

# PITUITARY DISORDERS

435 medicine teamwork

[ **Important** | **Notes** | Extra | **Editing file** ]

## lecture objectives:

- ⇒ Anatomy and physiology related to pituitary gland
- ⇒ Hypofunction of anterior pituitary: ACTH, LH and FSH, TSH, GH deficiency
- ⇒ Hyperfunction of anterior pituitary: ACTH, LH and FSH, TSH , GH oversecretion
- ⇒ Clinical diagnosis of each disorder
- ⇒ Management of each disorder
- ⇒ Posterior pituitary dysfunction: Diabetes insipidus

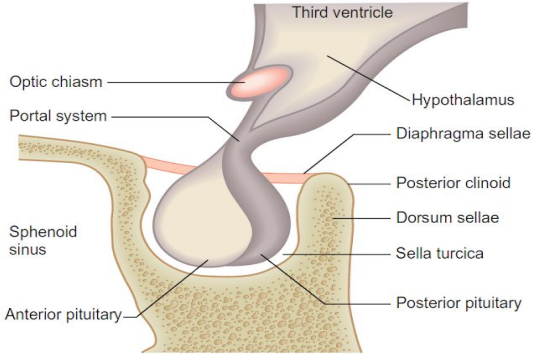
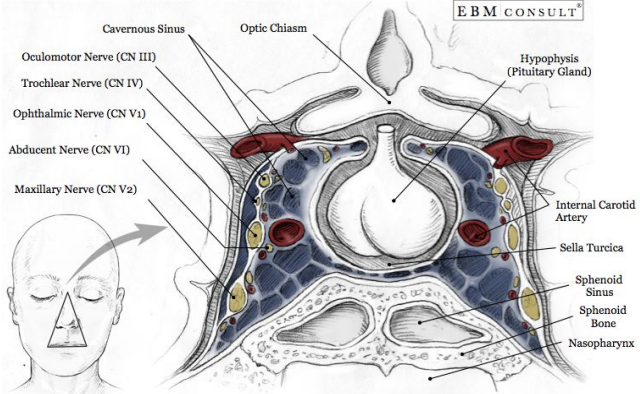
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References:

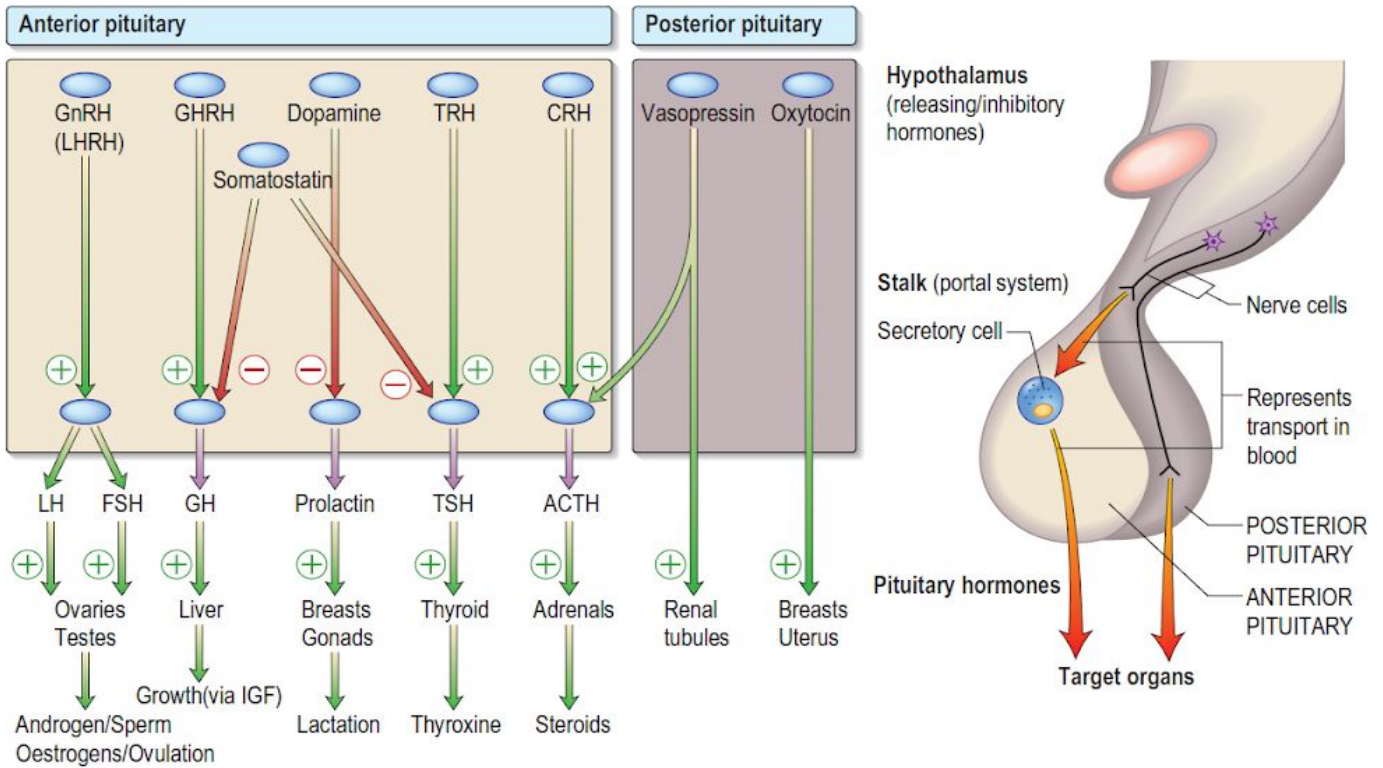
Dr.Aisha & Dr.Ryadh Slides+Davidson+Kumar+Master the bo

# Basic Review of Hypothalamus & Pituitary Gland

it's better to revise the basics from 435 endocrine physiology team :p <3

Anatomy of Pituitary Gland	
<ul style="list-style-type: none"> <li>- The gland is composed of two lobes, anterior and posterior, and is connected to the hypothalamus by the infundibular stalk, which has portal vessels carrying blood from the median eminence of the hypothalamus to the anterior lobe and nerve fibres to the posterior lobe.</li> </ul> <p><b>Anatomical relations to the pituitary:</b></p> <ul style="list-style-type: none"> <li>- The pituitary gland is <u>enclosed</u> in the sella turcica and bridged over by a fold of dura mater called the diaphragma sellae (Which is reflection of dura mater. It prevents the CSF coming from 3rd ventricle to sella turcica)</li> <li>- <u>Inferiorly</u>: sphenoidal air sinuses.</li> <li>- <u>superiorly</u>: optic chiasm.</li> <li>- <u>Laterally</u>: cavernous sinuses which contain the 3rd, 4th and 6th cranial nerves and the internal carotid arteries. (Important to know because if there is anatomical defect it will go to the lateral and effect the structure of cavernous sinus and temporal lobe,if temporal lobe gets affected the pt could present with epilepsy )</li> </ul>	 

Pituitary	Anterior (Adenohypophysis)	Posterior (Neurohypophysis)
Origin	Rathke's pouch (oral cavity)	Down growth of hypothalamic neural tissue
	#Development of pituitary cells is controlled by a set of transcription growth factors like: <b>Pit-1, Prop-1, Pitx2</b> (might be Mcq! If there is a defect in these transcription growth factor it will be in functional level rather than anatomical which is normal It will lead to miss certain cells so patients will present by hormonal deficiency)	
Hormones released	GH , LH, FSH, TSH, ACTH, Prolactin <b>Go Look For The Adenoma Please</b> ▶ <b>Note:</b> A compressive adenoma in pituitary will impair hormone production in this order ;))	Oxytocin, ADH
Hormones synthesis	Hormones are <u>synthesized</u> in anterior pituitary	Synthesized in hypothalamus and <u>stored</u> in posterior pituitary Remember ( <u>storage</u> not synthesis)
arterial supply	Superior hypophyseal(branches of internal carotid artery)	Inferior hypophyseal(branches of internal carotid artery)
venous drainage	(have the same venous drainage)hypophyseal veins → drain into cavernous sinuses	
Hypothalamic control	Hormonal signals (releasing and inhibitory hormones) ▶ <b>Note:</b> all releasing hormones are stimulatory except dopamine inhibits prolactin,so pituitary stalk lesion will cause hyperprolactinemia and decrease to all other ant.pit HORMONES	Neural signals



### More details about anterior pituitary hormones:

Hormone	GH	LH,FSH	PRL	TSH	ACTH
Original Cells	Somatotroph	Gonadotroph	Lactotroph	Thyrotroph	Corticotroph
Stimulators	GRH sleep , stress ,exercise , hypoglycemia(very strong stimulus for GH secretion) clonidine	GnRH	TRH TRH control both TSH and prolactin So in case of hypothyroid the TSH will be high as well as prolactin imp	TRH	CRH Stress
Inhibitors	IGF-1 Somatostatin(GHIH)	Testosterone, estrogen	Dopamine	T3, T4	Steroid
Target Gland	Liver & other tissues	Ovaries, Testes	Breast& other tissues	Thyroid	Adrenals
Trophic Effects	IGF-1 production, Growth induction, Insulin antagonism (GH exerts its activity indirectly through the induction of insulin-like growth factor (IGF-1), which is synthesized in the liver and other tissues, or directly on tissues such as liver, muscle, bone or fat to induce metabolic changes)	<b>FSH :</b> in male:stimulates testicular growth & spermatogenesis in women : it stimulates follicular growth production of estrogen & progesterone .  <b>LH :</b> stimulates ovulation in women and testosterone production in males	Milk Production ,can inhibit GnRH	T4 synthesis and secretion	Steroid production Androgen

# Pituitary Disorders

## Classification of diseases of the pituitary:

	Anterior pituitary	hypothalamus & posterior pituitary
<b>Masses</b> Any tumour could be either:	<b>Non-Functioning tumours:</b> هي نفسها ماتقرز هورمونز لكن ممكن اذا حجمها كبير تضغط عالتراكيب التي حولها فتطلع اعراض حسب الجزء المنضغط Pituitary Adenomas __ Pituitary incidentaloma __ Craniopharyngioma __ Metastatic tumours	
	<b>cause Hormone Excess:</b> Functioning Pituitary Adenomas: <ul style="list-style-type: none"> <li>- PRL secreting:Prolactinoma</li> <li>- GH secreting adenoma:Acromegaly</li> <li>- ACTH secreting adenoma:Cushing’s disease</li> <li>- Rare TSH-, LH-and FSH-secreting adenomas</li> </ul>	<b>cause Hormone Excess:</b> Syndrome of inappropriate antidiuretic hormone (SIADH)
	<b>cause <u>inadequate</u> hormone production:</b> hypopituitarism	<b><u>inadequate</u> hormone production:</b> Cranial diabetes insipidus

## Principles of endocrine investigation:

Timing of measurement
<ul style="list-style-type: none"> <li>- Release of many hormones is rhythmical (pulsatile, circadian or monthly), so random measurement may be invalid and sequential or dynamic tests may be required</li> </ul>
Choice of dynamic biochemical tests
<ul style="list-style-type: none"> <li>- Abnormalities are often characterised by loss of normal regulation of hormone secretion</li> <li>- If hormone deficiency is suspected, choose a stimulation test</li> <li>- If hormone excess is suspected, choose a suppression test</li> <li>- The more tests there are to choose from, the less likely it is that any single test is infallible, so avoid interpreting one result in isolation</li> </ul>
Imaging
<ul style="list-style-type: none"> <li>- Secretory cells also take up substrates, which can be labelled</li> <li>- Most endocrine glands have a high prevalence of ‘incidentalomas’, so do not scan unless the biochemistry confirms endocrine dysfunction or the primary problem is a tumour</li> </ul>
Biopsy
<ul style="list-style-type: none"> <li>- Many endocrine tumours are difficult to classify histologically(e.g. adrenal carcinoma and adenoma)</li> </ul>

#imaging and biopsy, are more frequently reserved for patients who present with a tumour.Surgical biopsy is usually only performed as part of a therapeutic operation

# Pituitary tumours

## Overview:

Pituitary tumours are the most common cause of pituitary disease, and the great majority of these are benign pituitary adenomas, usually monoclonal in origin. Symptoms arise as a result of:

- local effects of a tumour: → nonfunctional pituitary adenoma
- or inadequate hormone production: → hypopituitarism.
- or excess hormone secretion (functional pituitary adenoma): The Types of secretory adenoma are:
  - GH secreting Adenoma → causing acromegaly in adults and gigantism in children
  - Prolactin secreting adenoma → causing galactorrhoea or clinically silent
  - ACTH secreting adenoma → causing Cushing's disease and Nelson's syndrome.
  - Rare TSH-, LH- and FSH-secreting adenomas

### ►NOTE:

- Genetic-related to pituitary adenoma: MEN-1, Gs-alpha mutation, PTTG gene, FGF receptor-4 (might be MCQ!!)
- MRI is superior to CT scanning and will readily show any significant pituitary mass
- The First line treatment of all pituitary tumours is surgery, EXCEPT Prolactinoma medication is the first line (MCQ!!).

## Nonfunctional pituitary Mass:

### General Characteristics:

- Absence of signs and symptoms of hormonal hypersecretion.
- 25 % of pituitary tumor.
- Needs evaluation. Is it Microadenoma or Macroadenoma?
- Average age 50 – 55 yrs, more common in males.

### Presentation:

- NOTE: nonfunctioning adenomas are clinically silent at the stage of microadenomas and only become clinically evident at the stage of macroadenomas
- Asymptomatic, incidentaloma by MRI. Such small lesions are sometimes detected during MRI scanning of the head for other reasons – so-called 'pituitary incidentalomas'
- Macroadenoma could cause Symptoms of mass effects (mechanical pressure, or infiltration of, surrounding structures) :
  - Optic chiasm: causing a bitemporal hemianopia
  - Cavernous sinus with III, IV and VI cranial nerve lesions
  - Bony structures and the meninges: causing headache
  - Hypothalamic centres: obesity, altered appetite and thirst, precocious puberty in children
  - The ventricles: causing interruption of cerebrospinal fluid flow and hydrocephalus.

### Hormonal findings:

- GH, LH, FSH, TSH, ACTH: not high
- PRL : low or high or normal (HOW :O?!!! depends on the size of adenoma some are "totally silent" in that they result in neither hormonal excess nor clinical manifestations. BUT if it get enlarged it may disrupt dopamine delivery to the pituitary by compression of the pituitary stalk and, consequently, be accompanied by a modest degree of hyperprolactinemia, in the late stage if the adenoma invade the hypothalamus tissue here the pt will get LOW PRL level) اقرروها تعبت وانا قاعده يوم اقرأ ارتيكلز

### Treatment:

- **Observation:**
  - Annual follow up for 5 years and then as needed.
  - Visual field exam Q6-12 months if close to optic chiasm
  - observe for a slow growing tumor
- **Surgery:** (Indicated if symptomatic, mass effect, etc)
- **Adjunctive Therapy:** Radiation therapy to prevent regrowth of the remaining adenomatous

# Functional Pituitary Adenoma:

PRL secreting adenoma	GH secreting adenoma	ACTH secreting adenoma	TSH secreting adenomas	Gonadotroph secreting adenomas
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## prolactin secreting pituitary adenoma(prolactinoma)

<b>characteristic:</b>	<ul style="list-style-type: none"> <li>- Most common of functional pituitary adenomas</li> <li>- 25-30% of all pituitary adenomas</li> <li>- Some growth hormone (GH)-producing tumors also co-secrete PRL</li> <li>- Prolactinomas women:90% present with <u>microprolactinomas</u></li> <li>- Prolactinomas in men :60% present with <u>macroprolactinomas</u></li> <li>- <u>Late diagnosis in men compared to women</u></li> </ul>	
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<b>diagnosis :</b>	<p><b>C:Clinical Features:</b></p> <ul style="list-style-type: none"> <li>- <b>in women:</b> Hyperprolactinaemia stimulates milk production in the breast producing galactorrhoea (spontaneous flow of milk unassociated with childbirth or breast feeding), and inhibits GnRH causing oligo- or amenorrhoea &amp; infertility.</li> <li>- <b>in men:</b> decreased libido, subfertility and erectile dysfunction in men. If there is a pituitary tumour</li> <li>- there may be Mass-effect(mechanical pressure):causing headache and visual field defects( Bitemporal hemianopia).</li> </ul> <p><b>B:Biochemical(hormonal):</b></p> <ul style="list-style-type: none"> <li>- prolactin level (a very high level suggest a prolactinoma)</li> <li>- GH,LH,FSH,TSH,ACTH: normal or low</li> <li>- TSH: to rule out Hypothyroidism( primary)(bc high TRH levels as in 1ry hypothyroidism stimulate prolactin)</li> <li>- IGF-1: to rule out acromegaly co-secretion</li> </ul> <p><b>A:anatomical:</b>ct or mri of the pituitary(MRI is superior to CT scanning and will readily show any significant pituitary mass)</p> <ul style="list-style-type: none"> <li>&lt; 1 cm (microadenoma)</li> <li>&gt; 1cm (macroadenoma)</li> </ul>
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<b>Treatment:</b>	<ul style="list-style-type: none"> <li>- Medical is the first line ( Dopamine agonist 'bromocriptine')</li> <li>- Surgical(if tumor is causing pressure symptoms)</li> </ul>
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<b>DDx of hyperprolactinemia:</b>	<p><b>Pathological:</b></p> <ul style="list-style-type: none"> <li>- The commonest cause is a prolactin secreting pituitary adenoma (prolactinoma).</li> <li>- Other causes are primary hypothyroidism (high TRH levels stimulate prolactin).</li> <li>- drugs which interfere with dopamine secretion or action:( <b>phenothiazines , metoclopramide , methyl-dopa</b> ) <b>دليما تحي بالاختبار</b></li> <li>- polycystic ovary syndrome and acromegaly (co-secretion of prolactin with GH by the tumour).</li> </ul> <p><b>Physiological:</b> Mildly increased serum prolactin levels may be physiological and asymptomatic,could be due to:asleep, stress, pregnancy, lactation</p> <p><i>if the prolactin high in women the most imp thing is to roll out the pregnancy then hypothyroidism ,Chest wall stimulation or trauma CABG or breast surgery Because prolactin neural end in the breast and when it is stimulate it will secrete prolactin</i></p>
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## Growth Hormone secreting adenoma

### characteristic:

- represents 10% of pituitary adenomas
- it cause ACROMEGALY **اضخامة الأطراف** in adult ,and GIGANTISM **العقلقة** in childhood.

### diagnosis :

#### **C:Clinical Features:** مره بالاروسكي جانبوا صورة وسألوا عن الفيتشرز اوف اكروميغالي

- **due to compression of surrounding structures**(usually large macro adenoma more than 1 cm in size): **headache** , dizziness , **bitemporal hemianopia** **دائما يجيبونها بالاختبارات**
- **due to invasion and destruction of the pituitary:** → lack of secretion of other hormones
- **due to excess GH secretion:**
  - acral(extremities) enlargement → large thick hands & feet
  - soft tissue enlargement → visceromegaly
  - thick skin , oily and **sweaty**
  - generalized symptoms:fatigue,lethargy & sleepiness.
  - arthralgia & degenerative arthritis **worsen at night**
  - **carpal tunnel syndrome** **باختبارات الميدينس يجبون** يسألون عن اسباب الكريال تانل
  - **impaired glucose tolerance** & diabetes
  - **cardiovascular effects** :cardiomegaly and chf (Cardiac disease is a major cause of morbidity and mortality ,50 % died before age of 50)Diastolic dysfunction is an early sign of cardiomyopathy,HTN in 40%, LVH in 50%

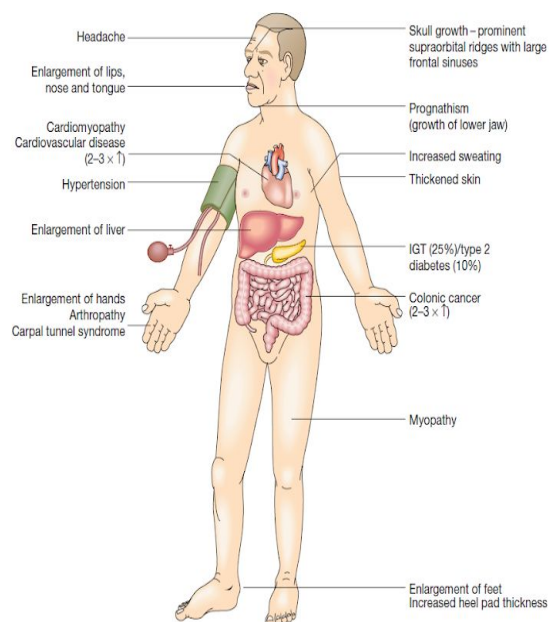


Fig. 20.29 Clinical features of acromegaly. (IGT = impaired glucose tolerance)

#### **B:Biochemica(hormonal):**

- A random GH level is not useful because there is wide physiologic fluctuation of GH levels.
- Screen(initial): measure IGF-1 → high in all patients with acromegaly,A normal serum IGF-1 concentration is strong evidence that the patient does not have acromegaly.
- Confirmatory Test : 75 g **oral Glucose tolerance test is diagnostic**. Normally glucose suppress GH levels. In normal people GH become undetectable. If there is a tumor GH will be high despite the injection of glucose.

#### **A:anatomical:**

- MRI or CT for the pituitary
- Echo:Diastolic dysfunction as an early sign of cardiomyopathy
- skull x-ray : thick heel pad  $\geq 22\text{mm}$

### Treatment:

#### **treatment is almost always surgery:**

1. surgical (trans-sphenoidal adenomectomy) Access to the pituitary is achieved through the nasal cavity, sphenoid sinus and sphenoid bone.

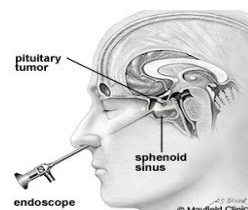
#### **other options used when surgery alone has failed to reduce GH and IGF-I levels to normal:**

2.radiotherapy

3. medical :

- somatostatin analogues(octreotide)AKA growth hormone inhibiting hormone
- GH receptor antagonist(Pegvisomant)

دكتور لما نعالجهم هل بيرجعوا ل حجمهم الطبيعي ويقصروا؟ لا يا ابني احنا مانعالجهم تجميلي احنا نعالجهم عشان نحميمهم من امراض القلب التي تعتبر المسبب الاول لوفاتهم.



### DDx of Acromegaly:

- 98% GH pituitary adenoma
- abuse of exogenous GH

## ACTH secreting adenoma

- ACTH adenoma → result in cushing DISEASE

#NOTE:Cushing's disease must be distinguished from Cushing's syndrome. Cushing syndrome is a GENERAL term which refers to the abnormalities resulting from a chronic excess of glucocorticoids whatever the cause, whereas Cushing's disease specifically refers to excess glucocorticoids resulting from inappropriate ACTH secretion from pituitary adenoma

- **C:clinical Features:** related to excess cortisol (discussed in adrenal lecture)

- moon face, central obesity, supraclavicular fat pad (previously known as buffalo hump (بشيرة من باب احتزام البشر)).
- Hirsutism, acne.
- stria, thin skin easily bruise → difficult IV cannulation, poor wound healing.
- DM
- irregular period.
- proximal weakness.
- recurrent infections.
- depression.
- Osteoporosis with vertebral fracture.
- Cardiovascular effects (HTN\_LVH\_Diastolic dysfunction. intraventricular septal hypertrophy\_ inverted T-wave)

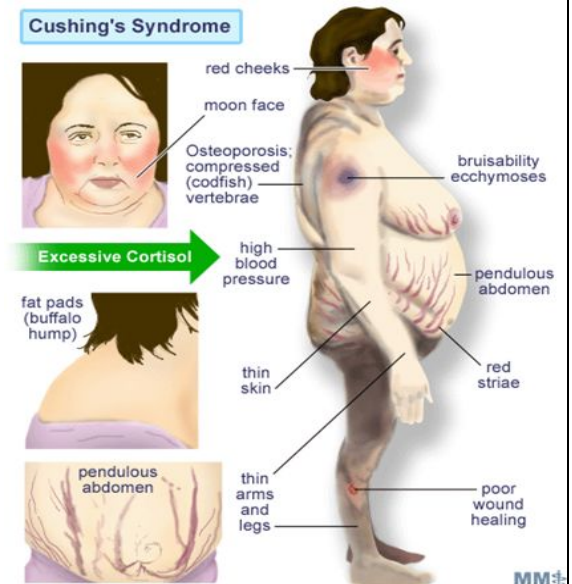
- **B:Biochemical:** (discussed in adrenal lecture)

- High cortisol, high ACH
- 24hrs for Urinary Free Cortisol (The Best)
- 1MG dexamethasone suppression test (At 11 pm and measure in morning if high so Cushing)

- **A: Anatomical:** MRI

- **Treatment:**

- first line Surgical → then Radiation to prevent regrowth of the remaining adenomatous



## TSH secreting adenomas

- Very rare < 2.8 %
- **Signs:** of hyperthyroidism (discussed in thyroid lecture)
- **Hormonal findings:** High TSH, Free T4, Free T3
- **Treatment:**
  - preop with anti-thyroid meds
  - Surgical resection of adenoma

## Gonadotroph secreting adenomas

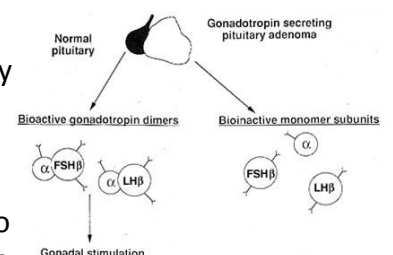
[For further reading not in your textbooks]

- Very rare
- **Clinical features:** Most of pt are middle-aged men who have a history of normal pubertal development and a normal fertility history, and by examination are normally virilized and have testes of normal size. They are brought to medical attention because of visual impairment, which is the result of the enormous size of the adenoma. While some pt have secondary hypogonadism because the adenomas are not secreting intact LH, but are compressing the normal gonadotroph cells and impairing LH secretion.

- **Hormonal findings:** hypersecretion of FSH, which is often accompanied by hypersecretion of FSH alpha-subunit (see the fig) \_ less often by hypersecretion of LH .

- **How to know whether high FSH result from menopause or gonadotroph adenoma in women?** in gonadotroph adenoma there will be in addition to high FSH, high estradiol, thickened endometrium and/or polycystic ovaries

- **Treatment:** gonadotroph cell adenomas treated first by surgery, to attempt to restore vision as quickly as possible, and then by radiation to prevent regrowth of the remaining adenomatous tissue





# Hypopituitarism:

## General Characteristics:

- Deficiency of hypothalamic-releasing hormones or pituitary hormones may be either selective or multiple. Multiple deficiencies usually result from tumour growth or other destructive lesions.
- **Panhypopituitarism** is a deficiency of all anterior pituitary hormones. Vasopressin and oxytocin secretion will only be affected if the hypothalamus is involved by either hypothalamic tumour or by extension of a pituitary lesion.

## Causes: seven Is

- **Infarction** : Sheehan's syndrome (pituitary infarction following severe postpartum haemorrhage)
- **Iatrogenic** : Radiation, surgery
- **Invasive** : **Large pituitary tumors (commonest cause)**, CRANIOPHARYNGIOMA
- **Infiltration** : Sarcoidosis, hemochromatosis
- **Injury** : head trauma
- **Infections** : TB once it cause basal meningitis
- **Idiopathic**

شيهان سيندروم: ما يحدث الإبحالة واحدة بعد أو أثناء الولادة إذا حدثت  
تعرس أو نزيف في الولادة لأن البيوتوتري يكون حجمها كبير أثناء الحمل  
وأصلا البلود سبلاي الي يجيها يعتبر ضعيف فاي نزيف خلال الحمل أو  
أثناء بيأثر عليها  
لكن انت باسم الله عليك إذا جاك نزيف لا قدر الله مارح يحصل لك شيهان  
سيندروم لان حجم البيوتوتري عندك مو كبير فما تحتاج الا كمية دم  
قليلة..  
##سيناريو على شيهان سيندروم: تجيك المريضة تقولك شوف انا جاني  
الطلق لما كنا بالبر و تعسرت الولادة علي وقعدت انز زرف يس الحمد لله  
الحين الولد طلع كويس وانا كويسه لكن فيه شي غريب لاحظته مع هالولد  
ماعصري لاحظته مع اخوانه العشرة الي قبله رالي هو اني ماني قادرة  
ارضع مايطلع مني حليب مهما حاولت: (جابولي طحينيه واكلت حليه  
عشان يطلع حليب بس ماش مايطلع شي :"  
##اول واهم علامة تدل ان البيوتوتري حصلها انفاركتشن هي  
inability to lactate in the lactating women

## Clinical Picture of Hypopituitarism (depends On Hormones Lost):

- Lack of FSH LH → Hypogonadism: amenorrhea
- Lack of TSH → hypothyroidism
- Lack of ACTH → adrenocortical insufficiency
- Prolactin deficiency → **failure of postpartum lactation (symptoms r noticeable in lactating women only)**
- Deficiency of GH → produces short stature in children **but in adults it is often clinically silent, although it may result in significant impairment in well-being and work capacity**
- If all of the above → **PANHYPOPITUITARISM**

## Investigations:

**Testing Ant.Pit.Function** (Each Axis Of The Hypothalamic-pituitary System Requires Separate Investigation):

- 1) **Clinical:** Hx and Px
- 2) **Biochemical studies:**
  - Baseline studies: TSH, ACTH, FSH, LH, prolactin, GH, Blood levels of IGF-1 may provide evidence of GH undersecretion.
  - Stimulation: TRH stimulation\_ GnRH(LHRH)stimulation\_ Insulin Tolerance Test (to Diagnosis ACTH and GH deficiency, in normal individual hypoglycemia will rise cortisol & Gh level )  
↑ this is a principle in endocrine: If hormone deficiency is suspected, choose a stimulation test
- 3) **Imaging:** CT, MRI

## Treatment:

- 1) **Remove the cause**
- 2) **Replacement Therapy (Depends On Hormone Lost):**
  - in secondary hypothyroidism give → Thyroxine
  - in secondary hypoadrenalism give → Hydrocortisone
  - GH deficiency give → GH analogues
  - Gonadotroph deficiency give → Testosterone monthly injections in **men**, Estrogen + progesterone in **women**  
▶ **BUT** If fertility is desired give → LH and FSH analogues (For induction of ovulation in **women**, spermatogenesis in **men**)

# شوف يا ابني بالزيبليسمنت ثيربي ما يحتاج نجيب الطباخ بكرة (ACTH, TSH, etc), و تنتظره لين يخلص الطبخه ابد ندلع نفسنا ونجيب الطبخه جاهزه (...cortisol, thyroxine, testosterone) يس حاله الي لازم نجيب الطباخ فيها: لما يكون الي عندهم نقص بالقونادوتروف حابين يجيبو بيبي فهنا اعطيهم الطباخ بكرة LH & FSH لانه بدون ما حيتم الاوفوليشن وتكوين السبيرم

Particular syndromes related to hypopituitarism are:(Kumar)

- **Kallmann's syndrome:** Congenital deficiency of GnRH.
- **Pituitary apoplexy** سكتة نخامية: rapid enlargement of a pituitary tumour due to infarction or haemorrhage. There is severe headache and sudden severe visual loss, sometimes followed by acute life-threatening hypopituitarism
- **'Empty sella' syndrome:** radiologically the sella turcica (the bony structure that surrounds the pituitary) appears devoid of pituitary tissue. In some cases, the pituitary is actually placed eccentrically and function is usually normal. In others there is pituitary atrophy (after injury, surgery or radiotherapy) and associated hypopituitarism.

20.62 Therapeutic modalities for hypothalamic and pituitary tumours				
	Surgery	Radiotherapy	Medical	Comment
<b>Non-functioning pituitary macroadenoma</b>	1st line	2nd line	–	
<b>Prolactinoma</b>	2nd line	2nd line	1st line Dopamine agonists	Dopamine agonists usually cause macroadenomas to shrink
<b>Acromegaly</b>	1st line	2nd line	2nd line Somatostatin analogues Dopamine agonists GH receptor antagonists	Medical therapy does not reliably cause macroadenomas to shrink Radiotherapy and medical therapy are used in combination for inoperable tumours
<b>Cushing's disease</b>	1st line	2nd line	–	Radiotherapy may be more effective in children than in adults and appears to cause less hypopituitarism in the long-term
<b>Craniopharyngioma</b>	1st line	2nd line	–	

## Hypothalamus & Posterior Pituitary Disorders

Diabetes insipidus	
<b>Types:</b>	<ul style="list-style-type: none"> <li>- <b>Central DI:</b> Decrease the amount of ADH</li> </ul> <p><b>other types not related to hypothalamus pituitary disorders:</b></p> <ul style="list-style-type: none"> <li>- <b>Nephrogenic DI:</b> renal resistance to ADH action</li> <li>- <b>Psychogenic DI:</b> is an excessive water intake seen in some patients with mental illnesses such as schizophrenia</li> </ul>
<b>Causes:</b>	<ul style="list-style-type: none"> <li>- <b>Nephrogenic DI:</b> ↓ K, ↑ Ca, lithium, Renal tubular acidosis-Sickle cell disease - Familial (mutation in ADH receptor)</li> <li>- <b>Central DI:</b> <ul style="list-style-type: none"> <li>○ disease of the hypothalamus: neurosurgery, head trauma, primary or secondary tumours, infiltrative disease (sarcoidosis, histiocytosis), vascular disease and 30-50% are idiopathic.</li> <li>○ rare with sheehan's: mild, undetectable degree</li> </ul> </li> </ul> <p>#NOTE: Damage to the hypothalamo-neurohypophysial tract or the posterior pituitary with an intact hypothalamus does not lead to ADH deficiency as the hormone can still 'leak' from the damaged end of the intact neurone.</p>
<b>Symptoms:</b>	<ul style="list-style-type: none"> <li>- Abrupt onset of polydipsia (as much as 15 L in 24 h) and polyuria</li> </ul>
<b>Investigations:</b>	<ul style="list-style-type: none"> <li>- <b>Urine:</b> ↑ urine volume ( 3 – 20 L/day )_ ↓ urine osmolality_ ↓ specific gravity</li> <li>- <b>Serum Na+:</b> usually high (bc ADH cause fractional excretion of Na in urine so lack of ADH result in high serum Na)</li> </ul> <p style="text-align: center;"><b>Water deprivation test</b> (To differentiate between CDI &amp; NDI)</p> <p>procedure:</p> <ol style="list-style-type: none"> <li>1. restrict p.o fluids or administer hypertonic saline to increase serum osmolality to 295-300 mosmol/kg (normal: 275-290)</li> <li>2. after a few hrs Measure the urine osmolality, then give vasopressin, after 3hrs measure urine osmolality</li> </ol> <p>interpretation:</p> <ul style="list-style-type: none"> <li>- <b>Central DI:</b> urine osmolality still low (before giving vasopressin), and returns to normal after administer vasopressin</li> <li>- <b>Nephrogenic DI:</b> exogenous vasopressin does not alter urine osmolality much</li> </ul>

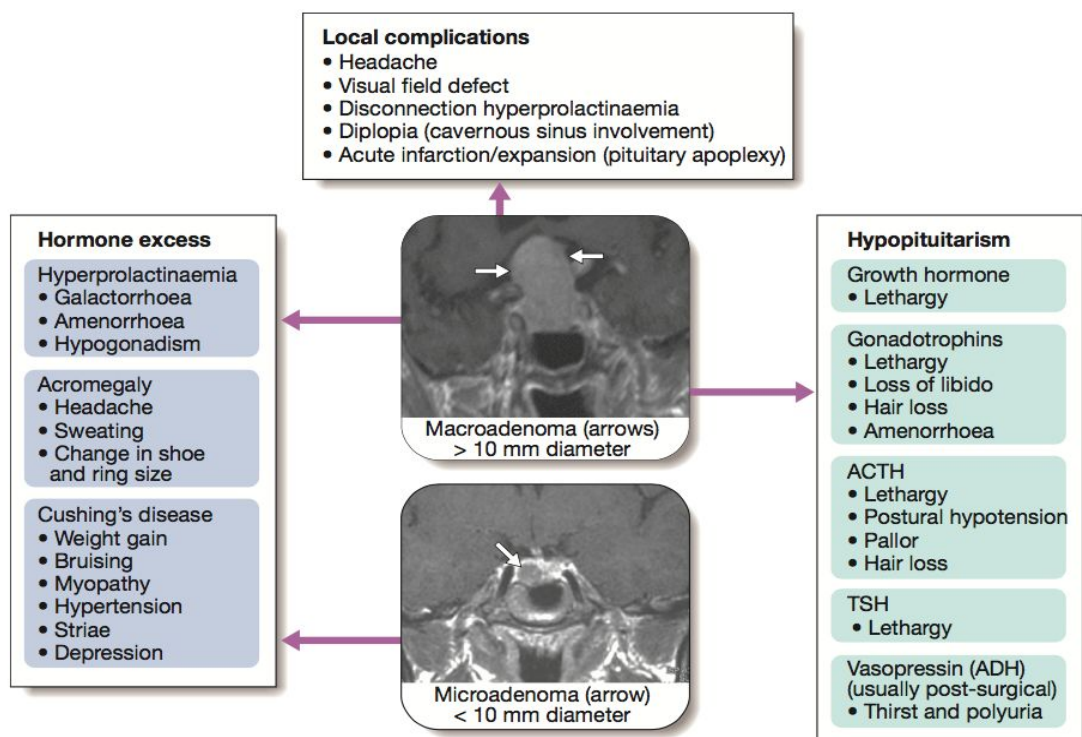
<b>Treatment:</b>	<ul style="list-style-type: none"> <li>- <b>Central DI:</b> DDAVP(Desmopressin Acetate):Synthetic analog of ADH <ul style="list-style-type: none"> <li>○ not catabolized by vasopressinase</li> <li>○ administered intranasally or p.o</li> <li>○ safe in pregnancy and breastfeeding</li> </ul> </li> <li>- <b>Nephrogenic:</b> <ul style="list-style-type: none"> <li>○ correct underlying cause</li> <li>○ hydrochlorthiazideare used to sensitize the renal tubules to endogenous vasopressin.</li> </ul> </li> <li>- <b>Primary Polydipsia:</b> psychiatric management</li> </ul>
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**Syndrome Of Inappropriate Antidiuretic Hormone** [not mentioned in the objective but within boy's slide]

<b>What is it</b>	a continued ADH secretion in spite of plasma hypotonicity and a normal or expanded plasma volume
<b>Causes</b> [Drug ,Brain,Lung & Cancer]	SIADH is caused by disordered hypothalamic–pituitary secretion or ectopic production of ADH: <ul style="list-style-type: none"> <li>○ CNS: meningitis ,head trauma, tumors</li> <li>○ Pulmonary: pneumonia,TB,small cell Ca(ectopic production of ADH)</li> <li>○ Drugs: Chlorpropamide ,Carbamazepine, Cyclophosphamide ,Vincristin</li> </ul>
<b>symptoms</b>	<ul style="list-style-type: none"> <li>- There is nausea, irritability and headache with mild Dilutional hyponatraemia</li> <li>- Fits and coma may occur with severe hyponatraemia</li> </ul>
<b>Findings</b>	<ul style="list-style-type: none"> <li>- Low serum sodium (Hyponatremia)&amp; ↑ urinary sodium</li> <li>- Low serum osmolality &amp; ↑ inappropriate urine osmolality</li> </ul>
<b>Treatment</b>	<ul style="list-style-type: none"> <li>- Removal of underlying cause</li> <li>- Restriction of fluid intake (0.5 – 1 L/day )</li> <li>- Demeclocycline inhibits the action of vasopressin on the kidney</li> <li>- If severe : I.V. hypertonic saline or normal infusion + Furosemide</li> </ul>

## Amazing Summary



**Fig. 20.27** Common symptoms and signs to consider in a patient with suspected pituitary disease.

# MCQs

**1) Which one of the following is the best initial management for patient with acromegaly?**

- a. Bromocriptine
- b. Radiotherapy
- c. Surgical removal of the adenoma
- d. Chemotherapy

**2) What is the cause of morbidity and mortality in acromegaly?**

- a. Cardiac diseases
- b. Hepatomegaly
- c. Splenomegaly
- d. Bone diseases

**3) A 30-year-old female patient came to the clinic with truncal obesity, purple striae, hypertension, diabetes and amenorrhea. What is the most common cause of her presentation?**

- a. Adrenal cortical adenoma
- b. Anterior pituitary adenoma
- c. Ectopic ACTH producing tumor
- d. Steroid or cortisone therapy

**4) A 28-year-old female Patient presented with hyperprolactinemia, what is the management?**

- a. Dopamine agonist
- b. Conservatives
- c. Surgery
- d. Dopamine antagonist

**5) Lady complaining of galactorrhea, amenorrhea, headache, what is the best explanation of her condition?**

- a. Cushing's syndrome
- b. Hyperprolactinemia
- c. Ovarian failure
- d. Hyperthyroidism

**6) What is the best treatment for microprolactinoma?**

- a. Surgery
- b. Dopamine agonist
- c. Radiotherapy
- d. Observation

**7) A 30 year old female was evaluated in the emergency department for her low blood pressure. She has been complaining of nausea, Vomiting, Abdominal pain and weight loss for the last 5 months. You found her serum ACTH and cortisol in the lower side. You decided to start her on steroid replacement. Which one of the following additional investigation would you request?**

- a. MRI pituitary
- b. Serum cortisol in the morning
- c. Growth hormone level
- d. Serum electrolytes

**8) A woman complaining of galactorrhea with 3mm pituitary adenoma, what is the appropriate treatment?**

- a. Dopamine agonist
- b. Surgical excision
- c. Supportive therapy

## Answer key:

1 (C) | 2 (A) | 3 (D) | 4 (A) | 5 (B) | 6 (D) | 7(A,not sure) | 8 (A)