SURGERY

- GASTROINTESTINAL SYSTEM
- ENDOCRINOLOGY

2

5.00

ENDOCRINE

• INDEX •

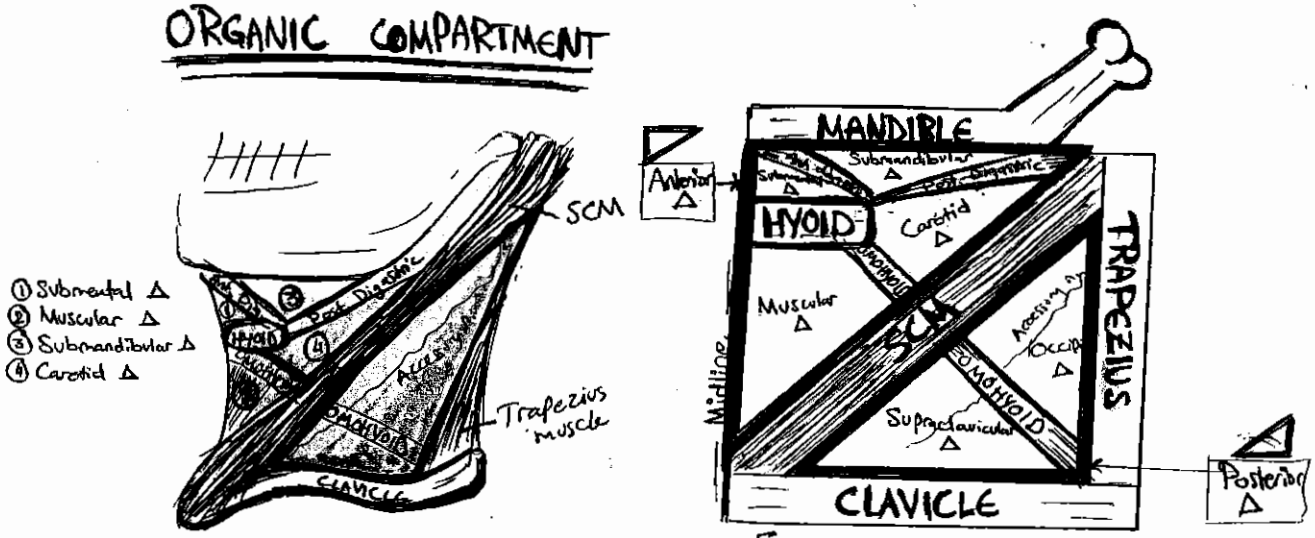
ANATOMY OF HEAD & NECK

Source : Dossier

NECK ANATOMY

- Anterior Compartment (ORGANIC) : 2 Δ (Ant. / Post.) separated by Sternocleidom.
- Posterior Compartment (MUSCULAR) — imp. in neurosurgery (ex. Disc surgery)

ORGANIC COMPARTMENT



[The Anterior compartment]
-Organic Δ-

Posterior Δ

- **Boundaries:** Ant. : Post. border of SCM (Sternocleidomastoid m.)
  Post. : Ant. border of Trapezius
  Inf. : Clavicle
- **Contents:** - L.N
  - Accessory n. (Innervates SCM & Trapezius)
  - Branches of the cervical Plexus
- **Subtriangles:** - Occipital Δ
  - Supraclavicular Δ

*** LAYERS: (From superficial → Deep)**

Skin — Subcut. tissue — Platysma — Superficial fascia — fat & L.N.

Muscular Δ — where the thyroid is present

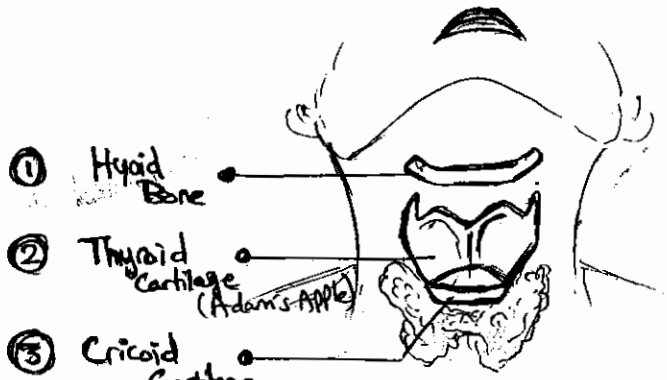
Contents: Strap muscles!

[Omohyoid
Sternohyoid
Sternothyroid
Thyrohyoid] They form the ant. coverage of the thyroid gland.

* Sternohyoid; lies superficial to sternothyroid m.

LANDMARKS IN THE MIDLINE

* Bony & cartilaginous landmarks:-



DEEP FASCIA OF THE NECK (Deep cervical fascia)

* 3 PARTS

① Pretracheal Fascia:

Fxn: holds the thyroid gland to trachea, esophagus & larynx

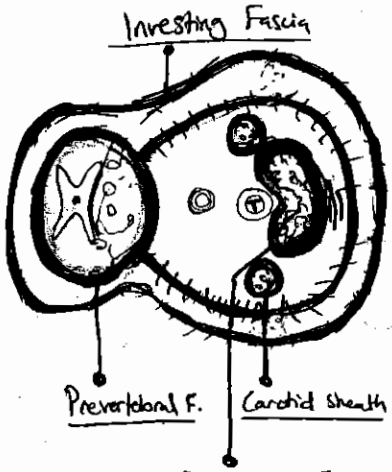
⇒ That's why the thyroid gland MOVES WITH swallowing!

② Prevertebral Fascia:

* Anything located ant. to the vertebra is located within this Fascia.
* Branches of cervical plexus run deep to it (the most imp. → Phrenic n. (C3,4,5))

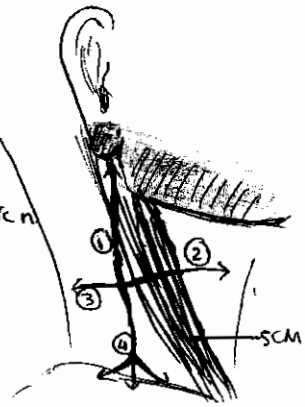
③ Carotid Sheath:

Contents: Common carotid art.
Internal Jugular Vein
Vagus n. (Posteriorly btw. art. & v.)



SENSORY INNERVATION OF THE NECK

It's mediated by the ant. cervical n. which comes from the cervical plexus.



* The branches emerges from **behind the SCM** forming a cross (+), in addition to the Phrenic n.

① **Greater Auricular n.** (Upward)
- * Innervates the skin of Parotid area & ear pinna
- * Runs along w/ ext. jugular v.

② **Ant. cervical n.** (AKA: Transverse cervical n.) (Ant.)

③ **Post. cervical n.** (Post.) — Innervates the Post. aspect of the neck.

④ **Supraclavicular n.** (downward)

* They're 3 in no. < ant. / Post. / Sup. } They innervate shoulder area.
- They share exit w/ Phrenic n. (Sharing C5)
  - * So Pts w/ gallbladder Problem will develop shoulder tip Pain as well!

PLATYSMA *NOTES*

- The platysma is attached to the clavicle & ribs (ant. chest) & superiorly, it's attached to the mandible & mastoid Post.
- It disappears in the midline
- It's innervated by the **cervical branch** of Facial n.
- It's also the muscle of facial expression
- Upon reaching the Parotid, it will slip to engulf the Parotid, forming a v. strong fascia (which is the **Parotid Fascica**)
  - → So any minimal swelling of Parotid dve to Parotitis or mumps will cause severe Pain (dve to strong fascia)
- The platysma also continues downward, it engulfs SCM also!

* Its not well developed in ♀, but in ♂ it's well-developed dve to the Process of shaving.

The End.

LYMPHATIC DRAINAGE OF HEAD & NECK

Source: Dossier 7

* It's important to know the primary drainage of each area in the head & Neck. **why?**
  bcz CA is transmitted first by lymphatic then by vessels.
* 1/3 of body's L.N are in the head & Neck !!
  ⇒ So it's rich in L.N & vessels → wounds heal quickly

L.N in Head & Neck

- **SUPERFICIAL GROUP** (Felt only under the skin)
  - Buccal
  - Facial
  - Preauricular (Parotid) – imbeded inside the Parotid g.
  - Auricular
  - Occipital
- **DEEP CERVICAL GROUP** (Around the internal jugular v.) → 6 Groups (Levels)
  - I
  - II
  - III
  - IV
  - V
  - VI

NOTES

- Site of inflammation/CA → Superficial L.N → Deep L.N
- If L.N is reddish & tender → THINK of inflammation
  Painless → THINK of malignancy.

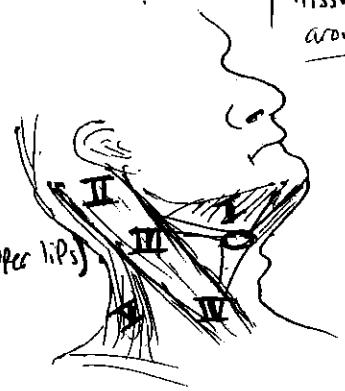
L.N are found in fatty tissue or plates around jugular.

DEEP L.N

(LEVEL 1) **GROUP I**

Ia) SUBMENTAL nodes → Drains → Midline (Tip of nose / mid of lower & upper lips)

Ib) SUBMANDIBULAR nodes → Drains → Nose, Sides of tongue



(LEVEL 2)

GROUP II UPPER Jugular n. (Jugulodigastric) – Behind the Post. belly of digastric

GROUP III MIDDLE Jugular n. (Jugular omohyoid) – Behind the omohyoid

GROUP IV LOWER Jugular nodes (Epithelio-cervical)

Below omohyoid

GROUP V Accessory

- Found in Posterior Δ
- Related to accessory nerve XI (hence the name!)

GROUP VI (Tracheo-esophageal or Paratracheal)

- Btw. trachea & cervical esophagus
- It drains thyroid & subglottic larynx

** So subglottic laryngeal CA (Below larynx) will metastasize to Paratracheal L.N.

NOTES

- Parotid L.N drains to → Buccal L.N to → Facial L.N

Deep L.N ← to ← Occipital L.N ← to ┘
(Submandibular L.N)

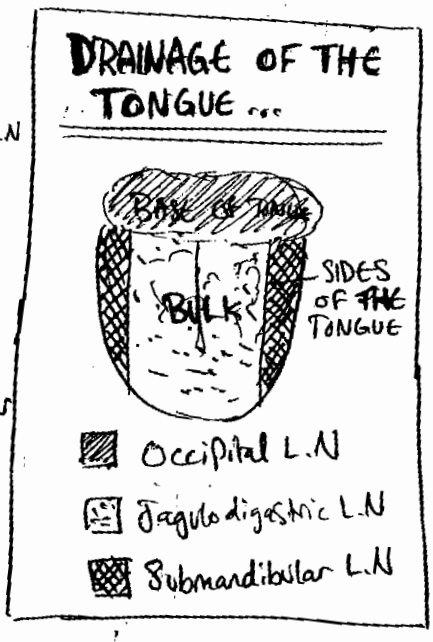
* Example: CA in the ear first metastasize to Parotid L.N then to submandibular then to deep cervical L.N

So drainage usually starts from superficial L.N → submandibular (I)

IV ← III ← Group II ←

⇓

This Pathway can be interrupted (or may be changed) by — Surgery
RTX
or enlargement (It will compress lymphatics)



NECK DISSECTION

TYPES
- RADICAL
- MODIFIED RADICAL
- SELECTIVE

① RADICAL DISSECTION

All L.N in levels I – V **PLUS**

Removal of Fat Plates
- SCM
- IJV
- Accessory n.

] muscle, vein, Nerve!

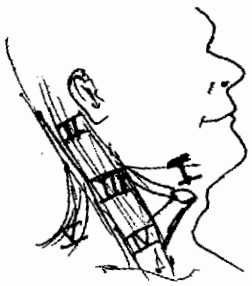
Indx:
- Extensive cervical involvement or matted L.N ω gross extracapsular spread.
- Invasion into SCM / IJV / or Accessory n.

② MODIFIED RADICAL DISSECTION.

Excision of L.N ω
SPARING ANY (or all) of non-lymphatic structures/

③ SELECTIVE DISSECTION

TYPES



- **Supraomohyoid**
  - Removal of Group I, II, III
  - ex. as in SCC (most of it are above omohyoid) so here, its effective in 70–80% of tumors of SCC
- **Anterior D.** (AKA ~~Extended~~ Supraomohyoid)
  - Removal of Group I, II, III, IV
- **Lateral**
  - Removal of Group II, III, IV
- **Posterolateral**
  - Removal of Group II, III, IV, V
- **Posterior**
  - Removal of Group V only (ex. Pharyngeal CA)
- **Central** (AKA median)
  - Removal of Group VI

COMPLICATIONS OF DISSECTION:

- If BOTH jugulars are removed → we'll have EDEMA
- If Accessory n. is removed → we'll have DROP shoulder (due to loose trapezius)
- If SCM is removed → Disfigurement (Cosmetic effect)
  - physiotherapy by exercising the other muscles.
  - It's removal won't affect the head movement.

Sentinal L.N

- It's the 1st L.N to drain the tumor
- To detect it, we inject the tumor w/ methylene blue & follow the stain till it reaches the FIRST L.N.
- Then, we excise the L.N & lay the Probe on it → It will give a peculiar sound (while if we point it to the neck → there will be No sound)
- → This confirms that we removed the SENTINAL L.N
- *It's sent to histoPathology to confirm negativity BUT if it's +ve → Neck Dissection is done!
- ***This method is only useful for small tumors! (as in breast surgery)

NOTES

- Supraclavicular L.N are found in supraclavicular fossa
  - → They're involved in malignancies of lung & breast NOT those of head & Neck!!
- Virchows L.N (Lt supraclavicular L.N) drains stomach & abd. CA
- Accessory L.N drains the Postnasal space (Group 5)
- Papillary thyroid CA will NOT metastasize above the hyoid or to Level I/II BUT drains to Group III/IV.
- Tonsils drain to Group II (Jugulodigastric L.N)

STAGING (TNM)

T_1 (< 3 cm)
T_2 (> 3 cm)] cure rate 90–95%

T_3 (> 6 cm)
T_4 (Bilateral)] cure rate 70–80%

[redacted]

- CT — accuracy 90%
- EUA (Examination Under Anesthesia)
- MRI — not routine (But better than CT)
- Bx
- FNA — usually performed on any enlarged L.N.

The End

BRANCHIAL ANOMALIES

Source : Dossier

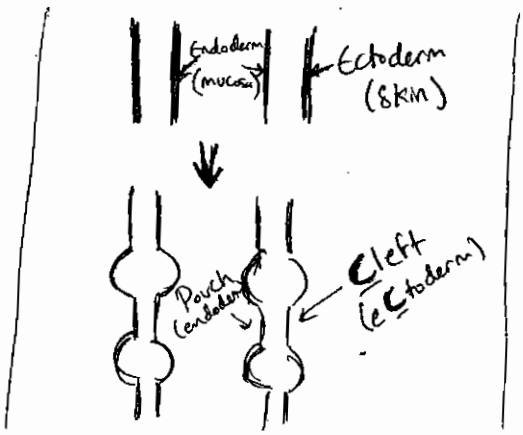
- **HEREDITARY/FAMILIAL** (Genetic)
  - Occurs due to abnormalities in the genes.
- **CONGENITAL**
  - Occurs due to failure in organs development & differentiation (Failure of organogenesis)
  - Usually occurs in the 1st trimester
  - Factors : Drugs / Radiation / Infan / Genetic abnormalities

NOTES

* Down Syndrome is a hereditary disorder BUT accompanied w some congenital anomalies in GIT & CVS.
* These disorders can be obvious at birth BUT can be delayed for years!

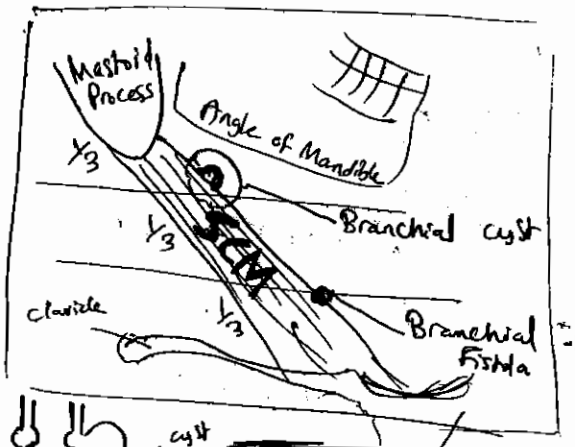
- During Embryogenesis, there will be BRANCHIAL ARCHES in the area of neck in the Pharynx
- Normally, these will disappear before birth w the **EXCEPTION** of 1st branchial cleft that will give → external Auditory Meatus
  - 1st Pouch that will give → Auditory (eustachian) tube
  - The area in btw. that will give → Tympanic membrane.
  - 1st arch → Bones of the middle ear.

DEVELOPMENT OF BRANCHIAL APPARATUS.



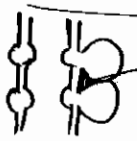
* If there are remenants after birth as fistula or ~~Cyst~~
  ↳ called **Vestigeal**
* The m.c site for Problems is the 2nd branchial app.

BRANCHIAL CYST

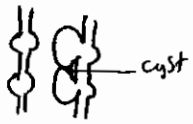


TYPES

- **Dermoid** (Ectoderm)
  - more common
  - derived from ectoderm
  - lined by skin
  - Contains Cholesterol (Yellow fluid → looks like Pus)



- **Mucous** (Endoderm)
  - derived from endoderm
  - lined by mucus membrane
  - Contains mucus sxn



DDX
- Parotid Swelling (superficial to SCM)
- Enlarged L.N (deep to SCM)
- Cold TB abscess (rare)
  - ↳ NO signs of inflam.

Site of Presentation

- In **Ant. Δ**, **deep** to SCM (so it disappears on muscle cxn)
- At the level of junction btw. upper & middle 1/3 of SCM

** Smooth surface & globular (Full of fluid!)
↳ can be aspirated

Age of Presentation

mostly in children. (BUT can appear at any age)

C/P

- It could be dormant & unnoticed, once there's inflammation (like sore throat or URTI) → enlarged lymph. & hyperxn
  ↓
  Enlargement of cyst!
- If inflammation was strong → suppuration & abscess formation.

Tt

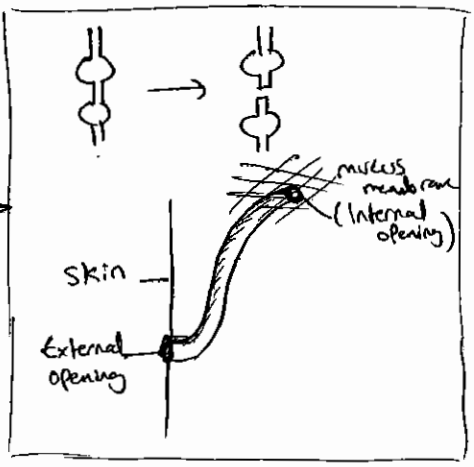
- If infected cyst → Give abx until infxn is resolved then do surgery
- If ineffective abx → do drainage & surgery

If the cyst was treated w I & D without removing the whole cyst ↓
① Recurrence (cos cyst)
② or fistula formation

BRANCHIAL FISTULA (Primary fistula)

.It's tract btw. 2 epithelial surfaces (ecto & endoderm) due to failure of 2nd branchial arch to grow caudally over the 3rd & 4th arches.

- During the development, Ectoderm grows & elongates more than endoderm → So tract won't be straight but OBLIQUE (see figure)



Site

Anterior Δ, it opens on the skin at junction btw. middle & lower 1/3 of SCM then extends as tract & open post. in the mouth in the supratonsillar region.

DDx

- Folliculitis (like Acne)
- Pilonidal Sinus

Age

Directly after birth
(But sometimes the opening is small & unnoticed!

on PE

ALWAYS feel the tract of fistula btw your fingers on PE → feels like **Firm, thin rope**

C/P

If Patent: mother brings her child c/o milk taken by mouth comes out from opening in the neck → easy dx!

If lumen of fistula is small (that could be obstructed) → Clear discharge & -rarely- Purulent discharge.

↳ Liable for infections!

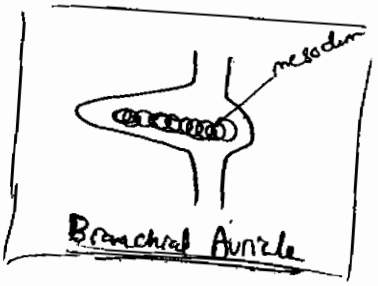
[BUT if the opening of fistula is obstructed → discharge won't come out even if it's infected.]

Tt Surgery! (By excision) – If incomplete → Recurrence!

CAUTION DON'T open it by Probe bcz this may damage the vessels & nerves in that area!

BRANCHIAL AURICLE (AKA Cervical Auricle)

— Occurs due to overProliferation of MESODERM that will manifest AFTER BIRTH as osseus or cartilage Protrusion.



The End
Erick Ephraim

NECK MASSES

APPROACH

— Pt Presenting @ neck mass ?

Hx

- **Pt Profile**
  - **Age**
    - <20 → THINK → Congenital > Infxn > Malignancy.
    - 20–40 → THINK → Benign thyroid swelling / Infxn / Inflam
    - >40 → THINK → Malignancy until proven otherwise!
  - **Gender** ♂ ×3 more risk to be malignant
  - **Occupation** (Ex.): In Benzene stations — CA of sinuses.
    In Crowded areas — TB
    Outdoor worker — Skin CA
    Radiation — Thyroid CA
- **Mass**
  - **Size** If >2 cm — must be investigated.
  - **Duration** (Roughly) If 7 days → THINK → Infxn
    7 months → THINK → CA
    7 years → THINK → Congenital
  - **No.** If multiple → THINK → Lymphoma
  - **Progression** If very rapid → THINK → bleeding in a cyst.
  - **Location** (Discussed Later): Midline vs. Lateral.
    & Ant. ▲ is more benign that Post. ▲
  - **Ass. Sx** Pain / URTI sx / Fever / wt loss.

The 7 CARDINAL SX:

1. Dysphagia
2. Odynophagia
3. Voice changes
4. Stridor
5. Speech disorder
6. Globus
7. Referred ear pain (via CN V, IX, X)

Facial n. invasion (manifested by Palsy) is an indicator of MALIGNANCY!

- **Aggrevating Factors** : ex. If ↑ size ⓦ Lemon / chewing THINK of submand/sublx obstruction (Stone)

- **PMHx** — ex. If hx of CA → most Probably its recurrence.
- **Social hx** :- Smoking (v.imp in head & Neck CA)
  It also ↑ risk of recurrence rate.
  - Alcohol
  - Travel hx
  - Animal Exposure.
  - Skin contact.
- **FHx** of Thyroid CA or MEN syndrome.

P/E — options:-

- Inspect all cutaneous & mucosal sites. — look for signs of inflammation.
- Examination under anesthesia (EUA)
- Indirect / Fibroptic Laryngoscopy.

Ulceration is CA until Proven otherwise!

* EXAMINATION of the mass:
  - Size / Site / Shape / Skin around it / Color / edges.
    Consistency (Soft - Firm - hard) / Fixation / Transillumination.

Inv*

- CBC (look for WBC ↑ ∴ diff.)
- FNA (if -ve → Repeat it)
- Contrast CT / MRI
- PET scan
- Triple endoscopy in metastatic CA
  1. Laryngoscopy
  2. Esophagoscopy
  3. Bronchoscopy
  + Blind bx

* Excisional bx is avoided ← UNLESS in lymphoma due to ↑ risk of distant metz

In Peds, we use U/S NOT CT/MRI
Why:
- Less radiation
- Less contrast exposure
- Less sedation

Here, CT done only if deep neck space infxn is suspected → retroperitoneal abscess.

CT Vs. MRI

CT	MRI
• To know the extent • Cystic vs. solid • Vascularity • Localize the tiny tumor • CT-guided bx	• Better for upper neck & skull base masses • Vascular delineation ∴ infusion.

DDx for Posterior ▲ Masses

If Solid : L.N

Cystic : – Cystic Hygroma
– Pharyngeal Pouch

Pulsatile : Subclavian Aneurysm.

MASSES
- CONGENITAL
- INFLAMMATORY / INFXN
- MALIGNANCY

CONGENITAL MASSES
- Midline
- Lateral

– Usually cystic
– Swell during URTI

Midline Masses

Sublingual Dermoid Cyst :

– It's congenital defect occurs during embryogenic development when skin layers don't grow probably together
– lined by epithelium
– Contents : Hair follicles / Sweat glands / Sebaceous cyst

Y3 of Congenital masses!

Thyroglossal Cyst

– It's failure of thyroglossal duct to obliterate after the embryological descent of thyroid from foramen cecum (at the base of the tongue) to the lower ant. neck.

on PE – moves ↑ swallowing (connected to ligament) & ↑ tongue protrusion (connected to hyoid)
– 50% present < 20 yrs old.

(imp.) — U/S. Should be done PREOP. to make sure that it's not the only functioning thyroid tissue.

— Rarely can transform to Papillary CA.

(!!!)

Sistrunk Procedure:

Resection of the cyst + the tract + Central Portion of Hyoid

Why? to ↓ Recurrence.

- Subhyoid Bursa
- Thymic Cyst
- Laryngocele
- Thyroid Nodule
- Pretracheal L.N
- Teratoma

Lateral Masses

- Branchial Cyst

1/3 of Congenital masses.

— The persistance of embryological Pharyngobranchial duct

— Can form cyst / sinus / fistula.

⟹ **m.c** from the (2nd) branchial cleft:

— C/P: Nontender, Fluctuant mass, ant. to SCM. ∵ a deep tract that travels btw. internal & external carotid art. to the tonsillar fossa.

⟹ If it was from the (1st) branchial cleft:

C/P: Near the angle of mandible or around the ear may be ass. ∵ facial n. / ear canal involvement.

RARE! ⟹ If it was from the (3rd) branchial cleft

C/P: Lower neck ∵ tracts that end in thyrohyoid membrane or Pyriform sinus. (4)

⏩ Carotid Art. Aneurysm.

⏩ Carotid Body Tumor

— locally invasive

on PE — Moves side by side (NOT up & down) ↔

— Transient carotid Pulse or can have its own Pulse

— Dx: Carotid Angiogram.

ttt: Surgical excision & Preop. embolization

⏩ Laryngocele.

⏩ Thyroid: MNG / Graves / Toxic MNG / Thyroiditis / Neoplasms.

Congenital Masses that can be midline or lateral:

* Cystic Hygroma

Lymph.-filled space arises from embryogenic remenant of the jagular Lymph. sac.

— NOT true cyst!
— Soft / Fluctuates / Transluminates / Lobular
— Painless
— Clear fluid
(ttt) Excision BUT ↑recurrence rate.

* Hemangioma

— Reddish-Bluish compressible mass
— Bruit on auscultation.
— ↑size w crying / Straining
— Maybe ass. w subglottic vascular malformation
— Grow rapidly in the 1st year of life.
— Slow involution at 18-24 m.
— 90% resolve w/out ttt ☺

Index for ttt
- Airway compromise
- Ulceration
- Eye Problems.
- Dysphagia
- Thrombocytopenia
- Cardiac failure

→ ttt by Steroids

* Pharyngeal Pouch

- Diverticulation in Pharyngeal mucosa.
- Bulge through weakness in the Pharyngeal constrictor muscle on the Lt side.
- common in elderly ♂
- C/P dysphagia
  Halitosis
  Swelling in the neck that gurgles.
- Dx Barium swallow.

* Lymphatic Malformation

on PE - Soft doughy
Compressable
Swell w/ URTI

Dx CT/MRI

tt Cosmetic or symptomatic relief;
* Complete excision is difficult/ b/c of its infiltrative nature.
So debulking maybe effective /sclerotherapy.

* Plunging Ranula

Ranula: is cystic mucosa extravasation from sublingual salivary gland.

Plunging: If extended through myelohyoid muscle.

(tt) Excision

INFXN / INFLAMMATORY MASSES

Cervical Adenitis.
- due to viral URTI
- Self-limited
- Generalized Lymphadenopathy

Suppurative bacterial lymphadenitis
- due to bact. (S. Aureus / Group A Strep.)
- Common in children.

Tx: IV abx
I&D if → Poor response to abx

Acute Mononucleosis
- due to EBV
- Young adults
- Involves cervical L.N
- Ass. w. fever / tonsillitis / hepatosplenomegaly

Inv: Monospot, EBV titers

Tx: Supportive!

Deep Neck space Infxn
- from dental infxn / tonsillitis / trauma / suppurative L.N
- m.c organisms: - Strep. & Staph.
  - oral anaerobic bact.

Ludwig Angina
Cellulitis of sublingual & submandibular spaces → compression of lymphatics → edema → airway obstruction.
Tx: Airway control + IV abx

Neck Abscess
CP: fever, Acute Neck swelling, Induration, Dysphagia / odynophagia / stridor, Redness / tenderness.

Tx: IV abx
I&D

Sialadenitis / Sialolithiasis

Other inflam.
- Sarcoidosis
- Kawasaki d.
- Low ant. midline mass → thyroglossal duct cyst

Other infxns
- Cat-scratch d.
- Atypical mycobact.
- HIV (diffuse hyperplastic lymphadenopathy)

➤ NEOPLASTIC MASSES

BENIGN:
- ✱ Paraganglioma: Vascular tumor from paraganglionic cells of aut. nervous syst.
  - ex. Carotid body tumor (m.c)
  - (tt) Surgical excision ± preop. embolization
- ✱ Lipoma
- ✱ Schwanoma
- ✱ Infiltrative Fibromatosis
- ✱ Neurofibroma
- ✱ Salivary gland neoplasms

MALIGNANT:
- ✱ Metastatic SCC (m.c) — adults.
- ✱ Lymphoma
- ✱ Thyroid CA
- ✱ Adenocarcinoma.
- ✱ Tonsillar SCC

● Location of mass is suggestive of Primary Site → based on Pattern of lymphatic drainage.

Lymphoma
- Hodgkin 85% → Painless cervical L.N
- Non-Hodgkin (dx) → ↓ surgery (tt) CTX ± RTX
- Bulky matted
- Painless

ex.
- Oral cavity CA — mtz to → Submandibular
- Lateral mt. SCC — mtz to → Level II, III
- Nasopharyngeal / Scalp mtz → Post.
- Papillary CA mtz → any level of the neck.

NOTE

Supraclavicular L.N usually from infraclavicular source; mostly from GIT — Virchow L.N / Scalene L.N.

Saba Faradat
The End.

SALIVARY GLANDS

Source: Dossier 25

- ANATOMY.
- INFECTIONS.
- MALIGNANCY.

2 GROUPS

- **MAJOR**
  - Parotid (The largest)
  - Submandibular
  - Sublingual (The smallest)
- **MINOR** — Distributed all over the oral cavity EXCEPT upper surface of the tongue.

Dryness of the oral cavity doesn't usually happen after removal of the major glands.
Why?
Bcz minor SG are enough to moisture the mouth.

CAUSES of xerostomia (Dry mouth)
- Sjögren's Synd. (Autoimmune)
- RTX (for tt of Head & Neck CA)

ANATOMY

① PAROTID GLAND — The LARGEST Salivary GLAND

Location: Located in the Preauricular area, wrapped around the mandibular ramus, superficial to masseter muscle.

****It has tail that extends to the neck overlying SCM.

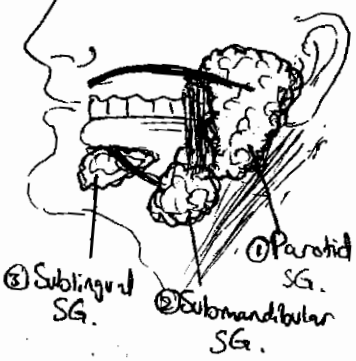
*** DDx for a mass BELOW the ear Pinna
- Parotid Gland (superf. to SCM)
- Deep cervical LN (deep to SCM)

diff. by Palpating the mass while in action.

Shape: Upside-down Pyramid

Fascia: enclosed w/in continuous strong capsule (Deep cervical fascia)

Lobes — Large **Superficial** (85% of the gland)
Small **Deep** (15% of the gland) — In close Proximity to the Pharynx.



Duct Stenson Duct
(Passes anteriorly & **enters** the buccinator at a sharp angle & **opens** opposite to the **2nd upper molar**)
↳ where we have to detect & palpate!

Sxn: Mainly **Serous**

Other Structures (Relations) The **Facial n.** Passes through the Parotid g. BUT WITHOUT innervating it & the nerve divides into **5** branches: — Temporal, Zygomatic, Buccal, Mandibular, Cervical } These divide the Parotid gland into superf. & deep lobes.

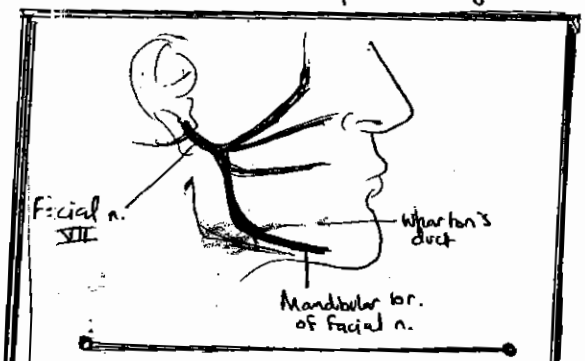
NOTE
Deep lobe of Parotid is in close Proximity to the Pharynx — so tumors in it are seen first through the mouth as intraoral Pharyngeal mass.
↳ 10% of Parotid CA are in the deep lobe.

② SUBMANDIBULAR GLAND

- **Location** Found in the submandibular
- **Duct** - Wharton's duct
  - —Opens at BOTH sides of frenulum
  - *So on P/E, inspect there! & do bimanual palpation of the duct, you might feel a **stone** (which can be operated through the mouth not the ✱ face → better cosmosis!)
- **Sxn** Mixed (mucoserous)
- **Relations** w/ mandibular branch of facial n. VII & w/ lingual n. (branch of mandibular branch of trigeminal)

NOTE

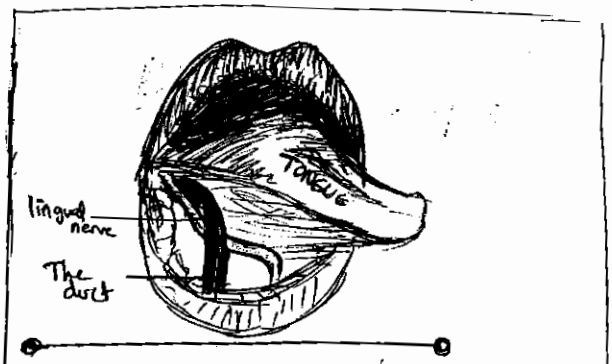
Submandibular gland is supported by **myelohyoid** muscle. So if it's lax (as in elderly) → leads to **Ptosis** of the gland — which appears as mass in the neck (NL mass!)



✱✱ Mandibular branch of Facial n. is related to the gland at the point it crosses the facial art. & v.

- The nerve is located **beneath** the **skin** & Platysma.

✱ If Be careful! might be injured during surgery.



Lingual n. crosses: the **LATERAL** surface of submandibular duct & then winding **BELOW** it, passes upward & forward on the medial side)

Then BOTH the lingual n. & the duct pass in the floor of the mouth where it (lingual n.) passes w. lingual art. & vein!

③ SUBLINGUAL GLAND — The SMALLEST of major salivary glands.

Location Lies deep to floor of oral mucosa (btw. mandible & genioglossus m.)

Duct Wharton's duct or separate duct

Sxn Mucinous **Relations** Lingual n.

Notes

- ✱ Enlargement appears as swelling in the floor of the mouth — **RANULA**
- ✱ Drained through 20 ducts!
  - most of them draining by submandibular duct.
  - others drain through the oral cavity.

RANULA

- Sublingual retention cyst arising from sublingual g.

Other definition

- Any cystic tumor of the undersurface of the tongue or floor of the mouth.

● COMPARISON BETWEEN SEROUS & MUCINOUS SXNS

TYPE OF SXNS	DENSITY	PROTEINS	IMMUNOGLOBULIN
SEROUS	Light	−ve	−ve
MUCINOUS	Thick	+ve	+ve

Sxns

PAROTID : (Mainly) Serous
SUBMAND. : Mixed
SUBLINGUAL : (Mainly) Mucinous
MINOR SG : Mixed

* Serous sxn means LESS possibility of stone formation compared to mucinous.

INFECTIONS OF THE SALIVARY GLANDS

- **ACUTE :**
  - **Viral** (ex. Mumps)
    - more common in children
    - Self-limited
    - Diffuse inflammation
    - May be ass. w Pancreatitis / orchitis / oophoritis
  - **Bacterial** (ex. Staph.)
    - Dryness of mouth is a risk factor
    - Ascending infxn
    - Seen in elderly
    - Post op - ↑ common in Parotid.

- **CHRONIC :**
  - Usually autoimmune
  - Inflammation causes destruction of glands.
  - 90% in ♀ (35-45) yrs
  - 60% ass. w SLE / RA / or Scleroderma.

① PAROTITIS (AKA : Parotid Sialadenitis)

CAUSES
- Viral — mumps
- Bacterial — Staph. (m.c)
- Autoimmune — Sjögren's Synd.

RF
- Dryness of oral cavity (Usually in elderly after major surgery)
- Loss of immunoglobulin inside gland (serous) — makes it more vulnerable to bact. → high mortality & morbidity

C/P
- Pain : as it's surrounded by dense capsule which will make a pressure on gland ⊕ its swelling] Severe Pain!
- Tenderness / hotness / Redness / ↑ swelling at site of Parotid w Fever

Dx

Mainly by hx & P/E

Mgt

. tt of ~~inflammatory~~ infxn → Antibiotics (↓ mortality from 50% → 20% BUT it's still high!

• Imaging studies (Done AFTER removal of infxn)
  - Plain X-Ray – If stone is radio-opaque
  - CT – If stone is radio-lucent
  - Sialogram – By injection of substance opaque on X-Ray.

• Invasive Procedures (Aspiration) – If complicated abscess.

② SUBMANDIBULAR GLAND INFXN

CAUSES mostly 2ry to OBSTRUCTION (Due to Stones)

C/P Typically, Swelling in the gland upon eating

(We eating ⊕ sxn of saliva BUT as the duct is obstructed → saliva will accumulate causing swelling!) → The swelling will disappear (Self-limiting)

Dx hx & P/E

X-Ray of the Floor of the mouth – for confirmation

~~tt~~ Surgical excision (Depends on the site of stone)

SITE OF STONE	INTRA-ORAL (Distal to lingual n.)	NEAR THE GLAND – at the hilum (Proximal to lingual n.)
SITE OF SURGERY	As the stone is DISTAL to nerve, its unlikely to injure it → so cut done INSIDE the oral cavity (better cosmetic app.)	As the stone is PROXIMAL to n., ↑ risk of injury so its preferred to do cut in the neck & remove all the gland!
EXCISION	Of the stone	Of the gland
ANESTHESIA	Local	General
COMPLICATIONS	Fistula (Post-op cuz it opens in the oral cavity)	Injury of mandibular branch of facial n.

Px

Submandibular gland has POOR recovery after infxn & Chronicity is common!

Beaded Duct

Alternating narrowing & dilation due to CHRONIC inflam.

Dx by CT & sialogram used to detect any small stones (appears as filling defect).

④

COMPARISON BETWEEN PAROTID & SUBMANDIBULAR STONES

	PAROTID GLAND	SUBMANDIBULAR GLAND
• STONE FORMATION	Rare due to ↓	Common due to ↓
• CAUSES	. Serous sxn . Duct is sloping down so movement of saliva is ↓ gravity . -ve suction by cavity produced by cheeks & gums	. Mucinous sxn forms a nidus for Ca^{+2} deposition & stone formation . Flow of saliva is against gravity . LONGER duct
• TYPE OF STONES	Radiolucent (NOT seen by X-Ray)	Radio-opaque — contains Ca^{+2} (Seen on X-Ray)

③ SUBLINGUAL & MINOR SG DISORDERS

- Minor mucus retention cysts develop in the floor of the mouth from obstructed minor SG or sublingual SG.
- Ranula is a term applied to mucus extravasation cyst arising from **sublingual gland**.
- ~~tt~~ of Ranula → Excision of cyst & affected gland.

TUMORS OF SALIVARY GLANDS

- Age group 40's (middle age) — BUT can occur at any age.
- MOST of them are **BENIGN**! ☺

80% of SG tumors arise in the Parotid Gland

- The m.c tumor of salivary glands is **Pleomorphic Adenoma** (70% of all tumors of SG)
- The smaller the gland → The higher risk to be malignant!

So the risk of malignancy (NOT the incidence) is as follows:- → Parotid 25% — Submandibular 50% — Sublingual 50% → minor 75%!

Warthin's tumor

Salivary tissue in a L.N

NEOPLASMS OF SG

BENIGN	MALIGNANT
• Pleomorphic adenoma (m.c) • Warthon's tumor (Adenolymphoma) • Tumor due to viral infxn! • Tumor due to bact. infxn! (cat scratch d.) • Monomorphic Adenoma	• Mucoepidermoid • Lymphoma • SCC • Adenoid cystic Adenoma • Acinar cell tumor

30 * In details *

Pleomorphic Adenoma

: Proliferation of Epithelial / Myoepithelial / Stroma tissue (resemble cartilage & bone)

- m.c tumor of the salivary glands (70%)
- BENIGN! ☺

histology:
- Doesn't have a complete capsule
- It has protrusions coming out of holes in the capsule.

* Recurs if not managed properly!
  - → Recurrence ↑ possibility to damage the facial n. on a 2nd operation.
  - → Recurrence requires 5 yrs from the time of OP.

CAUSES of Recurrence:
- Protrusions
- Multicentric tumor in 5% of cases.

imp.
! Can become malignant if NOT managed Properly AND remained > 10 yrs! (Risk of CA ↑ 1-2% per yr)
- Risk is 2-10%
- usually adenocarcinoma

gross appearance: Irregular / round-ovoid mass / well-defined borders

Warthin's Tumor (AKA Papillary cystadenoma)

occurs ONLY in Parotid!

PURELY BENIGN (Does NOT change to CA) — 2nd m.c benign tumor of the salivary gl.

- Usually appears at the tail of Parotid.
- cystic mass
- 10% Bilateral
- ass. w. smoking
- more in ♂ (90%)

MALIGNANT TUMORS

LOW GRADE
- Mucoepidermoid
- Acinar cell tumor

HIGH GRADE
- Mucoepidermoid
- Adenocarcinoma ♀ (rare, aggressive)
- SCC ♀
- Adenoid cystic adenomas.

MUCOEPIDERMOID
- The m.c malignant tumor
- 3 grades: LOW (majority) / MEDIUM / HIGH
- → ** It's specific for PAROTID GLAND

LYMPHOMA
Very common CA
2 forms: localized / Generalized.

SCC (Squamous Cell Carcinoma)

. The m.c.c of it in Parotid is **Metz** from skin (esp. in Australia & Icelands)

BUT in our countries it's more of Primary origin.

Adenocystic Adenoma — 2nd m.c malignant tumor

BUT
. m.c malignant tumor in the SUBMANDIBULAR & MINOR SG
. well defined BUT NOT capsulated
. It metastasizes to **NERVES** in 25–30% of case
So be sure there's no nerve injury!

has tendency for metz ex. as lungs!

. 3 TYPES
- Tubular — invades in 10% of cases nerves
- Cribriform
- Solid — The **WORST** type! (cuz it has the highest propensity to invade nerves — in 50% of cases)

The m.c tumor of **Parotid** in Children is HEMANGIOMA (non-epithelial tumor)

The m.c malignant tumors of the glands: –
Parotid — mucoepidermoid
Submandibular, Minor glands — Adenoid cystic Adenoma.

Modalities of tt (depends on the grade)

- **LOW GRADE**
  - CTX — in lymphoma
  - RTX
  - Surgery
- **HIGH GRADE**
  - Radical surgery
  - Neck Dissection
  - Postop. RTX

Surgery

. Total Parotidectomy: Removal of BOTH superf. & deep lobes w Preservation of facial n.

. Superficial Parotidectomy: Removal of superficial part of the Parotid (as in case of Low-grade mucoepidermoid)

. Partial Parotidectomy: Removal of the diseased segment (Enucleation)

. Radical Parotidectomy: Removal of the Parotid PLUS Facial n., muscle & fascia.

.Neck Dissection

Indx . HGSGT (High Grade Salivary Gland Tumor)

- LGSGT ẅ metz to L.N
- Ulceration of overlying skin
- Invasion of facial n.
- CA ẅ size > 5cm

.Postop. RTX Recurrence.

CTX is NOT effective! except in lymphoma

Indx same as Indx of Dissection.

Signs & Symptoms of Parotid malignant CA

1. Facial n. Palsy ẅ a mass (Th only significant indx of tumor)
2. Pain ẅ a solitary mass (Indicates advanced stage)
3. Mass Plus L.N involvement at the same side
4. Recurrence at the same or contralateral side is 100% indx of malignancy!

COMPLICATIONS OF SURGERY:

1. **Facial Nerve Palsy**
   - Neurapraxia; Injury of neural tissue BUT covering is still intact. — Regeneration occurs & it returns to NL.
   - Complete injury.
2. **Cosmetic Effects**
3. **Recurrence**
4. **Fistula** (c/p discharge – esp. ẅ eating)
5. **Frey's Synd.** (Gustatory Sweating)
   - c/p flushing, Pain, & diaphoresis in the auriculotemporal n. distribution initiated by chewing.
   - CAUSE: cutting the auriculotemporal n.

Nerve Injury	
Temporary	Permanent
30%-40% due to stretching of the n. during surgery. Needs 6m. to return to NL	Cutting n. completely (but) graft from greater auricular n. (But requires time to return to NL)

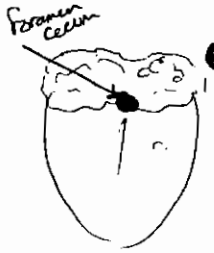
Alhamdulillah The End.

THYROID GLAND

Source: Recall Dossier Washington

EMBRYOLOGY

Endoderm



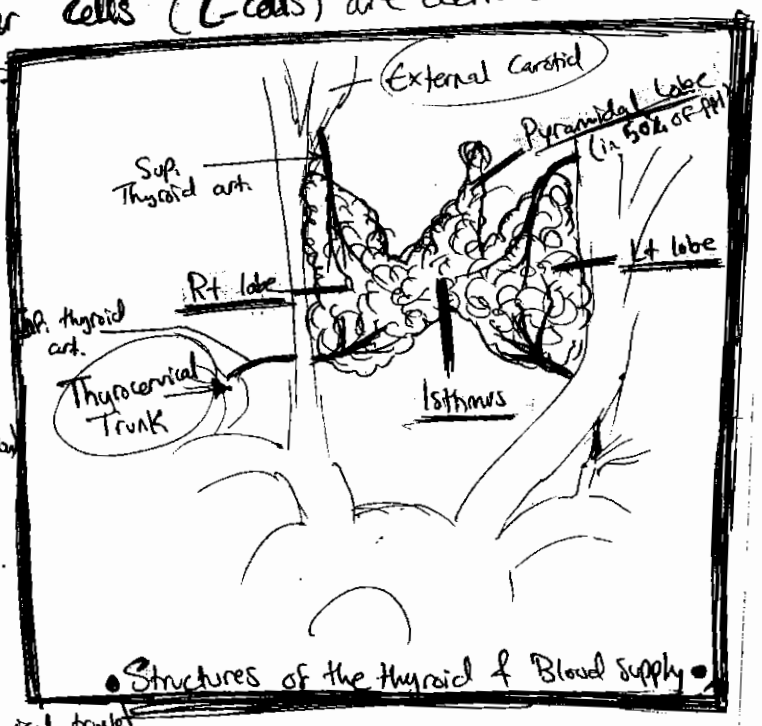
- The thyroid gland appears as an epithelial proliferation in the floor of the Pharynx at the base of the tongue at **Foramen Cecum**,
  So it starts from there & descends ant. to the hyoid bone & laryngeal cartilage (During migration, it remains connected to the tongue by a narrow canal, the thyroglossal duct!)

Thyroxin starts to be secreted by the embryo at 20 wks of gestation.

** **Parafollicular cells** (C-cells) are derived from neural crest

ANATOMY

- **Structures:**
  - 2 lobes
  - Isthmus
  - Pyramidal lobe (in 50% of Population)



• Structures of the thyroid & Blood supply •

- **Blood Supply**
  - Superior thyroid art. (1st br. of ext. carotid art.)
  - Inferior thyroid art. (Branch of thyrocervical trunk)
  - ± IMA (Innominate art.) — Rare! (3%) ↳ from aorta/innominate art.
- **Venous Drainage**
  - Superior thyroid v.
  - **Middle** thyroid v.
  - Inferior thyroid v.

The L.N group around Pyramidal lobe is called **Delphian** L.N group.

Tubercle of Zuckerkandl: The most Post. extension of the lat. thyroid lobes

The ligament that connects the thyroid to trachea is called → Lig. of **Berry**

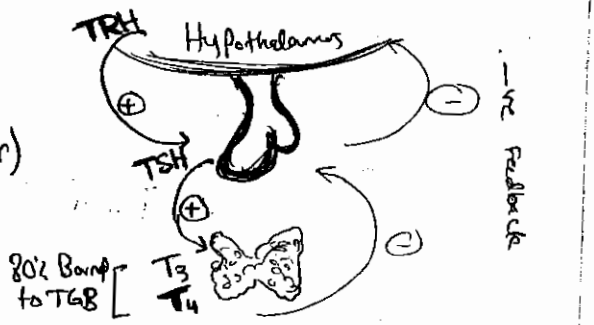
* During thyroidectomy, you first see the Platysma & then ant. jugular v.

⚠ Becareful during Surgery ..

- **Recurrent Laryngeal n.** → (found btw. trachea & esophagus) – Behind cricothyroid m.
  - 1 cm ant. or post. to inferior thyroid art.
  - If injured – unilat. → hoarseness; Bilat. → airway obst.
- **Sup. Laryngeal n.** → If damaged, Pt will have deeper & quieter voice (unable to hit high pitches!)

PHYSIOLOGY

- T_3 is the ACTIVE form.
- The m.c. site of conversion ($T_4 \rightarrow T_3$) is **PERIPHERAL** (liver)
- T_4 is purely from the thyroid ..



*Parafollicular cells (C-cells) secrete **Calcitonin**

- Screening by **TSH** (sensitive)
- NL values

TSH	0.5 – 5
T_4	9 – 19

} JUH lab values

*Levothyroxine is? T_4!
↳ $t_{1/2}$: 7 days

NOTES

- (INVX) . TFT
  . U/S
  . Uptake
  . Scan
  . FNA & Bx
- **UPTAKE** measure fxn (no.) while **SCAN** assesses anatomy (pic.)

THYROID NODULE

- % of PPl having a thyroid NODULE → 5%

- **DDX of a thyroid nodule:**
  - Adenoma / Hyperfxning adenoma
  - Multinodular goiter
  - Cyst
  - Thyroiditis
  - Carcinoma / Lymphoma
  - Parathyroid CA

- **Types of NON-thyroid masses:**
  1. **Inflammatory** lesions (lymphadenitis/abscess)
  2. **Congenital** lesions (thyroglossal duct - midline; branchial cleft - lat.)
  3. **Malignant** lesions (lymphoma / mets / SCC)

- **Studies done to evaluate a nodule**
  - U/S — Solid or cystic nodule
  - FNA — for cytology
  - ^{123}I scintiscan — Hot or cold nodule.

Index for Scan nodule is → ↓TSH

FNA is the DIAGNOSTIC test of choice for thyroid nodule.

% of **false -ve** result on FNA is 5%!

- **Approach**
  - Hx & P/E
  - Then,

imp.

Do **U/S**

- Non-suspecious → Consider **TSH**
  - NL/↑ → FNA
  - ↓ → **SCAN**
    - Hot
    - Cold → FNA
- Suspecious → FNA (Regardless size / type / TSH)

(HOT vs. COLD)

Hot — ↑I-uptake ≡ fxning / Hyperfxning

Cold — ↓I-uptake ≡ Non-fxning nodule

Notes

- **HOT** nodules are **NEVER** malignant ☺ So we don't do FNA for hot nodules.
- The MAJORITY of nodules are cold & the MAJORITY of cold nodules are BENIGN! BUT you should always r/o CA in cold nodules.

MULTINODULAR GOITER (MNG)

Plummer's disease is toxic MNG

MNG

NON-TOXIC (Simple MNG)

- Common, ♀ > ♂
- Asymptomatic (non-toxic), euthyroid w/ NL TSH
- **CP**: Goiter or pressure sx
- **Indx**
  - Symptomatic compression
  - Cosmetic
  - If you can NOT r/o CA
- **tt**
  - Ablation w/ I^{131}
  - or Bilateral subtotal thyroidectomy

Cold nodule in MNG has same risk of CA as a solitary cold nodule

TOXIC

- Fxning! ass. w/ thyrotoxicosis
  - ↓TSH
  - ↑T_4/T_3
- Scan shows ≥ 1 nodule

tt

- Antithyroid meds (temporary)
- Ablative I
- Surgery

Indx
- Compression Sx
- Refractory to I ablation

THYROID CA

- Evaluation (Suggestive for thyroid CA)

Hx:
- Neck Radiation
- FHx (Thyroid CA, MEN II)
- Extremes of Age (esp. children)
- ♂ > ♀

Signs:
- Single nodule
- Cold nodule
- ↑ Calcitonin levels
- Lymphadenopathy
- Hard, immobile nodule.

Sx:
- Voice change (Vocal cord Paralysis)
- Dysphagia
- Discomfort (in neck)
- Rapid enlargement.

25% of cold nodules are malignant

ONLY 1% of multinodular masses are malignant.

If pt came w/ thyroid nodule, hx of radiation BUT -ve FNA, most experts would REMOVE it surgically.

MOST thyroid CAs are EUTHYROID!

Work up
- **FNA** (the most imp.)
- US
- TSH
- Ca level
- CXR
- ± I^{123} scan.

If a pt has medullary HYPERPLASIA the risk of malignancy (Medullary CA) is 100%!!

Oncogens ass. w/ thyroid CA:
- Ras gene family
- RET Proto-oncogene

TYPES OF CANCERS

WELL-DIFFERENTIATED

- Papillary CA — 80%
- Follicular CA — 10%
- Hürthle cell CA — 5%
- Medullary CA — 5%

POORLY-DIFFERENTIATED

- Anaplastic 1-2%
- Lymphoma

Thyroid CA is more common in ♀ BUT if a nodule is found in ♂, there is higher risk in ♂ to be malignant!

• PAPILLARY CA

- The m.c thyroid CA (~80%)
- **Avg. Age**: <40 yrs (m.c btw. 30-40)
- **Gender**: ♀ > ♂ (2:1)
- **Environmental RF**: Radiation
- **Histology**: Psammoma Bodies
- **Route of spread**: Lymphatics
- **Rate of spread**: very Slow
- **I^{131} uptake**: Good uptake! (cuz it's differentiated)
- **Prognosis**: Good! (10-yr survival ~95%)

Papillary CA: (7 Ps)

1. **P**opular (m.c - 80%)
2. **P**sammoma Bodies
3. **P**alpable L.N
4. **P**ositive I^{131} uptake
5. **P**ositive Px ↓
6. **P**ostop. I^{131} scan (dx/tt)
7. **P**ulmonary mets

In Papillary CA, +ve L.N does **NOT** affect the Prognosis.

Tumor marker of Papillary CA is Thyroglobulin (tt)

IF <1.5 cm (some say <1 cm) **w/out** hx of radiation exposure ➡ Options:
- Thyroid lobectomy + isthmectomy
- Near-total thyroidectomy
- Total thyroidectomy

IF >1.5 cm (>1cm)/Bilateral/ +ve L.N /or hx of radiation exposure ➡ Total thyroidectomy

If Postop. (total thyroidectomy) TSH wasn't high enough, this means that there is remenant tissue! Postop I^{131} won't be effective

* Neck dissection: — only if +ve L.N
  - If lateral Palpable cervical L.N → Do **modified** neck dissection
  - If central → Do **central** neck dissection (selective)

Prophylactic neck dissection is NOT recommended in Papillary CA.

Postop. (after total thyroidectomy)

we don't give thyroxine replacement ~~tt~~ directly, we wait for 4-8 wks then we measure TSH, if it's sky high (>30), we start Iodine ablative therapy then we start thyroxine (1-1.5 mg/kg/day)

Postop I^{131} scan can locate residual tumor &distant mets that can be treated w ablative doses of Iodine.

• FOLLICULAR CA

~10% of all thyroid CA

- **Age:** Rare BEFORE age of 30!
- **Sex:** ♀ > ♂
- **Route of transmission:** Hematogenous (mostly to Bone, lung, liver) — more aggressive than Papillary
- **I^{131} uptake:** Good uptake ~~more its used~~
- **Histologic findings:** Capsular or blood vessel invasion
- **Prognosis:** worse than Papillary (10-yr survival: ~85%)
- FNA is NOT diagnostic — Tissue structure is needed.

(tt)
- Total thyroidectomy
- Postop. tt option: Postop. I^{131} (Ablation) Scan for dx & tt.

Follicular CA: 5 F's
Faraway mets (hematogenous)
Female (♀ > ♂ 3:1)
FNA — NOT it can NOT dx CA
Favorable Px ~85%
Favorable uptake

HÜRTHLE CELL CA

≡ It's a thyroid CA of the Hürthle cells (oxyphilic cells)
~5% of thyroid CA
Cell origin: Follicular cells
I^{131} uptake: NO uptake
Dx: FNA can identify cells BUT can be determined ONLY by histology! (like Follicular CA)
Route of mets: Lymphatic > Hematogenous
Px: 10-yr survival rate ~80%
(tt): Total thyroidectomy

MEDULLARY CA

Mnemonic:
MEN II found in MEDULLary

Medullary: 5 M's
- MEN II
- aMyloid
- Median L.N dissection
- Modified dissection (If +ve lateral L.N)

Calcitonin is a tumor marker

- The ONLY one that is **Parafollicular cell** in origin. (C-cell)
- ~5% of thyroid CA
- **Histology:** aMyloid (=Medullary)
- **I^{131} uptake:** Poor! (cuz it's Parafollicular)
- **Dx:** FNA
- **Sex:** ♀ as ♂ (1.5:1)
- Spread to L.N → Liver/Lung/Bone
- Ass. w/ **MEN II** (Pheo + Parathyroidism) → here, you should treat Pheo before treating the CA
- Ass. gene mutations: RET Proto-oncogene
- **Px:** 10-yr survival rate ~80% w/out L.N involvement; ~45% w/ L.N
- (tt): Total thyroidectomy + median L.N dissection (If +ve lateral L.N, do modified radical dissection)

What stimulation test can be done in medullary CA?
Pentagastrin stimulation Test

ANAPLASTIC CA

- **POORLY** differentiated.

~1% of all thyroid CA

Sex: ♀ > ♂

Histology: Giant cells, Spindle cells

I^{131} uptake: V. Poor uptake

Dx: FNA

MAJOR DDx → Thyroid Lymphoma which has much better Px!

Anaplastic is an undiff. CA arising in ~75% of Previously diff. thyroid CA. (most commonly from follicular CA!).

Tt Palliative!

If small tumor → Total thyroidectomy + RTX or CTX

If airway compromise → Debulking surgery & tracheostomy + RTX or CTX

Px BAD!

most Pts Present at stage **IV**

3% alive at 5 yrs! — They usually dive <u>win 6m.</u> after dx

COMPLICATIONS OF THYROID SURGERY

1. **Hemorrhage** (~6 hrs PostOP.) — CP SOB Postop.

Mgt ABC then Hematoma evacuation

2. **Hypocalcemia**

Usually transient, due to Parathyroid blood supply compromise.

* During surgery, Parathyroid gland should be Preserved & Blood supply must be Preserved.
So we usually take Parts of the Parathyroid gland & autograft it into the **SCM** or forearm.

3. **Recurrent laryngeal nerve injury** ~<1%

"Lateral aberrant rest" of the thyroid is **Papillary** CA of L.N from mets

DDx of Postop SOB after thyroidectomy

- Hematoma
- **Bilateral** recurrent laryngeal n. injury.

BENIGN THYROID DISEASE

HYPERTHYROIDISM

S&S ↑Appetite / wt loss
Tremor / Anxiety / restlessness / Palpitations
Heat intolerance
Diarrhea
Thrill over superior thyroid art.

GRAVE'S DISEASE

m.c.c of Hyperthyroidism.

Definition (Characterized)
Diffuse goiter w/ hyperthyroidism
Exophthalmos } characteristic!
Pretibial myxedema

CAUSE
Circulating antibodies that ⊕TSH receptors on follicular cells → deregulated production of thyroid hormone (i.e. ↑hyperthyroidism)

On PE
- Eye disease
  - Diplopia
  - Corneal ulcers
  - Proptosis
  - Chemosis
  - Periorbital edema
- Clubbing
- Pretibial myxedema

♀ ≫ ♂ 6:1

Dx
↑T_3, T_4
+ve anti-TSH receptors abs
↓TSH
Global uptake of I^{131} radiation
Diffuse (on scan)

MOA for PTU
⊖ incorporation of iodine into T_4/T_3
(by blocking Peroxidase oxidation of iodide → iodine)

tt

① Medical tt
options: - Iodide (for short-term usually given before surgery) to ⊖ hormone synthesis
- Propranol (symptomatic tt)
- **Propylthiouracil (PTU)**
- **Methimazole.**
- Steroids (↓ conversion)

② Radio-iodide Ablation ← tt of choice!
Absolute CI: Pregnancy / Newborn / Pt request / ↓RAI uptake
Relative CI: children & young adults (due to risk of long-term oncogenic effects.

③ Surgical tt — Bilateral subtotal thyroidectomy.

Index of surgery
- If you can't r/o CA
- Non-compliant Pt
- If Pt refuses radiation
- Failure of medical tt
- ~~Elderly~~
- Pregnant or child.

The MAJOR complication after radio-ablation & surgery is HYPOTHYROIDISM!

Other causes of hyperthyroidism :-

- **PLUMMER'S DISEASE** (Toxic adenom / Toxic MNG)

Risk factor: - Amiodarone intake

I^{131} Scan is diagnostic! → hot spot, the rest are suppressed

(tt) Radioablation or surgery (Lobectomy)

- **FACTITIOUS**
  - ↳ FINDINGS: ↓ Thyroglobulin
- **IODINE-INDUCED**
- **PITUITARY TSH-SECRETING ADENOMA**
- **TROPHOBLASTIC TUMOR** (molar)
- **STROMA OVARII**
- **NON-TOXIC GOITER** → usually benign, solitary

Pemberton's Sign

Large goiter causes plethora of head & when raising both arms.

HYPOTHYROIDISM

S&S
- Wt gain, edema
- Cold intolerance
- Menorrhagia
- Weakness
- Dry, thining of hair / dry skin
- constipation

CAUSES

m.c.c → • **Iatrogenic** (surgery or radio-iodine tt)

- Iodine insufficiency
- Hashimoto thyroiditis — Chronic autoimmune destructive lymphocytic infiltration in the thyroid.
  - ♀ >> ♂ (95% in ♀)
  - Antithyroglobulin Ab / Antiperoxidase Antibodies (Anti-TPO) / microsomal ab
  - Indx for surgery: • cosmetic • congestive SX
- Acute suppurative thyroiditis (Strep., staph. infxn) — Suspecious of malignancy. → Abx & surgical drainage
- Subacute thyroiditis (de Quervain) — rare
  - Viral
  - FNA: Giant cells
  - young ♀ after URTI (viral)
  - ↑ESR
  - Full recovery (tt is only supportive)
- Reidel's thyroiditis:
  - (Chronic like hashimoto)
  - Progressive inflammatory ass. in sclerosing cholangitis / mediastinal fibrosis.
  - tt: surgical decompression of trachea & thyroidectomy

Hashimoto = AUTO
AUTOimmune

Reidel's Thyroiditis
- Benign inflammatory thyroid enlargement w fibrosis of thyroid
- C/P painless large thyroid
- Fibrosis may involve surrounding tissues.

4.2

Uptake

DDx for ↑ uptake

- Grave's Disease
- Hot nodule
- TSH-secreting Pit. tumor
- hCG secreting tumor
- Iodine def.

DDx for ↓ uptake

- Thyroiditis
- Iodine excess
- Excess exogenous T4/T3
- Factitious hyperthyroidism (Hashitoxicosis)

Scan

Diffuse → THINK of Graves
Patchy → Plummer's
Localized → Toxic Adenoma.

The End.

PARATHYROID GLAND

EMBRYOLOGY

—Endoderm in origin

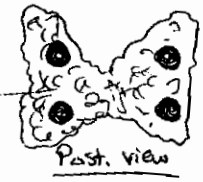
SUPERIOR Parathyroid gland → from the 4th pharyngeal Pouch
INFERIOR Parathyroid gland → From the 3rd Pharyngeal Pouch

* Long descend Pathway! So more incidence of ectopic gland.
  - If undescended → cranial to Sup. lobe of thyroid
  - excessive descent → mediastinum, other sites

ANATOMY

- **# of Glands** **MOST** of ppl have 4 Glands
  5% of ppl have 5 Glands (Think = 5y=5)
  10% of ppl have 3 Glands

- **Site** Posterolateral aspect of the thyroid


Post. view

SUPERIOR GLANDS

- 75% found on the junction btw. upper 1/3 & lower 2/3 at the Post. aspect of the thyroid lobe,
- **Superior** to the inferior thyroid art. (1 cm above it)
- **Posterior** to the recurrent laryngeal n. (so it's used as a landmark for recurrent laryngeal n., due to this, there's high risk of injury during surgery)

* It's the most final one! So in surgery don't remove it **UNTIL** you find the others

1% of pts have a PT gland in the mediastinum. (excessive descent)

INFERIOR GLANDS

The m.c site of "extra" gland is Thymus!

- Found inf./Lateral/or Posterior to the inferior Pole of the thyroid
- **Anterior** to the recurrent laryngeal n. (so it differs from superior glands which are Post. to the nerve)

Blood Supply

80% from the inferior thyroid art.

- If only 3 PT glands are found at surgery. Where can the 4th one be hiding?!
  - Thyroid gland (deep)
  - (mc) → Thymus / mediastinum
  - Carotid sheath
  - Tracheoesophageal groove
  - Behind esophagus

Types of cells

1. Fat cells
2. Cheif cells — Secrete PTH
3. oxyphil cells — Secrete PTH ONLY in hyperparthyroidism & hyperplasia. (Remember: OXyphils secrete extra PTH)

PHYSIOLOGY

NL Calcium value = 8.5 - 10.5

It's controlled by:

Note:
- PTH also
  - ↑ HCO_3^- excretion
  - ↑ Cl^- reabsorption
  - [Hyperchloremic acidosis]

1. **PTH**: Acts on BONE ⇒ ↑ Resorption of bone (+ osteoclasts) ⇒ ↑ Ca^{+2}, ↑ PO_4^{-2}

   Acts on KIDNEY ⇒ ↑ Ca^{+2} absorption, ↑ PO_4^{-2} excretion

   Acts on INTESTINE (via kidney) ⇒ ⊕ Vit D (by ↑ activity of 1α hydroxylase)

2. **Vitamin D**: Absorbed from small intestine

   25 $(OH)D_3$ → In liver

   (Active form) 1,25 $(OH)_2D_3$ → In kidney

Remember, we have 1 liver (so 1 (OH)) & 2 kidneys (2 (OH))

3. **Calcitonin**: PTH antagonist!
   ⊖ Bone & Kidney resorption of Ca^{+2} →→ so ↓ serum Ca^{+2}

NOTES

- PTH = 84 amino acids
  Active form
  C-Terminus & N-Terminus

NL values of PTH = 80
In 1ry HPT = 130-140
2ry = 400-800
3ry > 1000

- Ca^{+2} is absorbed in duodenum & Proximal jejunum.
- Bone is the largest RESERVOIR for Calcium in the body.
- Ca^{+2} in serum ⩽ 40% Bound to albumin, 50% Free, Rest is bound to Phosphate & citrate.
- Digeorge Synd.: Is congenital abscence of Parathyroid & Thymus glands.

HYPERPARATHYROIDISM

- 1ry Hyperparathyroidism — ↑ Sxn of PTH by PT. glands (Marked by ↑Ca^{+2}, ↓PO_4^{-2})
- 2ry Hyperparathyroidism — ↑ Serum PTH 2ry to Ca^{+2} wasting
- 3ry Hyperparathyroidism — Persistent hyperPTH AFTER correction of 2ry hyperPTH resulting in **autonomous** PTH sxn NOT responsive to Ve -ve feedback due to ↑Ca^{+2} levels.

PRIMARY (1ry) HYPERPTH

CAUSES:

Adenoma (m.c)	1 gland	85%
Hyperplasia	4 glands	10%
Carcinoma	1 gland	1%

In adenoma, ONLY 5% can be found in more that 1 gland but it's usually occurs in only 1

RF:
- FHx
- MEN I, IIa (Chromosome 11)
- Radiation

*Common in Postmenopausal ♀
*Mostly **sporadic**

X-Ray finding classic for HyperPTH is ↓ **Sub**periosteal bone resorption (usually in hand) = Pathognomic!

C/P: S&S of Hypercalcemia!

- **Stones** → Kidney stones (Nephrolithiasis)
- **Bones** → Bone Pain, Pathological ✳, Osteoporosis, Subperiosteal bone resorption (on X-Ray)
- **Groans** → Abd. Pain, weakness, Pancreatitis, Constipation, Gout
- **Psychiatric Moans** → Depression, Anaroxia, Wt loss, Anxiety, Emotional disturbances
- **Others:** HTN 10%, Polyuria / Polydypsia, Lethargy.

In Pt w 1ry HyperPT due to hyperplasia, ALWAYS r/o MENI & IIa

PT Carcinoma — rare
** 50% have **Palpable** neck mass
** Serum Ca^{+2} >15 mg/dL
* ↑ PTH
** Paralysis of recurrent laryngeal n. (Δ in voice)
** Hypercalcemic crisis.
Tumor Marker: hCG

Invx

FINDINGS: ↑ Ca^{+2} ↑PTH

- Phosphate
- Cr., alkaline phosphatase
- 24-hr urine collection (to r/o Fanconi)
- Cl^- : phosphate ratio > 33 (Rule of 33:1)
- PTH-level radioimmunoassay

IMAGING: **Systamibi scan** (^{99m}Tc)

PTH is controlled by:
1. Ca serum levels (ionized) — minute to min. control
2. # of chief cells

Fxns of Calcium
- Cxn & Sxn of most glands & muscles
- NMJ conduction
- 2nd messenger
- Coenzyme for many metabolic pathways
- Blood coagulation
- Mental activity

incl. **ttt**
- Initial medical ttt of hypercalcemia: IV Fluids & Furosemide (NOT thiazide)
- SURGERY!

If Adenoma → Surgically remove adenoma (Send for frozen section) & **biopsy** all abnormally enlarged PT glands (some experts bx all glands)

If Hyperplasia → Neck exploration removing **ALL** PT glands & leaving at least 30 mg of PT tissue placed in the forearm muscles (nondominant arm, of course!)

If Carcinoma → Remove carcinoma + ipsilateral lobe + all enlarged L.N (Modified radical neck dissection if LN mets)

* **Why place 30-40 mg of sliced PT gland in the forearm?**
To retain PT Fxn, IF HyperPT recurs, remove some of the PT gland from the easily accessible forearm.
[Advantage: Easier access!

Familial Hypocalciuric Hypercalcemia (FHH)
- Mild ↑ Ca, (↑PTH or NL PTH), **ASYMPTOMATIC**
- ↓ urine Ca
- Autosomal Dominant (AD) — mutation in Ca-sensing Receptors
- Labs: 24-hr urine collection: Loss of feedback inhibition
  Renal Ca/Cr clearance ratio — < 0.01 FHH; > 0.01 HyperPT
- Surgery is **NOT** indicated!

DDx of ↑ Ca:
Remember it as "**CHIMPANZEES**"
- **C**a overdose
- **H**yperparathyroidism / FHH / Hyperthyroidism
- **I**mmobility / Iatrogenic (thiazide)
- **M**etz / Milk alkali Synd.
- **P**aget d.
- **A**ddison / Acromegaly
- **N**eoplasm
- **Z**ES
- **E**xcessive vit. A
- **E**xcessive vit. D
- **S**arcoid

Complications of surgery

1. Postop. Hypocalcemia
   - Transient – If it's severe (<7.5), you should treat!
   - Persistant (After 6-8 wks) – tt: Calcium carbonate

Hungry Bone Synd.
= Severe hypocalc seen after surgical correction of HyperPT as chronically Ca-deprived bone aggressively absorb Ca^{+2}

If acute → IV Ca
If chronic → PO Ca. + vit D

2. Recurrent Laryngeal n. injury – unilateral → voice change
   Bilateral → Airway obstruction
3. Neck hematoma
4. Sup laryngeal n. injury.

Signs & Sx of Hypocalc.
1. Perioral Numbness
2. Parasthesia & Tetany
3. Chvostek sign
4. Tovressers Sign

SECONDARY (2°) HYPERPTH

CAUSES
- Renal Failure
- Vit. D def. (Rickets, Osteomalacia)
- ↓ GI absorption of Ca^{+2} ($\downarrow Ca^{+2}$)

Labs:
$\downarrow Ca^{+2}$
↑ PTH

tt
- Correct Calcium & Phosphate
- Correct the underlying cause (ex. Perform renal transplantation)

* NO ROLE for PT surgery!

TERTIARY (3°) HYPERPTH

= Persistent hyperPT AFTER correction of 2° HyperPT resulting from autonomous PTH syn NOT responsive & REFRACTORY to –ve feedback.

tt
- Correct Ca^{+2} & PO_4^{-2}
- Surgical op.: Remove all PT glands & reimplant in forearm if refractory to medical tt.

Bone diseases in hyperparathyroidism

- Renal osteodystrophy
- Osteoporosis
- Osteomalacia
- Osteitis Fibrosa Cystica
- Brown tumors.

Indx of surgery in ASYMPTOMATIC HyperPT

1. Age < 50 yrs
2. Pts who can't get appropriate F/U
3. Serum Ca > 1mg above NL range
4. Urine Ca > 400 mg/d
5. 30% ↓ Cr
6. Complications of HyperPT — Nephrocalcinosis / osteoporosis (T score < -2.5)

NOTES

RARE! - **Parathyromatosis**: • Multiple, small, hyperfxning masses
• found in neck & mediastinum
• due to spillage of otherwise benign PT tissue during **surgery**

* One of DDx of recurrent hyperPT **after** Parathyroidectomy!
* **Mgt** Surgical removal

imp. [• m.c.c of HYPERCALCEMIA in hospitalized Pts is → CA (Bone Mets)
• m.c.c of HYPERCALCEMIA in OUTPts is → HyperPT

The Goal.

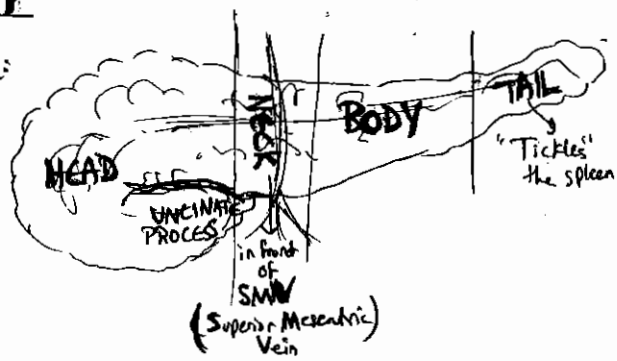
PANCREAS

EMBRYOLOGY

- Originates from the diverticula in the foregut endoderm.
- forms during the 5th – 6th wks of gestation.

ANATOMY

Structures:



Pancreatic Ducts
- Wirsung duct
- Santorini duct (=small)

Blood Supply
- Celiac Trunk → Gastroduodenal → Ant. Sup. Pancreaticoduodenal art.
  → Post. Sup. "
- Sup. mesentric art. (SMA) → Ant. Inf. Pancreaticoduodenal art.
  → Post. Inf. "
- Splenic art. → Dorsal pancreatic art.

TYPES OF PANCREATIC CELLS

- **ENDOCRINE CELLS** (Islets of Langerhans) — 50% of whole volume of Pancreas
  - **α-cells** Secretes **Glucagon**
    (Fxn) → Promotes the conversion of hepatic glycogen & ↑ Systemic glu level.
  - **β-cells** Secretes **Insulin**
    also secretes C-Peptide
    (Fxn) → Promotes glu transport into cells & ↓ Systemic glu level.
  - **D-cells** Secretes **Somatostatin**
    (Fxn) → ⊖ the release of GI hormones, gastric acid & small bowel electrolytes
  - **PP cells** Secretes Polypeptides / Vasoactive Intestinal Peptide (VIP)
- **EXOCRINE CELLS**
  Secretes digestive enzymes

PANCREATIC ISLET CELL TUMORS

- RARE!
- All are MALIGNANT **EXCEPT** insulinoma

● INSULINOMA

- Insulin-Secreting β-cell tumor of the Pancreas.
- m.c type of islet cell tumor (most of them are v.small tumors)
- **RULE OF 90%:**
  - 90% Benign
  - 90% < 1.5 cm (Small!)
  - 90% Solitary
  - 90% Intrapancreatic.
- Can be Part of **MEN I** / von Hippel Lindau disease.
- **C/P**: • Profound HYPOGLYCEMIA during fasting & after exercise
  - ✱ Neuroglycopenia (Anxiety/Tremor/Confusion/Obtundation)
  - ✱ Symp. response to hypoglycemia (Hunger/Sweating/Tachy)

●● Whipple triad to diagnose Insulinoma

1. Hypoglycemic Sx
2. Blood glu levels <50 mg/dL
3. Sx relieved after administration of IV glucose.

YOU should exclude Factitious hypoglycemia (insulin administration) & Postprandial reactive hypoglycemia.

✱ Dx is made by 72-hr fasting in hospital,
- inappropriate ↑ of plasma: ↑ insulin > 5 mol/mL
- ass. w/ hypoglycemia <50 mg/dL
- ↑ C-peptide ↑ Proinsulin.

Invx

- Dynamic CT scan at 5 mm intervals w/ contrast. (high-resolution CT)
- Endoscopic U/S
- Selective arteriography (THE BEST): (it will have excessive blood supply) — detects 50-60% of insulinomas
- Mamora test: ↓ glucose given → ↑ Ca^{+2} will ↑ all endocrine cells → so you see hyperfxning areas in insulinoma.

Remember, they are v.small tumors to a degree that 90% of insulinomas can't be seen on CT/or MRI!

(tt)

Surgical

— Remove the tumor.

* Pre-op give drugs → (Diazoide / Vasopamil / Octreotide)

* **Debulking Surgery** : Remove Part of tumor & leave the rest bcz it passes through vital structures.

Insulinoma — Notes

5% Malignant

10% ass. w/ **MENI** (usually those are multiple)

● **Indx of medical tt**

- Pts not candidate for surgery
- Preop. — to ↓ the hyperplasia & remove less tissue (as not to cause DM)

● **GASTRINOMA** — 2nd m.c islet tumor.

↳ G-cell tumor. ex Zollinger Ellison syndrome (ZES) (Part of MENI)

Remember, Parietal cells have 3 Receptors:
① Gastrin
② Histamine
③ Ach

* Premalignant

* Normally, there are NO G-cells in the Pancreas.

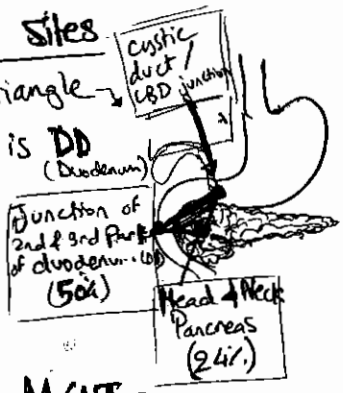
G-cells secrete **Gastrin** (Fxn: ↑ gastric acid & Pepsinogen Sxn)

● ZES is ass. w/ severe ulcers (refractory to medical tt) due to gastrin-mediated gastric acid sxn.

C/P
- Epigastric Pain
- Diarrhea
- Wt loss

Common Sites

- As a triangle →
- m.c site is **DD** (Duodenum)



THINK of ZES in any Pt w/ :-
① Recurrent, multiple ulcers or typically located (distal DD & jejunum) in ZES
② PUD refractory to medical tt
③ Complicated PUD (Bleeding / Perforation)
④ PUD w/ Significant diarrhea, obst.
⑤ PUD w/ HyperPT / nephrolithiasis / familial endocrinopathy

* It occurs in **MENI** :
- The m.c tumor in MENI is Gastrinoma (here, it's more common than insulinoma)
- Gastrinoma w/ MENI has benign course more than malignant course in comparison w/ Sporadic form.
- tt of Parathyroid (which is Part of MENI) can relieve gastrinoma.
- Usually multiple

Dx of ZES → Fasting serum gastrin ≥100 pg/ml (>500 is diagnostic)
Basal gastric acid output >15meq/hr (NL <10)

DDx
- gastric outlet obst.
- G-cell hyperplasia
- Renal Failure
- Atrophic gastritis.
- pt taking H2-Blockers or omeprazole (PPI)

* So you do Secretin stimulation test to exclude all other DDx.
↑ secretin ↑ gastrin

* Localization of gastrinoma:
- 80% win gastrinoma triangle!
- done by MRI/CT, Endoscopic U/S, scintography (If all other methods failed) (expensive but good for localization)

Tt
- Surgery (Rules for surgery:
  - Precise localization
  - Curative resection of the tumor.
- Medical by PPI IF pt NOT fit for surgery.

VIPoma
- Vasoactive Intestinal Peptide (VIP)-secreting tumor

2/3 is Pancreatic (>½ are malignant)

C/P: Secretory Watery Diarrhea, Hypokalemia, Achlorhydria / Hypochlorhydria

Dx: ↑VIP (>190), ↓K+

Tt
- Resection (usually occurs in distal Pancreas so we do distal Pancreatectomy)
- Medical (somatostatin analogue)

GLUCAGONOMA
- Glucagon-secreting α-cell tumor
- Presents as type II DM - Persistent Hyperglycemia
- Anemia / wt loss
- Hypoaminoacidemia
- Characteristic skin rash (Scalus Necrolytic Erythema)

Glucagonoma: Plasma Glucagon >1000

Tt: Resection

SOMATOSTATINOMA — RARE!
- D-cell secreting tumor
- usually in the head of Pancreas.

3 Ds
- Diarrhea
- DM
- Dilated GB w/ stone

Thank Glitch the End.

DIABETIC FOOT

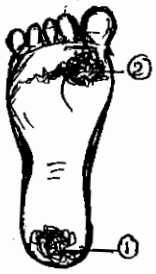
Source: Dossier 53
4. Washington

- 25% of all diabetic pts develop foot problems
- Diabetic foot is the m.c.c of admissions for diabetics.
- 50% of all causes of **AMPUTATIONS.**

CAUSE: The m.c.c is **undetected** or **untreated** trauma to the neuropathic foot.

SITES: (the most common)
① Heels
② The Plantar surface of the metatarsal heads.

*Wt is transmitted from Femur & tibia



PATHOPHYSIOLOGY

Multifactorial!

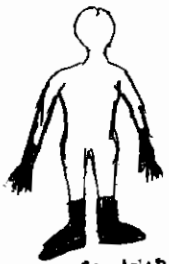
I) Peripheral NeuroPathy (The most imp. factor)

imp.

Sensory: loss of light touch/ vibration/ & Pressure Sensation

→ Causes harmful distribution of Pressure forces.

** Starts distally & migrate proximally in "Stocking" distribution (Parasthesia & Numbness), ↑ at night



"Gloves & Stocking" Distribution

Motor: Atrophy of the intrinsic muscles of foot
→ Collapse & loss of stability
→ Abnormal Pressure Points (shifts wt more on metatarsal heads)

⇒ This causes **DEFORMITIES**!
- Hallux Valgus
- Hammer toes (Claw toes) — Planter Flexion of DIP Joints
- Flat foot — collapse of medial arch.
- Overriding ingrowing nails

Mgt: Off-loading (to ↓ inflammation & accelerate repair)

*Ill fitting shoes cause Pressure on the dorsum of toes at the Post. interphalang. Joint.

Mechanism of claw toes
weakness of BOTH extensors & flexors, BUT usually flexors are stronger than ext. muscles → so pt will have claw toes!

Autonomic Neuropathy

● Failure of sweating → Inadequate lubrication
Mechanical breakdown ← Dry Skin ←
Fissures → Site of entry of Bacteria!

Pt Profile
- DM >10 yrs (usually type II),
Chronic hyperglycemia,
Alcoholic / Impaired vision /
Previous amputation

[2] Peripheral Vascular Disease (Vascular Insufficiency)

. Vessels will be blocked by atherosclerosis "Lead Pipe Artery"

. Failure of autoregulation in microcirculation so arterial blood will shunt Past the capillaries into venous circulation ... ↓ Blood Flow ↓ Healing.

(Dx) . Measuring ABI (Ankle-Brachial Index)
. Micro- & Macro-angiography.

AV Shunt
on PE: weak Pulse
warm / Pink.

Ischemia
on PE: weak Pulse
cold / Blue.

[3] Metabolic Hyperglycemia

* Mechanisms:
. ↑ Sorbitol — Damage of schwan cells — Nerve ischemia ↓ Impaired transmission!
. Intraneural accumulation of advanced Products of glycosylation
. ↓ Insulin — Delayed healing (bcz insulin is anabolic)

[4] Immune Deficiency

↓ BOTH Cellular & Humoral immunity.

[5] ↓ Growth Factors

like TGF-1, insulin-like GF

[6] Impaired Phagocytosis

weak healing Process.

[7] ↑ Metalloproteases

CHARCOT FOOT

m.c SITES
- Subtalar j.
- Ankle j.
- Interdigital j.

- Includes **Cartilaginous** Fibrillation & destruction & Subchondral / Endochondral **Bone** Formation.
- Also Fragmentation of Periarticular areas (midtarsal joints) & Subluxation (due to altered biomechanics) → Painless collapse of ligaments, joints & foot arches.

C/P

Acute : Swelling / hotness / & Pain — Mgt → Bisphosphonate
Subacute : Dislocation / Subluxation / & calcification on X-Ray
Chronic : Rocker-Bottom Deformity — Mgt → Immobilization / Amputation

Manifestations

① SKIN DISEASE

- **Dermopathy** (m.c) — m.c on shin, at tibia (due to atherosclerosis of small vessels.)
  - Brownish scar Preceded by red or blistering spots
  - Self-limiting
- Bullea / Blebs / Chilblain / & Blisters
- Nails abnormalities (difficulty cutting) — Thickening
  - Onchogryphosis (Ram's horn nail)
  - Onchomycosis — nail bed infxn is serious.
- Macerated webs (Tinea Pedis)
- Cellulitis (↑ESR, ↑WBC)

② SOFT TISSUE LESIONS

- Ulcers
- Necrotizing fasciitis — Serious / life-threatening
  - Swollen cyanotic foot w blisters on the skin + foul Smelling discharge.
- Gangrene — Blocked art. → cold & black, then dry out & shine ← infxn ← wet gangrene ← sepsis
- Abscess.

③ BONE

- Osteomyleitis
- Charcot Neuropathy
- Deformities

* Navicular bone is the most medial bone in the foot & the most affected by charcoal Changes!

The most imp. Complication is **AMPUTATION!**

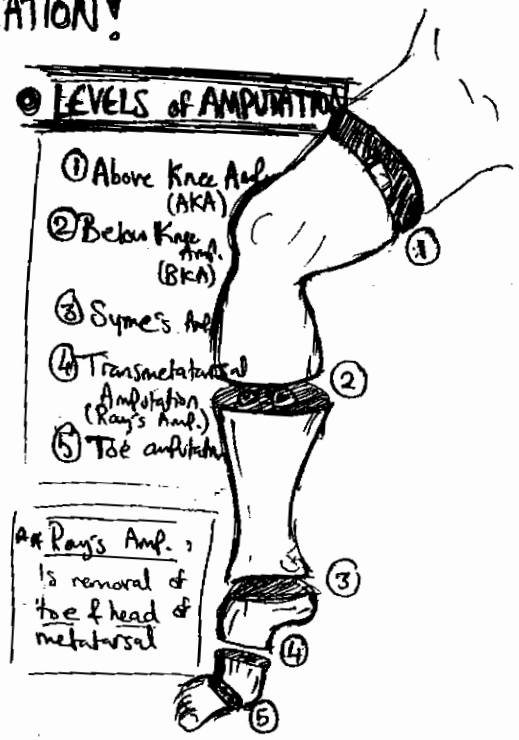
EVALUATION

P/E

** In the P/E of diabetic foot, you Should examine 4 Parts:- ① Lower 1/3 of leg
② Forefoot
③ Midfoot
④ Hindfoot

- **SKIN**: Comment on color (Cyanosed/Pale/hyperemic/dusky)
  - texture
  - Blister, Blebs, Bullea,
  - Hyperkeratosis (Skin response to ↑ Pressure to ↑ no. of cells)
  - Scaly (dryness)
  - Hair loss, Swelling
  - Dermopathy, Gangrene.
  - V. Thick callus.
- **HEEL**: Fissures / Ulcers / HyperKeratosis
- **NAILS**: Healthy / Trophic / Thickened (if ischemic)
  Onchogryphosis / Oncomycosis / Ingrowing nails
- **WEB SPACES**: Fissuring of the skin
- **BONE & JOINT DEFORMITIES**
- **ULCER** Examination (see below)
- **DISTAL PULSES** — Dorsalis Pedis (Against Navicular); Post. Tibial (Against Calcaneum) → m.c to be involved
- **NEURO EXAM.** (Sensation / Vibration / Proprioception)
- **L.N**

LEVELS of AMPUTATION



** Ray's Amp.: Is removal of toe & head of metatarsal

* Dusky Skin indicates Soft tissue Necrosis that lead to venous Congestion.

Pain indicates ischemia NOT Neuropathy.

ULCER EXAM. "SSS ME BF"

- Site / Shape / Size
- Margin / Edge (Border) (Skin around it)
- Base (You Palpate) / Floor (you inspect) [Comment on necrotic tissue / granulation tissue]
- Discharge & Smell
- Soft tissue swelling

** DONT forget to do the squeeze test. (4)

Signs of Infection In an ulcer

- Signs of inflammation (Redness / Hotness / swelling / loss of fxn)
- Foul smelling discharge
- Purulent sxns
- Presence of friable tissue
- Undermined edge!

→ for definitive dx of infxn take TISSUE ± (biopsy) culture

NOT from the surface (due to contamination)

Ulcer Classification (Wegner Classification)

0	Intact Skin
1	Superficial
2	Deep (to tendon / Bone / ligament)
3	Osteomyelitis
4	Gangrene of toe or forefoot
5	Gangrene of the entire foot

NOTE
We feel dorsalis Pedis Pulse lateral to the tendon of extensor hallucis longus

Invx

- Blood Sugar
- CBC
- ESR, CRP
- Urea, Electrolytes
- Tissue specimen
- X-Ray — to detect osteomyelitis or gas in soft tissue.

- 24% will heal in 12 wks
- 30% will heal in 20 wks

Tt

- Control Blood Sugar
- ↓ Pain (Analgesia) — give vit. B complex

****BEST Mgt is PREVENTION!**

- Daily inspection for signs of TRAUMA
- Attention to Hygiene.
- Off-loading orthotic devices (Casts / Diabetic Shoes)

- **Debridement** :- "Piece-meal debridement"
  - Debride all necrotic tissue
  - All surface should be bleeding
  - Wound edges should be healthy

→ Then according to the grade... (5)

Then Mgt according to the grade:-

* **LOW GRADE** : → Superficial / No hyperemia or swelling / No ischemia
  Mgt . Give abx topical or oral for 1wk
  Prognosis : → 100% complete recovery in 2-4 wks .

* **MODERATE GRADE** : → Pale / Necrotic tissue / Pus formation / Hyperemia BUT NO ischemia
  Mgt Admit the pt
  IV abx for 2-4 wks
  Px 80% will heal in 3 months.

* **HIGH GRADE** : Gangrene / Ischemia / Infected deep tendon & bone / Necrotizing infxn.
  Mgt Admit the pt
  IV abx for 4-6 wks
  Vascular reconstruction or Amputation
  Px 20% will heal in 12 months
  80% will need amputation !!

NOTES

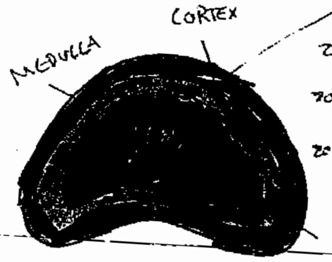
* Abx should cover Staph. Aureus / Staph. Epidermis / & Streptococcus
  (depends on wound culture)
  - 2nd generation cephalosporins
  - Wet-dry dressing
  - Aliginate Dressing → Minimizes contact of the wound → used for wounds that have a large amount of exudate
* If IV → give cephalosporins & Flagyl

* Graft & Flap → Should NOT be put on wt-bearing areas.

The End

ADRENAL GLAND

Intro.



CORTEX Remember GFR

- Zona **G**lomerulosa — Aldesterone "Salt" — Controlled by angiotensin II (RAAS) / [K+] — also ACTH
- Zona **F**asciculata — Glucocorticoids "Sugar"
- Zona **R**eticularis — Androgens (DHEAS) "Sex"

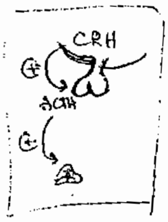
} Controlled by ACTH

*LH has **NO** effect on adrenal androgens.

MEDULLA

Catecholamines { Epinephrine, also norepinephrine & dopamine } controlled by autonomic nervous system.

*Notes



- CRH is secreted in response to stress / ↓ serum cortisol / & circardian rhythm, ⇒ it will ⊕ ACTH sxn
- ACTH follows a circardian rhythm, levels are highest at morning (around 6:00 am)
- ACTH ⊕ sxn of <u>all</u> cortex hormones BUT NO effect on adrenal medulla.

Cholesterol → it's taken up by the adrenal which forms the hormones.

- Progesterone
  - ↓ CYP 21 A2
  - 11-deoxy corticosterone
  - ↓ CYP 11 B1
  - **Aldesterone** "Salt"
- CYP 17 →
  - 17-OH Progestrone
    - ↓ CYP 21 A2
    - 11-deoxycortisol
    - ↓ CYP 11 B1
    - **Cortisol** "Sugar"
  - DHEA-S (95%)
    - ↓
    - **Testesterone** (small amount) → (Estrogen) (Aromatase)
    - "Sex"

Gene	Enzyme
CYP 21 A2	= 21-hydroxylase
CYP 17	= 17α hydroxylase
CYP 11 B1	11 β-hydroxylase

The End. (signature)

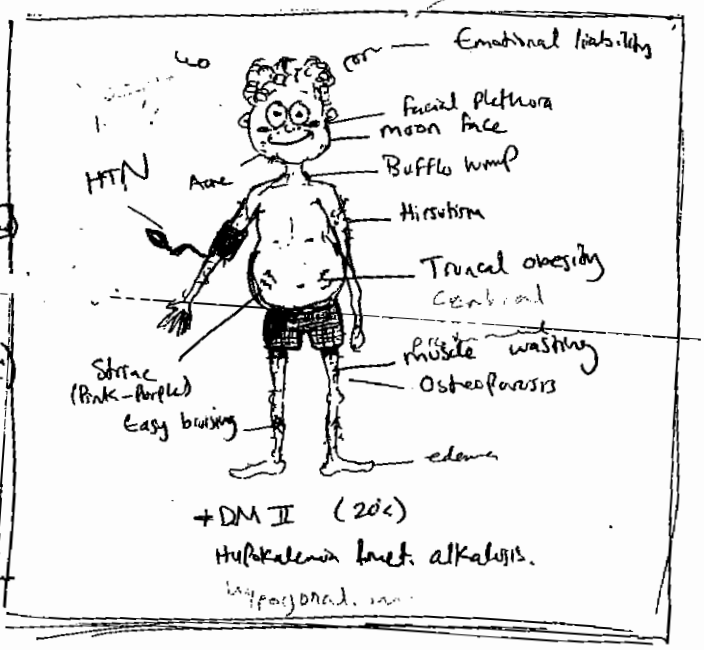
Cushing Syndrome

Source: Cecil Medicine Oxford 61

- General description for any ↑ cortisol

Sx

* See Pic. *



CAUSES [From the m.c → least common]

m.c.c
- Iatrogenic cortisol administration
- ACTH-secreting Pituitary adenoma ("Cushing disease")
- Ectopic ACTH-secreting tumor: (Bronchogenic/Pancreatic/Thymic Ca)
  If >60 yo spec is m.c.c!
- Non-Pit. related, bilateral adrenal hyperplasia
- Adrenal Tumors.

KEEP IN MIND!

* CAUSES of "PSUDOcushing":-
  - Obesity
  - Alcoholism
  - Depression

} Similar Phenotypic features
Slight ↑ 24-hr urine cortisol
&/or abnormal low-dose suppression test

* ACTH ⊕ son of cortisol, Aldesterone & Androgen

So in cushing disease, you may find ↑DHEA (& present w/ virilization)

While in adrenal adenoma, ↑cortisol
BUT ACTH, DHEA are ↓

* In true cushing, urine cortisol is significantly high!

Low-dose dexamethasone suppression test is used for SCREENING.

WORK-UP

Clinical Suspecion

↓

24 Urine free-cortisol (UFC)
or Low-dose dexa suppression test
or Night-time Plasma/salivary cortisol

NL → Cushing syndrome is unlikely.

Abnormal (make sure it's not Pseudocushing)
↓
Repeat test
↓ If still abnormal

We want to know if ACTH-dependent or not
Measure Plasma ACTH

If Low
↓
ACTH-independent
(Adrenal tumor most likely)
↓
Do Adrenal CT/MRI
↓
Tx Adrenalectomy ± CTx & RTx if carcinoma
'cures' if adenoma
but rarely cures in carcinoma.

If High / NL
↓
ACTH-dependent
(Cushing d. or ectopic)
↓
Do HIGH-dose dexa
(not done nowadays) cuz it might be suppressed in both

- If suppressed
  ↓
  Pituitary adenoma
  (Cushing d.)
  ↓
  Tx: trans-sphenoidal surgery
- If NOT suppressed
  ↓
  most likely ectopic
  ↓
  Tx: Surgery
  (if it's located & hasn't spread)

* Order also DHEA, ~~[illegible]~~
why?

Adrenal adenoma:
↓ACTH
modest ↑ DHEA

Adrenal carcinoma
↓ACTH
↑ DHEA + urine 17 ketosteroids

Cushing disease in the head, suppresses with high-dose dexa.

The End

PHEOCHROMOCYTOMA

Source: Medstudy Kaplan
63

- Is rare, usually benign tumors that arise from Chromaffin cells of the symp. nervous syst. (Sx are due sxn of catecholamines: Epineph, Norepi. + DA)

THE RULE OF 10s

10% extraadrenal (abd.), 10% Malignant, 10% in children, & 10% bilateral / 10% multiple, 10% not ass. w HTN (>Rt. side)

10% familial

DDx (which are more common than Pheo!)

- Labile essential HTN
- Anxiety
- Hyperthyroidism
- Hypoglycemia
- Menopausal flushing
- Carcinoid (rare)

Sx

PHEOchromocytoma
- Excessive sweating
- Headache
- Pressure ↑ (HTN)
- Palpitations & Tachycardia

RFs

(High Risk) Most imp.:
- **FHx of Pheo**
- **FHx of MEN II** or Neurofibromatosis or von Hippel-Lindau d.

(lower risk Pts)
- Combined HTN + DM
- Refractory HTN
- HTN in young person without FHx
- Adrenal incidentaloma
- Idiopathic dilated cardiomyopathy
- Hx of HTN during procedures (w ingestion of tyramine-containing food)

64

Dx debatable!

screening

for low-risk ppl: Fractionated metanephrines & catecholamines on 24-hr urine.

*Note: Antidepressants interfere so urine Pt sucks testing test.

for High-risk ppl: **Plasma** Fractionated metanephrine (sensitivity is ↑) but ↓specificity.

Fhx of Pheo

Fhx of MEN II, VHL incidentaloma = Cushing

VMA (vanillylmandelic Acid) was used before, but now metanephrines are preferred over VMA

IF you still suspect false -ive result, do clonidine suppression test. (will be still ↑ in Pheo) agonist

⇒ If results suggestive, do Abd. CT/MRI to find the tumor

Other less common tests: Radioactive trace I^{123} or MIBG / PET scan / Total body MRI / Genetic testing.

~~Rx~~

Preop: Give Antihypertensive (Combined α + β-blockers)

— Phenoxybenzamine (α-blocker) is preferred 2 wks prior to surgery THEN β-blocker is added 3 days before surgery

NEVER use β-blocker alone (coz it leads to unopposed α stimulation & Potential for hypertensive crisis)

Then Surgery (after stabilization of BP)

coz otherwise, outcome is poor if not stable BP

★ Notes

- > 1/3 of Pheo cause death prior to dx!
- Death is usually due to cardiac arrhythmias & stroke.

Adrenal Incidentaloma

Source: Medstudy

- mass > 1 cm, discovered by accident on an imaging study
- Most of them are non-fxning adenomas
- up to 15% of them have bilat. masses.

* If found to be malignancy → 50% chance of being metz!

- Wout a hx of CA: ~~[redacted]~~
  - → EXCLUDE a fxning tumor / Adrenal hyperfxn
  - → DETERMINE whether the mass is 1ry CA or metz

* Metz tends to be larger / bilat. / irregular / inhomog.

** ALL Pts w incidentaloma should undergo:

eval. 2. Glomerulosa for hyperaldo. — • BP & serum K+ (IF HTN, ↓K+ suspect hyperaldo. & do PAC, PRA)

eval. 2. Fascicul. for cushing — • 24-hr urine free cortisol or low-dose dexa suppression test

eval. medulla for pheo — • Plasma fractionated metanephrines

• ♀ w virilization or ♂ w feminization should have estrogens & androgens eval.

⟹ If results are NL AND mass < 4 cm; observe & repeat imaging in 3-6 m.

* Indx for Adrenalectomy of an incid.

1. Fxning tumor
2. mass > 4-6 cm
3. or imaging suspicious of CA.

(OT)
Content is imp. the more the life the most likely to be benign ☺

Gurbir Ghotra
The End

Multiple Endocrine Neoplasia
MEN

* All endocrine glands originate from endoderm.
* All ~~three~~ MEN are hereditary endocrine tumor syndromes & are Autosomal Dominant (AD)

MEN I AKA: Werner's Syndrome.

. Chromosome 11q13 (called through transcription factors)

Remember it as 3 Ps

- **Pituitary** (40% is present in MENI)

  (Types) - Benign Prolactin Producing Adenoma (m.c)
  - GH-secreting adenoma
  - ACTH-secreting adenoma
  - Nonfxning adenoma.

  (C/P) Headache / diplopia / Amenorrhea / Acromegaly / Galactorrhea

  (tt) MEDICAL: Bromocriptine (↓ tumor bulk)
  SURGICAL: Trans-sphenoidal hypophysectomy (definitive!)

- **Parathyroid** (100% is present in MENI)

  - The first one is usually detectable.
  - Generalized 4 glands hyperplasia.

  (tt) options: 3.5 gland removal
  or Removal of all of them & Autotransplantation in forearm.

  * Recurrence is 50%

- **Pancreatic islet cells** (50% present in MENI)

Types
- Gastrinoma (ZES) – m.c 50%
- Insulinoma 20%
- VIPoma
- Glucanoma
- Somatostatinoma

* Diffusly involved, ↳ islet cell hyperplasia, & multifocal tumor.

25% of ZES Pts have MENI

- Gastrinoma occurs in Prox. duodenum & gastrinoma ▲
- Multiple
- Always malignant ☹

NOTES

- They also can come ↳ adrenal cortex tumors to do INV
- We should screen all family members in their early teens.

MEN II AKA: Sipple's Syndrome.

Gain of fxn mutation in RET proto-oncogene which encodes a transmembrane tyrosine kinase receptor → tumorigenesis.

MENIIa [Remember it as MPH]

- Medullary Thyroid CA 100%
  - Early presentation
  - ↑ thyrocalcitonin (C-cells)
- Pheochromocytoma 30%
- Hyperparathyroidism 50%

MENIIb [Remember it as MMMP]

- Medullary thyroid CA 85%
  - –here, bilateral & v. aggressive
  - –Present early 1-2 yrs old
- Mucosal neuromas (ganglioneuromatosis) – 100%
  - –Neurofibromatosis
  - –multiple.

- Marfanoid body habitus

  Characteristic Physical appearance
  Hypergenthism of midface.
  Pes cavus / planum (Large arch of foot)

- Pheochromocytoma – 30%

* Prophylactic Thyroidectomy is indicated for all RET mutation carriers

  MEN IIa → At age of 5
  MEN IIb → At age of 1

Take Glitter.
The End.

70