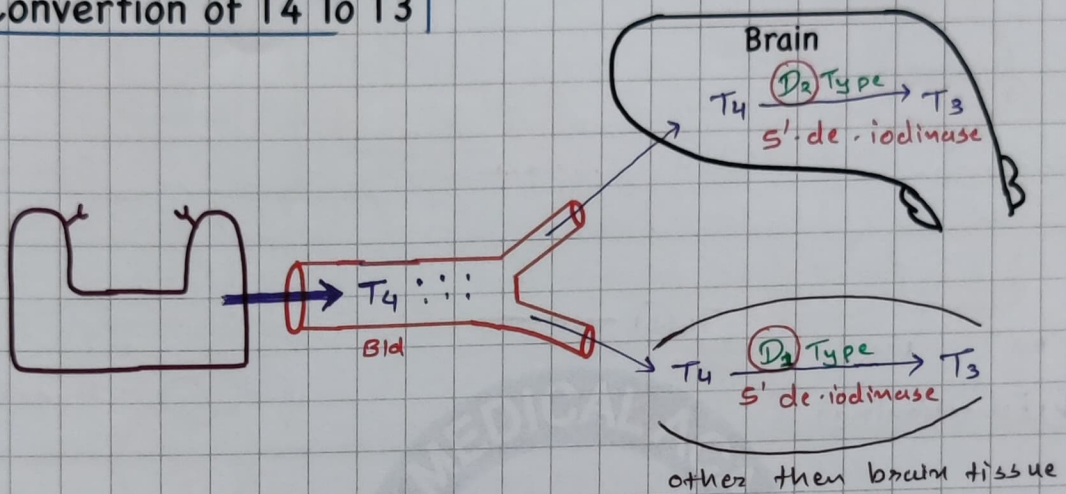
5'- de-iodinase → I⁻

T_3 active

T4 v/s T3

	T4	T3
Production	More	Less
Activity	Less	More
Half life	7 days	1½ day

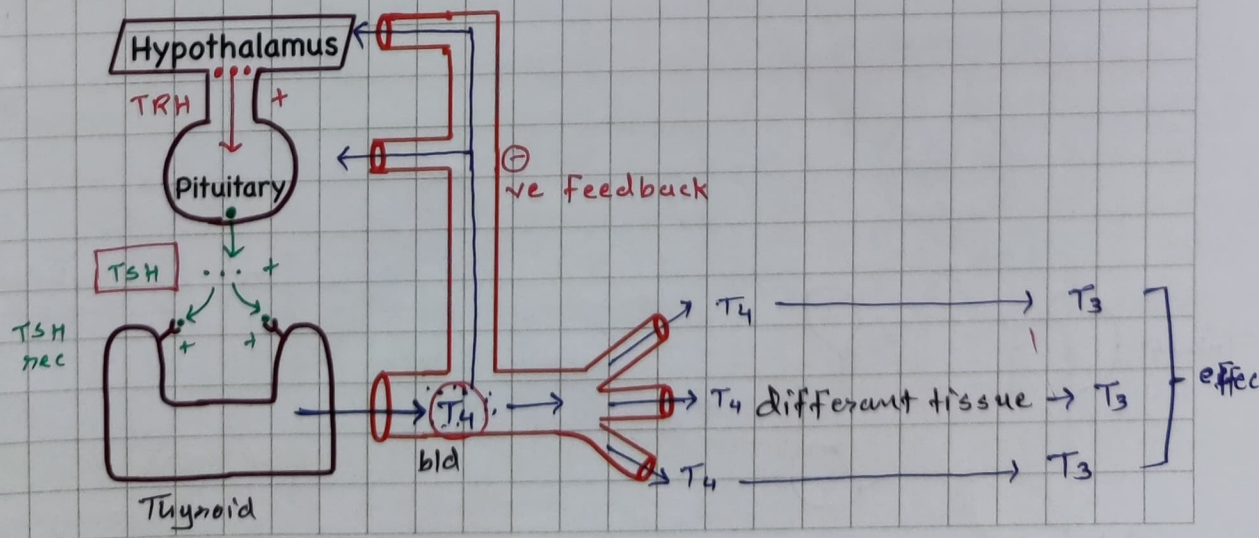
Convertion of T4 To T3



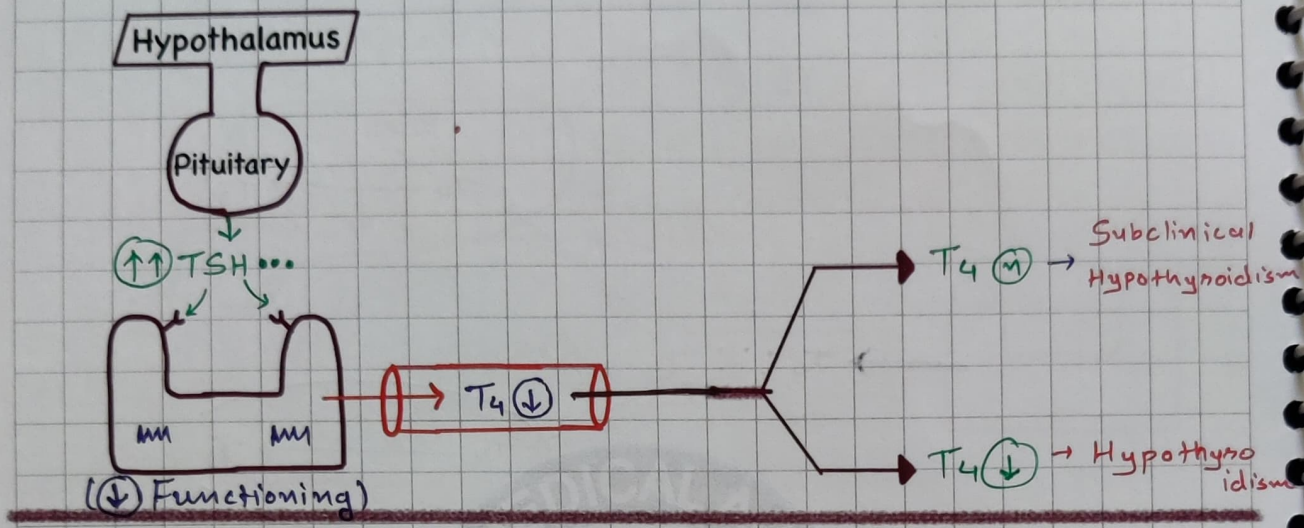
Effect of thyroxine

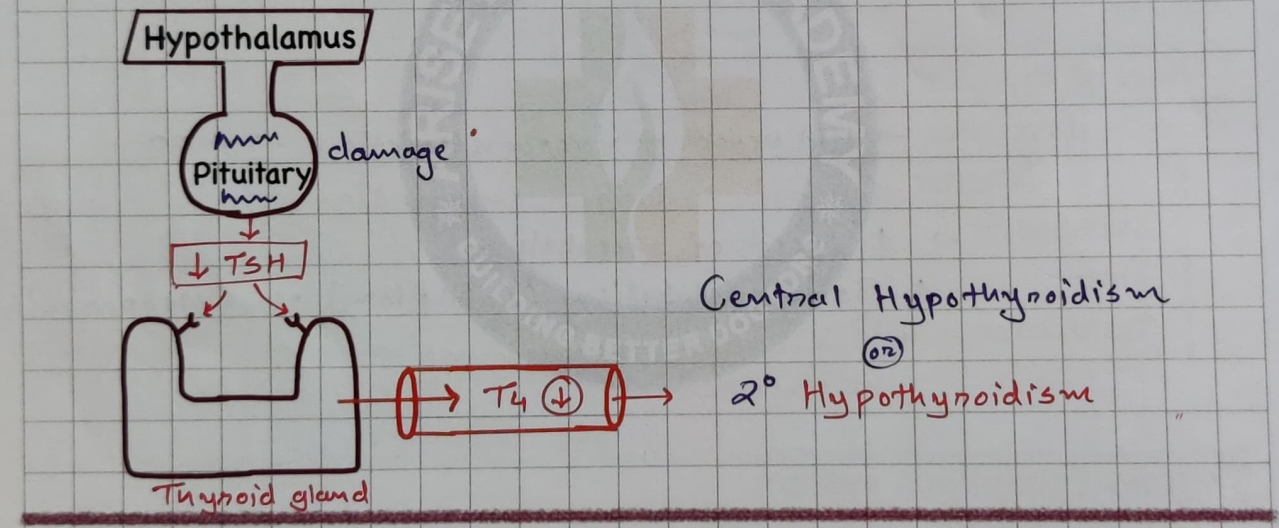
- On α recpetor → Bld vessel → Vasoconstriction → ↑ BP
- On β- receptor → Heart → ↑ HR → Palpitation
- Metabolism → ↑ speed of metabolism
- Bone → ↑ Bone metabolism (Catabolism) → Osteoporosis
- Testosteron → ↑ Catabolism of testesterone → Gyneecomastia

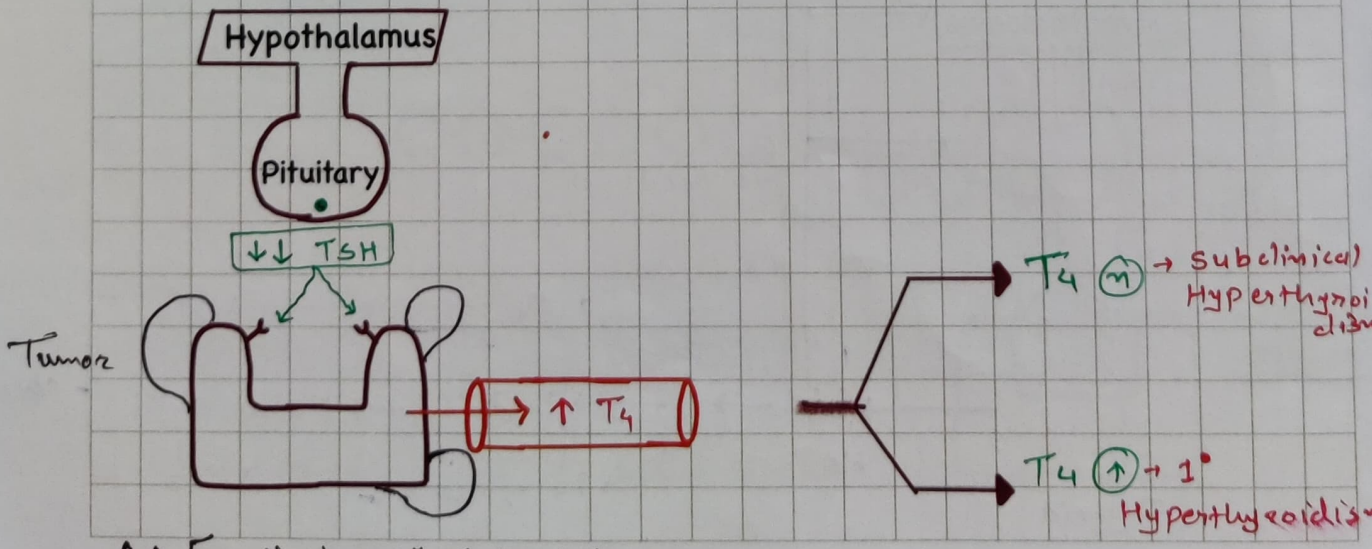
Thyroid FeedbBack system



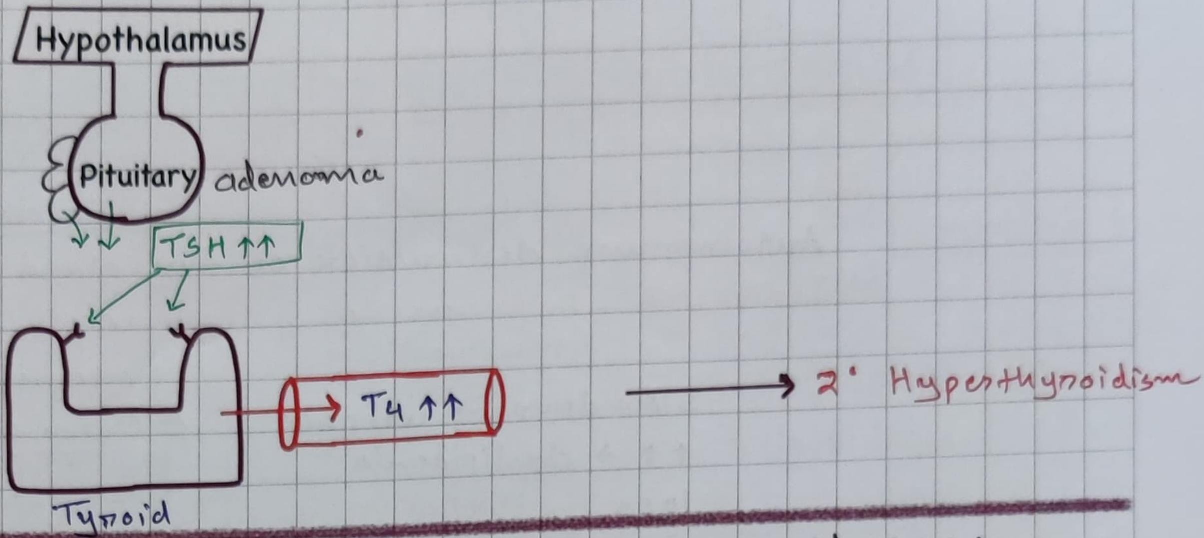
Chapter 5
Interpretation of thyroid profile test



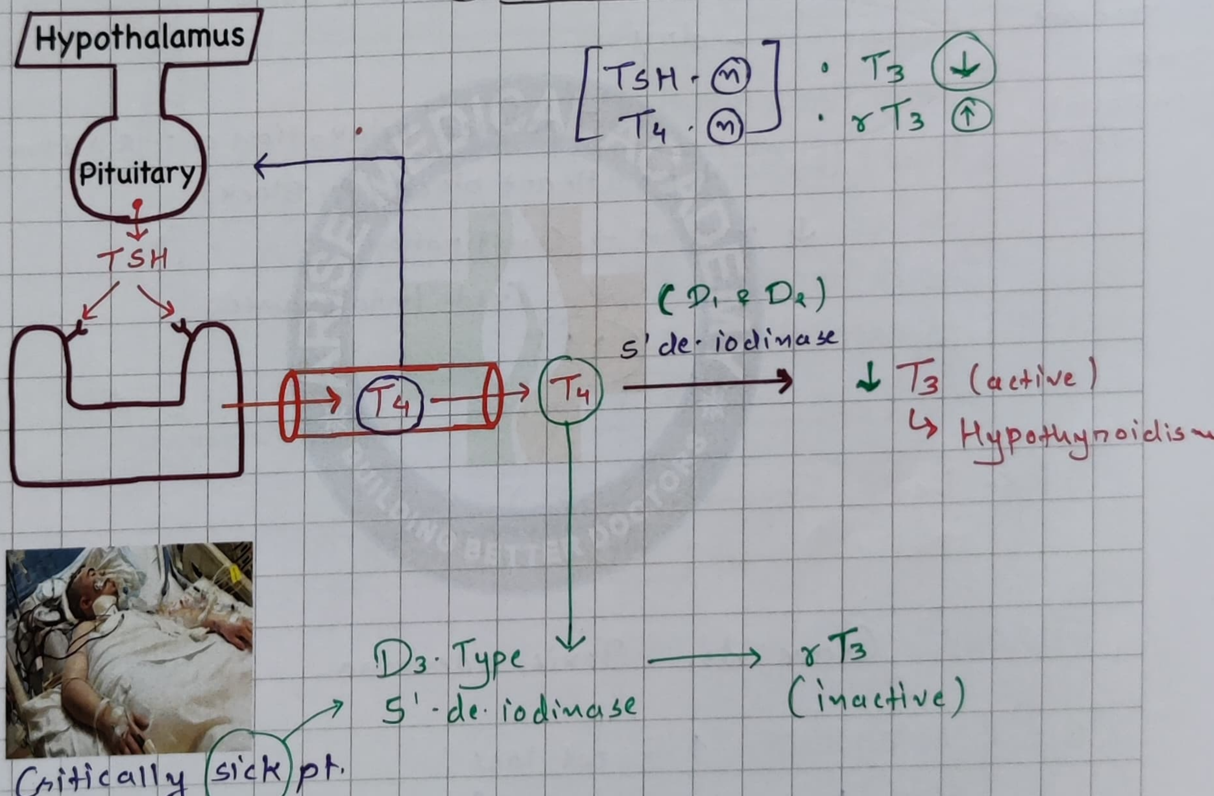




[↑ Functioning of T.gland]



⊛ - sick euthyroid syn :-



Lets practice

T4	TSH	Diagnosis
↓	↑	1° Hypothyroidism
Ⓝ	↑	subclinical Hypo.
↑	↓	Primary hyperthyroidism
↓	↓	★ 2° Hypo.
↑	↑	2° Hyper.
Ⓝ	↓	subclinical Hyper.
Ⓝ	Ⓝ	Sick euthyroid syn

Chapter 6
Hypothyroidism & Hyperthyroidism

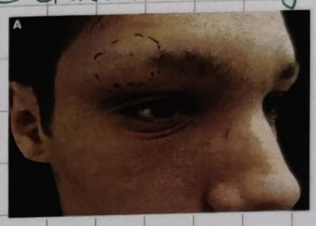
Hypothyroidism

Etiology – (MC) Autoimmune destruction of thy. gland → Hashimoto ds
↳ Iodine def., Selenium def., Sarcoidosis (Himalyas)

C/f.
- Metabolism – slow down → wt. gain
- Lipid level – ↑↑ → dyslipidemia
- Hair – loss
- Eyebrow – loss outer 1/3 of Eye brow
- Skin – dry
- Deep tendon reflex – delayed relaxation of DTR → Hung UP reflex
- Heart (ECG) – ↓ HR, Heart block (AV Block)
- GIT – ↓ motion → Constipation
- body temprature – ↓ – Cold intolerance
- Menstrual bleed – (↑↑)

every thing is slow down except Menstrual bleed.

Hertoghes sign
Queen annie sign



Rx – Levo. Tuynoxine (T4)

Hyperthyroidism

Etiology – Crave ds., Toxic adenoma

C/f.
- Metabolism – ↑ – wt. loss
- Skin – ↑ sweat – Moist skien
- Deep tendon reflex – ↑ Brisk
- Heart – HR ..↑../ SBP ..↑../ ..D..CMP/ Means lerman murmur
- Hands – Moist (↑ sweat), Fine tremor
- GIT – ↑ motility → (↑) stool Frequency
- body temprature – ↑ – Heat intolerance
- Menstrual bleed – (↓↓), oligomenorrhae

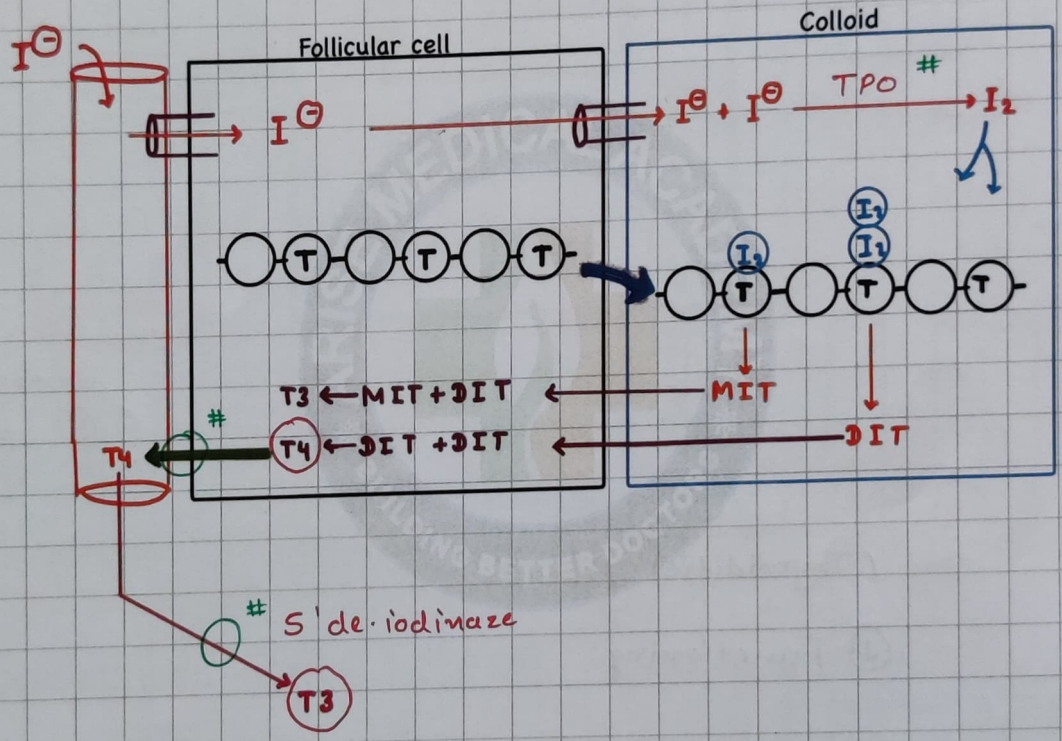
every thing going to speed up except Menstruation

Upper eye lid sign of hyperthyroidism

Sustained, ↑ contraction of upper eyelid (Abadie sign)
↓
Upper rim of sclera is visible (Dalrymple sign)

→ on downward gaze → lag in upper eyelid movement (Von graFe sign)

Managment of hyperthyroidism



So

1. Thyroid peroxidase inhibitor - Thiomides → Carbimazole, Methimazole, Propylthiouracil (PTU)
2. Harmone release inhibitor - Iodine → Lugol iodine, KI
3. Peripheral convertion inhibitor → β-# / steroid / thiomide

---

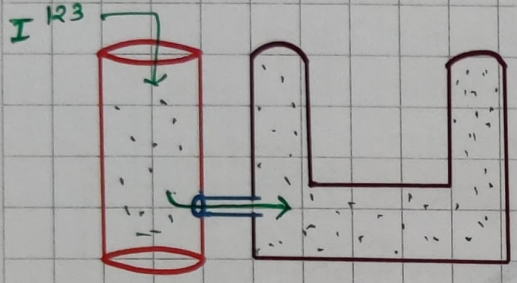
★ Safest in pregnancy for Hyperthyroidism → PTU

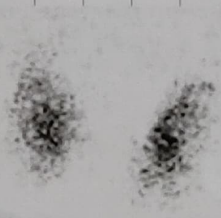
Chapter 7
RAIU & Thyroid disorders

Radioactive iodine uptake test (RAIU) → Anatomical Functioning of thyroid gland

For RAIU - I^{123} ..... is used.

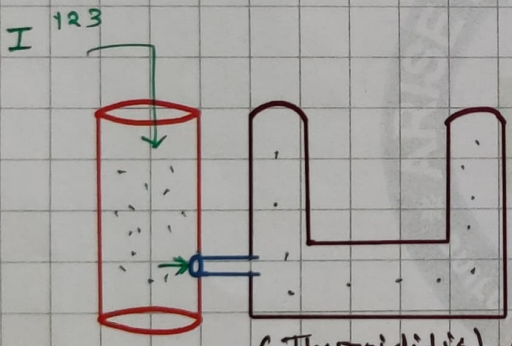
Mechanism of RAIU -





RAIU

Normal



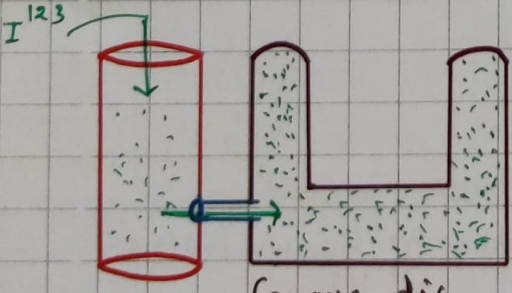
(Thyroiditis)

↓

↓ Functioning

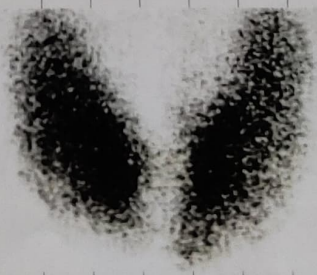
RAIU

Homogenously ↓ uptake

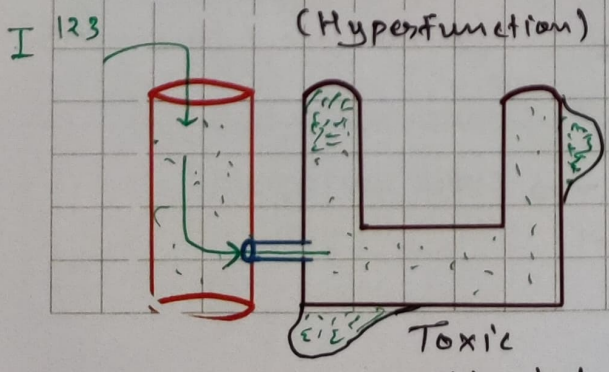


Grave dis.

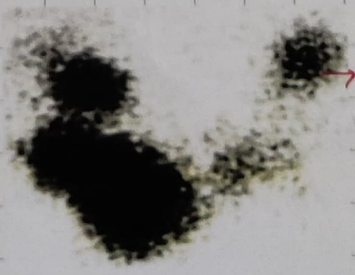
(Hyperfunction)



Homogenously ↑ uptake



Toxic multinodular goiter

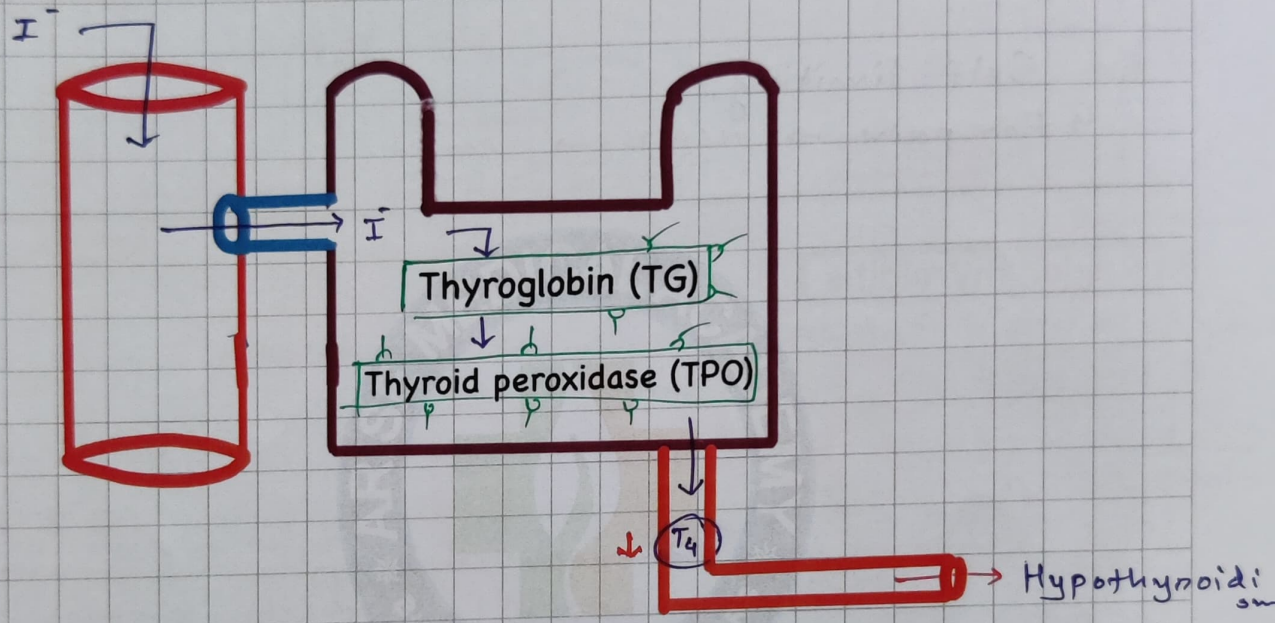


→ Hot Nodule

Thyroid Pathology

1. Hashimoto thyroiditis

Pathogenesis - Autoimmune → Anti-TPO Ab (or) Anti Tg Ab



Associated with - HLA DR ③ / ⑤ → ass. ≥ DM, M.gravis, addison ds.

C/f - initially - Hyperthyroidism
- later Stage (main) - Hypothyroid

Increased risk of - ↑ Risk of Marginal B cell lymphoma

Biopsy - Hurthel cell

Rx- Levo thyroxine × life long

Painful thyroid gland only seen in this

↑

2. Subacute thyroiditis or dequervein thyroiditis

Pathogenesis → Viral illness (flu) —after ①/② week→ Reactive inflammation of thyroid

↓

Subacute thyroiditis

↓

Painful Thyroid gland

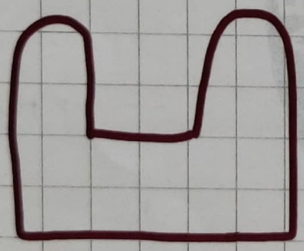
C/F - initially - Hyperthyroid
- later → Hypothyroid → Normal

Rx- Self limiting.
↳ For pain → NSAIDs → steroid

---

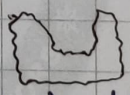
3. Reidel thyroiditis

Pathogenesis



Thyroid gland

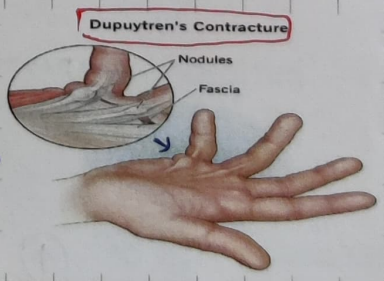
idiopathic fibrosis →



↓ size / ↓ function of Thyroid gland

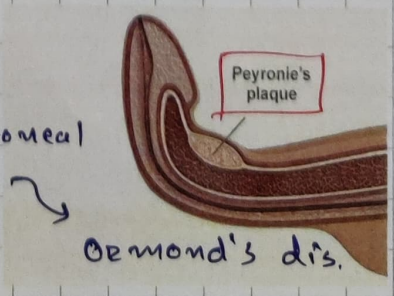
C/f- directly → Hypothyroid

Associated with - Fibrosis → Palm →



→ Penile Fibrosis

→ Retroperitoneal Fibrosis →

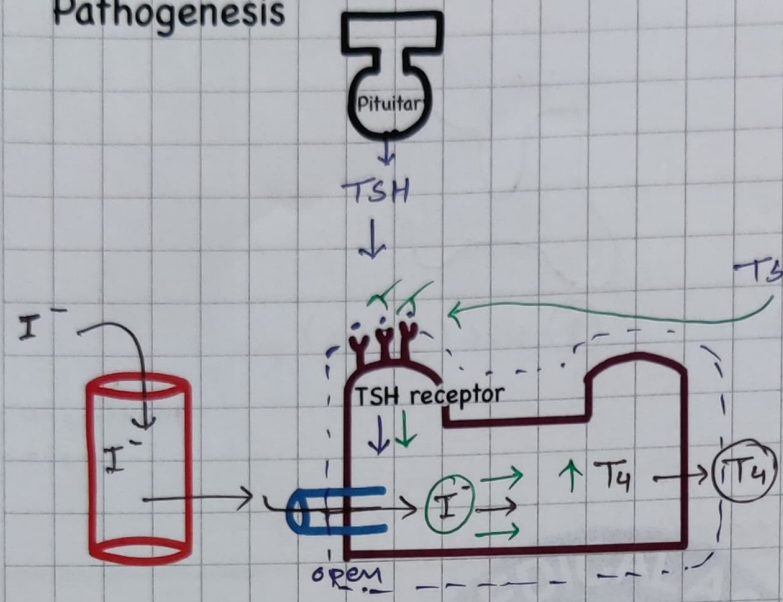


Ormond's dis.

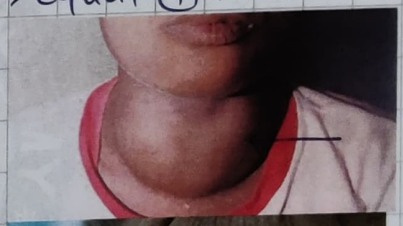
Rx - for hypothyroidism - levothyroxine
- to decrease fibrosis- - Temoxifen

4. Grave disease

Pathogenesis



[Hyperfunctioning of thyroid gland] → equal ↑ in size of T. gland



↓

Goiter

Smooth

Associated with-

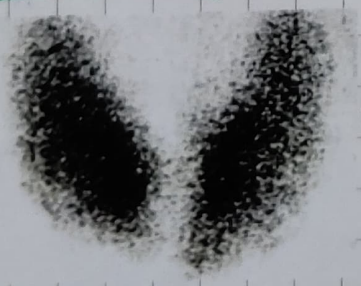
↓

GAG accumulation → Retro orbital →

[Glucosamino-glycan]

Proptosis

RAIU Test



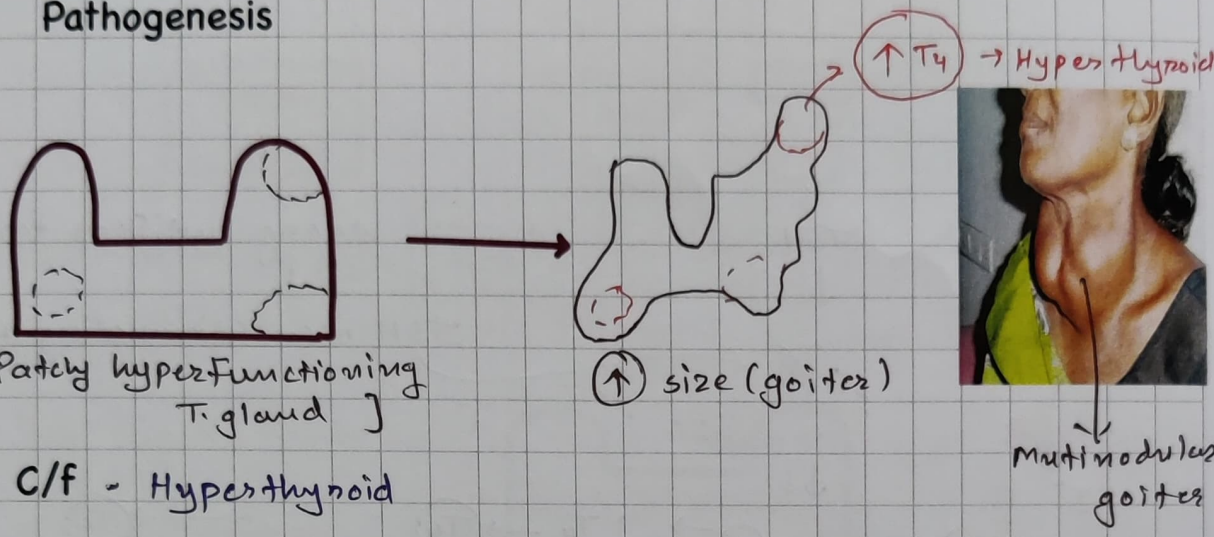
Homogenously ↑ uptake

Pretibial myxoedema

Rx - antithyroid Medication

5. Toxic multinodular goiter

Pathogenesis



C/f - Hyperthyroid

Rx - antithyroid Medication

---

6. Thyroid storm → Sudden severe ↑ in thyroid Horman level.

Pathogensis → accidental injection
→ during Sx manipulation of thyroid gland

C/f - HR - ↑ - Palpitation
BP - Hypotension
Body temp. - ↑↑ (Sudden) Sensorium - altered

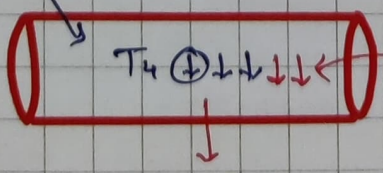
Rx- anti-thyroid ↔ iv steroid + Iodine
iv β #

---

7. Myxedema coma → sudden sever ↓ of thyroid Hormone

Pathogenesis

→ Hypothyroid
↓
defaulter



infection
↑ consumption of thyroid Hormo-

C/f - HR - ↓

BP- ↓ - shock

Body temp.- ↓ - cold

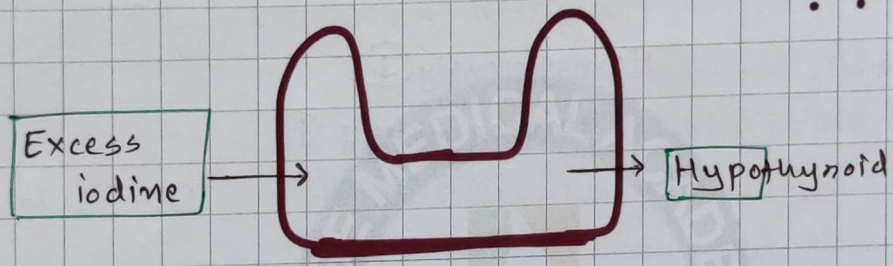
Sensorium - altered

S.glucose..... ↓ S.Na +..... ↓

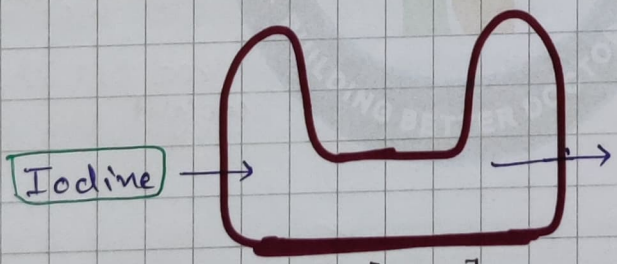
Rx- (IV) Tyroxine

Wolf- chaikoff effect

Extra point -



Jod basedow effect



Nurse

deliberatly → Thyroxine intake

for wt. loss

- wt. loss
- ↑ T_4
- Palpitation
- tremae
- ↑ Body temp

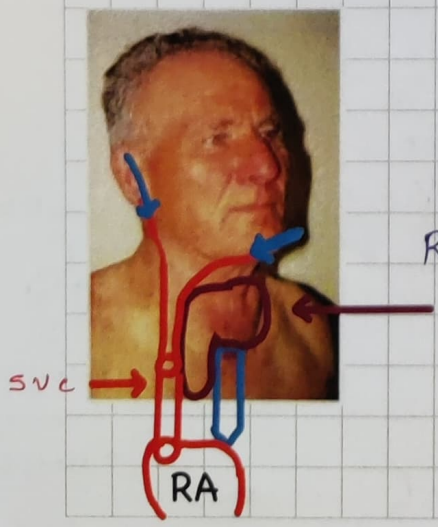
[Factitious thyrotoxicosis]

(or)

Thyrotoxicosis factitia

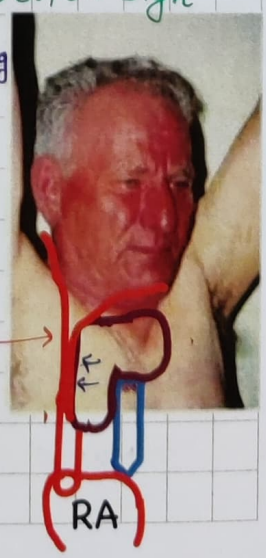
Summary

1. More active T3 form .
2. more production of T4 form.
3. Calcitonin is secreted by Parafollicular c-cell cell.
4. MCC of hypothyroidism Hashimoto thyroiditis
5. DTR - In hypothyroidism Hung up reflex
   - in hyperthyroidism Brisk
6. upper rim of sclera is visible is called as dalrymple sign.
7. thyroid peroxidase inhibitor drugs thyomide
8. most safe anti thyroid medication in pregnancy PTU
9. RAIU in a) thyroiditis Homogenously ↓
   b) Grave disease Homogenously ↑
10. hashimoto thyroiditis - HLA DR3/5 // Anti TPO Ab / anti TG Ab
    -Bx Hurthel cell
11. viral illness f/b pain in neck (tender thyroid) - subacute thyroiditis
12. fibrosis of thyroid gland Reidal thyroiditis .
13. Excess Iodine leading to hypothyroidism -c/a- wolf chaikoff effec
14. Delibrate ingestion of thyroxine to loose ~~wait~~ weight c/a Thyrotoxicosis factitia

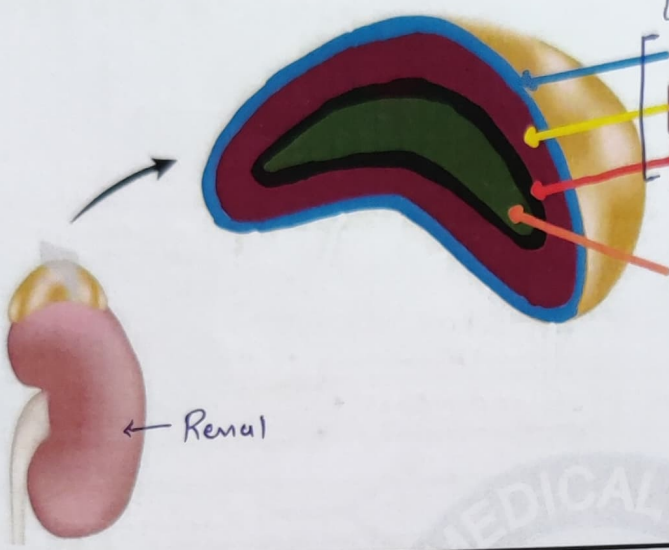


Pemberton sign

- on raising arm



Chapter 8
Basics of Adrenal Gland



- Aldosteron
- Cortisol
- Sex Hormone → Diffydro epiadrostrom sulphate (DHEAS)
- Norepinephrin / epinephrin

Increase in Aldosteron - Hyperaldosteronism

Increase in Cortisol - Cushing syn.

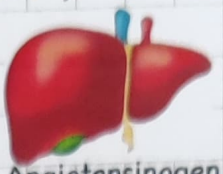
Increase in NE/E - pheo chromo cytoma

Slow (Gradual) decrease in all adrenal haromones - Addison ds.

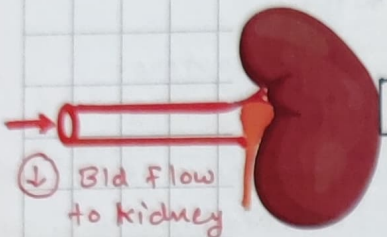
Rapid (acute) decrease in all adrenal harmones - Addisonian crisis

RAAS

~~RASS~~ system (Renin - Angiotensin - Aldosteron-System)



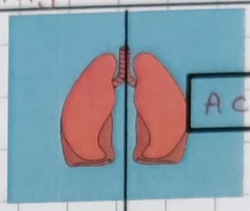
Angiotensinogen



↓ Bld flow to kidney

JG cell activate → Renin →

Angiotensin - I



Angiotensin - II

Blood vessel

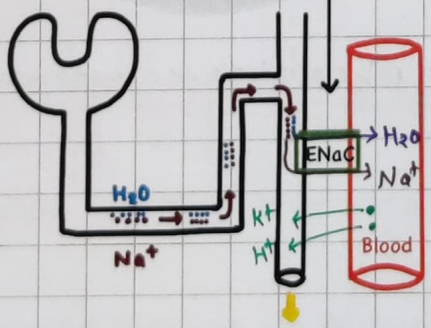
↑ BP ← Vasoconstriction

Z. glomerulosa

Aldosterone

Aldosteron Rec.

Activation OF E.Nac



Gain of H₂O & Na⁺

↑ BP

So loss of K⁺ & H⁺ in urine

↑ ALDOSTERON - leads to S.Na+ ↑

S.K+ ↓

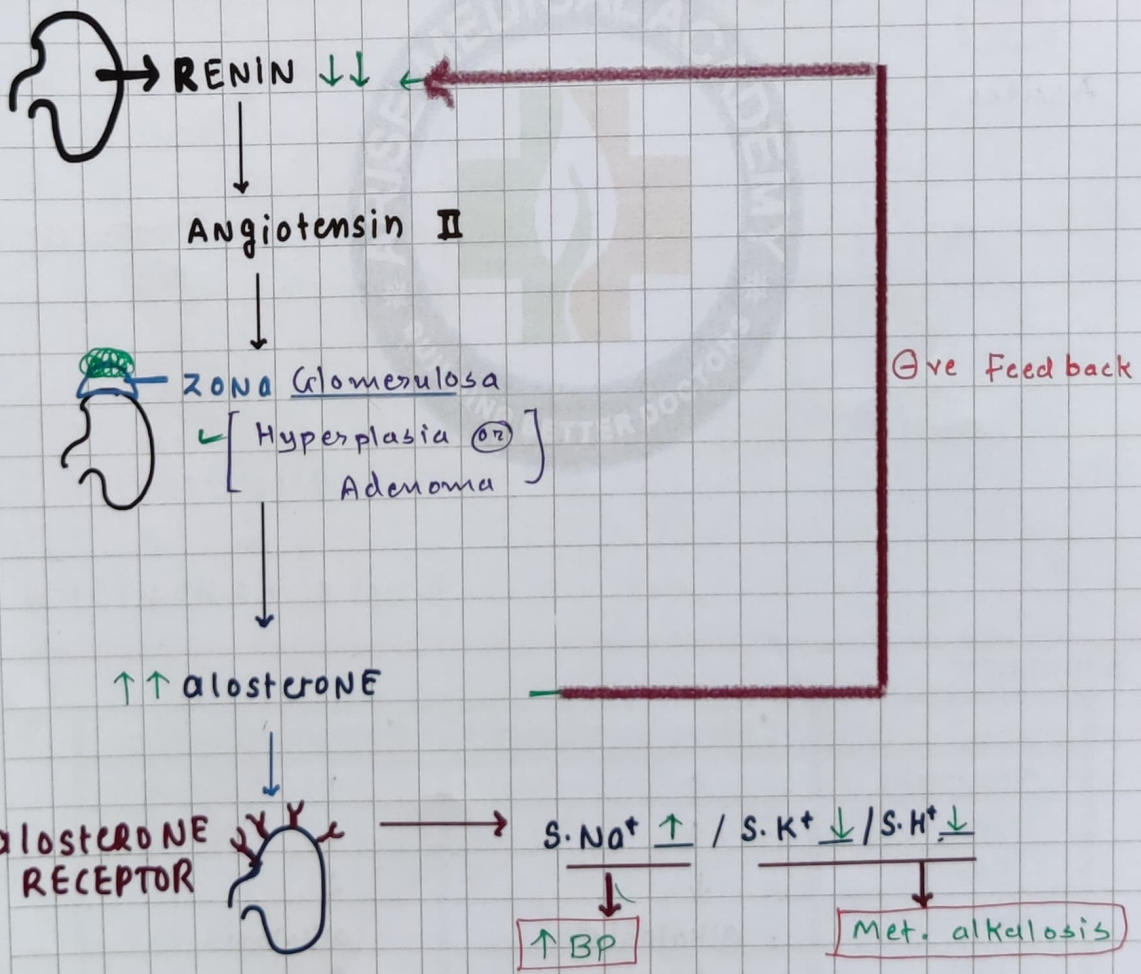
S.H+ ↓

Chapter 9
Hyperaldosteronism

Definition → ↑ Aldosteron so - S.Na + ↑ / S.K+ ↓ / S.H+ ↓
From zona Glomerulosa of adrenal gland.

Etiology
- Adrenal Pathology → 1° Hyperaldosterone
  - (mc) Hyperplasia of z. glom.
  - Adenoma of z. glom. ↓ Conn syn.
- Extra adrenal patho → 2° Hyperaldosterone

1° hyperaldosteronism



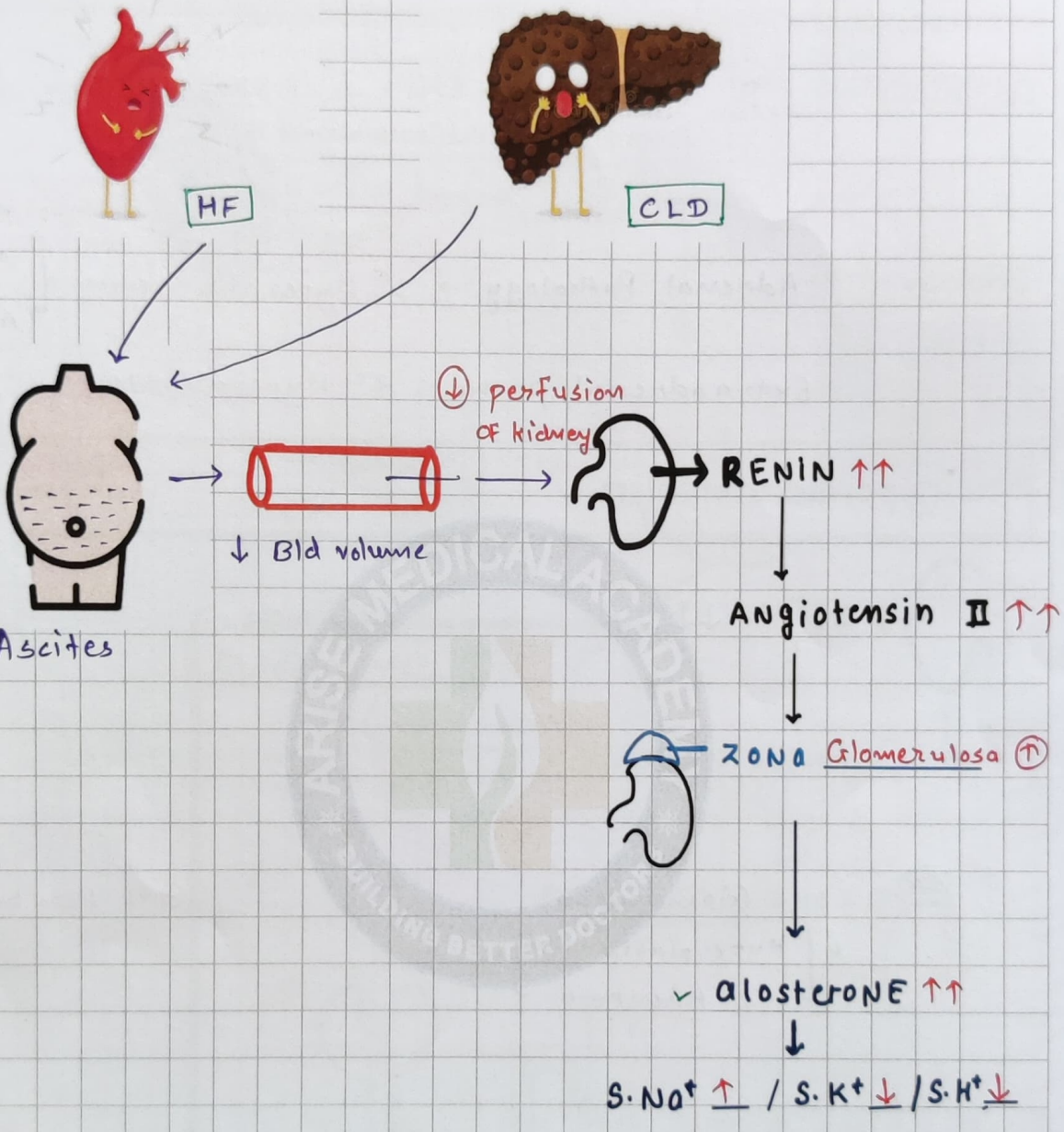
IOC - Saline infusion test

Rx - Aldosteron rec. antagonist - Spironolactone (S/E Gynecomastia)
- Eplerenone

* Renin → ↓↓ in 1°
↑↑ in 2°

* Pedel edema → ⊖ in 1°
⊕ in 2°

2° hyperaldosteronism

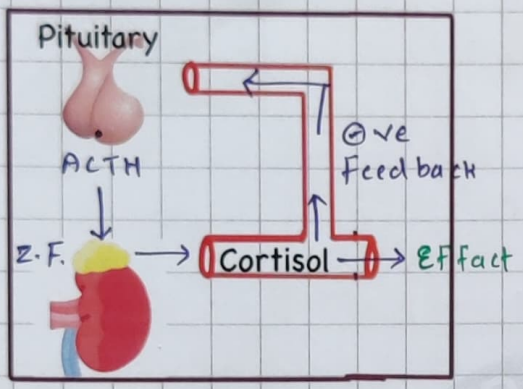


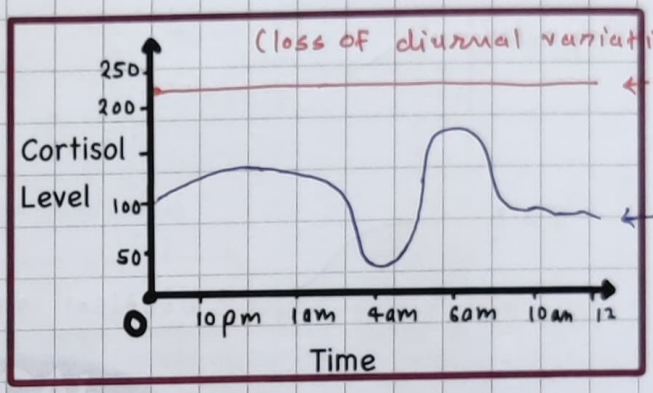
Summary

	1° hyperaldosteronism	2° hyperaldosteronism
Aldosterone	↑	↑
S.Na+	↑	↑
S.K+	↓	↓
Metabolic	alkalosis	alkalosis
HTN	↑	↑
Renin	↓	↑
Pedel edema	Absent	Present
Rx	Spironolectone	Rx the cause. + Spironolactone

Chapter 10
Cushing Syndrome

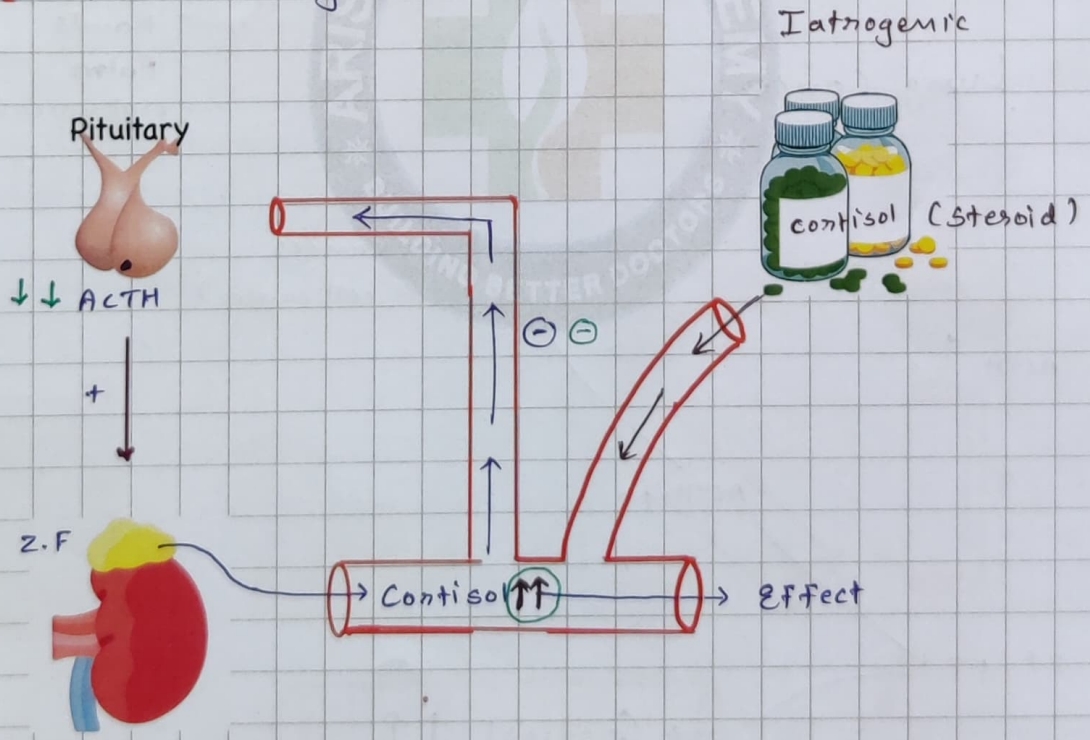
DEFINITION - increase in Cortisol [Z. Fasiculata]





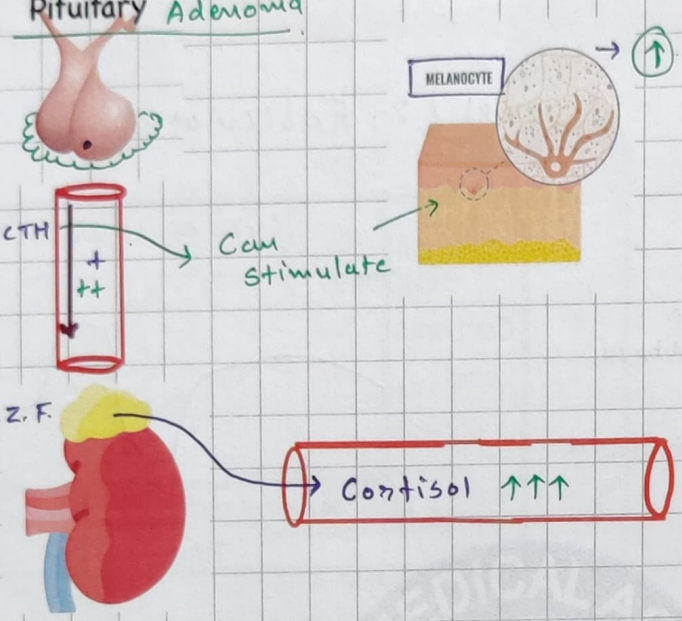
Etiopathogenesis

1.- Mcc - Iatrogenic

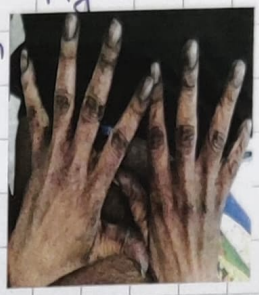


Cushing ds.

2.- Pituitary Adenoma → Leading to cushing sy



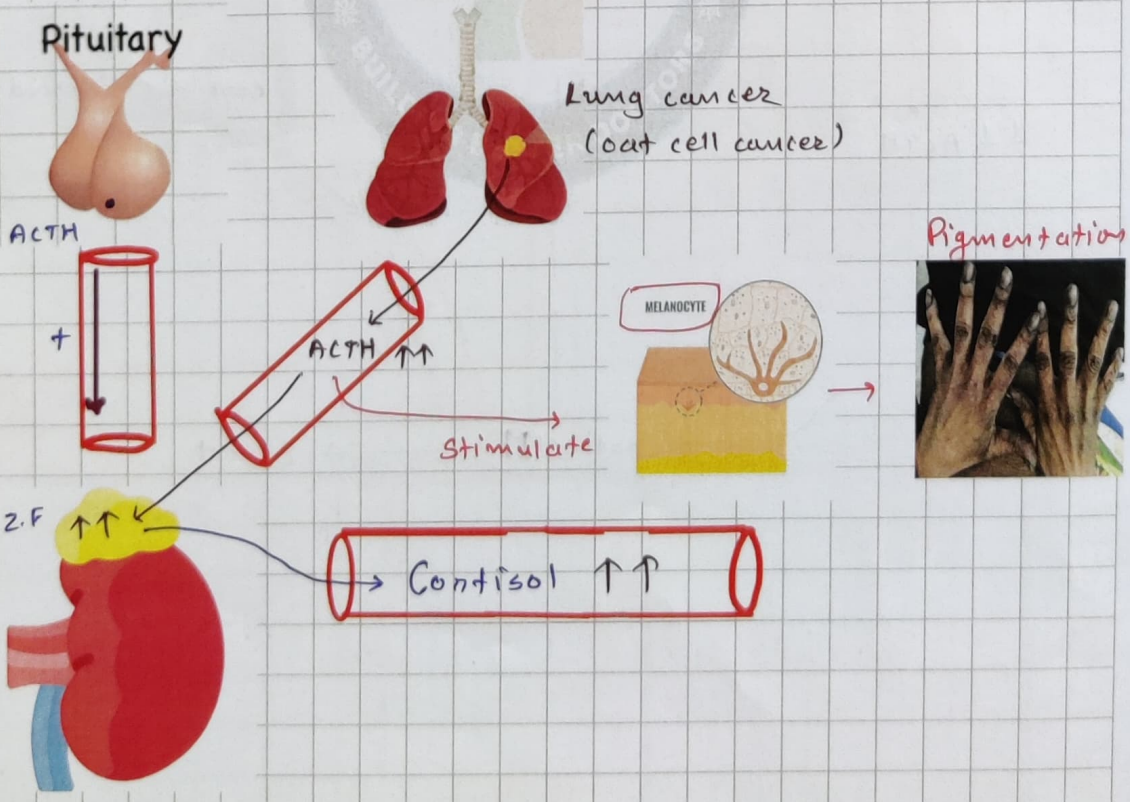
Pigmentation



Pigmentation

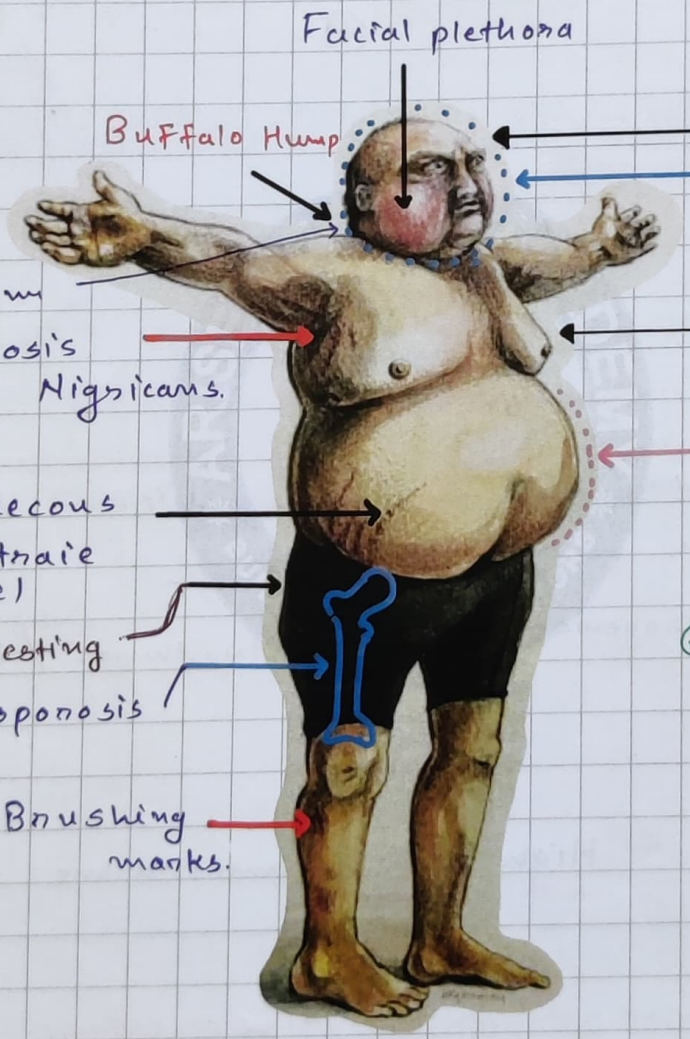
- Hands
- Palm
- oral mucosa

3. Lung Cancer



	Iatrogenic	Pituitary tumor	Lung cancer
Cortisol	↑	↑	↑
ACTH	↓	↑	↑
Pigmentation	No	+	+
Headache	–	+	–
Hemoptysis	–	–	+

C/f -



depression
Low mood /
Moon like Face
Gynecomastia
Truncal obesity
(MC / earliest C/F)

⭐ Lemon on Stick app.

Blood sugar - ↑↑ ; acanthosis Nigricans

BP - ↑↑

Imp

Work up → Cushing suspected

1. Screening test of choice → 24 hr urine cortisol level ↑

1 Tab of Dexamethasone → 11 Pm

2. IOC → Low dose, overnight, dexamethasone suppression test

(+)

To differentiate between iatrogenic v/s pitutory tumor / lung cancer

3. ACTH level

- Low → iatrogenic
- High → Pitu. tumor / Lung Ca.

4. High dose dexamethasone Suppression test

Managment-

1. Cortisol receptor antagonist- Mifepristone

2. Cortisol synthesis inhibitor -
   - Etomidate
   - Ketoconazole
   - Mitotane, Metyrepesone

3. In Cushing Disease to decrease release of ACTH from pituitary tumor - Somatostatin analoges → Octeriotide

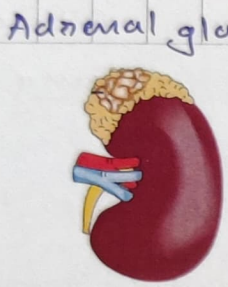
Summary of cushing syndrome

1. M/C/C of cushing syndrom - Iatrogenic
2. 2nd MCC of Cushing syndrome Pituitary adenoma c/a Cushing ds.
3. earliest pathological change of cushing syndrome - Loss of diurnal variation of contisol level
4. MC C/F - Truncal obesity
5. Blood sugar ↑ and BP ↑
6. pigmentation of skin is seen if cause of cushing syn is Pituitary tumor (↑ ACTH)
   lung cancer (↑ ACTH)
7. Screening test of choice - 24 hr urine contisol
8. IOC → overnight low dose dexamethasone supp. test
9. To differentiate between pituitary adenoma v/s lung cancer -
   ↳ High dose dexa. supp. test

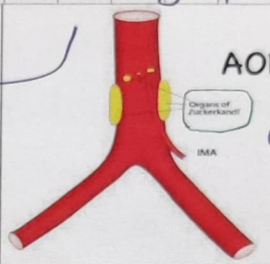
Chapter 11
Pheochromocytoma

Definition- Tumor of Chromaffin cell found in Adrenal Medulla / Sympathetic ganglia

Etiopathogenesis



Adrenal gland → Chromaffin cell tumor



Sympathetic ganglia (extra-adrenal) organ of Zuker candle

↑↑ NE / Epinephrine

metabolism

Normetanephrin + Metanephrin → Vanillyl Mandelic acid [VMA] → Urine

---

10 % Pheochromocytoma are extra adrenal.
10 % Pheochromocytoma are Malignant .
10 % Pheochromocytoma are bilateral. (adrenal gland)

Rule of 10

C/f - ↑ activity of sympathetic system

Δ episodic

- α..rec → Vasoconstriction → HTN (MC) ↳ Headache
- β..rec → ↑ HR → Palpitation
- sweat gland → Profuse sweating

+

↑ metabolism so wt loss

Investigation -

screening test of choice - Urine metanephrin level

IOC- plasma fractionated metanephrin level

For localization - MRI abd.

Rx → α & β # → 1st α # —F/B→ β #
(Phenoxybenzamine) (Propranolol)

RxOC- Sx excision of tumor

Summary of PCC

A/C/A - Chromaffinoma

RULE of 10

Rule of 50 - HTN

Rule of 9- - Burn

M/C extra-adrenal site → Organ of zuker candle

M/C- c/f - Headache (episodic) ✓✓

Screening test - Urine Metanephrine level

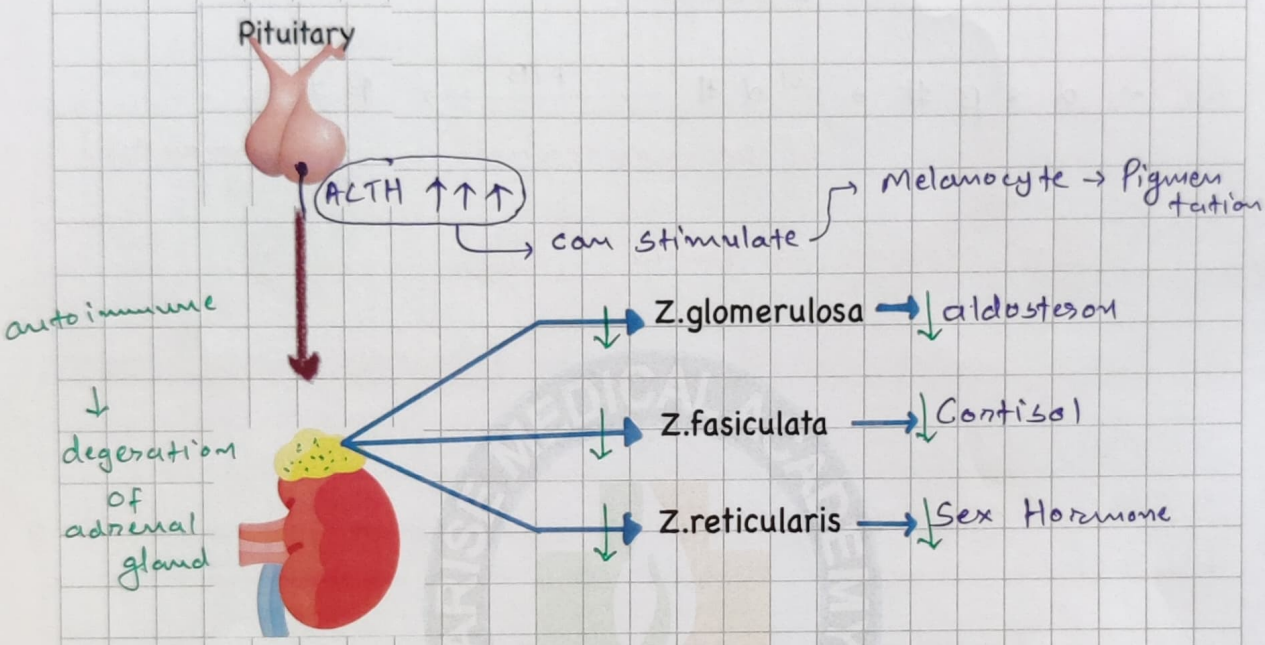
IOC - plasma frac. " "

RX - α blocker f/b β blocker .

Chapter 12
Adrenal Insufficiency

1 adrenal insufficiency / Addison ds.

Etiopathogenesis



M/C/C- worldwide- Autoimmune
- India - TB of adrenal gland
- HIV patients - CMV of adrenal gland infection

C/f - due to decrease in aldosterone - S.Na+.....↓/ S.K+.....↑/S.H+↑...

-due to decrease in cortisol - Hypoglycemia
-due to decrease in sex harmone - (↓) Libido
-due to increase in ACTH - Pigmentation

IOC - ACTH (Cosyntropin) stimulation test
Rx. -DOC - Hydrocontisone
- For salt craving- Fludrocontisone

Water house Friedrichson syn.

ACUTE ADRENAL INSUFFICIENCY (or) / Addisonian Crisis

Pathogenesis

- Meningococcal meningitis → Patechiea (↓ PLT)
- Dengue Fever → Patechiea (↓ PLT)
- HIV + severe pneumonia

acute bleeding in adrenal gland

↓

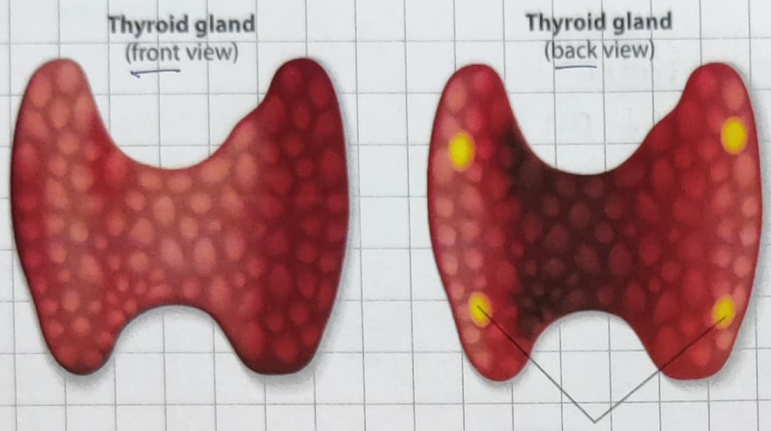
Acute shutdown of adrenal gland

↓

Shock

DOC - IV Hydrocortisone

Chapter 13
Physiology of parathyroid gland



Parathyroid glands

Secretes

Parathyroid Hormone (PTH)

Bone

Activation of osteoclast

Cutting of bone

Ca^{+2}

PO_4^{-2}

Blood

Ca^{+2}

PO_4^{-2}

Ca^{+2}

Blood

Urine

PO_4^{-2}

Net effect of PTH - S.Ca+ ↑ / S.po4 ↓ / ALP ↑

Chapter 14
Parathyroid Pathology

1° hyperparathyroidism

Definition - increase in PTH due to d/t Parathyroid gland - adenoma

Etiopathogenesis

Excess PTH ↑↑

Excess activation of osteoclast

↑ Cutting of bone

Ca^{+2}
PO_4^{-2}

BID

Ca^{+2} & PO_4^{-2}

↑↑ Ca^{+2}

BID

Hypercalcemia

Urine → PO_4^{-}

Constipation

Bone Pain

Psychiatric symptom

Renal stone

Normal

1° Hyper PTH → Subperiostal resorption of bone

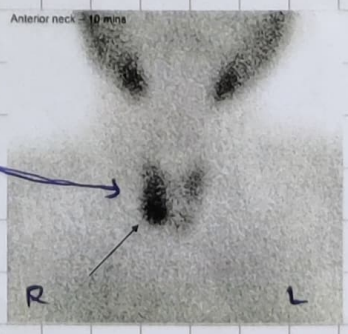
Normal

1° Hyper PTH → Salt & Paper appearance

⊛ 1° Hyper PTH :

Investigation - PTH.....↑..... / S.Ca+.....↑..... / S.PO4.....↓..... / ALP.....↑.....

For localization of tumor - Tech ^{99} Sestamibi scan



DOC - Ca^{++} Binder → Ibandronate (Biphosphonate)

RxOC- Sx excision of tumor

2° Hyperparathyroidism

Definition → ↑↑↑ in PTH due to Pathology other than Parathyroid gland.

Etiopathogenesis →

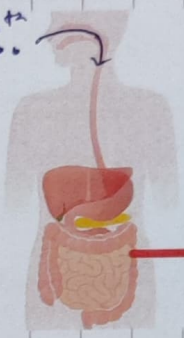
Most active form

↓ Absorption of mainly Ca^{+2}

↓ Calcitriol

CKD

Ca^{+2}



Bld

Ca^{+2} ↓↓

Stimulate @ Trigger

Parathyroid gland

PTH ↑↑

↑↑ activation osteoclast

Gutting of bone (Vertebra)

← Ca^{+2} & PO_4^-

Blood

PO_4 Excretion will not occur

↓

PO_4 ↑

Investigation - PTH.....↑..... / Vit D.....↓..... / S.PO4.....↑..... / ALP.....↑..

S.Creatinine.....↑..... / S. Ca+2.....↓↓

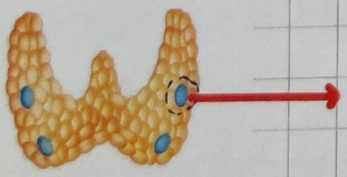
CxR → Rugger Jersy Pattern
(Renal osteodystrophy)

Rx-
↳ Treat the cause

Hypoparathyroidism

Etiopathogenesis- .....↓↓..... in PTH

Due to ⇨ Autoimmune distruction of PTH gland
↳ accidental removal of parathyroid gland during thyroid Sx
↳ Digeorge Syndrome (Ch. 22q 11 defect)
↳ Congenital absent of PT gland



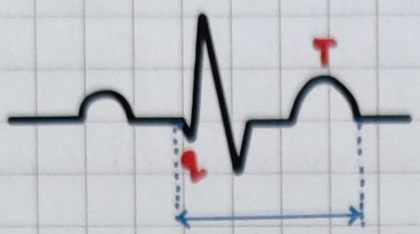
↓ PTH → Ca^{+2} ↓↓ Hypocalcemia

C/f

Chvostek sign.

Trosseau sign

ECG

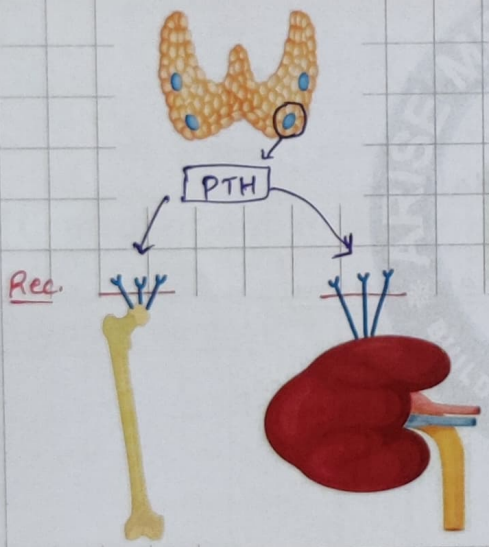


S Ca+2 α $\frac{1}{\text{QT interval}}$ (↑)

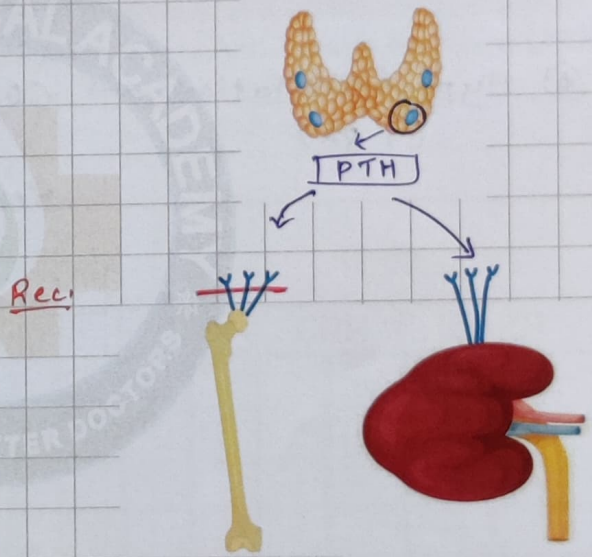
Rx →
- Calcium gluconate

Extra point → dlt GNAS gene mutation

Pesudo-hypoparathyroidism



Pseudo-Pesudo-hypoparathyroidism



Summary

1. effect of PTH ---S.Ca+2 ↑ and S.PO4 ↓
2. 1° hyperParathyroidism - MCC - Adenoma
   - Xray skull - Salt & paper app.
   - PTH ↑ S.Ca+2 ↑ S.PO4 ↓
3. 2° hyperParathyroidism - MCC - CKD
   - Bone defect c/a - Renal osteodystrophy
   - PTH ↑ S.Ca+2 N↓ S.PO4 ↑

4. Hypoparathyroidism - PTH ↓ S.Ca+2 ↓ S.PO4 ↑

Carpo-pedal spasm with BP Cuff c/a - Trosseau sign.

* Troisier sign--- Stomach cancer involving (L) Supraclavicular LN.

5. PTH - Both bone and kidney resistant - Pseudo hypo PTH
- only bone resistant → Pseudo Pseudo Hypo PTH
- associated with GNAS gene mutation.

---

(4) Hyperventilation (↑ RR) → CO_2 wash out

↓

Respi. alkalosis

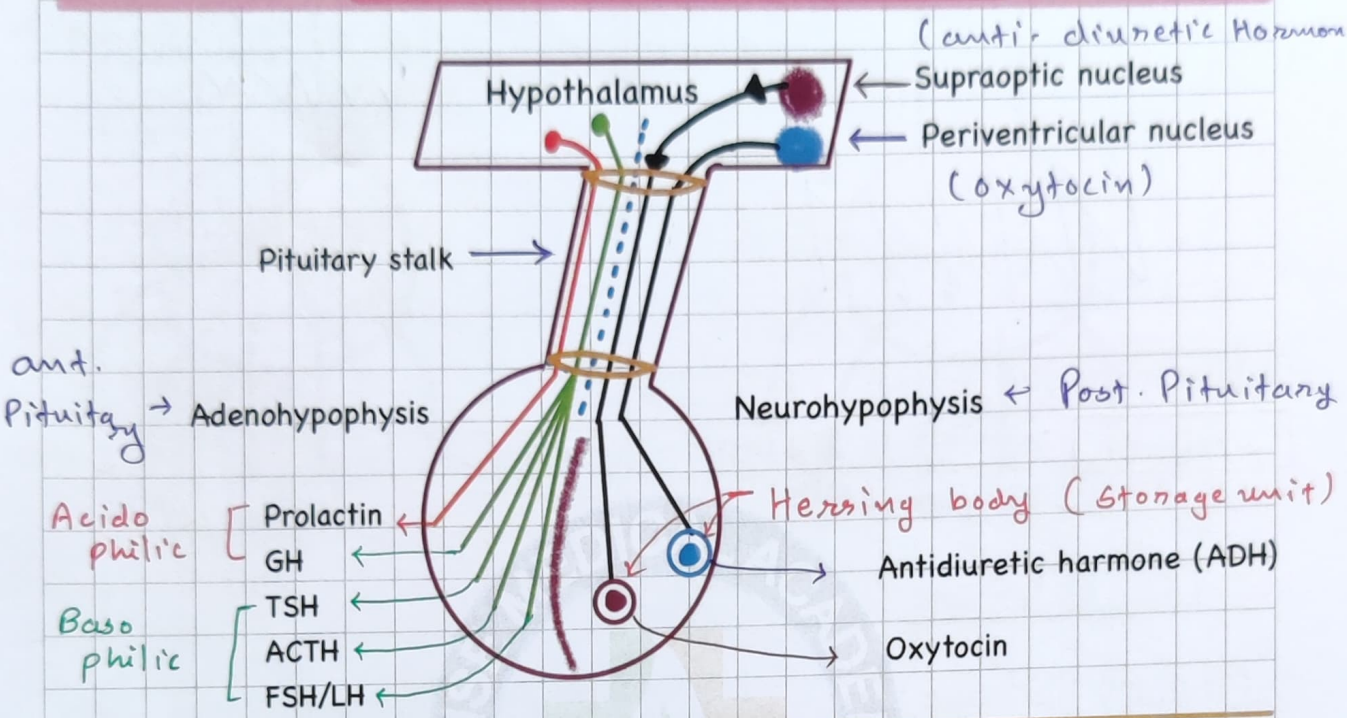
↓

Carpo-pedal spasm

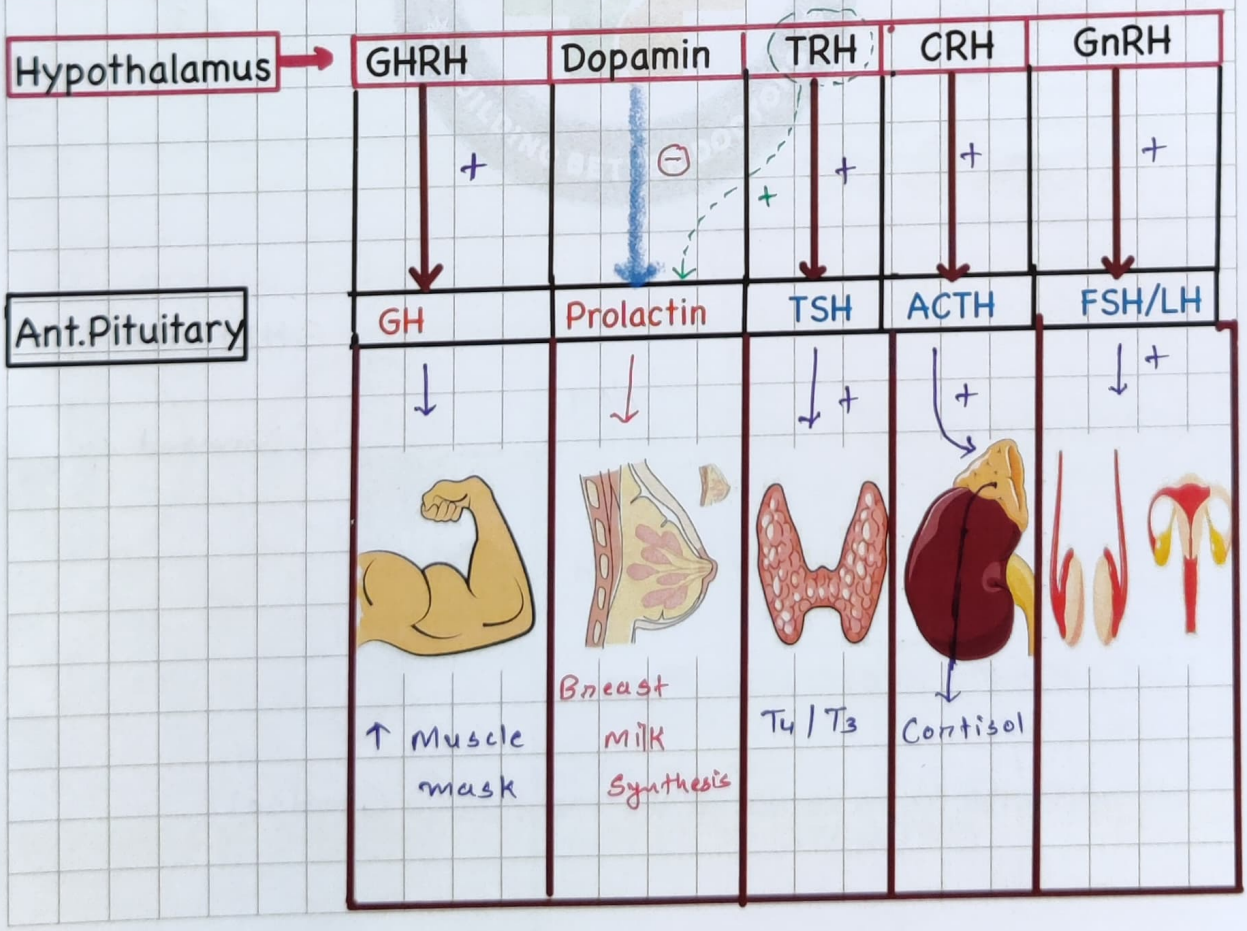
↓

(Rx) - Rebreathing of CO_2

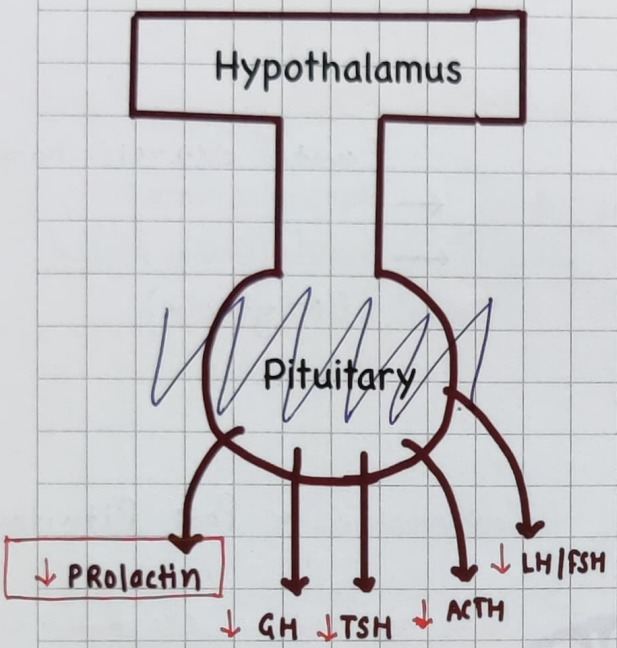
Chapter 15
Basics of pituitary Gland

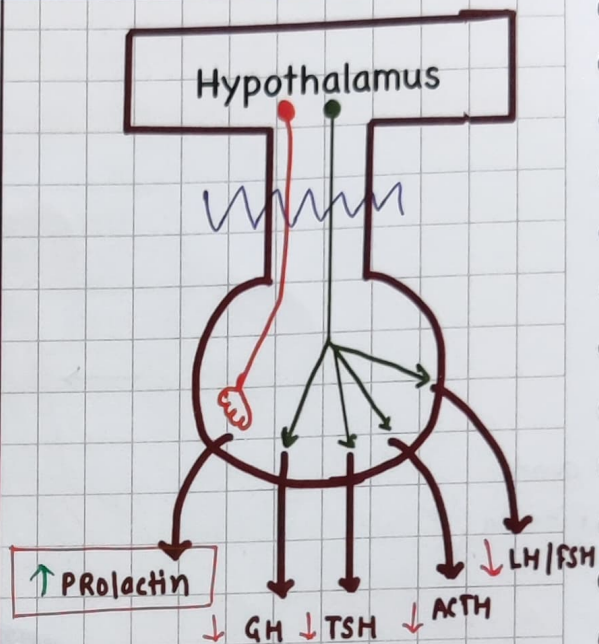


Control of ant. Pituitary



Pituitary injury v/s pituitary stalk injury





Pituitary harmone level - ↓↓

Prolaction level ↓↓

Pituitary harmone level - ↓↓

Prolaction level ↑↑

Extra points for pituitary injury

1. Pituitary injury if due to obstetric cuase → SHEEHAN Syn.
2. Pituitary injury if due to Non-obstetric cuase → Simmond syn

1 st harmonen to decrease - GH

Last harmone to decrease - TSH

1st harmone to be replaced via treatment- Cortisol

Chapter 17
Pituitary disorders

Hyperprolactinemia

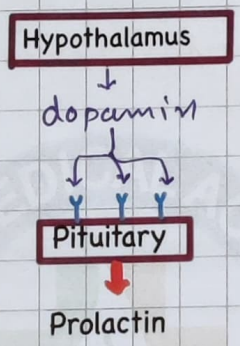
Definition - ..... ↑↑↑ ..... level of prolactin

Etiopathogenesis-

(MCC) 1. Prolactinoma

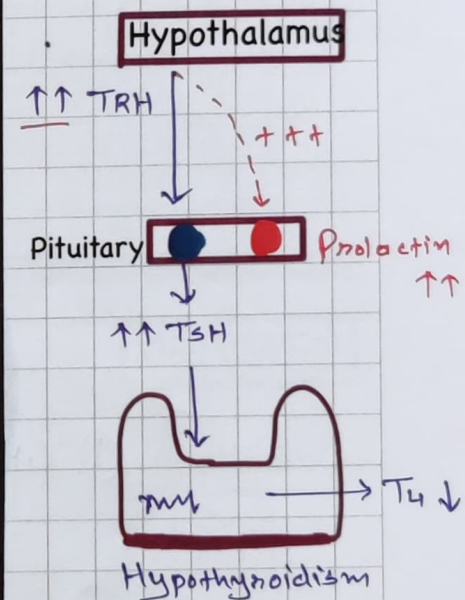
↑ Prolactin

2. drugs → # activity of dopamin

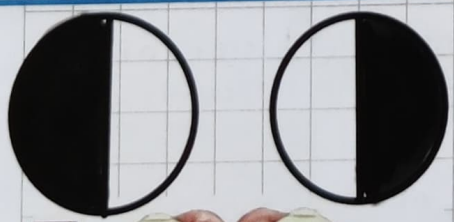


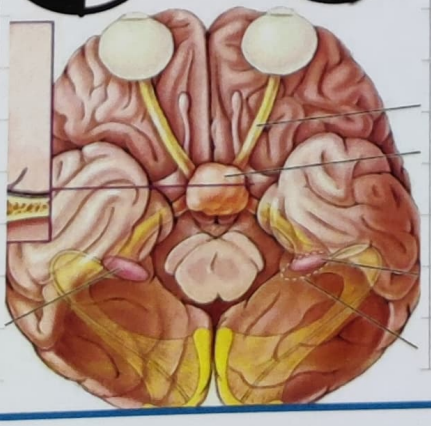
Eg. of drug
- Haloperidol
- Metachlopromide
- chlorpromazine

3 1° Hypothyroidism



C/f- increased prolactin - 1. ↑ Breast milk → Galactorrhea
2. Headache
3. Bitemporal hemianopia
(dlt compression of optic chiasma)





→ < 1 Cm → Microadenoma
→ > 1 Cm → Macroadenoma

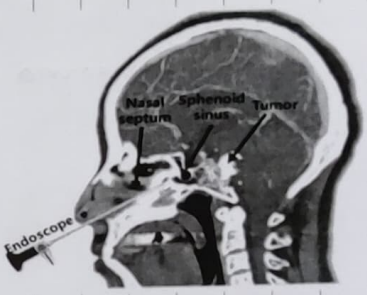
4. ↓ Menstruation

screening test - S. Prolactin level (↑)
IOC - MRI brain → For prolactinoma

Rx- 1. Dopamin agonist → Cabergolin (teratogenic)

2. In pregenancy → Bromocriptin

3. If compression symptom-
↓
Trans sphenoidal resection



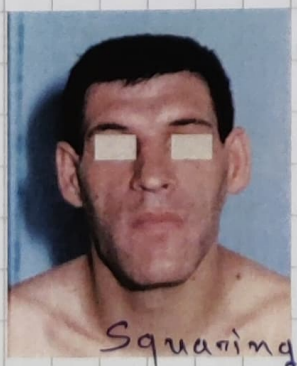
Acromegaly

Definition - (↑) GH after puberty

- Increase in GH before puberty is called as - Gigantism

Clinicopathogenesis

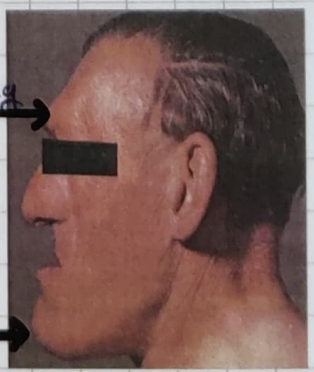
Increased GH - .....↑↑↑..... Somatomedins - (↑) size of Terminal bones



Squaring of mandible

Frontal bossing

Prognatism



↑ size of colonic polyp → Colon Cancer

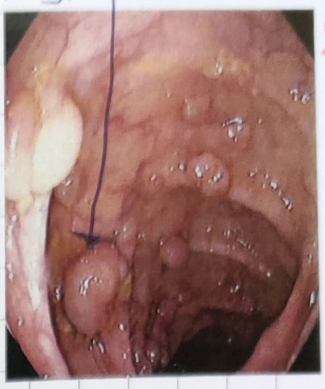
Facial feature Coarse

Voice - low pitch

Hand and ring size ↑
↳ shoe size ↑

Hands looks like Spade

- Hepatomegaly
- Macroglosia (↑ Tongue)

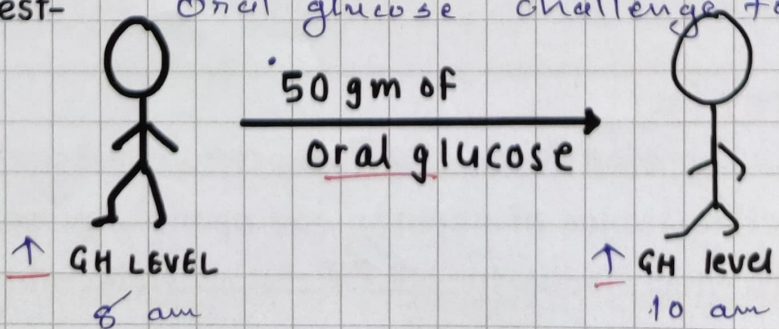


↑ GH →  → ↑ IGF-1 → ↑ Blood sugar
↳ DM

↑ GH →  → ↑ muscle mass

1981 1982 1983 1984

- Screening test of choice - IGF1 levels ↑
- Confirmatory test- Oral glucose challenge test



For size of tumor → MRI brain

Rx- if resectable tumor - Trans sphenoidal resection

If non-resectable - initial treatment - octreotide → ↓ release of GH

Best drug - GH rec. antagonist → Pegvisomant

Extra point For acromegaly :-

Xray foot

↓

↑ in size of Fat pad (> 2.5 cm)

Heel pad thickness

GH receptor anomaly

↓

Laron dwarfism

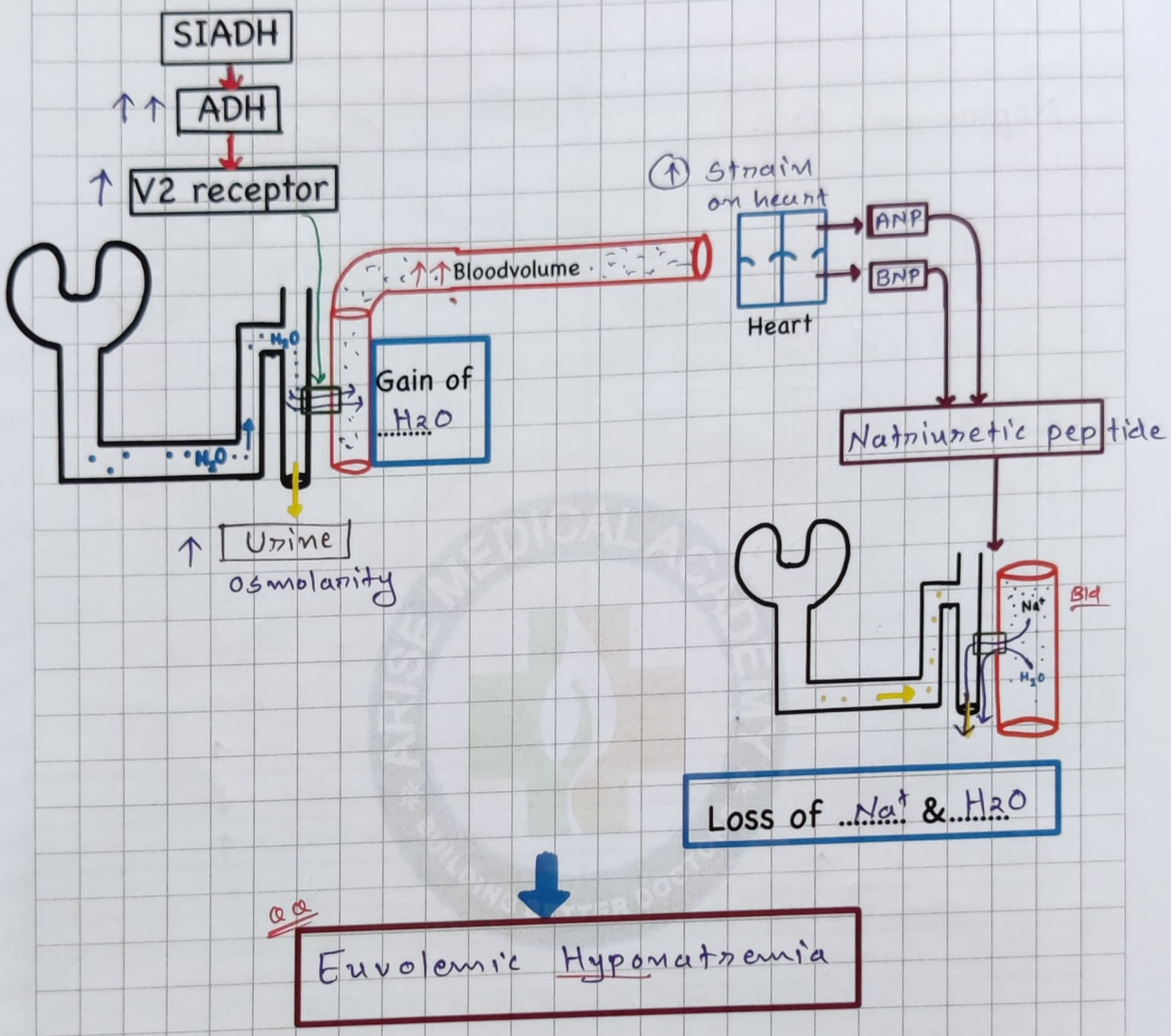
SIADH (↑ ADH) → Syndrome of inappropriate secretion of antidiuretic harmone (ADH)

Etiology - H - Head injury (MCC)
E - Ectopic (Lung cancer)
L - Lung abscess
D - Drug - vincristin / SSRI / cyclophosphamide

Physiology fact

* ADH is secreted from Post. pituitary acts on V2 receptor in collecting tubles of nephron and opens Aquaporin II channels which leads to ↑↑↑ water absorption from Urine.

Pathogenesis



ANP- atrial natriuretic peptide
BNP- brain natriuretic peptide

C/f - depends on severity of Hyponatremia (↓Na+) →
- Fatigue
- Muscle pain
- Altered sensorium
- seizure

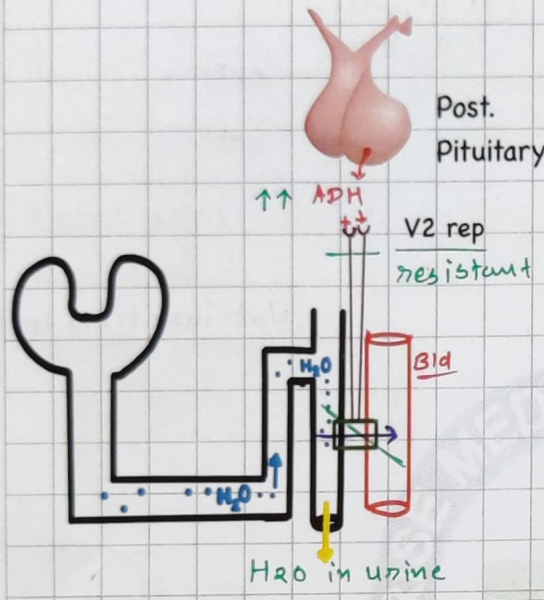
→ Investigation -
urine osmolarity ... ↑↑
Plasma osmolarity ... ↓↓

IOC - water loading test

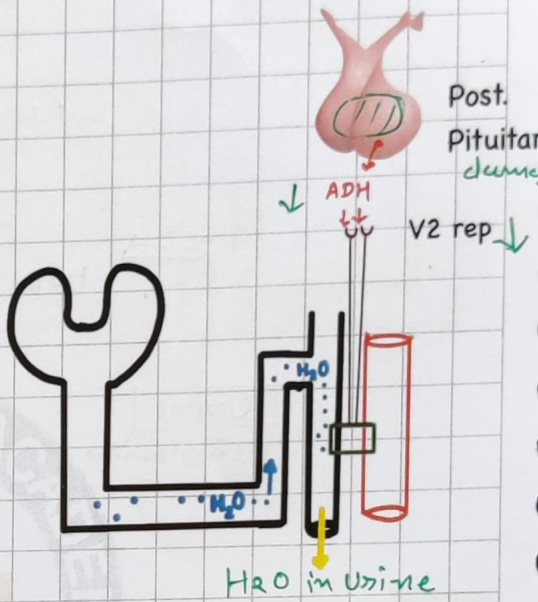
DOC - V2 rec. #
↓
[-Vaptan]
(demeclocyclin can also be used)

Diabetes Insipidus

Nephrogenic D.I.



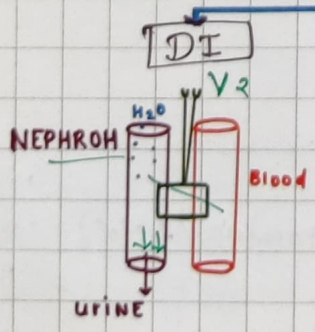
Central D.I.



Nephrogenic D.I.	Central D.I.
Urine amount – ↑	Urine amount ↑
Urine osmolarity ↓	Urine osmolarity ↓
Plasma osmolarity ↑	Plasma osmolarity ↑
ADH level ↑ ✓	ADH level ↓ ✓
IOC – water deprivation test	
DOC Thiazide (↑ sensitivity of rec.)	DOC - Desmopressin

Extra point

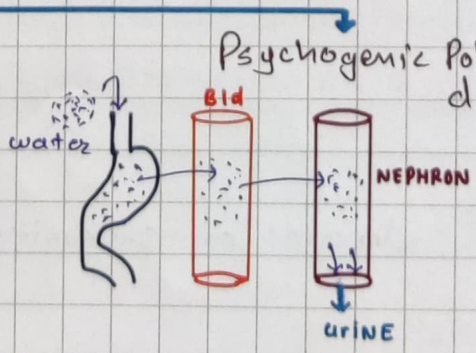
Polyuria (3lit./day or >40ml/kg/day)



Urine volume ....↑

Urine osmolarity ....↓

Plasma osmolarity.. ↑



Urine volume ....↑

Urine osmolarity ....↓

Plasma osmolarity.. ↓

Extra point
DM + DI + Optic atrophy - Wolfram Syn

Endocrine neoplasia

- **Insulinoma** - tumor of ...B... cell so ...↑↑... insulin → ↓ Glucose level

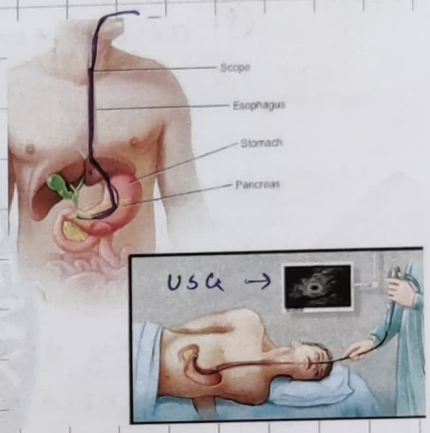
C/f - Hypoglycemia (Palpitation / sweating / drowsy)
- Blood sugar ↓↓
- Glucose will ↓ Symptoms

Whipple Δ

Ioc - Fasting insulin/glucose ratio.

PYQ For localizing tumor - endoscopic USG
- MRI abd.

Rx - Excision of tumor



- B-cell hyperplasia is called as- Nesidioblastosis

VIPOMA - ↑ VIP
(VIP - vaso active intestinal peptide)

A/c/a - WDHA Syn. (PYQ)
- watery diarrhea
- Hypokalemia (K+) / Cl⁻ ↓
- Metabolic acidosis

C/f -

also c/a = Pancreatic cholera

Glucagunoma - tumor of α.....cell

C/f - blood sugar ↑ → DM

4D's - skin - Necrolytic erythema migrans.

Gastrinoma/ zollinger elison syndome

Tumor of G..... cell. M/c site- duodenum

C/f-

↓

↑ Gastrin

↓

Doc- ↑ acid secretion → Acid peptic ds.

↳ PPI

Multiple endocrine neoplasia (MEN)

MEN 1 (wermer syn)	MEN 4
• Pituitary tumor • Parathyroid tumor • enteropancreatic tumor overall mc enteropancreatic tumor Gastrinoma M/C pancreatic tumor Insulinoma	• Pituitary tumor • Parathyroid tumor • testicular / ovarian tumor
MEN 2A (Sipple Syn)	**MEN 2B (MEN 3)**
• Medullary ca of thyroid • Pheochromocytoma • Parathyroid tumor	• Medullary ca. of thyroid • Pheochromocytoma • Marfanoid Habitus Megacolon

Summary -

1. Pituitary gland injury - prolactin level ↓
2. Pituitary stalk injury - prolactin level ↑
3. ~~Prolactinoma~~ - mcc - Prolactinoma

Hyperprolactinemia -DOC - Cabergolin

-DOC in pregnancy - Bromocriptin

4. ACROMEGALY - voice - Low pitch

@ - MC cancer - Colon cancer

Screening test - IGF-1 level ↑

IOC - oral glucose challenge test

Xray foot - ↑ Heel pad thickness

Rx- initial - octreotide

- BEST- Pegvisomant

5. SIADH

MCC - Head injury

PATHOgenesis - euvolumic Hypo natremia.

Urine osmolarity - ↑

IOC - water loading test

DOC -Vaptan..

6. Diabetes insipidus

Central - ADH level - ↓

Nephrogenic - ADH level - ↑

Urine osmolarity ↓

IOC water deprivation test

rx - central - Desmopressin

- nephrogenic - Thiazide

7. Insulinoma

C/f - whipple's triad

IOC - Fasting insulin/glucose ratio

FOR localization -- endo usg > MRI

8. Glucagunoma - skin finding - Necrolytic erythema migrans

9 VIPOMA- c/f - watery diarrhea

K+. ↓ / cl-↓

You're braver than you believe, stronger than you seem, and smarter than you think.

Best wishes

④ Metabolic Syndrome / syn (x) :-

① Diagnostic criteria

- BP > 130/85
- Fasting Blood sugar > 100 mg/dl
- TG > 150
- HDL < 50 (male)
  < 40 (Female)
- Abd. circumference > 102 cm (male)
  > 88 cm (Female)

→ 3/5 (+) → Metabolic syn

⑤