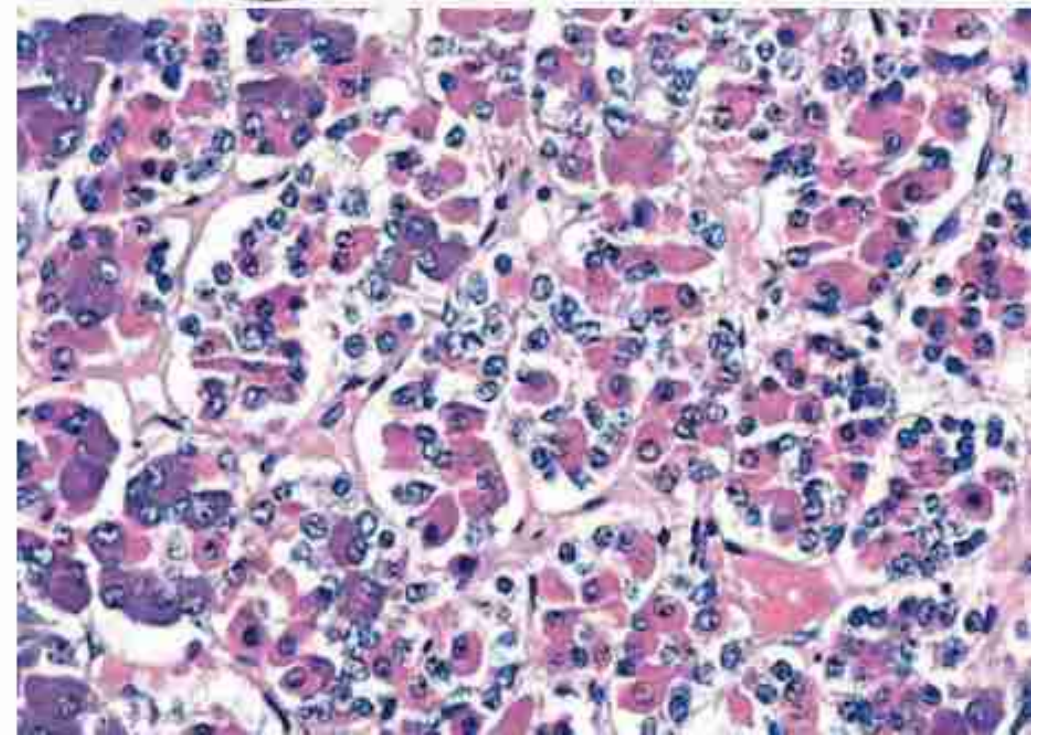
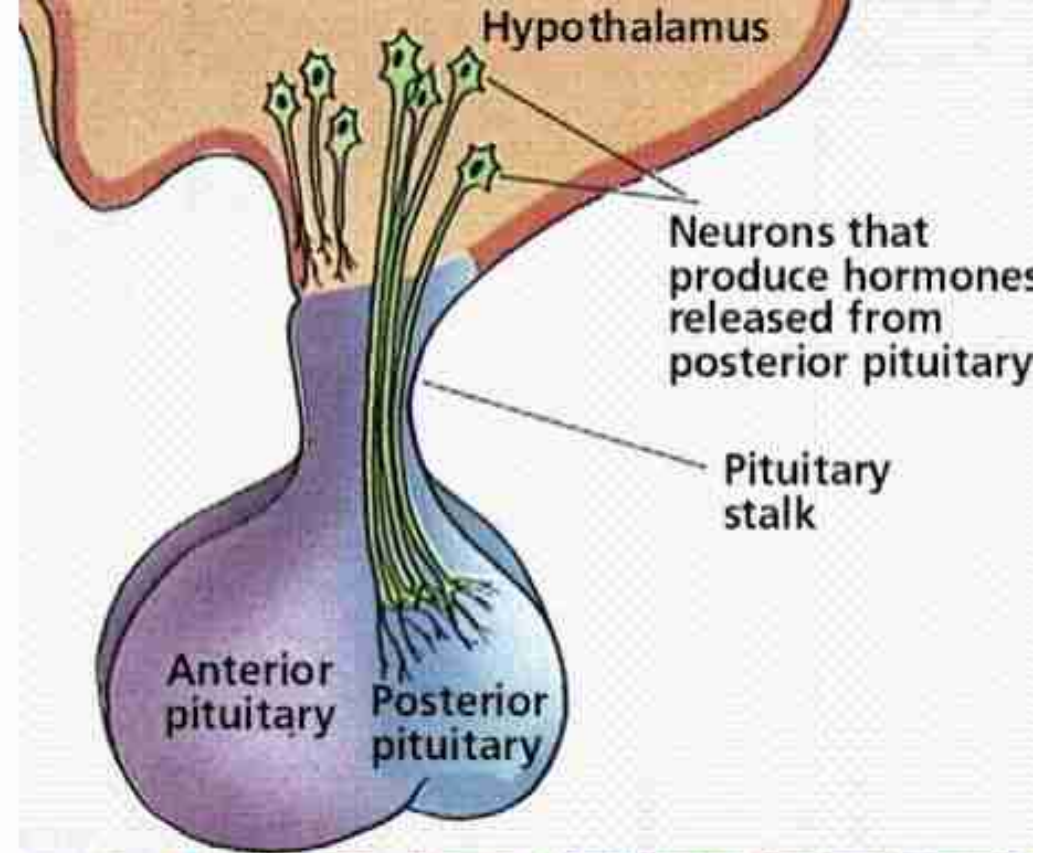
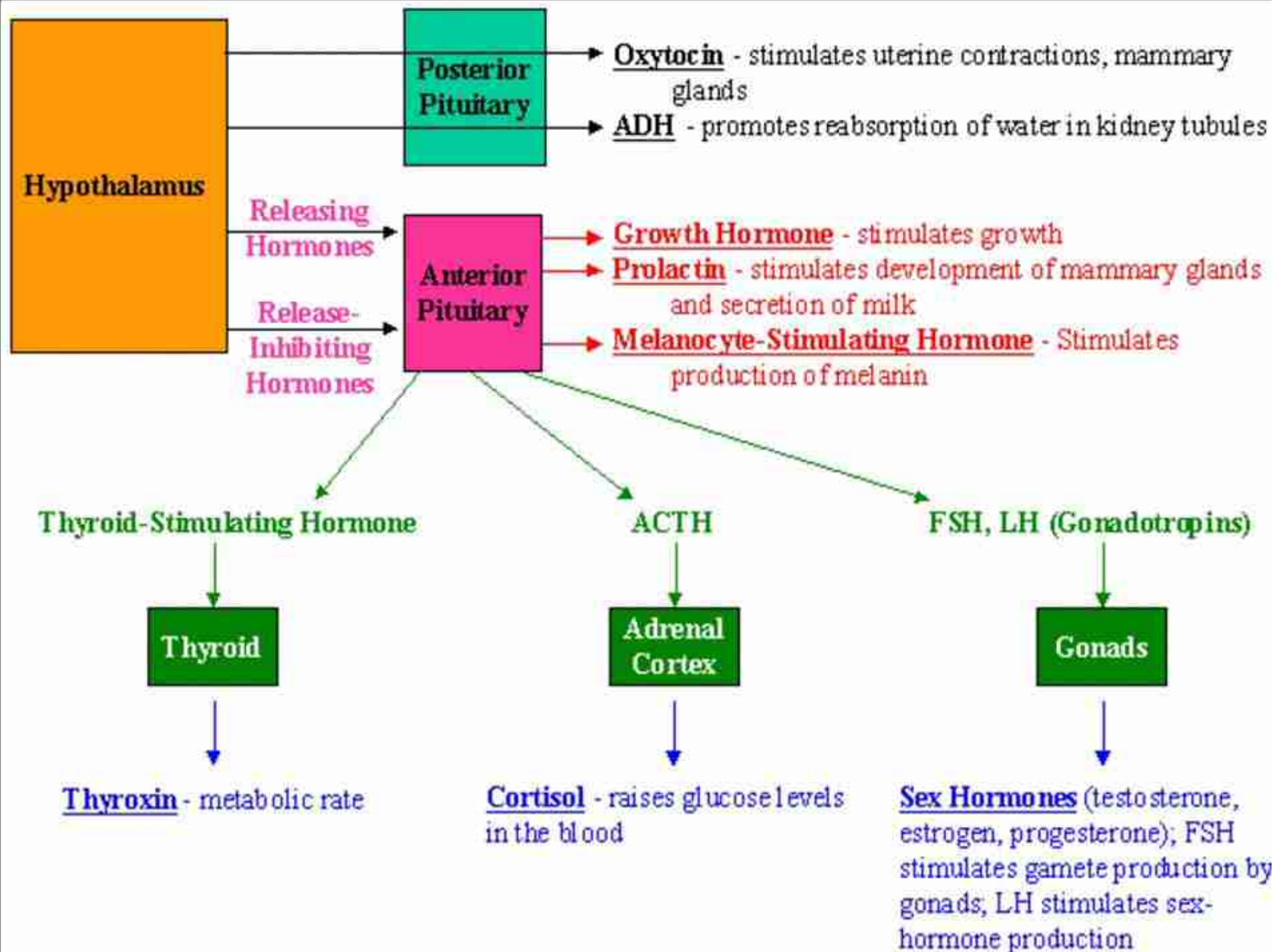


PITUITARY GLAND PATHOLOGY

- Anterior pituitary
- Posterior pituitary





Neoplasms

- Adenoma

Functional disorders

- Hyperfunction
- Hypofunction

PITUITARY ADENOMA

- Functional
- Non- functional
- May be part of MEN-1 syndrome
- Carcinoma & metastasis - rare

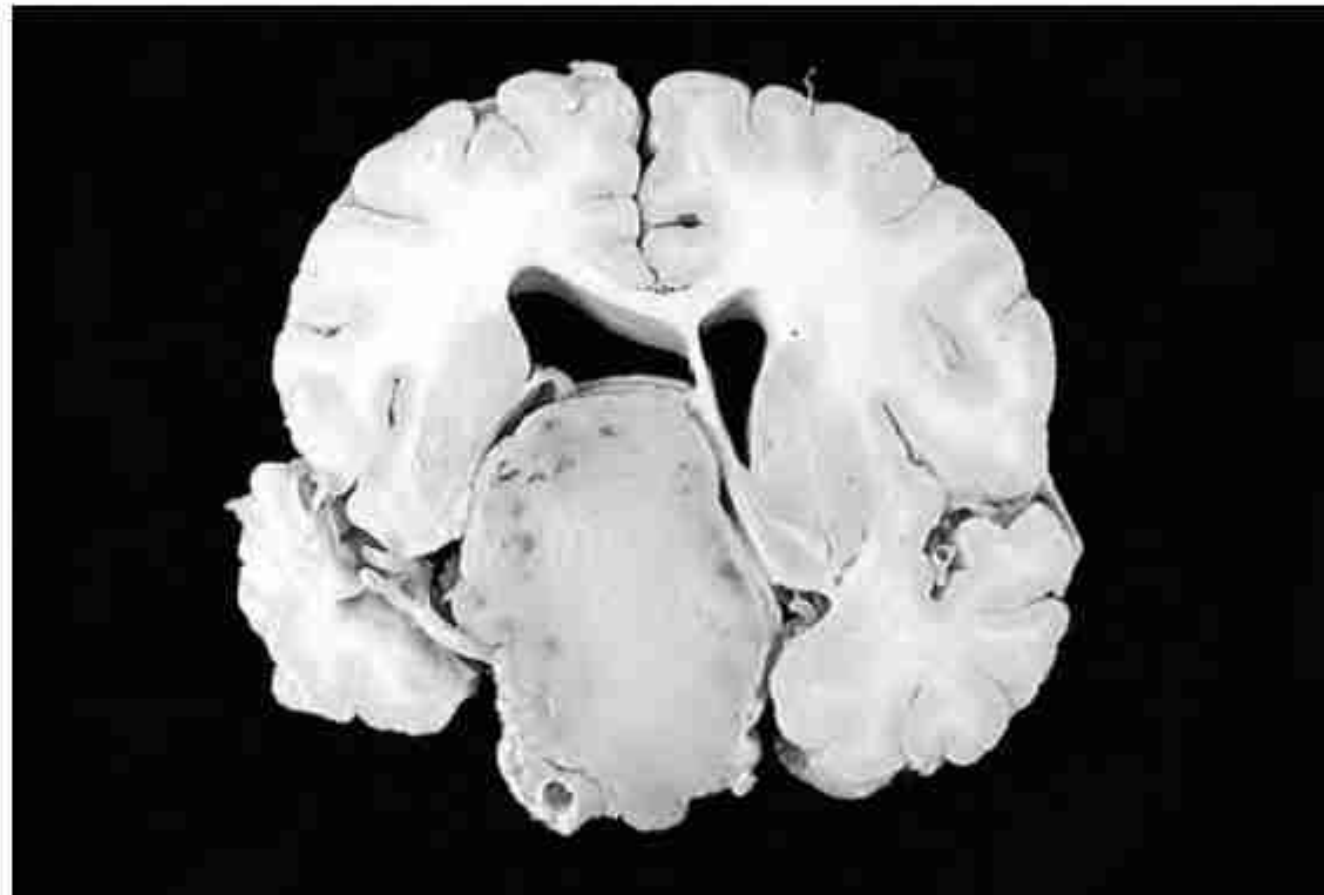
Symptoms

- Hyper/ Hypopituitarism
- Mass effects-
 - Visual field defects- bitemporal hemianopia
 - CN palsies
 - Raised ICT

PITUITARY adenoma	Hormone	SYMPTOMS
Lactotroph adenoma (Prolactinoma)	Prolactin	Females- Galactorrhea, amenorrhea Males- Sexual dysfunction
Somatotroph adenoma	GH	Children – Gigantism Adults- Acromegaly
Thyrotroph adenoma	TSH	Hyperthyroidism (High TSH levels)
Corticotroph adenoma	ACTH, POMC derived peptide (MSH)	Cushing syndrome (Cushing disease)
Gonadotroph adenoma	FSH, LH	Hypogonadism, mass effects

Gross

- Micro adenoma- $< 1\text{cm}$
- Macro adenoma- Up to several cm
- Soft, spherical, encapsulated



CRANIOPHARYNGIOMA

- Benign neoplasm arising from remnants of Rathke pouch
- Children & young adults
- Resembles ameloblastoma microscopically
- Local invasion & compression

Anterior Pituitary - Hyperfunction

➤ 3 Syndromes

1. Gigantism & Acromegaly –
 - Somatotroph Adenoma





2. Hyperprolactinemia – Prolactinoma

Females

- Amenorrhea – Galactorrhea Syndrome
- Infertility

Males

- Loss of libido

3. Cushing Syndrome – Corticotroph adenoma (Cushing disease)

Anterior Pituitary - Hypofunction

- **Hypopituitarism**
- When >75% of anterior pituitary is damaged
- 2 Syndromes

PANHYPOPITUITARISM

PITUITARY DWARFISM

PANHYPOPITUITARISM

- **SHEEHAN Syndrome –**
Post partum pituitary necrosis
 - ❖ Failure of lactation
- Pituitary apoplexy –
 - sudden hemorrhage
- Trauma/ surgery

Pituitary Dwarfism

Severe GH Deficiency in children

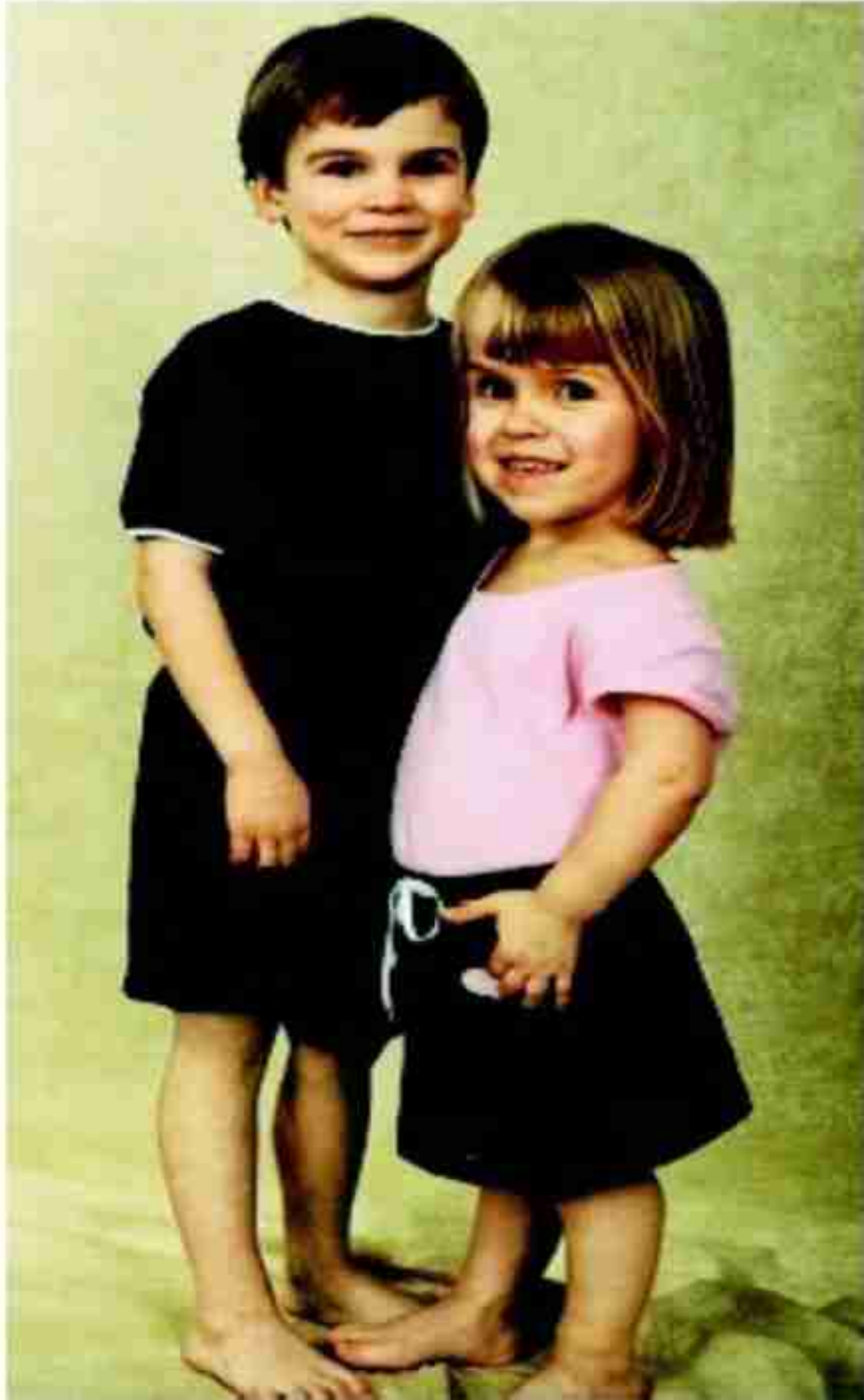
Causes

1. Inherited- AR ; most commonly
2. Pituitary adenoma/ Craniopharyngioma
3. Infarction/Hemorrhage

Clinical features- Pitutary dwarf

- Normal mental state for age
- Proportionate
- Poorly developed genitalia
- Delayed puberty
- Episodes of hypoglycemia





Posterior Pituitary - Hypofunction

DIABETES INSIPIDUS (CENTRAL)

Reduced ADH

Polyuria, Polydipsia

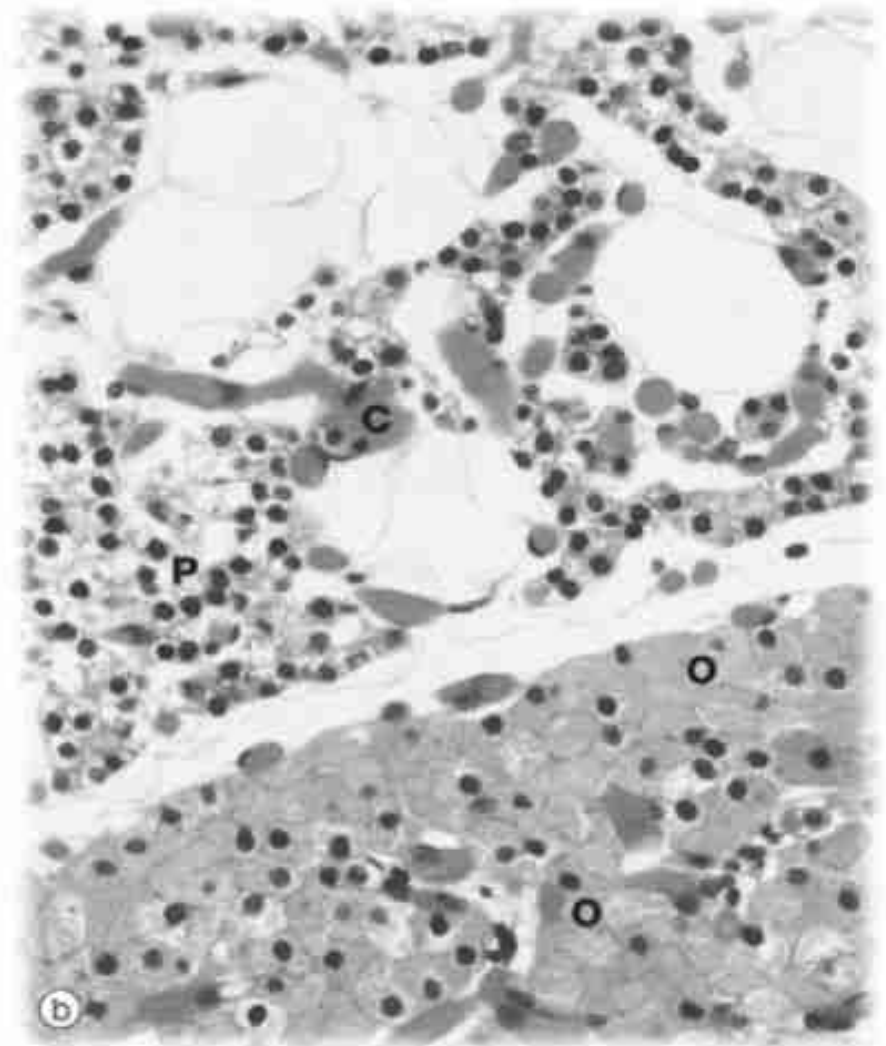
Causes

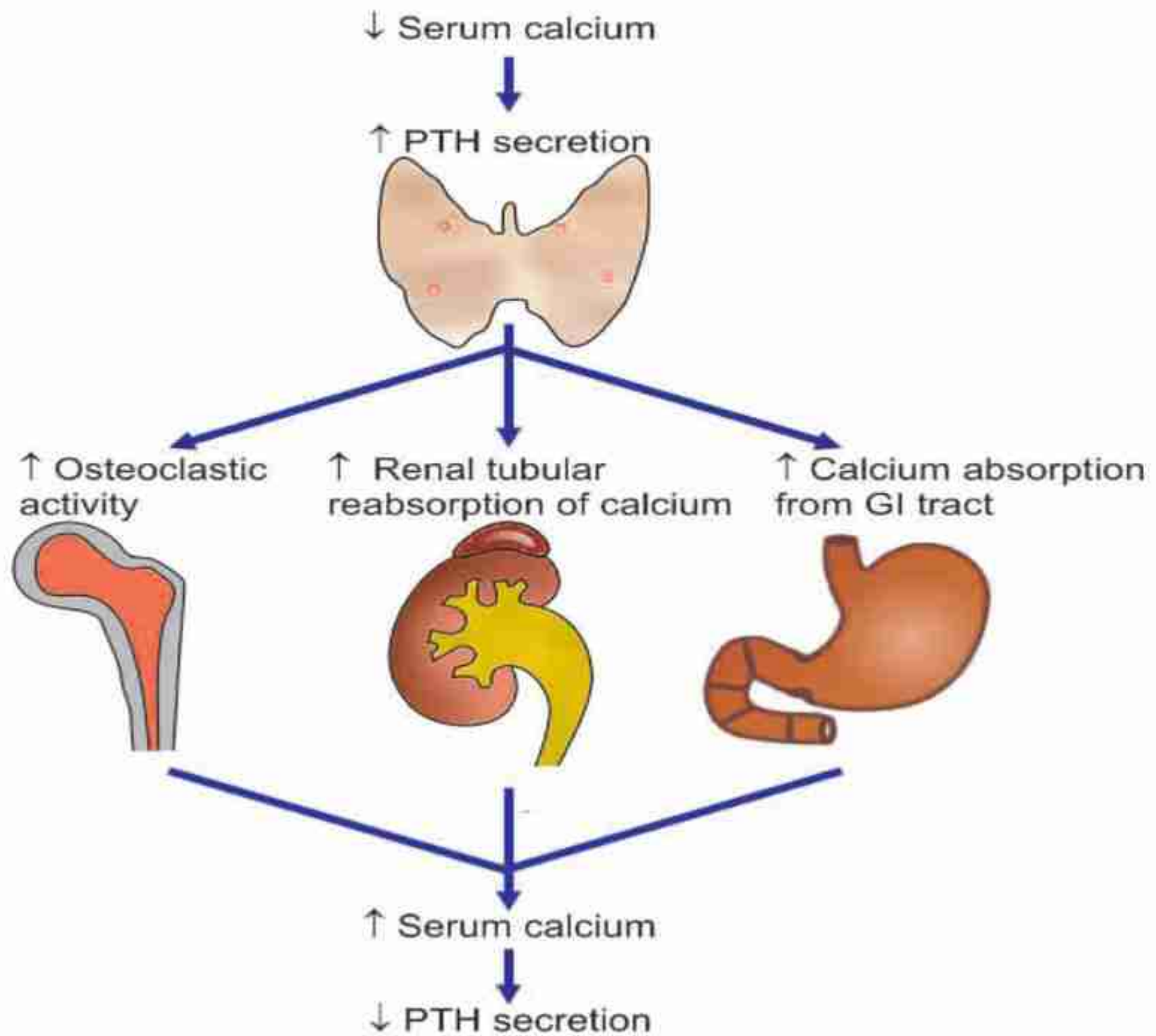
Surgery, Radiation

Head injury, Inflammatory/ neoplastic conditions

PARATHYROID GLANDS

- 2 Superior & 2 Inferior
- Each 35 – 40 mg
- Parenchymal cells + stromal fat
- 3 Cell types
 - Chief cells – PTH
 - Oxyphil cells
 - Water clear cells





PATHOLOGY

Functional

Hyperfunction

Hypofunction

Neoplasms

HYPERPARATHYROIDISM

3 Types

Primary

Secondary

Compensatory increase in PTH release

Tertiary

Complication of secondary

Persistent PTH release inspite of removal of cause

Primary Hyperparathyroidism

Causes

Parathyroid Adenoma – 80%

Primary hyperplasia - 10 -15 %

Parathyroid carcinoma – 5%

Females (3:1)

Association with MEN -1 & MEN 2A

Etiology

Parathyroid

- Adenoma(80%)
- Hyperplasia (15%)
- Carcinoma (2-3%)

Biochemical abnormalities

- i. Elevated levels of parathyroid hormone
- ii. Hypercalcaemia
- iii. Hypophosphataemia
- iv. Hypercalciuria

Clinical features

- **Renal** - Nephrolithiasis (Stones)
- **Bone** - Excessive bone resorption
 - Osteitis fibrosa cystica
 - Brown tumor of hyperparathyroidism



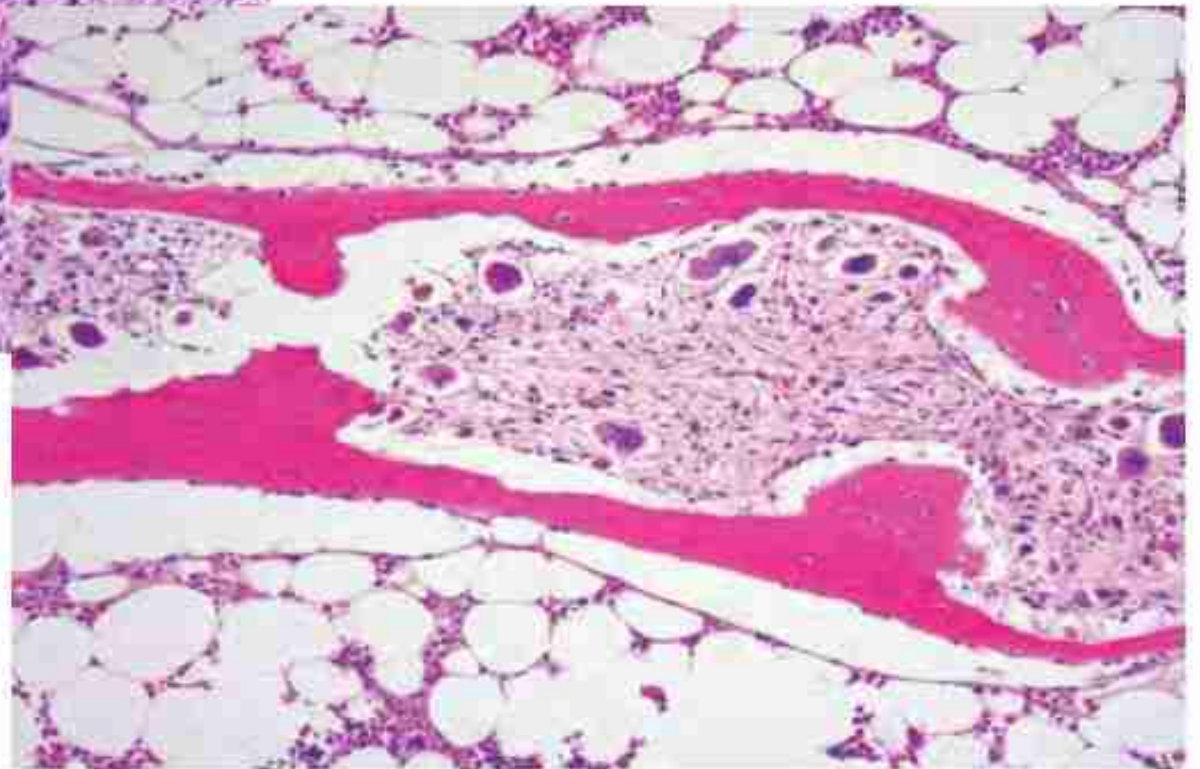
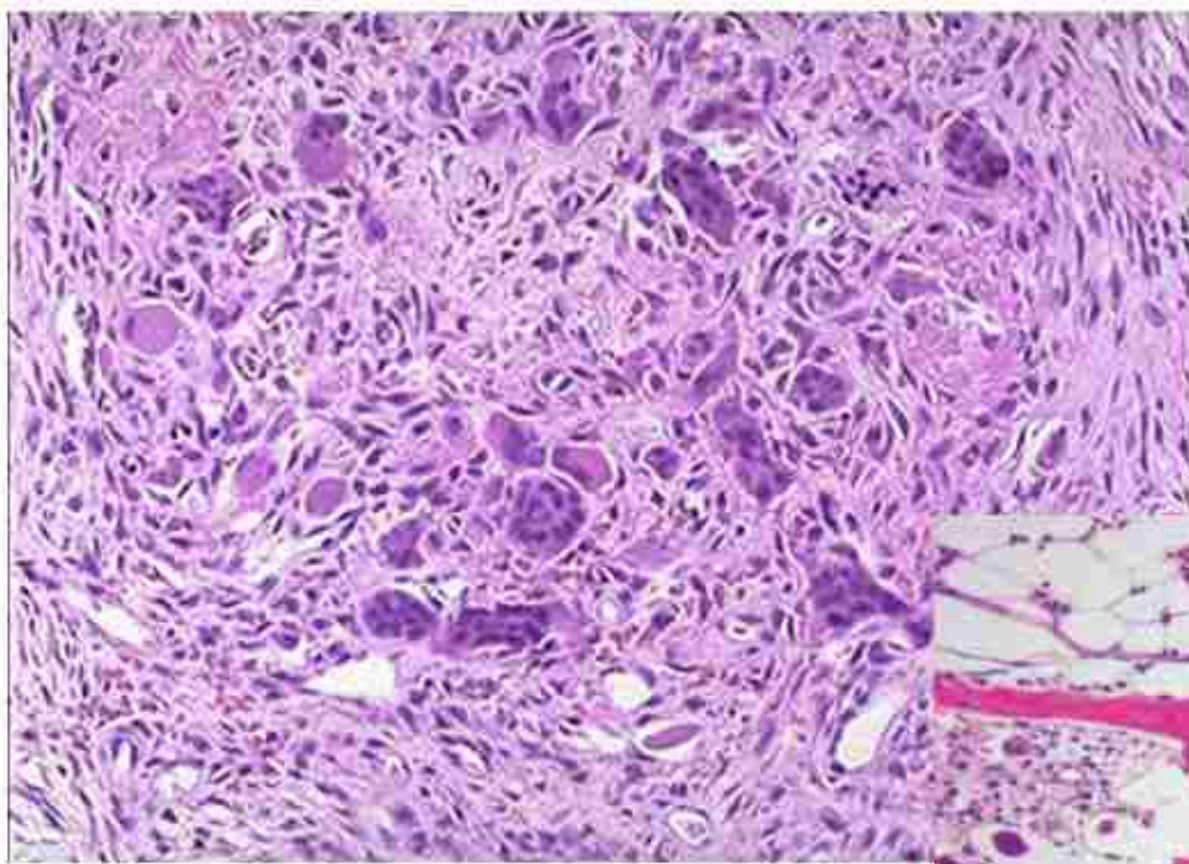
Clinical features

- **Neuropsychiatric** – Depression, lethargy, seizures
- **GIT** – gall stones, peptic ulcers
- **Metastatic calcification**- Stomach, Lung, myocardium, Blood vessels, Eye
- **Cardiac** – HTN, Mitral & aortic valve calcification

Clinical presentation

“ Painful **bones**
Renal **stones**
Abdominal **groans**
Psychic **moans** ”

BROWN TUMOR OF HYPERPARATHYROIDISM



BROWN TUMOR OF HYPERPARATHYROIDISM

SECONDARY HYPERPARATHYROIDISM

- Chronic renal failure - retention of phosphate - $\downarrow$ Ca -
-> $\boxed{\begin{smallmatrix} \uparrow \\ \text{A} \\ \text{AFS} \end{smallmatrix}}$ PTH (Most common)
- Vit. D Deficiency - $\downarrow$ Ca - $\boxed{\begin{smallmatrix} \uparrow \\ \text{A} \\ \text{AFS} \end{smallmatrix}}$ PTH
- Intestinal malabsorption syndromes

TERTIARY HYPERPARATHYROIDISM

- Develops from secondary hyperplasia after removal of the cause of secondary hyperplasia
- Autonomous

HYPOPARATHYROIDISM

- Less common
- Primary –
 - ❖ Surgery
 - ❖ Autoimmune
- Pseudohypoparathyroidism
 - ❖ End organ resistance
 - ❖ Normal PTH

Clinical Features- hypocalcemia

- **Neuromuscular irritability & tetany**
 - Circum-oral numbness & paresthesias
 - Carpopedal spasm
 - Chvostek sign, Trousseau sign
- **Cardiac conduction defects**
- **Ocular cataracts & Dental abnormalities**
- **Intracranial calcifications**

PARATHYROID TUMORS

Rare

- Adenoma

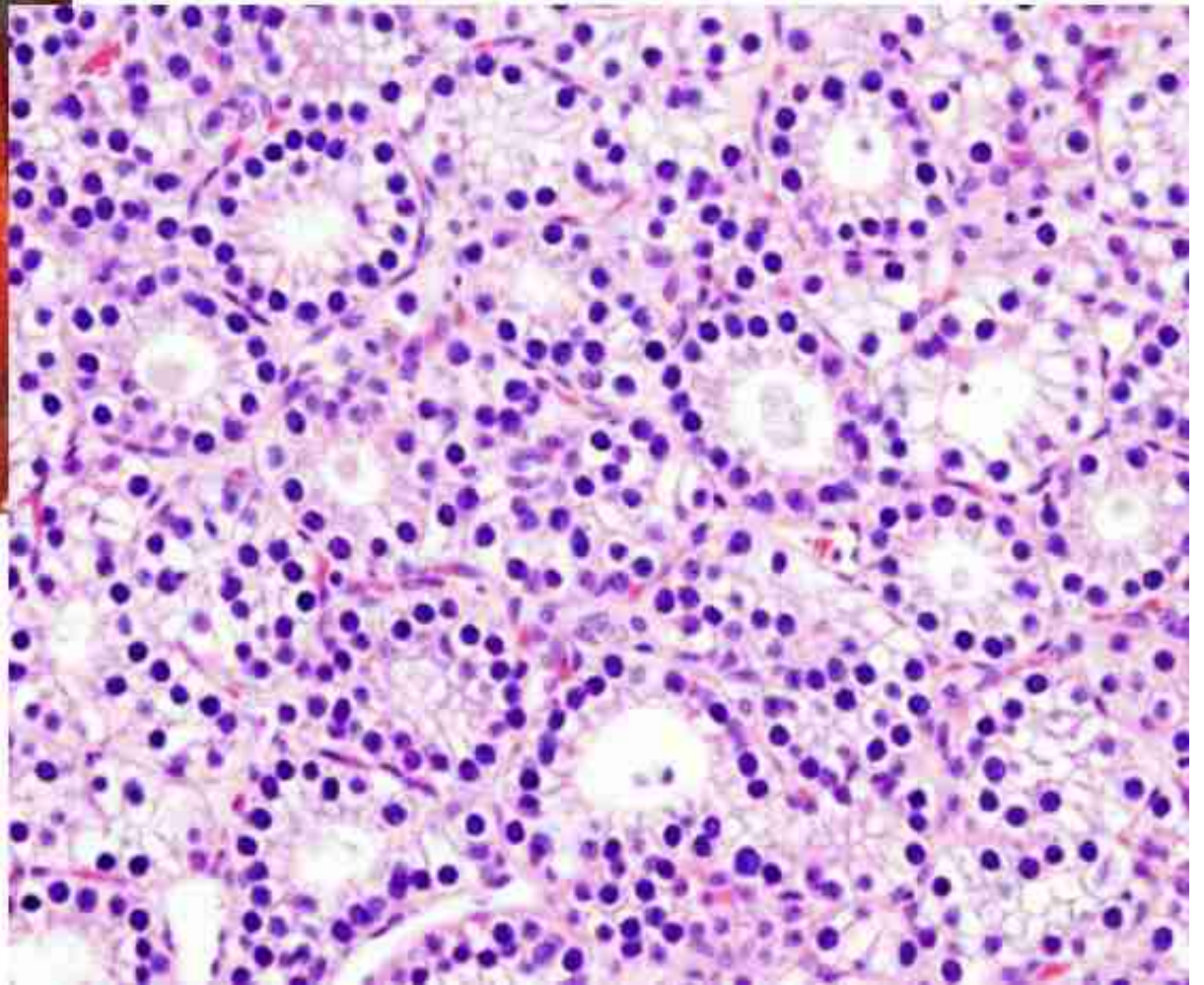
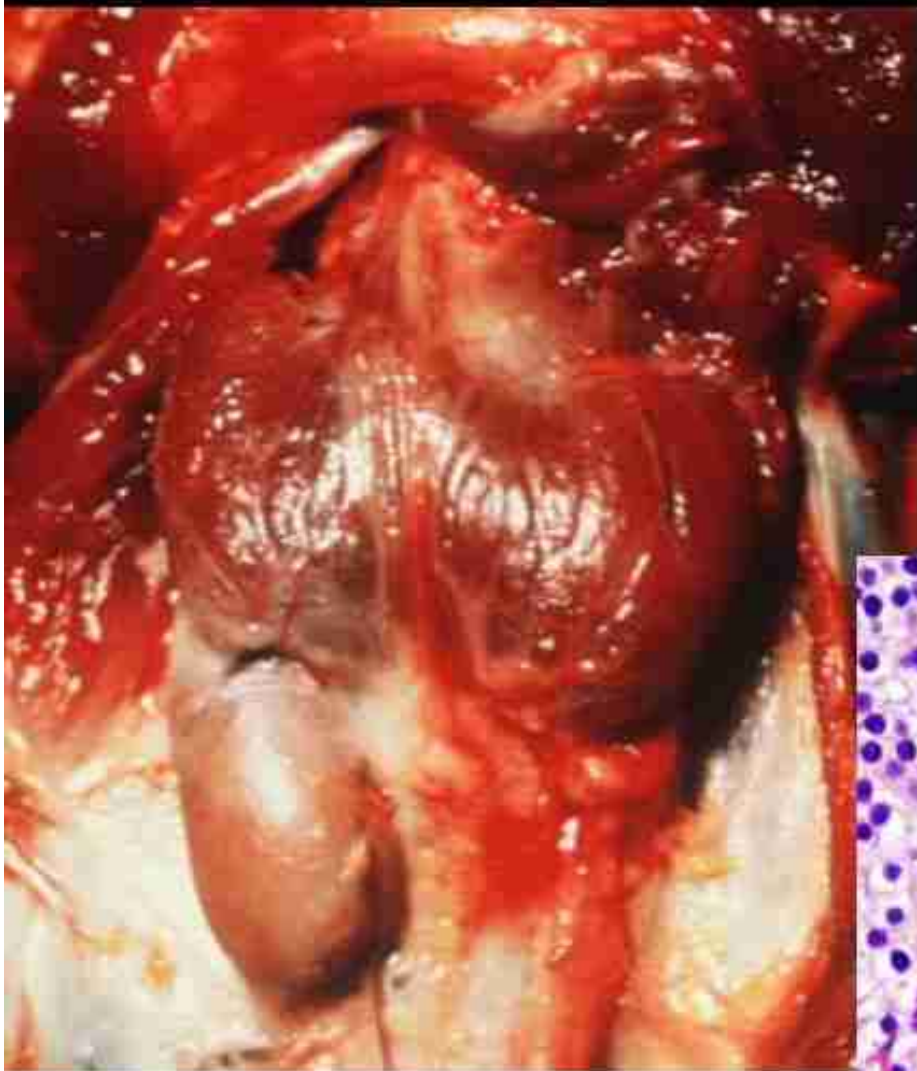
- Carcinoma

Presents with features of hyperparathyroidism

Carcinoma –

- Invasion of adjacent tissues

- Metastasis



MULTIPLE ENDOCRINE NEOPLASIA (MEN) SYNDROME

MEN Syndromes

- Inherited disorders --> Germline mutations
- Proliferative lesions (hyperplasia/ neoplasms) of **multiple** endocrine organs

Cardinal features

- Younger age
- Synchronous/ Metachronous
- Multifocal even in one organ
- Preceded by an asymptomatic stage of hyperplasia
- More aggressive & recur

Classification of MEN syndrome

- **MEN-1** - “menin” tumor suppressor gene
- **MEN-2 –RET proto oncogene**
 - MEN-2A
 - MEN-2B

MEN-1 (Wermer syndrome)

- **Parathyroid- hyperplasia/ adenoma**
- **Pancreas - endocrine tumor** (Zollinger
Ellison syndrome -gastrinoma
Recurrent hypoglycemia - insulinoma)
- **Pituitary - Prolactinoma**
- **Duodenum – gastrinoma**

MEN-2A (Sipple syndrome)

- Medullary Carcinoma thyroid (**100%**)
- Pheochromocytoma

Multifocal, C-Cell hyperplasia

- Parathyroid hyperplasia

MEN-2B

- Medullary Thyroid carcinoma
 - Pheochromocytoma
 - Neuroma - skin, oral mucosa, respiratory & GI tract
 - Marfanoid habitus
-
- NO primary hyperparathyroidism
 - RET mutation with single AA change distinct from MEN-2A

Importance

- More aggressive & recur
- Genetic screening of family members for RET mutations (MEN-2)
- Prophylactic early thyroidectomy

Thank You!

