MEDICINE 435

AATERIOR PITUITARY DISORDERs

OBJECTIVES :

- Earn about all the hormones coming from anterior pituitary.
- Increase on one hormone : the clinical conditions like cushing's, acromegaly, hyperthyroid, hyperprolactinemia and associated symptoms with it.
- And hormonal labs you need for each disorders.

FOR FURTHER EXPLANATION, CORRECTION OR IF YOU WANT THIS LECTUER IN OTHER FORMAT CONTACT US ON
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مصدر مذاكرة

Revised by
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IMPORTANT NOTES EXTRA MNEMONICS EDITING FILE

The first part of the lecture is anatomy and embryo revise your previous info. + the bold are mentioned by the doctor at least don't miss what in bold ^^

Anterior Pituitary Development

is recognizable by **4- 5th wk** of gestation ➔ Full maturation by **20th wk**

1. **From Rathke's pouch, Ectodermal evagination of oropharynx**
2. Migrate to join **neurohypophysis**

Then :

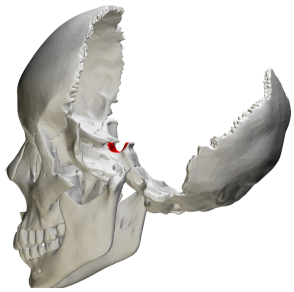
Portion of Rathke's pouch *will give us* : **Intermediate lobe**

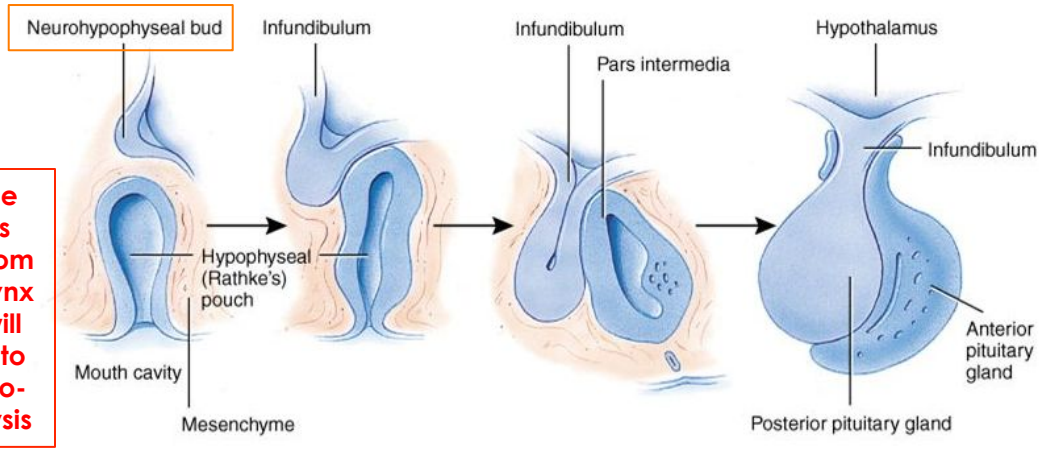
Remnant of Rathke's pouch cell in oral cavity ,
will give us **pharyngeal pituitary**

Lies at the base of the skull as **sella turcica**

Roof is formed by **diaphragma sellae**

Floor by the roof of **sphenoid sinus**





Here the rathke's pouch from oropharynx which will migrate to the neuro-hypophysis

This pic extra !

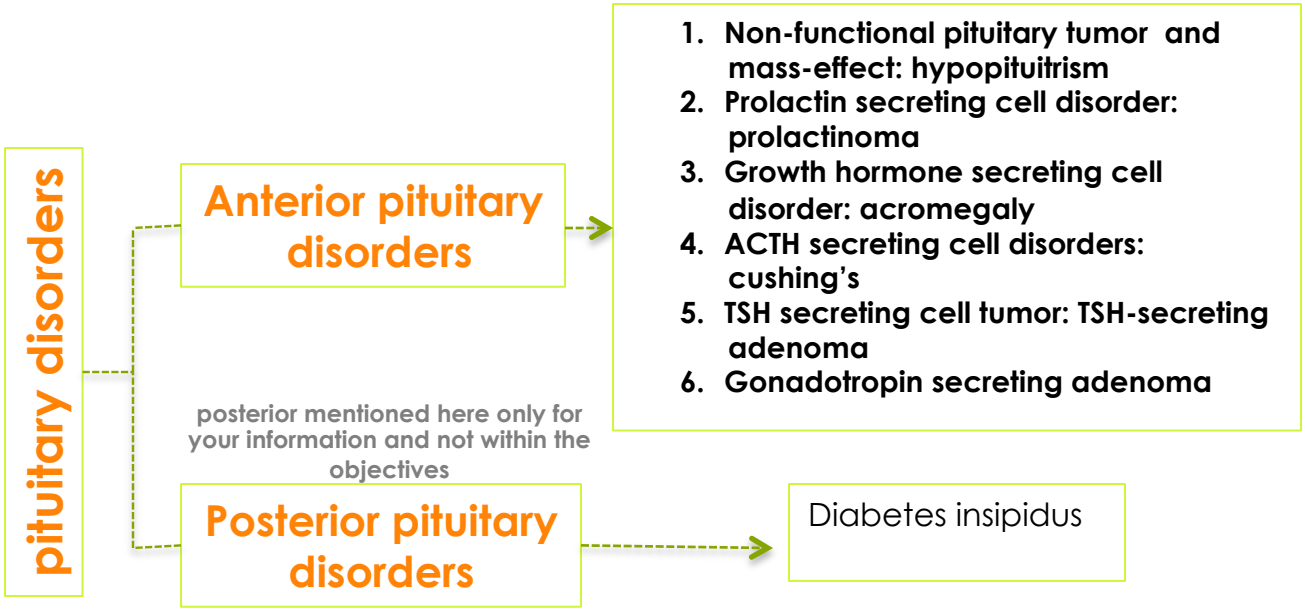
Anterior lobe Function

① Growth Hormone **(GH)**
② prolactin **(PRL)**
③ Thyroid Stimulating Hormone **(TSH)**
④ Luteinizing hormone **(LH)**
⑤ Follicle Stimulating Hormone **(FSH)**.
⑥ adrenocorticotrophic hormone **(ACTH)**

Anterior Pituitary Function cont.

The doctor went through the whole table !

	Corticotroph	Gonadotroph	Thyrotroph	Lactotroph	Somatotroph
Hormone	POMC, ACTH	FSH, LH	TSH	Prolactin	GH
Stimulators	CRH, AVP, gp-130 cytokines	GnRH, Estrogen	TRH	Estrogen, TRH	GHRH, GHS
Inhibitors	Glucocorticoid	Sex steroids, inhibin	T3, T4, Dopamine, Somatostatin, GH	Dopamine Used as a treatment	Somatostatin, IGF-1, Activins
Target Gland	Adrenals	Ovary, Testes	Thyroid	Breast and other tissues	Liver, bone and other tissues
Trophic Effects	Steroid production	Sex Steroid, Follicular growth, Germ Cell maturation	T4 synthesis and secretion	Milk Production	IGF-1 production, Growth induction, Insulin antagonism



Posterior lobe Function

posterior mentioned here only for your information and not within the objectives

① Oxytocin

② VASOPRESSION (ADH) = ↑ secretion ➔ SIADH + ↓ secretion ➔ Diabetes insipidus

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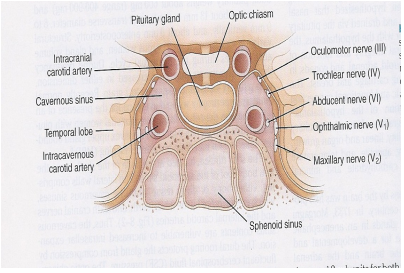
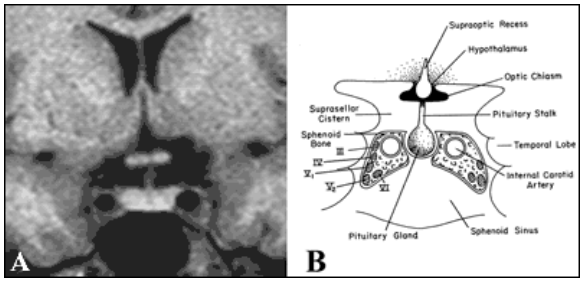
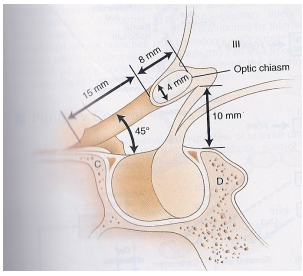
Relations of pituitary gland

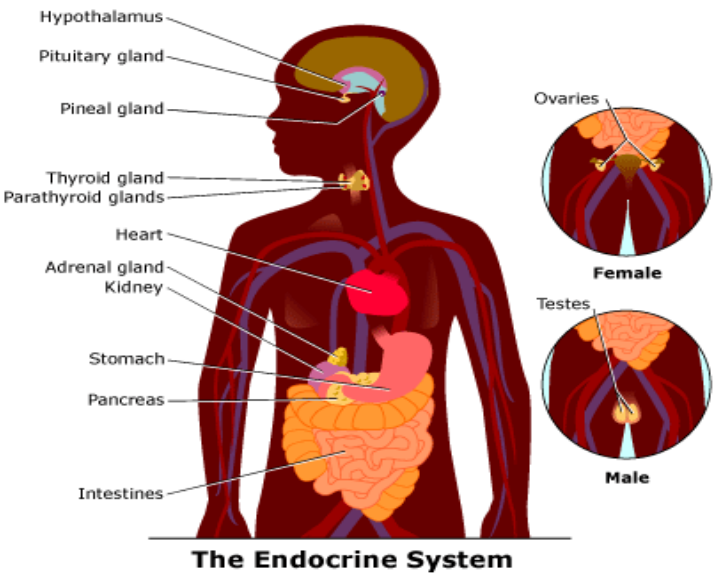
- ★ Pituitary stalk and its blood vessels pass through the diaphragm.
- ★ Lateral wall by cavernous sinus containing III, IV, VI, V1, V2 cranial nerves and internal carotid artery with sympathetic fibers.
- ★ Both adjacent to temporal lobes.
- ★ Pituitary gland measures 15 X 10 X 6 mm, weighs 500 mg but about 1 g in women.
- ★ Optic chiasm lies 10 mm above the gland and anterior to the stalk.
- ★ Blood supply : *superior, middle, inferior hypophysial arteries (internal carotid artery)* running in median eminence from hypothalamus.
- ★ Venous drainage: to *superior and inferior petrosal sinsuses to jugular vein*







Endocrine system



Pathological scenarios (in case of pituitary adenoma):

- If the tumor grows in an up direction; it will compress the optic chiasm and causes bi-temporal hemianopia.
- - If the tumor grows down, it will destroy the sphenoidal bone, and CSF will be released from the nose, because the tumor destroyed the diaphragma sellea, so no protection.
- - If the tumor grows laterally, it will affect the cranial nerves that pass through cavernous sinus (3,4,5 and 6).

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★ Disorders of Pituitary Function ★

① **Hypopituitarism**
- ★ **Central hypoadrenalism, hypogonadism, hypothyroidism or GH deficiency.**
- ★ **Panhypopituitarism.**

If all of them got affected

② **Hypersecretion of Pituitary Hormones**
- ★ **Hyperprolactinemia**
- ★ **Acromegaly** increase in GH
- ★ **Cushing's Disease** increase in ACTH

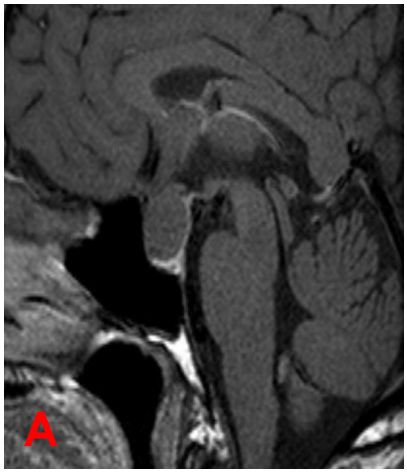
★ Etiology of Pituitary Masses ★

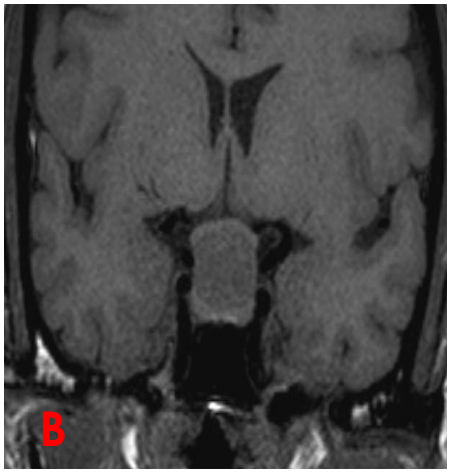
Etiology of Pituitary-Hypothalamic Lesions

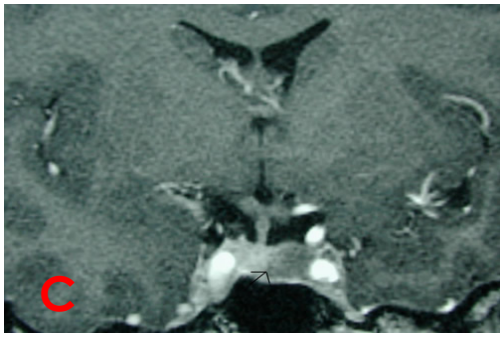
1. Non-Functioning Pituitary Adenomas
2. Endocrine active pituitary adenomas
   - Prolactinoma
   - Somatotropinoma
   - Corticotropinoma
   - Thyrotropinoma
   - Other mixed endocrine active adenomas
3. Malignant pituitary tumors: Functional and non-functional pituitary carcinoma
4. Metastases in the pituitary (breast, lung, stomach, kidney)
5. Pituitary cysts: Rathke's cleft cyst, Mucocoeles, Others
6. Empty sella syndrome
7. Pituitary abscess
8. Lymphocytic hypophysitis -Anti-bodies attacking lymphocytes
9. Carotid aneursym

Adenomas are the most important ones!

-A&B are showing a very big adenoma that compresses the optic chiasm.
-C is not showing an adenoma, but it shows something else.







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★ Evaluation of Pituitary Masses ★

Normally, it's a small adenoma that will cause NO harm!
الدكتورة قالت إن لو فحصتنا راح تلاقي كم بنت عندها!

★ Pituitary adenoma: 10 % of all pituitary lesions
★ Genetic-related
★ MEN-1, Gs-alpha mutation, PTTG gene, FGF receptor-4
★ Pituitary incidentaloma: 1.5 -31% in autopsy (prevalence)
★ 10 % by MRI most of them < 1 cm

The doctor skipped this table .. Its up to you either to go through it or not ^^

Table 1. Functioning Adenomas: Clinical Disease and Medical Therapy

Clinical disease	Hormone produced by tumor	Estimated frequency (%)	Medical therapy
Acromegaly	Growth hormone	5–10	Somatostatin analog (octreotide) Growth hormone receptor blocker
Cushing's disease	ACTH	10–15	Ketoconazole (blocks cortisol synthesis)
Gonadotroph	FSH, LH	5	None
Prolactinoma	Prolactin	20–30	Dopamine agonist (bromocriptine, cabergoline, pergolide)
Null cell	None	20–25	None
Thyrotropic	TSH	<3	Somatostatin analog (octreotide) Propylthiouracil
Other (including mixed cell adenomas)	None	20	None

ACTH = adrenocorticotropic hormone, FHS = follicle-stimulating hormone, LH = luteinizing hormone, TSH = thyroid-stimulating hormone.

First\Non-Functional pituitary lesion (adenoma) NFPA:

★ **Absence of signs and symptoms of hormonal hypersecretion.**
★ **25 % of pituitary tumor.**
★ **Needs evaluation either micro or macroadenoma.**
★ **Average age 50 – 55 yrs. old, more in male**

Presentation of NFPA:

① **As incidentaloma by imaging.**
② **Symptoms of mass effects (mechanical pressure).** –on optic chiasm
③ **Hypopituitarism (mechanism).**
④ **Gonadal hypersecretion.**

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Treatment:

Surgery if indicated If it's hypo

- ✓ recurrence rate 17 % if gross removal, 40 % with residual tumor
- ✓ predictors of recurrence: young male, cavernous sinus invasion, extent of suprasellar extention of residual tumor, duration of follow up, marker; Ki-67

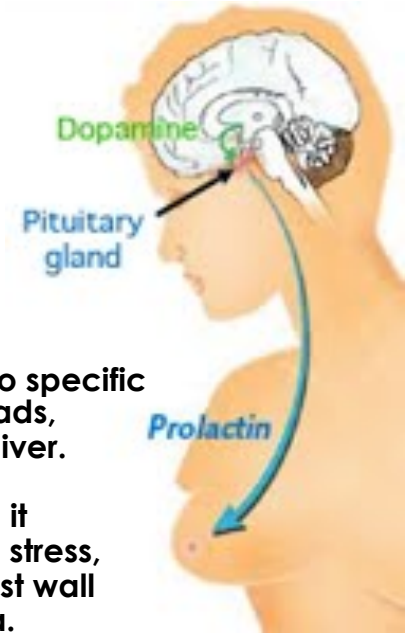
Observation

with annual follow up for 5 years and then as needed, visual field exam Q 6-12 month if close to optic chiasm. Slow growing tumour

Adjunctive therapy:

- ✓ **Radiation therapy**
- ✓ **Dopamine agonist**
- ✓ **Somatostatin analogue**

Second\Functional pituitary lesion FPA:



Prolactin:

Prolactin is the only pituitary hormone that is inhibited by hypothalamus.

- ★ Human prolactin is a 198 amino acid polypeptide.
- ★ **Primary function is to enhance breast development during pregnancy and to induce lactation.**
- ★ **Prolactin also binds to specific receptors in the gonads, lymphoid cells, and liver.**
- ★ **Secretion is pulsatile; it increases with sleep, stress, pregnancy, and chest wall stimulation or trauma.**

Secretion of prolactin is under tonic inhibitory control by **dopamine**, which acts via D2-type receptors located on lactotrophs.

Prolactin production can be stimulated by the hypothalamic peptides, thyrotropin-releasing hormone (TRH) and vasoactive intestinal peptide (VIP)

Other causes for prolactin secretions:
Lactation and chest or breast surgery.

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Causes of Hyperprolactinemia :

Hypothalamic Dopamine Deficiency

- ★ Diseases of the hypothalamus(including tumors, arterio-venous malformations, and inflammatory processes
- ★ Drugs (e.g. alpha-methyldopa and reserpine)

Defective Transport Mechanisms

- ★ Section of the pituitary stalk
- ★ Pituitary or stalk tumors

Lactotroph Insensitivity to Dopamine

- ★ Dopamine-receptor-blocking agents: phenothiazines (e.g. chlorpromazine), butyrophenones (haloperidol), and benzamides (metoclopramide, sulpiride, and domperidone)

Stimulation of Lactotrophs

- ★ Hypothyroidism- increased TRH production (acts as a PRF)
- ★ Estrogens: stimulate lactotrophs
- ★ Injury to the chest wall: abnormal stimulation of the reflex associated with the rise in prolactin that is seen normally in lactating women during suckling

Other causes for hyper-prolactinemia:
-Sleep disturbances.
-Hypothyroidism, because it will increase t3 and t4 secretions, thus it will increase prolactin.

Work up of Patient with Hyperprolactinemia :

- ★ **In females, pregnancy must always be ruled out**
- ★ **Get a TSH- hypothyroidism is another common cause of elevated prolactin:**
- ★ **Obtain detailed drug history- rule out medication effects**
- ★ **Rule out other common causes including:**
  - ✓ **Nonfasting sample**
  - ✓ **Nipple stimulation or sex**
  - ✓ **Excessive exercise**
  - ✓ **History of chest wall surgery or trauma**
  - ✓ **Renal failure**
  - ✓ **Cirrhosis**
- ★ **If no cause determined or tumor suspected, consider MRI, especially if high prolactin levels (> 100 ng/mL)**

-Patient must be fasting.
- **The 3 most common causes you first think of are:**
  - **-Lactation**
  - **-Pregnancy**
  - **-Hypothyroidism**

REMEMBER
Not all hyperprolactinemia is due to a prolactinoma !

Prolactinomas :

- ★ Most common of functional pituitary adenomas
- ★ 25-30% of all pituitary adenomas
- ★ Some growth hormone (GH)–producing tumors also co-secrete PRL
- ★ Of women with prolactinomas- 90% present with microprolactinomas
- ★ Of men with prolactinomas- up to 60% present with macroprolactinomas

- Increased prolactin will decrease estrogen and progesterone in women, and testosterone in males.

Clinical Features of Hyperprolactinemia/Prolactinoma :

① **Women** may present with oligomenorrhea, amenorrhea, galactorrhea or infertility.

① **Men** often have less symptoms than women (sexual dysfunction, visual problems, or headache) and are diagnosed later.

③ **In both sexes**, tumor mass effects may cause visual-field defects or headache. Because it compresses the dura matter, or the temporal lobe.

Management of Prolactinomas :

- ★ Medical therapy: Dopamine agonist First in line! It will supress prolactin and shrinks the tumor
- ★ Surgical resection If it's big in size
- ★ Radiation therapy 3rd line

Growth hormone (GH) :

① Pituitary tumor as mass effect ➔Growth hormone deficiency

② Hyperfunctioning mass ➔ Acromegaly

① Growth hormone deficiency

Diagnosed in children and adult

NOTE: ↓ GH in children → dwarfism. On other hand, ↓ GH in adults → The growth is not effected. Instead, there're metabolic effects. For example, no breakdown of fat → Obesity, particularly truncal obesity (أي Central obesity = في البطن)
NOTE: ↓ GH usually die from CVS complications

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Diagnosis of GH-deficiency :

Diagnosis here is not very important, just know that GH is not reliable because it is pulsatile (not constant through out the day. Hence, if we measure it in the morning it will be different from that of the night & vice versa). So, what can we measure instead? IGF-1; because it is stable during the day.

- ✓ **GH, IGF-I level**
- ✓ Dynamic testing: clonidine stimulation test, glucagon stimulation, exercise testing, arginine-GHRH, insulin tolerance testing
- ✓ X-ray of hands: delayed bone age
- ✓ In Adult: Insulin tolerance testing, MRI pituitary to rule out pituitary adenoma

management :
GH replacement

In adults: it is not necessarily being given, unless they have complications of its deficiency, such as being depressed, socially isolated,…etc (simply when they suffer from the metabolic effects of its deficiency).
On the other hand in children: GH replacement is highly neede

② Acromegaly

Clinical picture and presentation (all of them are important).

- ✓ GH level (not-reliable, pulsatile)

GH level is not the choice since its level varies during the day.

- ✓ IGF-I > **IGF-1 is the choice, because it is constant during the day.** أهم واحد
- ✓ 75 g OGTT tolerance test for GH suppression **we might do it as confirmational test, but not very imp**
- ✓ Fasting and random blood sugar, HbA1c
- ✓ Lipid profile
- ✓ Cardiac disease is a major cause of morbidity and mortality
- ✓ 50 % died before age of 50
- ✓ HTN in 40%
- ✓ LVH in 50%
- ✓ Diastolic dysfunction as an early sign of cardiomyopathy

To diagnose acromegaly, we can also give him sugar. Normal GH is suppressed by hyperglycaemia, if there is exssive GH it will not be suppressed and its levels will remain high. Keep in mind that hyperglycaemia will suppress both cortisol + GH.

management :

Medical treatment : Somatostatin analogue
Surgical resection of the tumor

Any treatment (with exception of prolactinoma) , we should start the surgical treatment as a first-line treatment and medical treatment as a second line treatment.

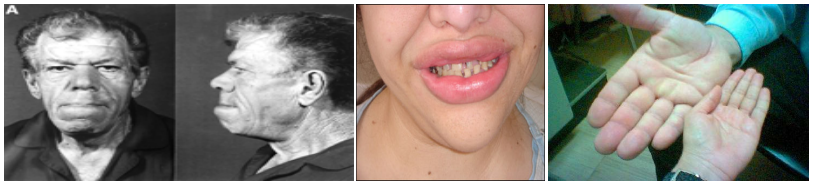

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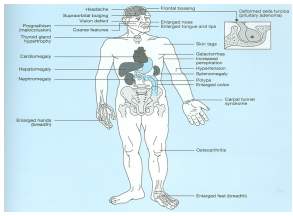
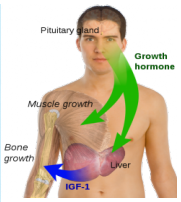
Table 1. Clinical Features of Acromegaly.

Local tumor effects	**Visceromegaly**
Pituitary enlargement	Tongue
Visual-field defects	Thyroid gland
Cranial-nerve palsy	Salivary glands
Headache	Liver
Somatic systems	Spleen
Acral enlargement, including thickness of soft tissue of hands and feet	Kidney
Musculoskeletal system	Prostate
Gigantism	**Endocrine and metabolic systems**
Prognathism	Reproduction
Jaw malocclusion	Menstrual abnormalities
Arthralgias and arthritis	Galactorrhea
Carpal tunnel syndrome	Decreased libido, impotence, low levels of sex hormone–binding globulin
Acroparesthesia	Multiple endocrine neoplasia type 1
Proximal myopathy	Hyperparathyroidism
Hypertrophy of frontal bones	Pancreatic islet-cell tumors
Skin and gastrointestinal system	Carbohydrate
Hyperhidrosis	Impaired glucose tolerance
Oily texture	Insulin resistance and hyperinsulinemia
Skin tags	Diabetes mellitus
Colon polyps	Lipid
Cardiovascular system	Hypertriglyceridemia
Left ventricular hypertrophy	Mineral
Asymmetric septal hypertrophy	Hypercalciuria, increased levels of 25-hydroxyvitamin D_3
Cardiomyopathy	Urinary hydroxyproline
Hypertension	Electrolyte
Congestive heart failure	Low renin levels
Pulmonary system	Increased aldosterone levels
Sleep disturbances	Thyroid
Sleep apnea (central and obstructive)	Low thyroxine-binding–globulin levels
Narcolepsy	Goiter

This schedule was mentioned by the doctor, she mentioned from it the following as examples:
Big nose, prognathism (low jaw prominence), large lips, spaces between teeth due to the growth on mandible, hand will be enlarged because both bones and soft tissue will be enlarged (change the size of his shoes, gloves, rings as a consequence), Osteoarthritis (cartilage that seprated bones from each other to reduce friction during movement will be enlarged → less friction is provided) prominent preorbital ridge, hyperlipidemia, hypertrophied heart (Hence, heart failure is a risk factor to die)
In both ↑ or ↓ GH, heart failure is a major risk of die.
If ↑ GH → all the body will be enlarged, and there is organomegaly (splenomegaly, hepatomegaly,…etc).
If ↓ GH → only central (in the trunk) obesity في البطن.



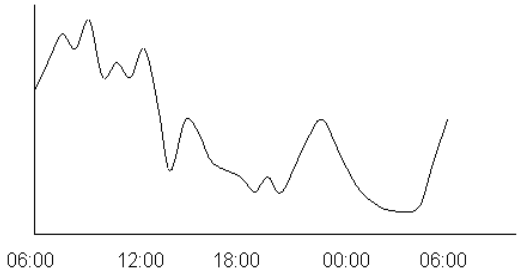


Note that IGF-1 control the growth of bone, muscles, soft tissue.

ACTH (adrenocorticotrophic hormone) :

HPA-axis :

- ★ 2nd adrenal insufficiency
- ★ glucgocorticoid replacement
- ★ Circadian rhythm of cortisol secretion
- ★ Early morning cortisol between 8-9 am



الكورتيزول يرتفع في الصباح وينخفض في الليل، الصباح 500 والليل تقريبًا 200-300، لو كان بالليل 500 هذا غير طبيعي.
What is the function of cortisol? One of the stimulus of its release is hypoglycemia to protect you from coma. It will vasoconstrict your blood vessels to prevent shock by maintaining BP. Another stimulus for its release is physiological stress, for example during illness, high levels of cortisol is normal. Also, if you have hypotension it will be realse to vasconstrict blood vessels and ↑ BP. If we don't have cortisol we will die if we have and stress due to illness.

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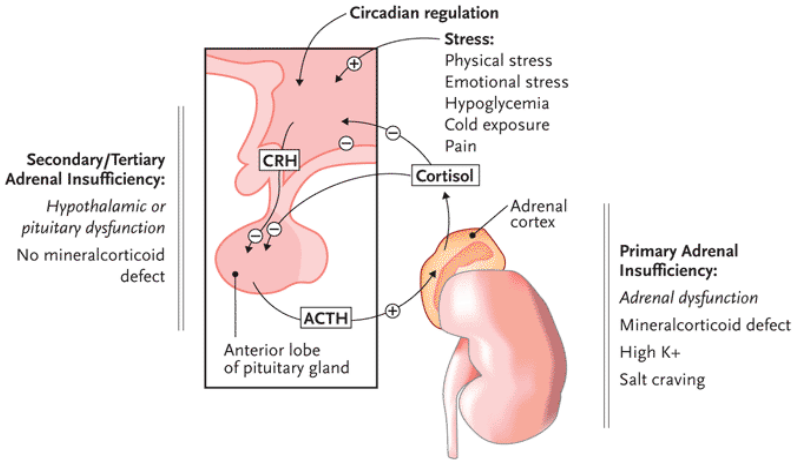
ACTH-disorders :

① Hypoadrenalism

- ✓ Nausea
- ✓ Vomiting
- ✓ Abdominal pain
- ✓ Diarrhoea
- ✓ Muscle ache
- ✓ Dizziness and Weakness
- ✓ Tiredness
- ✓ Weight loss
- ✓ Hypotension



Management of hypoadrenalism :
Cortisol replacement.
BUT you have to double the dose during illness.

- **Check this picture, because you have to differentiate between primary and secondary hypoadrenalism.**
  - **Primary: addison's disese.**
  - **Secondary: pituitary problems.**

If it was due to pituitary problem, call it secondary hypodrenalism insuffeccincy but don't call it addison, because addison is problem in adrenal gland (primary).

② ACTH-Adenoma

ACTH-Adenoma may be clinically silent or may cause hyper cortisolism , manifested clinically as Cushing syndrome, because of the stimulatory effect of ACTH on the adrenal cortex

(↑ cortisol→ acne, haristum, abnormal stria, Cervical back fat bag)+ Thin extremities but obesity is seen in the centrally (in the trunk, abdomen), skin is thin → skin can be seen easily with bruising), rounded face, gyncomastia in men).

HPA-axis (excessive cortisol)

- ★ 80 % HTN
- ★ LVH
- ★ Diastolic dysfunction, intraventricular septal hypertrophy
- ★ ECG needed: high QRS voltage, inverted T-wave
- ★ Echocardiogram preop
- ★ OSA: 33% mild, 18% severe. Needs respiratory assessment and careful use of sedative during surgery
- ★ Glucose intolerance in 60%, control of hyperglycemia
- ★ Osteoporosis with vertebral fracture➔ positioning of patient in OR (50 %), 20 % with fracture
- ★ thin skin ➔ difficult IV cannulation, poor wound healing

- In chushing's, they usually die from CVS.
- Normally, cortisol levels goes down by night, but in Cushing it will be still high all the day long due to excessive release of it. (Impaired circadian rhythm).
- - Crushing's → ↑ blood glucose levels → ↑ DM.

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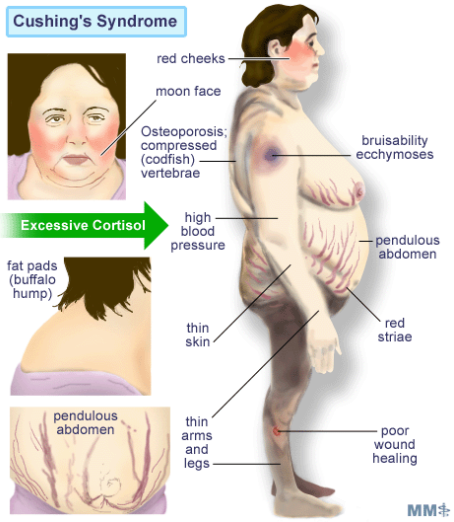
Cushing's-Management

- ✓ Surgical resection of pituitary
- ✓ Medical Treatment

NOTE: Start with surgery as the first line of treatment



TSH (Thyroid Stimulating Hormone) :

① TSH- Hypothyroid

Central Hypothyroidism

- ✓ **Low TSH.**
- ✓ **Low free T4 and T3.**

In hypothyroidism everything is low (metabolism, memory, concentration..)

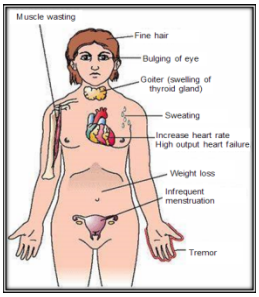
TSH and free T3&T4 are low if it was central (secondary) pituitary or hypothalamus affected like in pituitary adenoma .
However, if it was primary (thyroid gland problems) TSH is going to be high, and T4 and T3 will remain the same (low).

Management of Central Hypothyroidism:

- ✓ Thyroxine replacement.
- ✓ Surgical removal of pituitary adenoma if large.

In hypothyroidism, there's menorrhagia = excessive menstrual bleeding).
In hyperthyroidism: there's oligomenorrhea : less bleeding.

② TSH-hyperthyroid



Central : TSH, T4&T3 all high > problem in the pituitary
Primary : TSH low , T4&T3 high > problem in the thyroid gland

Management same as hypothyroid + anti-thyroid drug to control T3&T4

③ TSH-Producing adenoma

- ★ Very rare < 2.8 %
- ★ Signs of hyperthyroidism
- ★ High TSH, FT4, FT3

Treatment preop with anti-thyroid meds
Surgical resection of adenoma
Medical therapy: Somatostatin Analogue

(っ´ω`)/(╥ω╥) one more page, you can do it !

LH (Luteinizing hormone) &FSH (Follicle Stimulating Hormone) :

Gonadotroph Adenoma Very rare

Laboratory finding :

- ✓ High FSH and Low LH ;
- ✓ High serum free alpha subunit .
- ✓ High estradiol , thickened endometrium and polycystic ovaries .

Usually hypogondasim, pregnancy issues, infertility.

Management of Gonadotroph Adenoma:

- ✓ Surgical resection if large
- ✓ Radiation therapy

IMPORTANT NOTE: only hyperprolactinoma medical treatment is first line of treatement, the rest surgical treatment is the first line of treament.

☆ Assessment Of Pituitary Function ☆

Thanks to 434 team ^^

Clinical

Take the history: Galactorrhea ? Loss of libido ? Visual disturbance ?
Cardiac and respiratory assessment

Biochemical

TSH, FT4, FT3, LH, FSH,
Prolactin, GH, IGF- I,Testosterone, Estradiol

Anatomical

Pituitary MRI

THE END

MCQs

1) The best measurement to diagnose GH-deficiency is ;

A) GH level
B) IGF-1 level
C) TSH
D) LF T

2) Pituitary tumor as mass effect lead to ;

A) Acromegaly
B) Criticism
C) Growth hormone deficiency
D) Cushing

3) The first line to manage Prolactinomas is:

A) Surgical resection
B) Radiation
C) Medical therapy

4) All hyperprolactinemial is due to a prolactinoma ;

A) True
B) False

5) Which nerve will be compressed 1st when a pituitary gland enlarges?

A) Opthalmic
B) Oculomotor
C) Maxillary
D) Abducent

6) What is the complication of SIADH?

A) Oedema formation
B) Dehydration
C) Hypertension
D) Excess urine formation

7) What is the best description of a pituitary tumour?

A) Enlargement of pituitar
B) Neoplasm located in the sella turcica
C) Malignancy of the pituitary gland

8) When during the day ACTH & Cortisol are high in amount?

A) After waking up
B) Last few hours before waking up
C) During sleep
D) Before sleeping

9) Which organ is dysfunctional if the endocrine disorder is a secondary disease?

A) Adrenal cortex
B) Pancreas
C) Hypothalamus
D) Pituitary gland

* Primary endocrine disease inhibits the action of downstream glands/ target endocrine glands.
* Secondary endocrine disease is indicative of a problem with the pituitary gland.
*Tertiary endocrine disease is associated with dysfunction of the hypothalamus and its releasing hormones.

10) In pituitary tumour, the pituitary stalk will be compressed. Which of the following is the side effect of the compressed pituitary stalk

A) Headache
B) Vomiting
C) Prolactinemia
D) Increased ADH secretion

1)A, 2)C, 3)C, 4)B, 5)B, 6)Both A&C, 7)B, 8)B, 9)D, 10)C

SAQs

❖ **To diagnose hyperprolatinemia first we should exclude..?**

-Lactation
-Pregnancy
-Hypothyroidism

❖ **An insulin tolerance test is done to evaluate GH hyposecretion. Assuming that the patient has enough GH reserves, what happens when an insulin is injected to a patient with GH deficiency?**

GH serum level rises

❖ **In hypothyroidism, the cause can be evaluated by simply giving a TRH injection. If TRH is given, and after that TSH serum level is discovered to be normal, where is the defect that is causing the disease?**

Hypothalamus

IF given TRH injection, TSH normal: Hypothalamic defect. If TSH remain low: Pituitary defect.

❖ **Which hormone has a positive feedback mechanism?**

Oxytocin & LH

❖ **An oral glucose tolerance test is done to evaluate GH hypersecretion. Assuming that this is not a tumour, what happens when an the patient is placed in a hyperglycemic state?**

GH serum level decreases

In hyperglycemic state -> GH is inhibited.

❖ **To rule out tumour, in hyperthyroidism, T3 suppression test is done. Large doses of T3 is given. What will happen to the level of TSH?**

Decreased

T3 suppression test:
Large doses of T3 given – negative feedback to hypothalamus & anterior pituitary – normal: TSH suppressed.
If TSH not suppressed: Tumour (autonomous)

Source : female and male slides

FOR FURTHER EXPLANATION, CORRECTION OR IF YOU WANT THIS LECTUER IN OTHER FORMAT CONTACT US ON
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THANK YOU !

FOR CHECKING OUR WORK ^^

AND

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