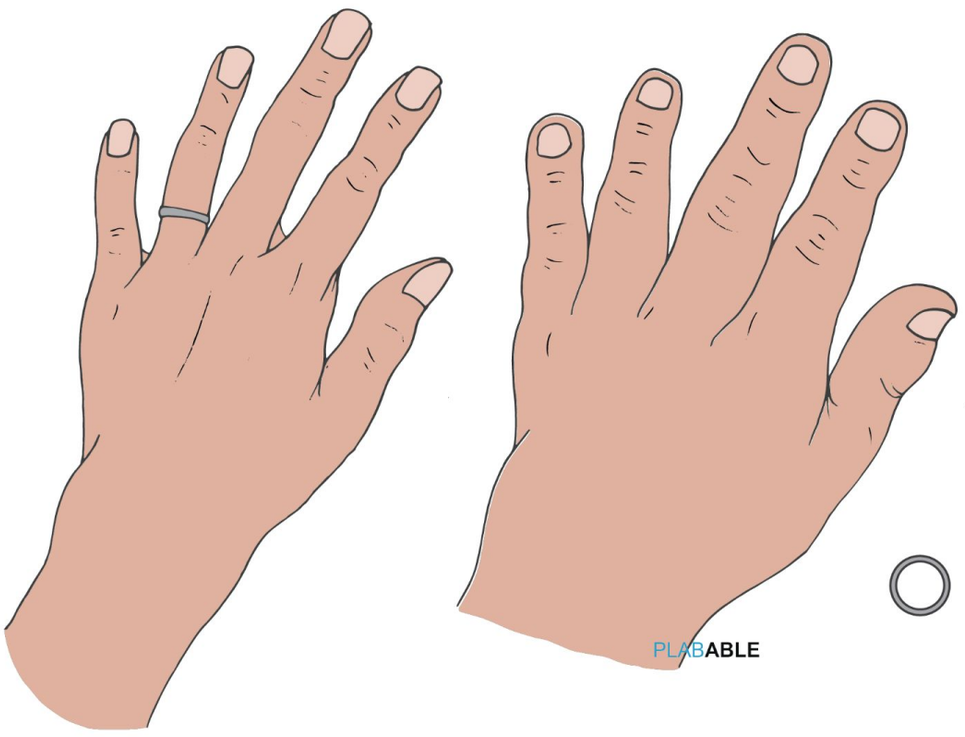
PLABABLE

GEMS

VERSION 4.8

ENDOCRINE



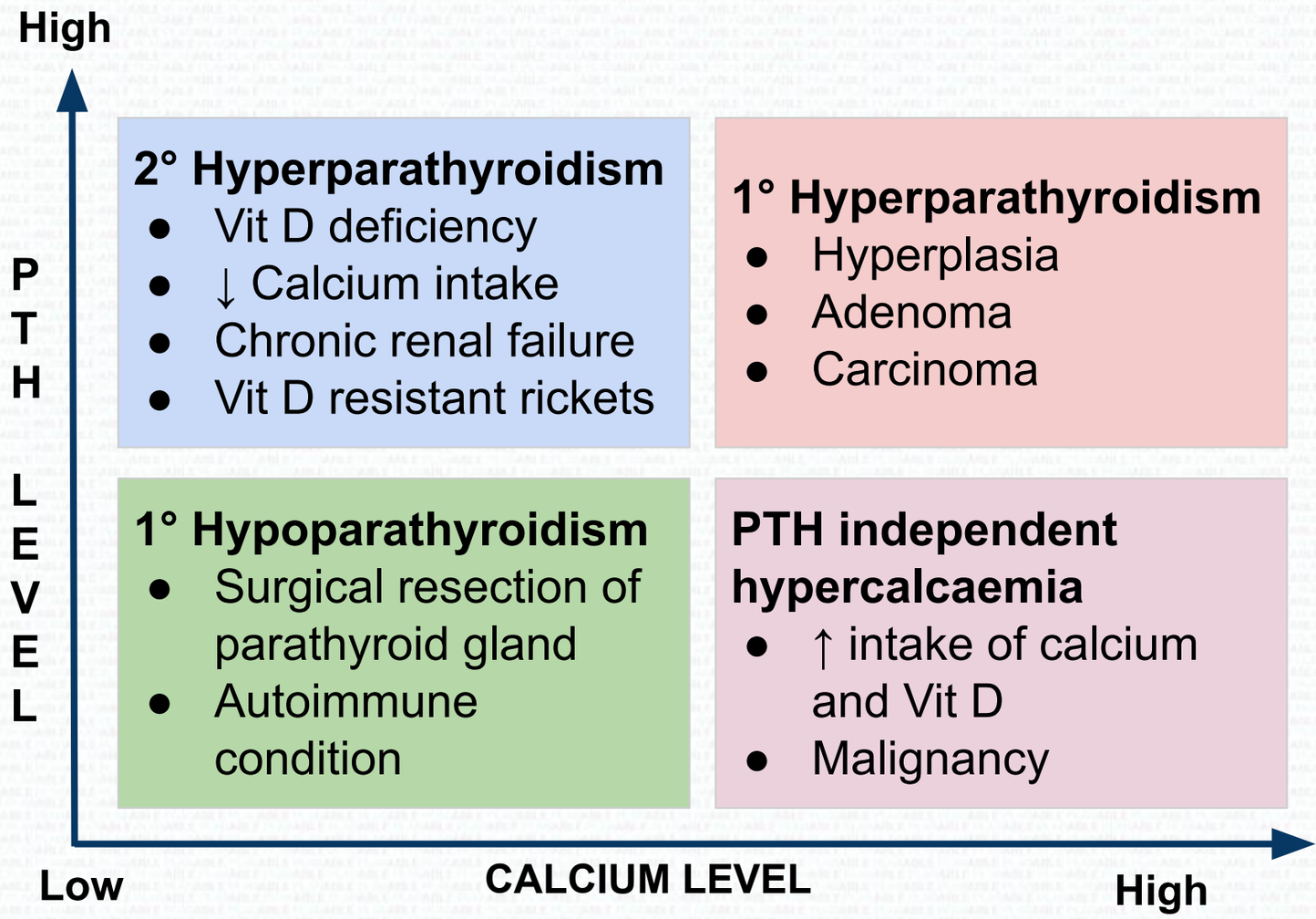
Hypercalcaemia

Mnemonic	Symptoms
Bones	Painful bones especially seen in primary hyperparathyroidism
Stones	Renal stones
Groans	• Constipation • Pancreatitis • Vomiting • Nausea
Psychiatric moans	• Lethargy • Easy fatigue • Depression

Causes

- **Primary hyperparathyroidism**
- Malignancy - **Multiple myeloma**
- Sarcoidosis
- Hyperthyroidism
- Prolonged immobilization
- Thiazides

Hypercalcaemia



Treatment

- Immediate: **0.9% NS** to increase urinary excretion of calcium
- Loop diuretic: **Furosemide** to increase calcium excretion
- Bisphosphonates: **Zoledronic acid** to reduce bone resorption
- **Denosumab** to reduce bone resorption

Hypocalcaemia

Symptoms "CATs go numb"

- **C**onvulsions
- **A**rrhythmia
- **T**etany and carpopedal spasm
- Paraesthesia (perioral, fingers and toes)

Causes

- **Low PTH:**
  - Parathyroid destruction by surgery, metastases or amyloidosis
  - Autoimmune
- **High PTH:**
  - Vit D deficiency
  - PTH resistance - Pseudohypoparathyroidism
- Hyperventilation
- Acute pancreatitis

Hypocalcaemia

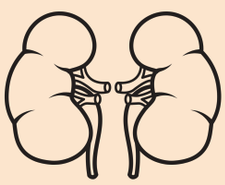
Signs

- **Chvostek's sign** - Tapping of facial nerve
- **Trousseau's sign** - Carpopedal spasm following BP cuff inflation
- Prolonged QT interval

Treatment

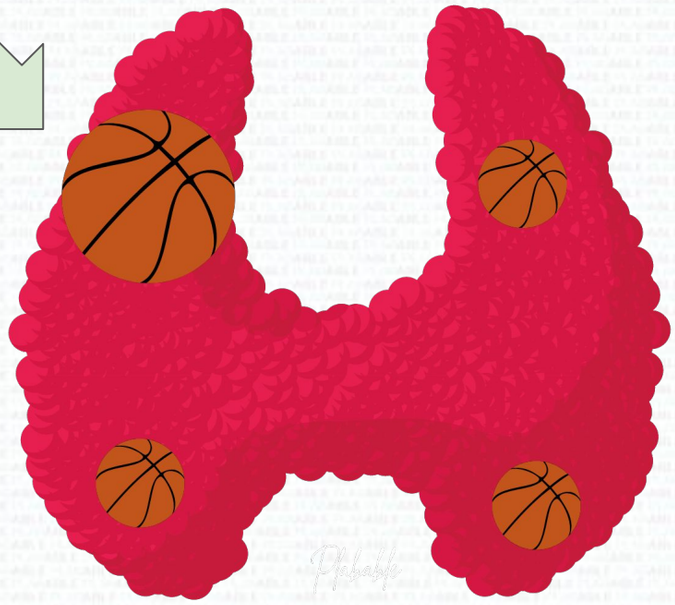
- **Symptomatic:** IV **calcium gluconate** 10%
- **Long term:** Oral calcium supplements with vit D

Hyperparathyroidism

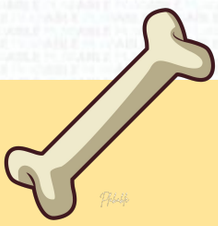
	Cause	Labs	Others
Primary 	Parathyroid adenoma (mostly) Hyperplasia Carcinoma	↑ PTH ↑ Calcium ↓ Phosphate	Associated with MEN 1 and MEN 2a Treatment is surgery
Secondary 	**CKD** causes parathyroid hyperplasia due to long standing hypocalcemia	Low-normal calcium ↑ PTH ↑Phosphate in CKD	Treatment with calcium and Vit D
Tertiary 	Autonomous production of PTH following long standing chronic kidney disease	↑ PTH ↑ Calcium ↑ Phosphate	Surgery is definite treatment Cinacalcet

Primary Hyperparathyroidism

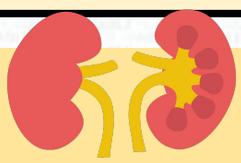
Most of the time caused by a **parathyroid adenoma**



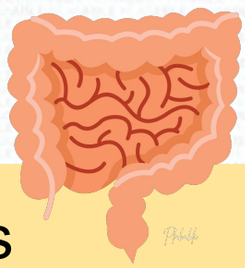
PTH increases



Bone resorption which increases serum calcium levels



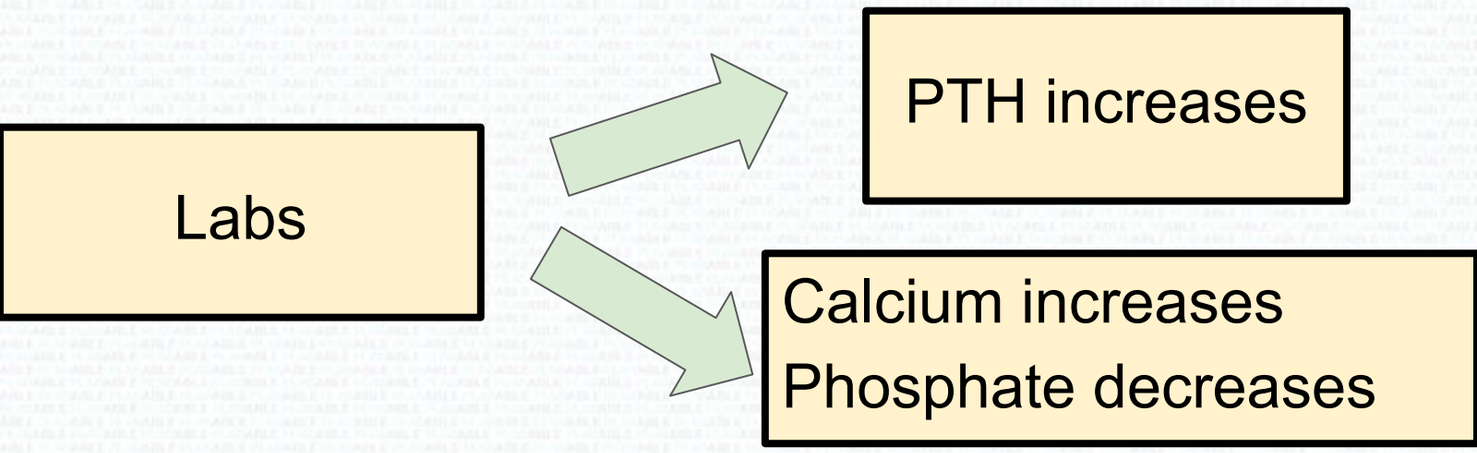
Reabsorbs calcium from kidneys but excretes phosphate



Absorbs calcium from intestine by increasing production of activated vitamin D

Calcium increases
Phosphate decreases

Primary Hyperparathyroidism



Most patients are asymptomatic and found incidentally on blood test. Others have only mild symptoms. Rarely do they present with severe hypercalcaemia

Calcium ↑

Causes symptoms of hypercalcaemia

PTH ↑

Resorption in bone can cause osteoporosis and fragility fractures

Primary Hyperparathyroidism Management

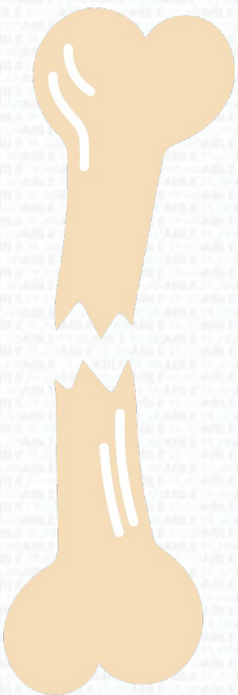
Cinacalcet	Only suitable for patients who cannot undergo surgical management
Bisphosphonates	Used in patients with increased risk of fracture but but should not be used for chronic hypercalcaemia of primary hyperparathyroidism
Parathyroidectomy	Best for most patients especially if symptomatic or have presence of end organ damage (fragility fractures, renal stones)

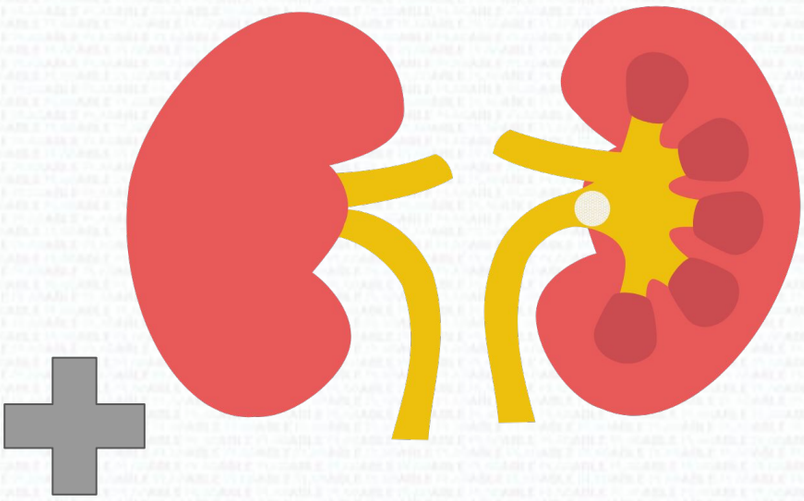
Primary Hyperparathyroidism Management

Remember

History of

- Fragility fracture
- Kidney stones





Laboratory evidence of primary hyperparathyroidism

=

Time to perform a **parathyroidectomy**

Secondary Hyperparathyroidism

For any patient with chronic renal failure with low calcium levels and high PTH

↓

Think secondary hyperparathyroidism!

Treatment

MAINLY with VITAMIN D supplements

VITAMIN D supplements:

Cholecalciferol → *Treats vitamin D*

Alfacalcidol → *Treats vitamin D in patients with severe renal impairment*

Other things to consider based on calcium levels and phosphate levels:

If calcium is low → Add in calcium supplements *(this can be part of vitamin D supplements e.g. Adcal-D3 or Calcichew D3)*

If phosphate levels are high → Add in phosphate binders

Secondary Hyperparathyroidism

For any patient with chronic renal failure with low calcium levels and high PTH

↓

Think secondary hyperparathyroidism!

Memory tool:
SH-CKD

Caused by

Secondary **H**yperparathyroidism

Chronic **K**idney **D**isease

Diabetes Mellitus

	Type 1 (insulin deficiency)	**Type 2** (insulin resistance)
Risk factors	Family history	Obesity, Family history, H/O gestational diabetes, physical inactivity
Diagnosis	**Symptomatic patients:** One abnormally high fasting glucose or one abnormally high HbA1c **Asymptomatic patients:** Needs two test showing abnormal values (e.g 2x high HbA1c **OR** 2x high fasting glucose **OR** 1x high HbA1c + 1x high fasting glucose)	

Diabetes Mellitus

	Type 1 (insulin deficiency)	**Type 2** (insulin resistance)
Treatment	**Insulin** therapy	**Metformin** (first line) Reinforce advice on lifestyle and ADD another drug if HbA1c > 58 mmol/mol

Diabetes Mellitus Type 2 Management

Metformin first line

↓

Recheck HbA1c in 3 to 6 months

↓

If rise to 58 mmol/mol or higher

↓

Reinforce advice on lifestyle
+
Add another diabetic drug if appropriate

What if both options of reinforce advice on lifestyle and add another diabetic drug are seen in the options in a patient with a high BMI and a HbA1c of 58 mmol/mol who is already on metformin?

↓

Pick reinforce advice on lifestyle

See question code EN 4808 and EN 4809

Diabetes Mellitus Type 2 Management

Metformin first line

↓

Recheck HbA1c in 3 to 6 months

↓

If rise to 58 mmol/mol or higher

↓

Reinforce advice on lifestyle
+
Add another diabetic drug if appropriate

↓

Which drug to start?

Any oral antidiabetic drug depending on the patient profile

Example 1

SGLT-2 inhibitors cause weight loss, so would be good for a high BMI patient

Example 2

Sulfonylurea has a high risk of hypoglycaemic events so you would NOT want to use this for a lorry driver

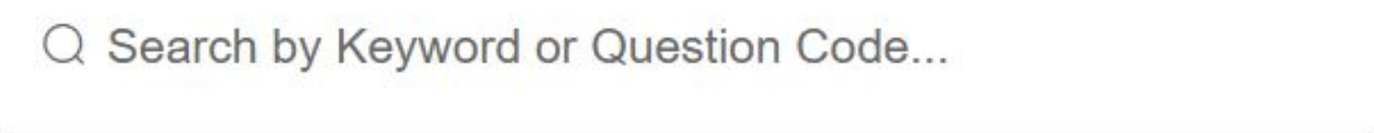
Diabetes Mellitus Type 2 Medication

PLABABLE Tip

We know many candidates struggle with diabetes mellitus type 2 medications. If you struggle, the best way to understand them is to attempt all of the questions in relation to them together and read the explanations thoroughly.

You can attempt the questions together by searching “diabetes mellitus type 2 medication” on the search bar.

The search bar looks like this



Diabetes Complications (Acute)

DIABETIC KETOACIDOSIS (DKA)

- Common in type 1 DM
- Metabolic acidosis and +ve urine ketones
- **Treatment:** IV 0.9% NS, Insulin infusion with dextrose and electrolyte monitoring

HYPEROSMOLAR HYPERGLYCAEMIC STATE

- Seen in type 2 DM
- High plasma glucose level, high serum osmolarity without significant ketoacidosis
- **Treatment:** 0.9% NSl and electrolyte correction

Diabetes Complications (Chronic)

DIABETIC NEPHROPATHY

- Proteinuria
- Microalbuminuria
- Treatment to control HbA1c
- ACEi to prevent progression

INCREASED RISK OF CVD

- Reduce risk by proper glycemic and BP control
- Statins

DIABETIC RETINOPATHY

Microvascular changes:
Aneurysms, hard exudates, haemorrhages, cotton wool spots, and neovascularisation

Treatment:

- **Early:** Glycaemic control
- **Late:** Panretinal photocoagulation, intravitreal steroids and anti- VEGF

Diabetes Complications (Chronic)

DIABETIC NEUROPATHY

- Glove and stocking neuropathy
- Burning, numbness and tingling sensation in the extremities
- Tight glycemic control and prevention of foot trauma

DIABETIC FOOT ULCER

- Glycemic and BP control, Smoking cessation
- Wound management
- Foot interventions to prevent ulcer formation such as well-fitting footwear

Diabetic Ketoacidosis (DKA) Presentation

Presentation

- Polyuria
- Polydipsia
- Dehydration
- Sweet smelling breath
- Altered mental status or even coma
- Most common in **type 1 diabetes**

Signs

- Dry mucous membranes
- Hypotension
- Tachycardia

Precipitating causes: Infection, stoppage of insulin, drugs such as steroids and thiazides

Diabetic Ketoacidosis (DKA) Investigations

Diagnostic criteria for DKA

- Capillary blood glucose > 11 mmol/L **or** known diabetes mellitus
- Capillary ketones above 3 mmol/L **or** urine ketones ++ or more
- Blood gas → pH < 7.3 **and/or** bicarbonate < 15 mmol/L

Glucose

Ketones

Gas
→ Metabolic acidosis

Remember, for the blood gas, you can do either **arterial** or **venous** blood gas *(venous is preferred mostly because it is easier)*

Diabetic Ketoacidosis (DKA) Investigations

Treatment

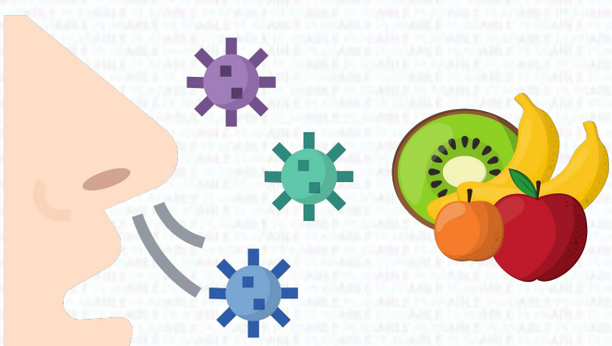
- **0.9% normal saline**
- **IV insulin infusion** → 10% glucose when glucose falls below 14 mmol/L
- Hypokalemia → KCl infusion

Complications

- Cerebral oedema
- Pulmonary oedema
- Cardiac arrhythmia due to electrolyte imbalance

High sugar level

Fruity breath (Acetone)

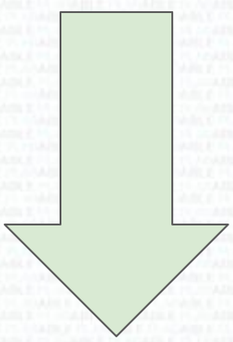


Diabetic Ketoacidosis (DKA)

Lets run through some examples

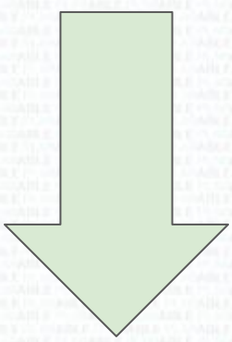
Type 1 diabetic + thirsty + confused + abdo pain + vomiting + ketones in urine + high capillary glucose

What is the next test to do?



Blood gas

What is the next management?



Start 0.9% normal saline

MODY

Brain trainer:

A 25 year old with polyuria and polydipsia presents to the GP surgery. A HbA1c was done and came back at 60 mmol/mol (<48). His fasting blood glucose is 10 mmol/L (<7). His father and grandmother had some form of diabetes. What is the most likely diagnosis?

➔ **Maturity onset diabetes of the young (MODY)**

What is the most appropriate management?

➔ **Refer to diabetic clinic**

MODY is managed in secondary care. Be careful NOT to choose oral hypoglycaemics or lifestyle changes as the most appropriate management in primary care for cases where MODY is suspected.

If referral is not an option, then sulphonylureas would be your best pick since majority of MODY mutations are sensitive to sulphonylureas

Latent autoimmune diabetes of adulthood (LADA)

Latent autoimmune diabetes of the adult (LADA) is a variant of type 1 diabetes which develops much slower than type 1 diabetes.

It develops slowly and often mistaken for type 2 diabetes mellitus since it develops in adulthood.

Test to request for LADA:

GAD antibody testing

This test will help distinguish LADA from type 2 diabetes

MODY Vs LADA

Maturity onset diabetes of the young (MODY)

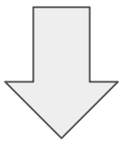
- Age < 25
- Strong family history
- Refer to endocrinology (not managed in primary care)
- Genetic counselling (like all genetic test) before performing the genetic test

Latent autoimmune diabetes of adulthood (LADA)

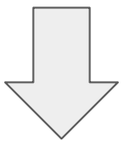
- Age 30 to 50 years old
- Like type 1 diabetes but occurs late in life
- GAD antibody test positive

Stress Hyperglycaemia

Raised blood sugar levels after infection/stress/trauma/surgery



Cause?



Stress Hyperglycaemia

Options can also state this as **“normal stress reaction”**

Resolves spontaneously within hours

Blood tests performed 6 weeks later show normal values

Glycosuria

Brain trainer:

A non-diabetic patient after surgery has glycosuria. What is the most likely diagnosis?

➔ Stress hyperglycemia

Infection or stress→cortisol→hyperglycaemia

Pre-Diabetes

Brain trainer:

What laboratory values are found with impaired glucose intolerance?

➔ **Fasting → 5.5 to 6.9 mmol/l**
➔ **2 hr post-prandial → 7.8 to 11.0 mmol/l**

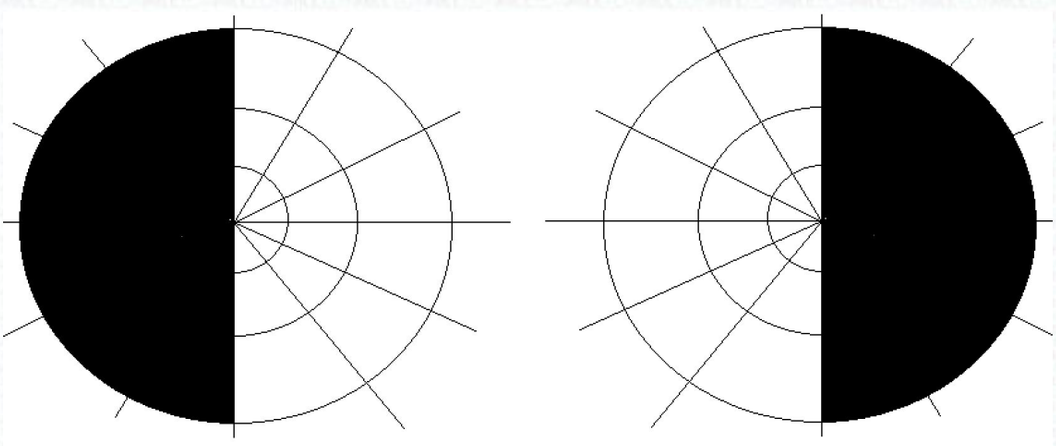
Hyperprolactinaemia

Causes

- **Prolactinoma**
- Hypothyroidism
- Drugs: antipsychotics (risperidone & haloperidol) and domperidone
- Brain injury

Presentation

- **Females:** Inhibits FSH and LH causing menstrual irregularity (amenorrhea) and galactorrhoea
- **Males:**
  - Secondary hypogonadism
  - Reduced libido
  - Gynecomastia
  - Erectile dysfunction
- **Visual symptoms:** Bilateral hemianopia
- Headache

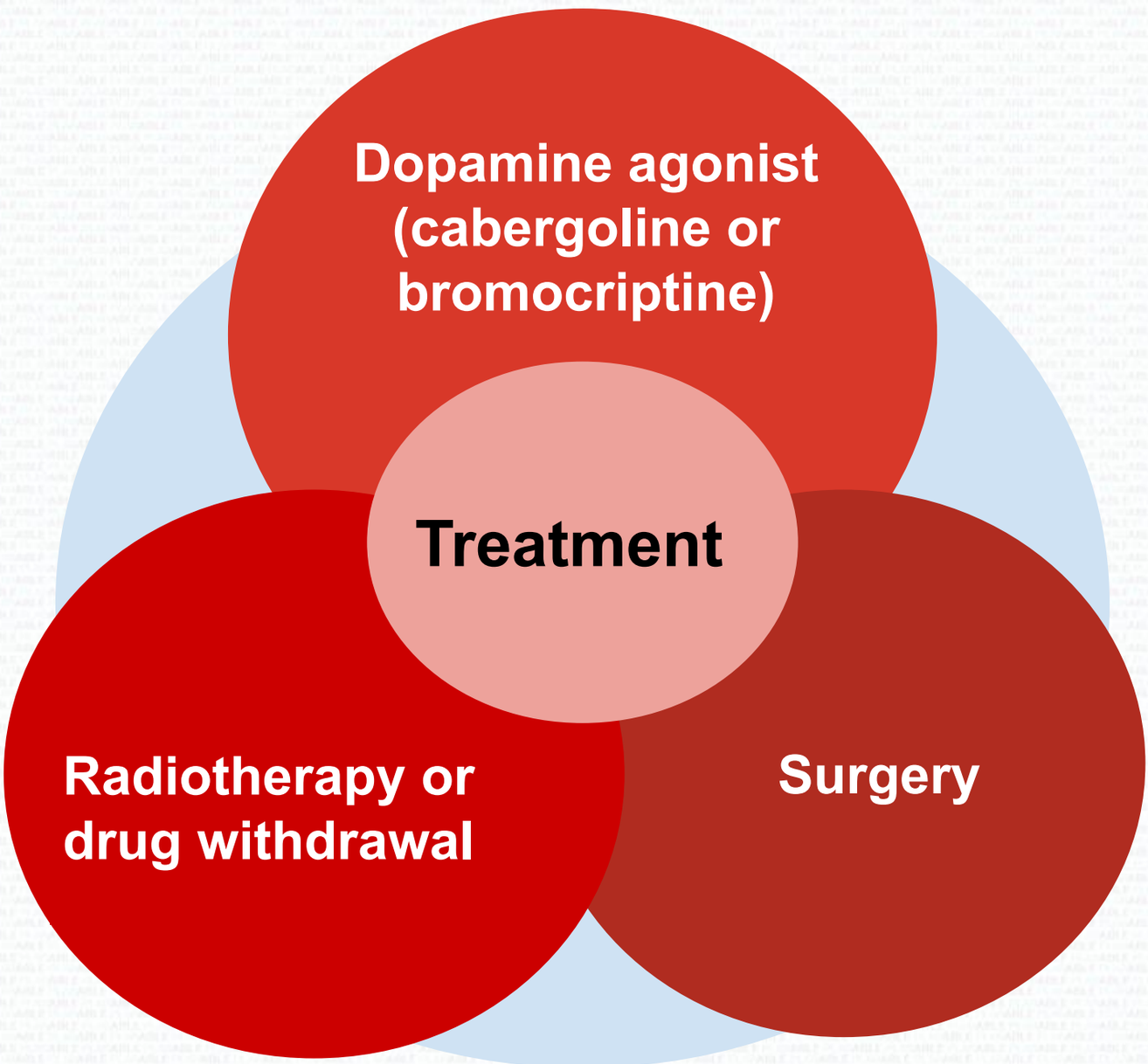


Bitemporal Hemianopia

Hyperprolactinaemia

Investigations

- ↑ serum prolactin level
- Pituitary MRI
- Visual field testing



Thyroid Disorders

Hypothyroidism	Hyperthyroidism
Decreased appetite but weight gain	Increased appetite and weight loss
Cold intolerance	Heat intolerance
Constipation	Diarrhoea
Bradycardia	Palpitations and tachycardia
Menstrual irregularities - Menorrhagia	Menstrual irregularities - Oligomenorrhea
Tiredness and lethargy	Irritability and weakness
• Deep hoarse voice • Reduced libido	• Tremor • Increased sweating • Brisk reflex • Lid lag • Palmar erythema

Thyroid Disorders

	Hypothyroidism	Hyperthyroidism
Causes	**Primary:** **(low T4 and high TSH)** Autoimmune - Hashimoto's thyroiditis Iodine deficiency Drugs - Amiodarone and antithyroid medications Congenital hypothyroidism **Secondary:** **(Low TSH and T4)** • Hypopituitarism • Isolated TSH deficiency	**Graves disease** Antimicrosomal antibodies against thyroid peroxidase and Antithyroglobulin antibodies Toxic nodular goitre De Quervain's thyroiditis Exogenous thyroid ingestion

Thyroid Disorders

	Hypothyroidism	Hyperthyroidism
Treatment	**Thyroxine (T4) supplementation** If due to iodine deficiency then replacement of iodine	**Anti-thyroid drugs** • **Carbimazole** Bone marrow suppression and cutis aplasia (pregnancy) • **Propylthiouracil** Liver failure Radio-iodine therapy Thyroidectomy

TSH and T4 levels

	T4	TSH
Hypothyroidism	Low	High
Hyperthyroidism	High	Low
Subclinical Hypothyroidism	Normal	High
Subclinical Hyperthyroidism	Normal	Low

Subclinical Hypothyroidism

Definition → TSH is high but the T4 is within normal range

Always start by repeating TSH blood test in 3 months

If TSH is raised but below 10miU/L (on 2 test 3 months apart):

If symptomatic + below 65 years of age then consider levothyroxine

If TSH is raised above 10miU/L (on 2 test 3 months apart):

Consider levothyroxine

Hyperthyroidism In Pregnancy

Pre-pregnancy and first trimester:

- Propylthiouracil

2nd, 3rd trimester + post-pregnancy

- Carbimazole
- Partial thyroidectomy (in 2nd trimester if carbimazole is not effective)

This means if a pregnant woman is on PTU in her first trimester, she would be switched to carbimazole in her 2nd trimester.

Notes in pregnancy:

- Lowest possible dose should be prescribed
- Radioiodine therapy contraindicated
- Block and replace regime not suitable

We would like to highlight this point! Block and replace means administering carbimazole and levothyroxine. This combination can be used outside of pregnancy but it is not suitable in pregnancy!

Hyperthyroidism In Pregnancy

What would you pick if you had a second trimester pregnant woman with hyperthyroidism with positive TPO and a heart rate of 113 beats/minute?

Carbimazole and levothyroxine? or **PTU?** or **Propranolol?**

Answer → PTU

Carbimazole is used in second trimester (preferably over PTU), but NEVER together with levothyroxine since the block and replace regime is NOT suitable in pregnancy.

Propranolol will help with her heart rate but will not help with the overactive thyroid. Besides, she is not complaining of palpitations

So PTU is the most appropriate option given the options

Of course, look out for caveats. If the same woman has **palpitations** and the question specifically ask you for the best medication to pick to **manage her symptoms** → Pick **Propranolol**

Thyroid Crisis

Brain trainer:

What is the management for thyroid storm?

➔ Propylthiouracil
➔ Palpitations → beta-blocker
➔ If infective etiology → broad-spectrum IV antibiotics

Postpartum Thyroiditis

Brain trainer:

What medication is indicated to control the symptoms of postpartum thyroiditis?

➔ **Beta-blocker (palpitations + sweating)**

PodsForDocs

Check out our podcast episode '*The Thyroid (Butterflies & Storms)'* to further solidify your knowledge on the topic.

Click on the image below to head to our PodsForDocs podcast page to find out more.

We also have a dedicated PodsForDocs WhatsApp group which you can join via the Study Group tab.

Enjoy!

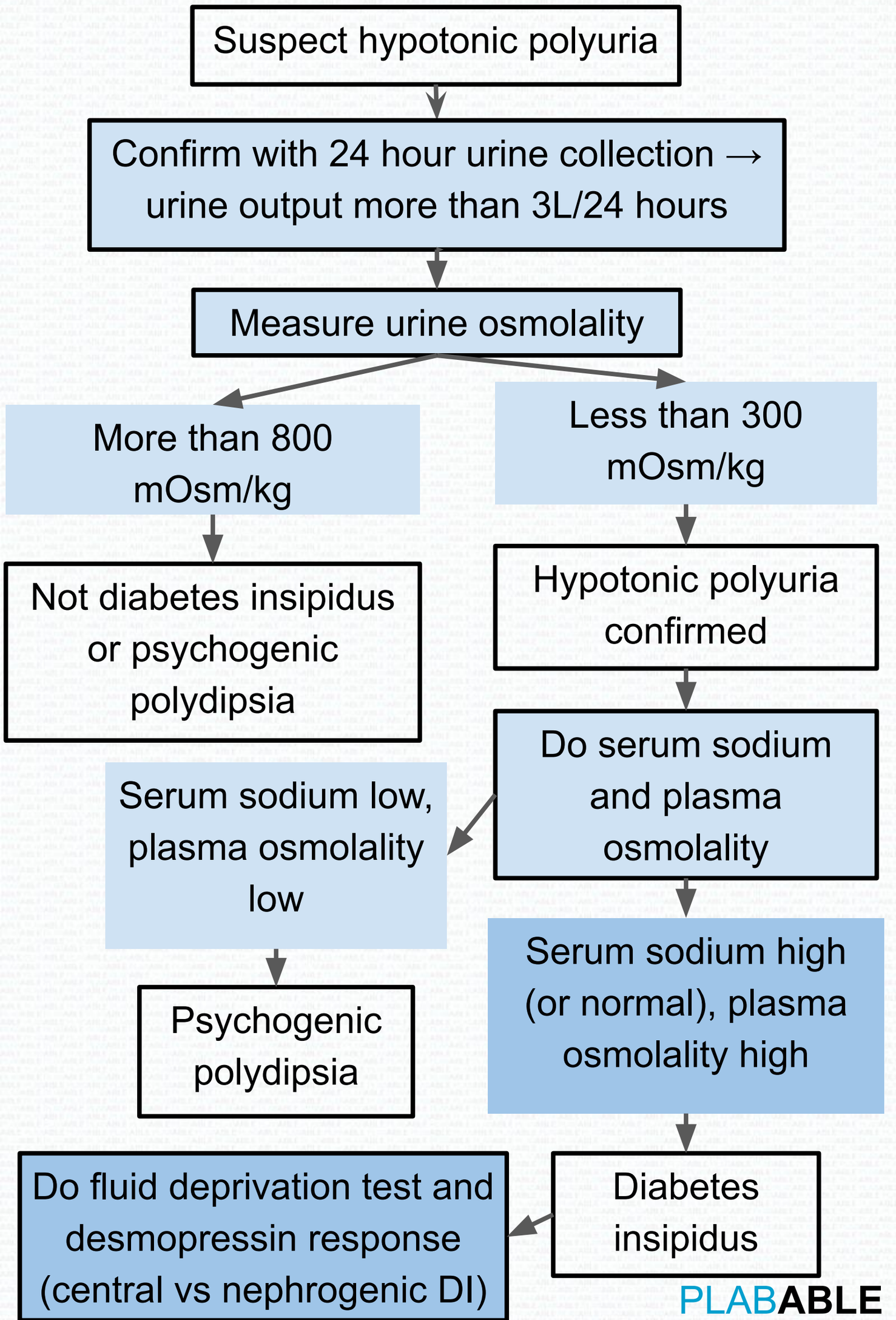
ADH Disorders

	Diabetes insipidus (decreased ADH)	**SIADH** (excess ADH)
Symptoms	**Polyuria** (>3L of urine/day) Polydipsia	Hyponatraemia - CNS and muscular in nature
Causes	**Cranial:** Decreased secretion of ADH ● Craniopharyngioma ● Head injury ● Sarcoidosis and tuberculosis ● Infections: Meningitis and encephalitis ● Post-radiotherapy **Nephrogenic:** ADH resistance ● Lithium ● Renal tubular acidosis ● CKD ● Idiopathic	Small cell lung cancer Meningitis and encephalitis Drugs: SSRIs Chlorpropamide Oxytocin Amitriptyline

ADH Disorders

Treatment	
Diabetes Insipidus	**SIADH**
Central: Desmopressin **Nephrogenic:** High dose desmopressin with or without thiazide and NSAIDs	• Fluid restriction (mild cases) • Demeclocycline • Tolvaptan

Hypotonic Polyuria



Hypotonic Polyuria

Urine Osmolality		
	Cranial DI	**Nephrogenic DI**
Without intervention	<300	<300
Water deprivation	<300	<300
Desmopressin injection	>800	<300

Because desmopressin can still work on the kidneys to reduce water that is eliminated in the urine so urine osmolality increases

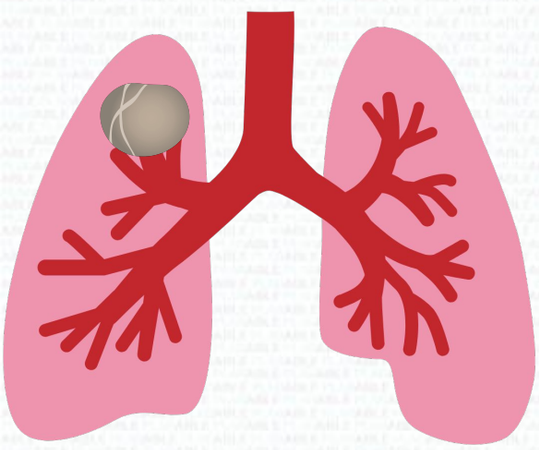
Because desmopressin cannot work on the kidneys due to improper response of the kidney to ADH, therefore, urine osmolality continues to be low

If this was a case of **psychogenic polydipsia**, his urine osmolality would increase as he undergoes water deprivation

SIADH Causes

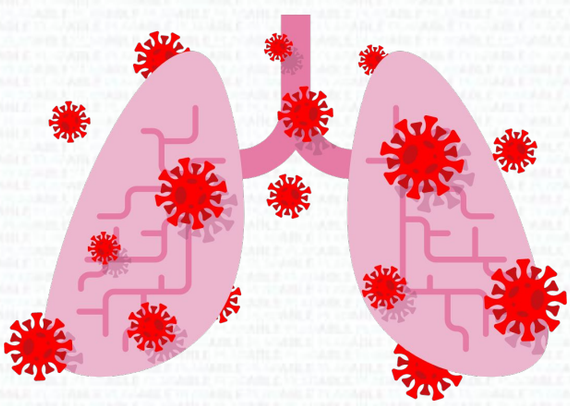
Here are some examples of conditions that can cause SIADH

CNS causes which includes meningitis, stroke, tumours



Neoplastic diseases which includes small cell lung cancer

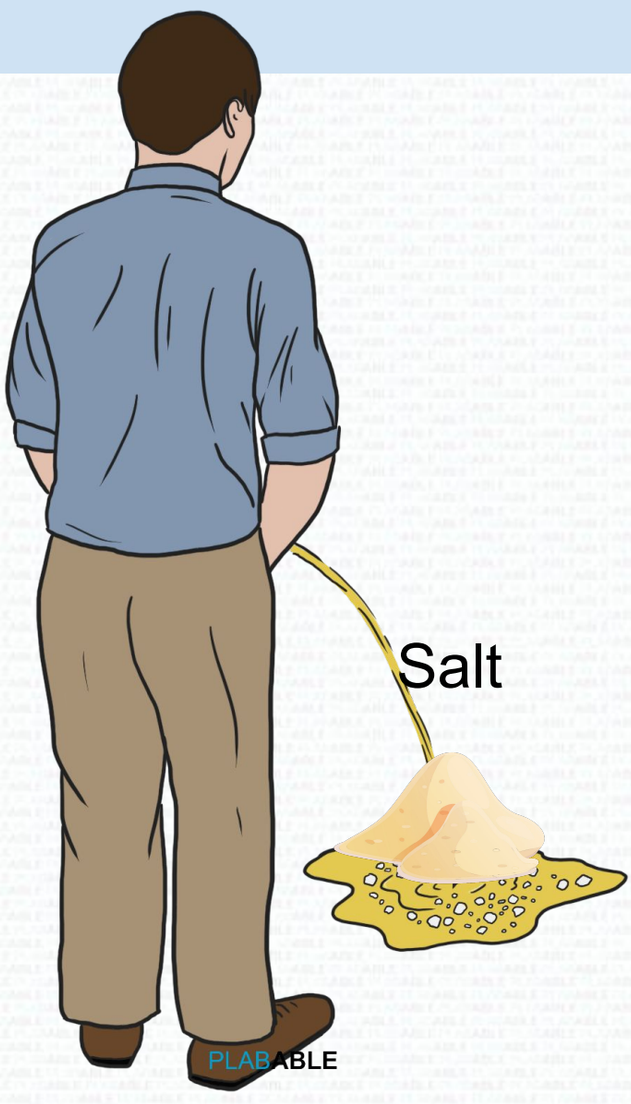
Chest infections (pneumonia, tuberculosis)



SIADH Investigations

Urine Osmolality	Urine sodium	Serum sodium	Plasma osmolality
High	High	Low	Low

Think of it as peeing salt in the presence of insufficient salt in the blood

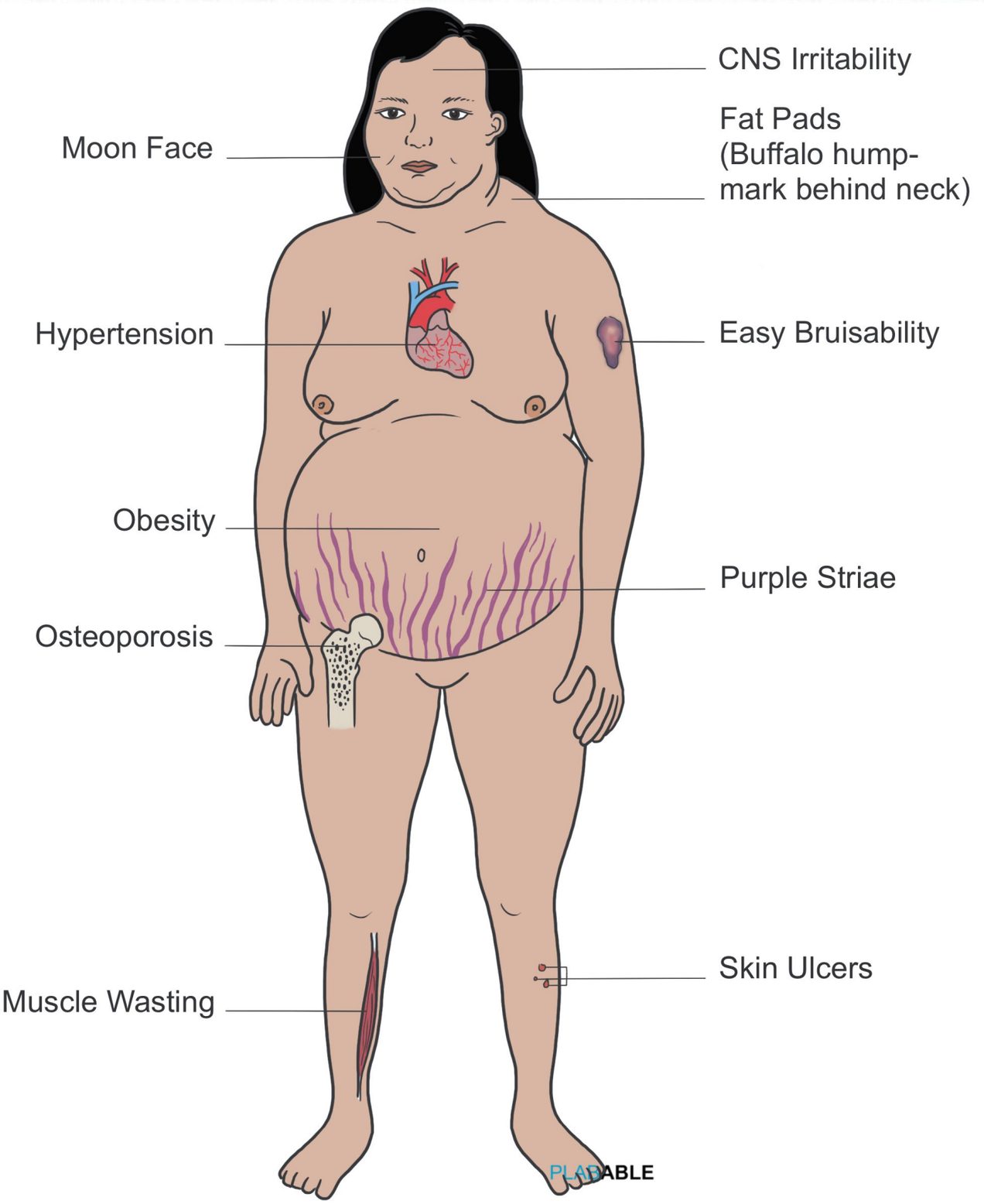


Cushing's Syndrome

Prolonged exposure to either endogenous or exogenous cortisol

ACTH dependent	ACTH independent
Cushing's disease (ACTH from pituitary)	Adrenal adenoma
Ectopic ACTH producing tumours	Adrenal carcinoma
Excess ACTH administration	Exogenous glucocorticoid administration

Cushing's Syndrome



Cushing's Syndrome

Investigation

To confirm cushing's syndrome:

- 24-hour urinary free cortisol
- Low-dose dexamethasone suppression test

To identify the cause:

- Plasma ACTH: High - ACTH dependent
- Plasma ACTH: Low - ACTH independent

If ACTH is high to differentiate between pituitary and ectopic cause:

- High-dose dexamethasone test
- MRI pituitary

If ACTH is low:

CT or MRI scan of abdomen to look for adrenal tumour

Treatment

- **Medications** to inhibit cortisol synthesis
  - Metyrapone
  - Ketoconazole
  - Mitotane
- **Surgery** - Pituitary tumour
- Removal of exogenous corticosteroid

Pheochromocytoma

High levels of catecholamine production

Symptoms and signs

- Palpitations
- Profuse sweating
- Anxiety
- Sense of doom
- Headache
- Tremor
- Hypertension

Look for the young patient with hypertension or the middle age man with many hypertensive medication coming in with EPISODIC headaches

Investigation

- 24-hour urinary metanephrines - first test to perform
- If above not available pick, 24-hour urinary catecholamines or VMA
- CT abdomen to locate the tumour once the test above are seen as abnormal

Pheochromocytoma

Associated with

- Multiple endocrine neoplasia (MEN 2A and 2B)
- Neurofibromatosis
- Von Hippel-Lindau syndrome

Treatment

Surgery after preoperative alpha-blocked with phenoxybenzamine

Adrenal Insufficiency

Primary adrenal insufficiency/Addison's disease

- Autoimmune cause
- Infections and haemorrhage
- Congenital adrenal hyperplasia
- Drugs: ketoconazole

Secondary adrenal insufficiency

- Hypothalamic and pituitary failure
- Long term steroid medication

Clinical features

Acute: Hypotension, shock, abdominal pain and vomiting

Chronic: Fatigue, weakness, weight loss, salt cravings, syncope, vomiting, and hyperpigmentation in buccal mucosa and lips (primary)

Labs

- **Low sodium and high potassium (in Addison's disease)**
- Early morning cortisol - Low
- ACTH: high in primary and low in secondary
- ACTH stimulation test: Cortisol does not rise

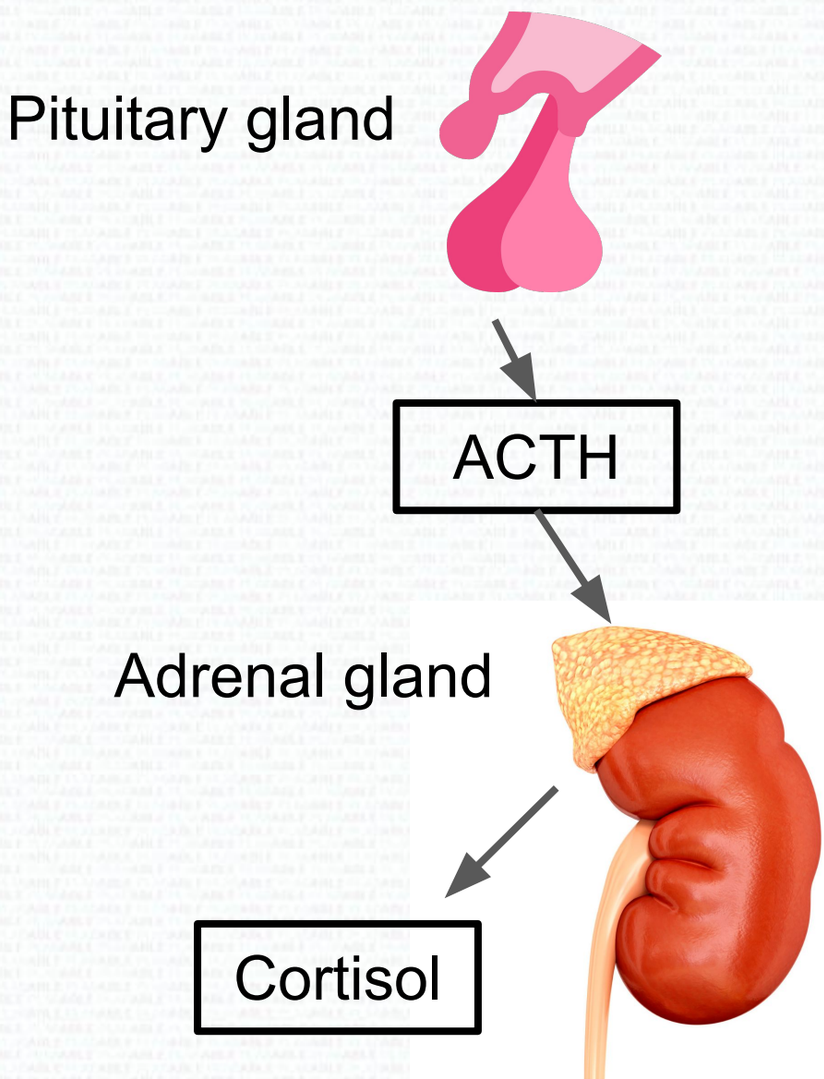
Treatment

- Hydrocortisone (Glucocorticoid)
- Fludrocortisone (Mineralocorticoid)

Adrenal Insufficiency

Breaking down to understand better

Normal physiology



Adrenal Insufficiency

Breaking down to understand better

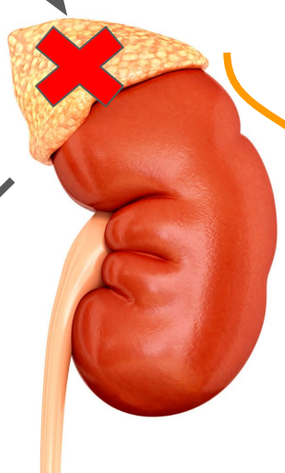
Primary Adrenal Insufficiency
(also known as Addison's disease)

Pituitary gland

Negative feedback increases ACTH

ACTH

Destruction of adrenal cortex



Cortisol low

Also aldosterone low

Which results in hyperkalaemia and hyponatraemia

Adrenal Insufficiency

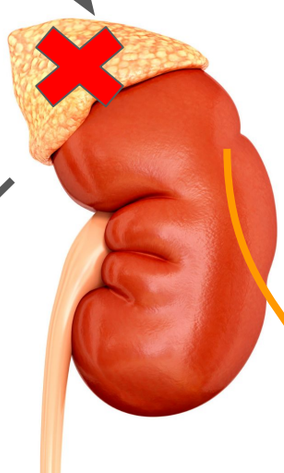
Breaking down to understand better

Secondary Adrenal Insufficiency

Inadequate pituitary or hypothalamic stimulation

ACTH low

Adrenal gland



Cortisol low

Aldosterone unaffected since aldosterone is regulated primarily by renin-angiotensin system which is independent of pituitary or hypothalamus

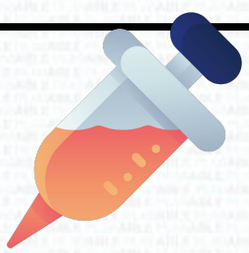
Synacthen Test

Test for

Primary Adrenal Insufficiency
(also known as Addison's disease)

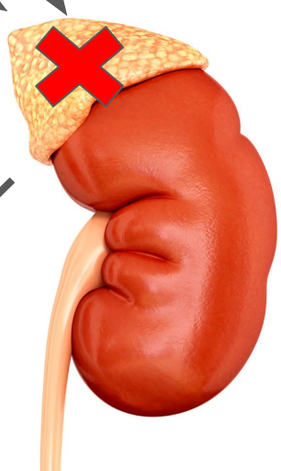
Pituitary gland

Artificial ACTH called **synacthen** is given intramuscularly



ACTH

Destruction of adrenal cortex



Cortisol low

Cortisol is measured at 30 minutes, In a normal person, the level of cortisol should double. If the cortisol level is still low, then it is Addison's disease.

Addison's Disease

Plabable tip

Hyperkalaemia + ***Hyponatraemia***

The combination of **hyperkalaemia** and **hyponatraemia** in the exam, usually means addison's disease.

There are not many other causes of this combination that will be tested at your level

Adrenal Insufficiency

Brain trainer:

What is the most common cause of Addison's disease in the UK?

➔ **Autoimmune**

Adrenal Insufficiency

Brain trainer:

A patient with Addison's disease has recently fallen ill with a cough and cold. Her observations are stable. What is the most appropriate advice?

➔ **Double the dose of hydrocortisone**

You do not need to double the dose of fludrocortisone as hydrocortisone has some mineralocorticoid activity. Just doubling hydrocortisone would suffice.

Addison's Disease (Primary adrenal insufficiency)

Remember that not all Addison's disease presents to the Emergency Department with an acute presentation of Addisonian crisis.

Most present with early presentations to primary care

Clinical features of early Addison's disease:

- Tired, malaise
- Dizzy on standing (postural hypotension)
- Hyperpigmentation
- Salt craving
- Nausea, abdominal pain
- No strength in limbs (difficulty climbing stairs)

Biochemical findings:

- Hyponatraemia
- Hyperkalaemia
- Blood glucose - borderline or low

This picture usually sparks more investigations such as 9am cortisol

- If < 100 nmol/L, diagnosis very likely
- If 100 - 400 nmol/L, refer for short synacthen test
- If > 400 nmol/L, diagnosis unlikely

Addisonian Crisis

Causes

- Withdrawal of chronic steroid therapy
- Infection or stress in a patient with chronic adrenal insufficiency

Clinical features

- Shock (confusion, postural hypotension, tachycardia and oliguria)
- Abdominal pain (may be severe enough to mimic an acute abdomen)
- Hypoglycaemia

Labs

- Cortisol and ACTH level
- FBC, U&Es, blood glucose
- Cultures (blood/urine/sputum)

Treatment

- IV fluids if shocked
- IV hydrocortisone 100mg
- Hypoglycaemia → IV glucose infusion
- If the condition improves in 72 hours → Switch to oral steroids
- If an adrenal pathology is identified → Fludrocortisone may be indicated

Hyperaldosteronism

Primary hyperaldosteronism or Conn's syndrome:

- Adrenal adenoma
- Adrenal hyperplasia
- Adrenal carcinoma

Secondary hyperaldosteronism

- Renin producing tumour
- Renal artery stenosis

Clinical features

- Hypertension
- Hypokalemia
- Metabolic alkalosis

Investigations

- Renin and aldosterone levels
- CT/MRI
- Adrenal venous sampling (gold standard for primary)

Treatment

- Aldosterone antagonist - spironolactone
- Surgery: adrenalectomy

PodsForDocs

Check out our podcast episode '*Add, Subtract & Don't Conn'* to further solidify your knowledge on Addison's disease, Cushing's syndrome and Conn's syndrome!

Click on the image below to head to our PodsForDocs podcast page to find out more.

Sheehan Syndrome

Postpartum hypopituitarism due to pituitary necrosis as a result of intra-partum or post-partum haemorrhage causing hypotension

Symptoms

- Absence of lactation
- Amenorrhea or oligomenorrhea
- Hypothyroidism
- Adrenal insufficiency

Labs

- Low TSH and T4
- Low ACTH and cortisol
- Low LH and FSH
- Low estrogen

Treatment is with specific hormone replacement

Note: Pituitary apoplexy due to pituitary tumour presents with acute headache, visual field defect and symptoms of hormone deficiency

Amenorrhoea

Primary: Never had menses till 13 years without secondary sexual characteristics and 15 years with secondary sexual characteristics

Causes

- Constitutional delay
- Ovarian failure
- Hypothalamic failure
- Kallmann’s syndrome and Imperforate hymen
- Congenital adrenal hyperplasia

Secondary: Absence of menses for 6 months in someone who have had menses before

Causes

- Pregnancy and lactation
- Menopause
- Premature ovarian failure and PCOS
- Pituitary and hypothalamic disease, and hyperprolactinemia

Amenorrhoea

Investigation

- **Primary ovarian failure:** FSH and LH increased
- **PCOS:** LH:FSH ratio is increased
- **Hyperprolactinemia:**
  - Pituitary tumour (MRI)
  - ↑ Serum prolactin level
- Hypothyroidism:
  - Low T3
  - Low T4

Treatment

- Hormone replacement therapy - ovarian failure
- Treatment focussed on specific condition

Amenorrhoea

Reading FSH, LH and prolactin if given in the exam

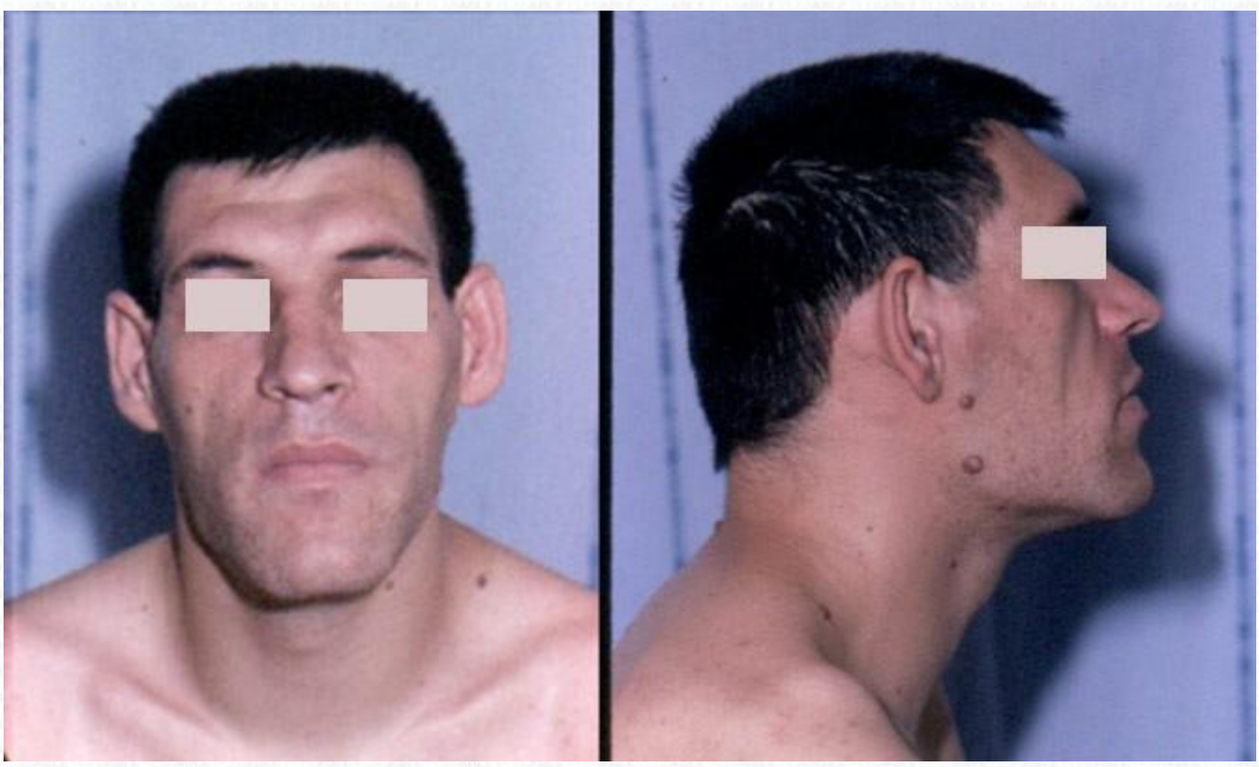
	FSH and LH	Prolactin	Extra info
Hypothalamic amenorrhoea	Low	Low	GnRH low Look for the very low BMI
PCOS	LH:FSH ratio is high (more than 3:1)	Normal	Although LH:FSH ratio is high, it is not used as part of the diagnostic criterion because of its inconsistency. FAI (Free Androgen Index) is used as part of the diagnostic criteria instead
Prolactinoma	-	Very high	-
Premature ovarian failure, menopause	Raised FSH	-	-
Post-pill amenorrhoea	Low-normal	Normal or mildly raised	Usually low-normal levels of FSH, LH or oestrogen

Acromegaly

↑ **Growth hormone** secretion due pituitary tumour

Clinical features

- Enlargement of hands and feet
- Frontal bossing
- Macroglossia
- Prognathism (enlargement of jaw)
- Coarse facial features
- Hypertension, cardiomyopathy and arrhythmias
- Type 2 diabetes mellitus



Acromegaly

Labs

- **IGF-1 (screening)**
- Oral glucose tolerance test to confirm
- MRI scan of pituitary

Treatment

- **Trans-sphenoidal surgery** is the treatment of choice
- Somatostatin analogues - octreotide
- GH receptor antagonist - pegvisomant
- Radiotherapy

Metabolic Acidosis

Arterial pH <7.35 and plasma bicarbonate <22 mmol/L

H+ excess:

- Ketoacidosis - Starvation and DM
- Lactic acidosis - Heart failure, Drugs
- Methanol, Salicylate and Ethylene glycol poisoning
- Renal failure and Type 1 RTA

Loss of bicarbonate:

- Diarrhoea
- Type 2 RTA
- Drugs: acetazolamide

Respiratory compensation immediately by hyperventilation

Renal compensation by increased bicarbonate absorption or H+ excretion

Metabolic Alkalosis

Arterial pH > 7.45

Causes

- Vomiting (loss of H+)
- Hyperaldosteronism
- Hypokalemia (To compensate K+ shifts outside and H+ shifts inside the cell)

- **Respiratory compensation** occurs immediately and involves decreased respiratory rate to increase PCO2
- **Renal compensation** occurs late and involves increased excretion of bicarbonate

Respiratory Acidosis & Alkalosis

Respiratory acidosis	Respiratory alkalosis
Hypoventilation ($PaCO_2$ > 45 mmHg)	Hyperventilation ($PaCO_2$ < 35 mmHg)
• Severe asthma or COPD • Suppression of respiratory center by drugs such as opioids • Obesity hypoventilation syndrome	• Panic attack • Aspirin toxicity (mixed respiratory alkalosis and metabolic acidosis)

Pseudogout

Brain trainer:

A 29 year old woman presents with a swollen, painful right knee joint.She also complains of constipation, feeling cold and weight gain. Joint aspiration of her knee joint will show what finding?

➔ **Positive birefringent crystals**

Hypopituitarism

- Pituitary adenoma → Most common cause
- Other causes include tumours, infections, stroke, radiotherapy, trauma, Sheehan's syndrome

LH ↓, FSH ↓
Amenorrhoea, infertility

GH ↓
Usually no symptoms in adults

TSH ↓
Hypothyroidism symptoms

ACTH ↓
Fatigue, hyponatraemia, hypotension

Prolactin ↓
Absent lactation

The hormones that are usually affected first are on top

Meaning LH, FSH followed by GH are the hormones to be deficient first

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