STEM-S



NEET-SS

# ENT

GROUP

# Essential Notes

PRESENTED BY STEM-S

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# Neuroanatomy



## Classical Brainstem Vascular Syndromes

| Syndrome | Anatomical Site | Vessels Involved | Cranial Nerve Deficit | Long-Tract / Cerebellar Signs |
|---|---|---|---|---|
| Weber Syndrome | Ventral Midbrain (Basis Pedunculi) | Paramedian branches of PCA | Ipsilateral CN III palsy (ptosis, dilated pupil, "down and out") | Contralateral hemiplegia (Corticospinal tract) |
| Benedikt Syndrome | Midbrain Tegmentum | Paramedian branches of PCA | Ipsilateral CN III palsy | Contralateral involuntary tremor & choreoathetosis (Red nucleus) |
| Claude Syndrome | Midbrain Tegmentum & Peduncle | Branches of PCA | Ipsilateral CN III palsy | Contralateral cerebellar ataxia (Cerebellothalamic tract) |
| Millard-Gubler | Ventral Caudal Pons | Pontine circumferential arteries | Ipsilateral CN VI & LMN CN VII palsies | Contralateral hemiplegia (Corticospinal tract) |
| Wallenberg | Lateral Medulla | PICA or Vertebral Artery | Ipsilateral Horner, facial pain/temp loss (V), dysphagia (IX, X) | Contralateral body pain/temp loss (Spinothalamic) |

## Anterior Skull Base & Chiasmal Lesions

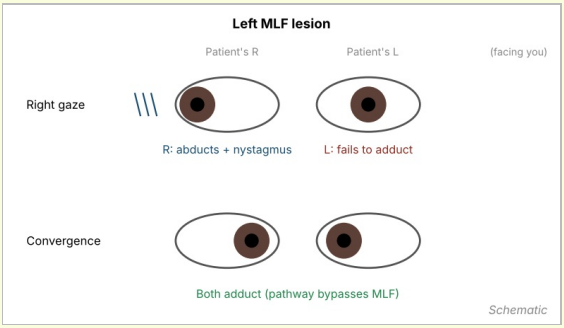
| Condition | Primary Anatomic Site | Classic Visual Field Defect | Key Associated Findings |
|---|---|---|---|
| Pituitary Macroadenoma | Sella Turcica / Central Inferior Chiasm | Bitemporal Hemianopia (starts superior temporal) | Endocrine dysfunction (amenorrhea, galactorrhea, acromegaly) |
| Tuberculum Sellae Meningioma | Optic Nerve-Chiasm Junction | Junctional Scotoma (Ipsilateral central loss + Contralateral superior temporal defect) | Anterolateral skull base dural tail on MRI |
| Olfactory Groove Meningioma | Anterior Cranial Fossa / Cribriform | Foster-Kennedy Syndrome (Ipsilateral optic atrophy + Contralateral papilledema) | Ipsilateral Anosmia |

## Facial Nerve Topographical Tests

| Topographical Test | Nerve Branch Tested | Anatomic Branching Site | Clinical Deficit When Impaired |
|---|---|---|---|
| Schirmer's Test | Greater Superficial Petrosal (GSPN) | Geniculate Ganglion | Reduced lacrimation (Dry Eye) |
| Stapedial Reflex Test | Nerve to Stapedius | Mastoid Segment (Proximal) | Hyperacusis (Loss of dampening) |
| Taste Testing / Salivary Flow | Chorda Tympani | Mastoid Segment (Distal) | Loss of taste (Anterior 2/3 tongue) |

## Ocular Motor & Brainstem

- Compressive CN III (PComA aneurysm): painful complete palsy WITH fixed dilated pupil, ptosis, "down and out" → emergency; Rx: urgent clipping / coiling (prevent SAH)
- Ischaemic CN III (diabetic): ptosis, diplopia, down and out, pupil spared; Rx: control glucose, BP, risk factors; resolves in 8–12 weeks
- CN IV palsy: vertical diplopia, worse depressing the adducted eye (stairs, downhill, reading); head tilt away from the affected side
- CN VI palsy in raised ICP = false localizing sign (stretch over petrous apex; no pontine / cavernous lesion)
- FEF lesions: destructive (stroke) → eyes look TOWARD the lesion | irritative (seizure) → AWAY from the lesion
- INO (MLF): ipsilateral adduction failure + contralateral abducting nystagmus + convergence preserved
  - Bilateral INO in a young adult = MS; unilateral in elderly = lacunar stroke
  - Rx: IV methylprednisolone (MS relapse) or secondary stroke prevention




*INO, left MLF lesion*

- Parinaud (dorsal midbrain): dorsal tegmentum, pretectum, riMLF; pineal tumour (pinealoma, germinoma) or aqueductal stenosis
  - Upgaze palsy + convergence-retraction nystagmus + light-near dissociation
  - Rx: biopsy / resection, CSF diversion (ventriculostomy), RT / chemo by histology

**Heads up:**

Pupil-sparing CN III = diabetic, conservative. Pupil-involving CN III = PComA aneurysm until angiography says no.

**Heads up:**

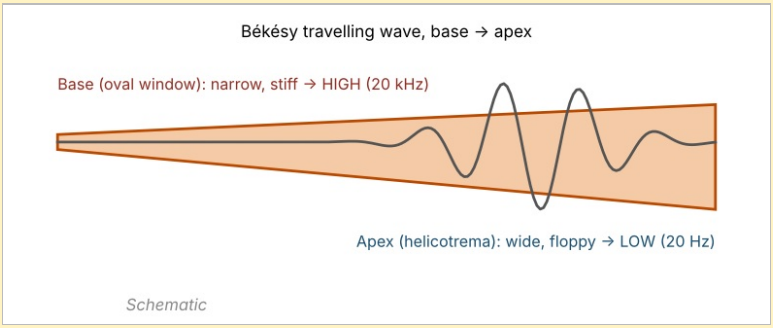
Vertical diplopia, trouble with stairs / reading, head tilt = CN IV (superior oblique).

| Vestibular Structure | Sensory Organ | Gelatinous Matrix | Primary Stimulus / Function | Specific Nerve Innervation |
|---|---|---|---|---|
| Semicircular Canals (Lateral, Superior, Posterior) | Crista Ampullaris | Cupula (lacks calcium carbonate crystals) | Senses angular acceleration and rotational movements | Lateral & Superior → Superior Vestibular N.; Posterior → Inferior Vestibular N. |
| Otolith Organs (Utricle & Saccule) | Macula | Otolithic membrane containing Otoconia ($CaCO_3$) | Senses linear acceleration and gravitational pull | Utricle → Superior Vestibular N.; Saccule → Inferior Vestibular N. |

**Heads up:**
Superior vestibular n. = utricle + lateral + superior canals. Inferior = saccule + posterior canal.

## 4 Transduction & Central Pathway

- Békésy travelling wave (Nobel 1961): base → apex; base narrow, stiff → high (20 kHz) | apex wide, flexible → low (20 Hz)
- Basilar-tectorial shear bends stereocilia toward the tallest (kinocilium) → mechanically gated $K^+$ channels (TRPA1 / TMC1) open → $K^+$ in (driven by +80 mV) → depolarization → $Ca^{2+}$ entry → glutamate onto afferents




*Tonotopy along the basilar membrane*

**Heads up:**
Recruitment = cochlear, not retrocochlear. Acoustic neuroma: no recruitment, SISI 0–20%, marked tone decay, OAE preserved, rollover.

- BERA (within 10 ms of click): I distal cochlear n. | II proximal cochlear n. | III cochlear nucleus | IV SOC | V lateral lemniscus + inferior colliculus (biggest peak); wave I present, II–V absent = retrocochlear block
- Infant screening: OAE first (fast, OHC, no sedation) → BERA to confirm after a fail
- Malingering: PTA is subjective → use BERA, stapedial reflex, Stenger (feigned unilateral loss)

## 7 Genetics of Hearing Loss

- > 50% of childhood congenital SNHL is genetic

Hereditary hearing loss
Non-syndromic (~70%): AR ~80% (GJB2) | AD ~18% (DFNA) | X / mito ~2%
▼
Syndromic (~30%): AD Waardenburg, BOR, NF2 | AR Pendred, Usher, JLN | X Alport

- Non-syndromic (~70%): AR ~80% (GJB2 / connexin 26, DFNB1) | AD ~18% (DFNA: KCNQ4, COCH, TECTA) | X-linked / mitochondrial ~2% (DFNX2 POU3F4; 12S rRNA 1555A>G)
- Syndromic (~30%): AD Waardenburg, BOR, NF2 | AR Pendred, Usher, Jervell & Lange-Nielsen | X-linked Alport
- GJB2 (connexin 26), DFNB1, 13q12: 50% of AR non-syndromic SNHL; gap junctions recycle $K^+$ back to the stria
- Mitochondrial 12S rRNA (1555A>G, 1494C>T): ribosomes hypersensitive to aminoglycosides → profound irreversible loss even after one dose
- DFNX2 / DFN3 (POU3F4): X-linked, stapes fixation + wide subarachnoid-perilymph communication → no stapedectomy (gusher)

| Syndrome Name | Inheritance | Primary Non-Auditory Features | Specific Gene / Locus | Key Exam Differentiator |
|---|---|---|---|---|
| Waardenburg Syndrome | Autosomal Dominant | Dystopia canthorum, heterochromia iridum, white forelock, patchy hypopigmentation | PAX3 (Type I), MITF (Type II), SOX10 (Type IV) | Most common autosomal dominant syndromic SNHL; presence (Type I) vs absence (Type II) of dystopia canthorum |
| Branchio-Oto-Renal (BOR) | Autosomal Dominant | Branchial cleft cysts/fistulae, preauricular pits, renal malformations | EYA1, SIX1 | Combined conductive, SNHL, or mixed loss with renal structural defects |
| Neurofibromatosis 2 (NF2) | Autosomal Dominant | Bilateral vestibular schwannomas, juvenile posterior subcapsular cataracts, meningiomas | NF2 gene on chromosome 22q12 (Merlin / Schwannomin) | Hallmark is bilateral acoustic neuromas presenting in 2nd/3rd decade |
| Pendred Syndrome | Autosomal Recessive | Euthyroid goiter developing in puberty, Mondini dysplasia, enlarged vestibular aqueduct | SLC26A4 (chromosome 7q31) encoding Pendrin ($I^-/Cl^-$ transporter) | Second most common autosomal recessive syndromic SNHL; abnormal perchlorate discharge test |
| Usher Syndrome | Autosomal Recessive | Retinitis pigmentosa (progressive night blindness & visual field loss) | MYO7A (Type 1B), CDH23 (Type 1D) | Most common cause of congenital deaf-blindness; Type I has severe SNHL + vestibular loss |
| Jervell & Lange-Nielsen | Autosomal Recessive | Prolonged QT interval on ECG, recurrent syncopal attacks, risk of sudden cardiac death | KVLQT1, KCNE1 (Potassium channel genes) | High risk of sudden death; requires immediate beta-blocker / ICD therapy |
| Alport Syndrome | X-Linked Recessive (85%) | Progressive glomerulonephritis, hematuria, renal failure, anterior lenticonus | COL4A5 (Type IV collagen) | Hematuria and renal failure paired with progressive SNHL in males |

**Heads up:**

Usher = commonest AR syndromic SNHL (deaf-blind, RP). Pendred = second (goitre + EVA, SLC26A4).

# External Ear



**Heads up:**
Diabetic + greenish discharge + EAC floor granulation + facial palsy = MOE (Pseudomonas). Always biopsy the granulation to rule out SCC of EAC.

**Heads up:**
MOE vs CSOM: MOE has an intact TM with granulation on the EAC floor. CSOM (safe type) has a perforated TM with granulations in the middle ear.

Diabetic / immunocompromised with otalgia + otorrhea

↓

Otoscopy: granulation at EAC floor (bone-cartilage junction)

↓

EAC biopsy (exclude malignancy)

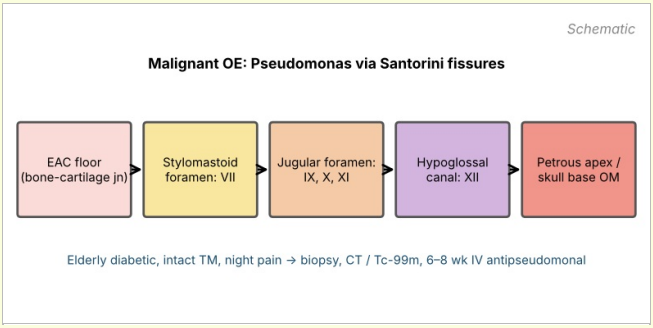
↓

CT temporal bone (bony erosion) / Tc-99m scan (osteoblastic activity)

↓

High-dose systemic antipseudomonal (IV ciprofloxacin / ceftazidime)

+ glycemic control




*Pathological spread of MOE*

## 5 Comparative Tables

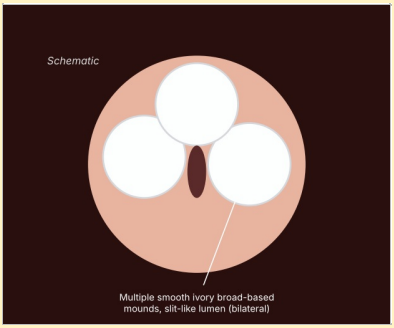
**Table 1: Inflammatory and infectious EAC conditions**

| Condition | Etiology / Pathogen | Host / Risk | Cardinal Sign | TM & Canal | Primary Management |
|---|---|---|---|---|---|
| Acute diffuse OE | Pseudomonas aeruginosa (90% bacterial) | Water exposure, cotton bud use, phone headsets | Positive tragal sign (severe pain on tragal pressure) | EAC edema, erythema, purulent debris; TM intact | Topical antipseudomonal drops + gentle aural toilet |
| Malignant OE (NOE) | Pseudomonas aeruginosa (Aspergillus in AIDS) | Elderly diabetics, immunocompromised | Granulation at EAC floor (bone-cartilage junction) + facial palsy | Bony EAC erosion; skull base extension; TM intact | 6-8 weeks IV antipseudomonal therapy + glycemic control |
| Otomycosis | Aspergillus niger or Candida albicans | Humid climate, prior broad-spectrum antibacterial drops | "Wet newspaper" / "black pepper" conidiophores or cotton-wool mass | Diffuse fungal hyphae; intense pruritus; TM intact | Mechanical aural toilet + topical clotrimazole 1% |
| Perichondritis of pinna | Pseudomonas aeruginosa | Auricular trauma, high cartilage piercing, burns | Painful, red, swollen pinna sparing the earlobe | EAC normal; cartilage ischemia risk | Systemic IV antipseudomonal + I&D if abscess |
| Ramsay Hunt syndrome | Varicella zoster virus | Reactivation in CN VII geniculate ganglion | Conchal vesicles + LMN facial palsy + otalgia | Herpetic eruptions in concha / EAC; CN VII paralysis | Oral acyclovir / valacyclovir + systemic corticosteroids |

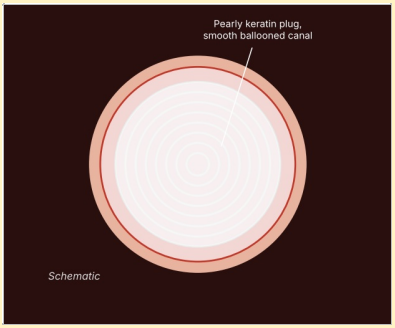
# External Ear



- Exostosis: multiple smooth, round, ivory, broad-based mounds from anterior, posterior and superior walls of deep EAC → slit-like lumen.
- Keratosis obturans: pearly-white packed keratin plug widening the bony EAC; smooth ballooned wall; intact thick TM in the distance.

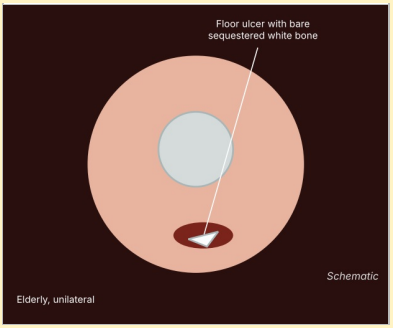



*Exostosis*




*Keratosis obturans*

- EAC cholesteatoma: ulcer in inferior or posterior wall with a solitary bare sequestered fragment of white necrotic bone.
- Sade 2: focal depression of posterosuperior pars tensa draped over the long process of incus, no collapse onto promontory.
- Tos 3: pars flaccida pocket above the short process of malleus disappearing under the scutum, with partial blunting / bony defect of outer attic wall.




*EAC cholesteatoma*

### Ossicular reconstruction classifications

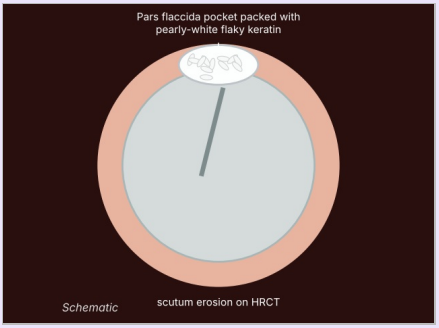
| System | Type | Anatomical Criteria | Surgical Reconstruction / Placement |
|---|---|---|---|
| Wullstein | Type 1 | Intact ossicular chain; TM perforation | Myringoplasty (graft over intact malleus / ossicles) |
| Wullstein | Type 2 | Malleus handle eroded; incus intact | Graft placed directly over the incus |
| Wullstein | Type 3 | Malleus and incus eroded; stapes intact | Graft placed directly over stapes suprastructure |
| Wullstein | Type 4 | Stapes suprastructure absent; footplate intact | Graft placed on stapes footplate (round window shield) |
| Wullstein | Type 5 | Fixed stapes footplate | Fenestration of horizontal semicircular canal (historical) |
| Austin | Type A | Malleus handle present | Stapes suprastructure present |
| Austin | Type B | Malleus handle present | Stapes suprastructure absent |
| Austin | Type C | Malleus handle absent | Stapes suprastructure present |
| Austin | Type D | Malleus handle absent | Stapes suprastructure absent |

**Heads up:**
Malleus handle present + stapes suprastructure absent = Austin B. Graft directly onto the incus = Wullstein 2.

## 3 Cholesteatoma & Life-Threatening Otogenic Complications

### Cholesteatoma mechanics

- Abnormal accumulation of keratinizing squamous epithelium in middle ear / mastoid.
- Histologically non-neoplastic but locally invasive: osteolytic enzymes (collagenases, acid phosphatases, TNF-α) erode bone.
- Primary acquired: deep pars flaccida (attic) retraction pocket from chronic negative pressure; trapped keratin forms an expanding matrix.
- Secondary acquired: after an existing perforation; migration theory (EAC keratinizing epithelium migrates in through the perforation margins) is most accepted.
- Hallmark (primary): scanty, persistently foul-smelling discharge, progressive CHL, attic retraction pocket with flaky white keratin.




*Primary acquired cholesteatoma: attic pocket with keratin*

# Inner Ear



## Acute vestibular syndrome workup

Acute continuous vertigo + nausea + postural instability

↓

Otoscopy + audiometry

(hearing normal → vestibular neuritis; SNHL → labyrinthitis)

↓

Bedside HINTS: positive vHIT / catch-up saccade,

unidirectional horizontal-torsional nystagmus, no vertical skew

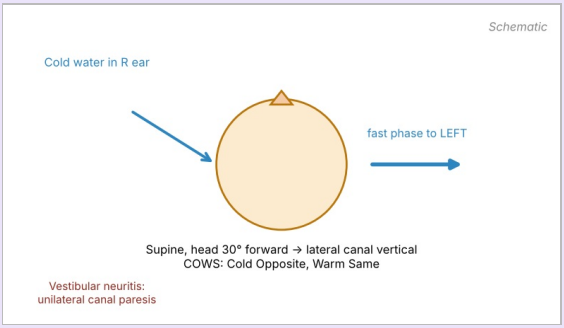
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Caloric test (supine, head tilted 30° forward)

→ UNILATERAL CANAL PARESIS

## Caloric testing mechanics

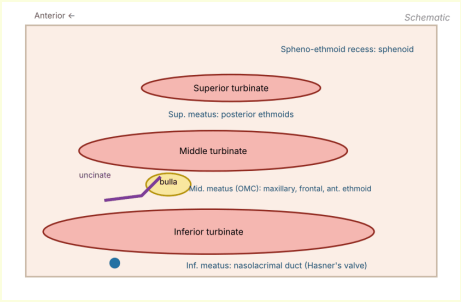
- Tests the lateral (horizontal) canal.
- Supine, head tilted 30° forward → lateral canal becomes vertical → temperature-induced convection currents act maximally on the cupula.
- COWS: Cold → Opposite, Warm → Same. Cold makes endolymph fall (fast phase to opposite side); warm makes it rise (fast phase to same side).
- Vestibular neuritis: significant unilateral canal paresis (reduced / absent response) on the affected side.



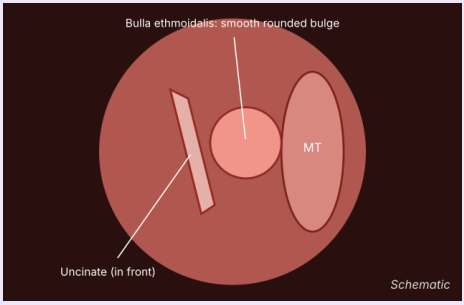

*Caloric test and COWS*

## Lateral nasal wall

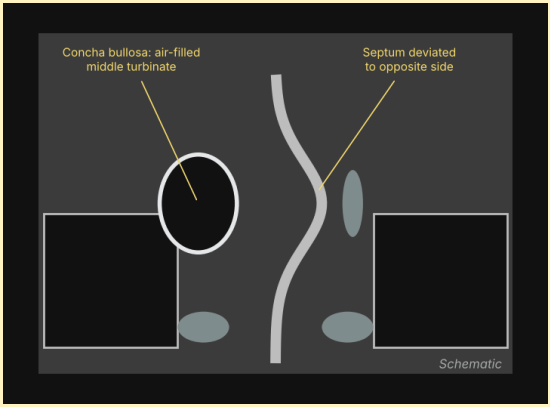
- Three or four scroll-like turbinates (conchae) with their meatuses.
- Inferior meatus: below the inferior turbinate; nasolacrimal duct ends here at Hasner's valve.
- Middle meatus: main territory of FESS; houses the osteomeatal complex (OMC).
- – Bulla ethmoidalis: largest, most prominent and most constant anterior ethmoid cell; rounded bulge on the lateral wall; posterior boundary of the hiatus semilunaris; key FESS landmark. Hyper-pneumatization can compress the infundibulum and block maxillary drainage.
- – Uncinate process: thin sickle-shaped ethmoid bone projecting posteroinferiorly; anterior-inferior boundary of the ethmoidal infundibulum.
- – Hiatus semilunaris: crescent groove bounded inferiorly by the uncinate and superiorly by the bulla; drains maxillary, frontal and anterior ethmoid sinuses into the middle meatus.
- – Concha bullosa: pneumatized middle turbinate; when large it deviates the septum to the opposite side and blocks the ipsilateral OMC.
- Superior meatus: below the superior turbinate; drains posterior ethmoid cells.
- Sphenoethmoid recess: above and behind the superior turbinate; drains the sphenoid sinus.




Lateral nasal wall: turbinates, meatuses and drainage




Bulla ethmoidalis behind the uncinate (endoscopic view)

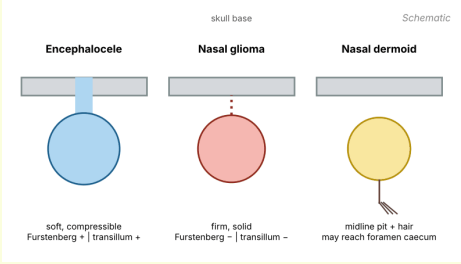



Concha bullosa on coronal CT

# 3. Midline Congenital Masses & Anterior Skull Base Defects

## Embryology

- In early fetal life a dural projection passes through the prenasal space (between unossified nasal bones and the cartilaginous nasal capsule) via the foramen caecum.
- Failure of dural involution or incomplete obliteration of ectodermal-neuroectodermal tracts → three midline anomalies: encephaloceles, nasal gliomas, nasal dermoids.




Encephalocele vs glioma vs dermoid

Congenital midline nasal mass

- Intracranial connection
  - compressible + transilluminant → ENCEPHALOCELE (Furstenberg +)
  - non-compressible dural extension (frontoethmoidal)
- No intracranial connection
  - firm mass, no impulse → NASAL GLIOMA (Furstenberg −)
  - subcutaneous cyst with punctum / hair → NASAL DERMOID (hair extruding)

## Encephalocele

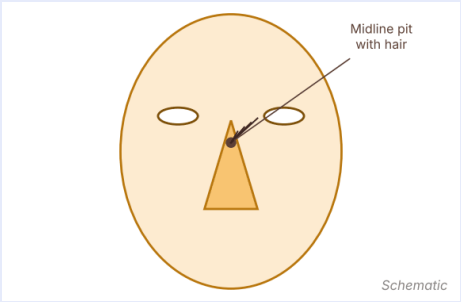
- Herniation of intracranial contents (meninges ± brain) through a skull base defect: most often nasofrontal / frontoethmoidal junction or cribriform plate.
- Mechanism: incomplete closure of the fonticulus frontalis or foramen caecum → CSF + meninges (meningocele) or brain (encephalocele) herniate.
- **Classic findings:**
- – Midline compressible swelling at the nasal root, nasofrontal region or intranasally.
- – Positive Furstenberg sign: enlarges and pulsates with crying, coughing or jugular compression (Queckenstedt) from transient ICP rise.
- – Positive transillumination (fluid-filled CSF sac).

**HEADS UP**

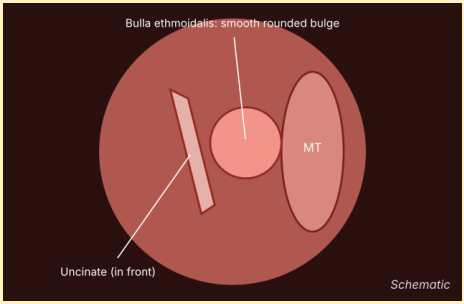
No biopsy or needle aspiration of a nasal mass before neuroimaging. Risk: CSF fistula, ascending meningitis, tension pneumocephalus.

## Endoscopic and surgical anchors

- Bulla ethmoidalis: smooth, rounded, balloon-like bony bulge immediately behind the uncinate on entering the middle meatus.
- Nasal dermoid punctum: single clean midline pit on the nasal bridge with pathognomonic hair extrusion.

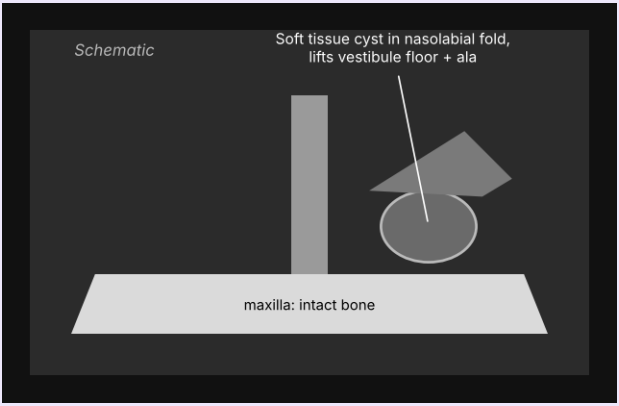



Nasal dermoid punctum




Bulla ethmoidalis

- Nasolabial cyst: photo or coronal CT showing a smooth soft-tissue mass in the incisive fossa / nasolabial fold elevating the vestibule floor and lateral alar rim, with underlying bone completely intact (non-odontogenic soft-tissue cyst).




Nasolabial cyst

## 5.3 Orbital complications: the pediatric emergency

- Ethmoid sinusitis in young children spreads into the orbit through the paper-thin lamina papyracea or congenital dehiscences.

**HEADS UP**

In a child with acute ethmoid sinusitis, high fever and periorbital signs, blood cultures or eye discharge swabs are NOT the next step. Do an immediate CT of the PNS and orbits to rule out a subperiosteal abscess needing surgical decompression.

5-year-old with ethmoid sinusitis + high fever
+ periorbital edema / conjunctival chemosis
▼
Emergency non-contrast CT scan of PNS & orbits with contrast
(crucial next step: separates periorbital cellulitis from orbital cellulitis or subperiosteal abscess)
▼
IV broad-spectrum antibiotics ± surgical / endoscopic drainage
(indicated if CT confirms subperiosteal abscess, vision deteriorates, or no improvement in 24–48 hours)

## 5.4 Cavernous sinus thrombosis (CST)

### What's going on

- Lethal septic venous thrombosis from retrograde spread of infection from the "danger area of the face", the paranasal sinuses (ethmoid/sphenoid) or the nasal cavity, via the valveless ophthalmic veins and intercavernous sinuses.
- The cavernous sinus contains CN III, IV, VI, the ophthalmic (V1) and maxillary (V2) divisions of CN V, and the internal carotid artery with its sympathetic plexus.
- Septic stagnation gives rapid venous congestion, orbital back-pressure and progressive cranial nerve deficits.

### Clinical signs

- Abrupt onset, fast progression: severe headache, high spiking fevers, proptosis, marked eyelid edema, conjunctival chemosis.
- Bilateral orbital involvement: unilateral signs soon become bilateral through free venous intercommunication across the anterior and posterior intercavernous sinuses.
- Cranial nerve palsies, sequential: CN VI (abducens) is hit FIRST and worst. It runs freely in the central venous lumen next to the ICA, whereas CN III, IV, V1 and V2 are protected in the lateral dural wall.
- Sympathetic dysfunction: inflammation of the plexus around the intracavernous ICA can give Horner's syndrome (ptosis, miosis, anhidrosis).

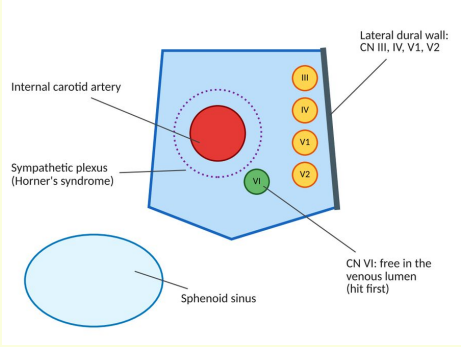



Fig. 7. Cavernous sinus, coronal section: where each cranial nerve sits. Schematic

**HEADS UP**

A favourite stem: "All of the following are features of CST EXCEPT simultaneous involvement of CN III, IV and VI at onset." CN VI goes first, and the others follow sequentially, not all three together at presentation.

## 8. Master Comparative Matrices

### Matrix 1: Antrochoanal polyp vs ethmoidal polyps

| Feature / Differentiator | Antrochoanal Polyp (ACP) | Ethmoidal Polyps |
|---|---|---|
| Anatomic Site of Origin | Maxillary sinus antrum (passes through ostium into choana) | Ethmoidal sinus air cells (middle meatus) |
| Number & Symmetry | Solitary and Unilateral | Multiple and Bilateral |
| Age Predilection | Children and young adults | Adults |
| Etiology | Non-allergic (chronic sinonasal infection) | Allergic, eosinophilic, AERD / Samter's triad |
| Best Visualization Site | Posterior Rhinoscopy (hanging into nasopharynx) | Anterior Rhinoscopy (filling nasal cavities) |
| Response to Topical Steroids | NO ROLE (Completely ineffective) | First-line medical therapy (highly effective) |
| Recurrence Rate | Low (if antral attachment is excised) | High (frequent post-surgical recurrence) |

### Matrix 2: Histopathological & clinical differentiators of granulomatous diseases

| Condition | Etiologic Agent / Marker | Diagnostic Histopathology / Cytology | Key Clinical Differentiator |
|---|---|---|---|
| Rhinoscleroma | Klebsiella rhinoscleromatis | Mikulicz cells (foamy histiocytes) & Russell bodies (eosinophilic plasma cells) | Hebra nose; 3 stages (catarrhal, granulomatous, sclerotic) |
| Rhinosporidiosis | Rhinosporidium seeberi (Aquatic Protistan) | Large sporangia containing thousands of endospores | Endemic in South India; reddish polyp with white dots |
| Wegener's (GPA) | c-ANCA (PR3-ANCA) positive | Necrotizing vasculitis with granulomatous inflammation | Saddle nose deformity + renal glomerulonephritis |
| Sarcoidosis | Elevated Serum ACE | Non-caseating epithelioid granulomas | Apple-jelly nasal mucosal nodules |
| Atrophic Rhinitis | Klebsiella ozaenae | Squamous metaplasia & submucosal endarteritis | Roomy cavities, greenish crusts, offensive odor (ozena) |

### Matrix 3: Paranasal sinus fungal disease classification

| Fungal Subtype | Patient Immune Status | Primary Fungal Organism | Characteristic CT / MRI Findings | Key Management Strategy |
|---|---|---|---|---|
| Allergic Fungal (AFRS) | Immunocompetent, Atopic | Dematiaceous (Bipolaris, Curvularia) | CT: Double density sign (hyperattenuation); MRI: T2 signal void | FESS + Post-op Oral/Topical Steroids |
| Fungal Ball (Mycetoma) | Immunocompetent | Aspergillus spp. | CT: Maxillary hyperdense mass with punctate calcifications | FESS alone (No antifungals required) |
| Acute Invasive (AIFR) | Immunocompromised (DKA, Neutropenia) | Mucorales, Aspergillus | CT/MRI: Bony destruction, fat-plane obliteration, necrosis | Emergency Surgery + IV Amphotericin B + Reverse Immunosuppression |

| Condition | Primary Cause | Visual / Palpatory Finding | Pain / Tenderness | Swab / Pillar Pressure Response |
|---|---|---|---|---|
| Acute follicular tonsillitis | Group A β-hemolytic Streptococcus | Swollen tonsils with yellow crypt spots | Severely painful | Pus easily expressed by pillar pressure |
| Keratosis pharyngis | Epithelial hyperkeratosis | Hard, yellowish-white adherent dots | Non-tender / painless | Cannot be wiped off; no pus expressed |
| Vincent's angina | Borrelia vincentii + Fusobacterium | Unilateral gray slough & ulceration | Moderately painful | Slough wipes off leaving bleeding surface |
| Chronic parenchymatous tonsillitis | Lymphoid hyperplasia | Massively enlarged tonsils meeting at midline | Non-tender | No pus expressed on pillar pressure |
| Infectious mononucleosis | Epstein-Barr virus (EBV) | Exudative tonsillitis + splenomegaly | Painful sore throat | Associated with generalized lymphadenopathy |

## 4 Deep Neck Space Infections & Airway Emergencies

### The big picture

- Pharyngeal or dental suppuration breaches local boundaries and tracks along fascial planes.
- Spaces communicate with major neurovascular structures and the mediastinum → rapid life-threatening spread.
- Hallmark: fever, severe trismus, asymmetric neck / pharyngeal swelling with airway compromise.

Assess: trismus, drooling, stridor

Threatened airway / Ludwig's angina
→ *EMERGENCY TRACHEOSTOMY*
→ *broad-spectrum IV antibiotics + drainage*

Stable airway

→ contrast CT neck

→ target-specific surgical drainage + IV antibiotics

### Peritonsillar abscess (quinsy)

- Pus between the tonsil capsule and superior constrictor; follows acute tonsillitis.
- Severe unilateral sore throat, high fever, trismus (internal pterygoid spasm), "hot potato" voice, drooling, referred otalgia (IX).
- Exam: bulging soft palate and anterior pillar, uvula deviated to the opposite side.
- Parapharyngeal extension: after drainage, worsening trismus, spiking fever, tender swelling behind the angle of mandible = pus through the superior constrictor into the parapharyngeal space.

| Feature | Acute Epiglottitis | Acute Laryngotracheobronchitis (Croup) | Laryngeal Diphtheria |
|---|---|---|---|
| Etiology | H. influenzae type b (bacterial) | Parainfluenza virus type 1 | Corynebacterium diphtheriae |
| Peak age | 2-6 years (and unimmunized) | 6 months - 3 years | Unvaccinated children / adults |
| Anatomical site | Supraglottis | Subglottis | True vocal cords & subglottis |
| Clinical signs | Toxic, high fever, drooling, tripod stance, muffled voice | Non-toxic, barking seal-like cough, low-grade fever | Dirty gray pseudomembrane, brassy cough, systemic toxicity |
| X-ray sign | "Thumb sign" (lateral view) | "Steeple sign" (AP view) | None (pseudomembrane on direct view) |
| Complications | Sudden complete airway collapse | Secondary bacterial tracheitis | Toxic myocarditis, palatal paralysis, late laryngeal stenosis |

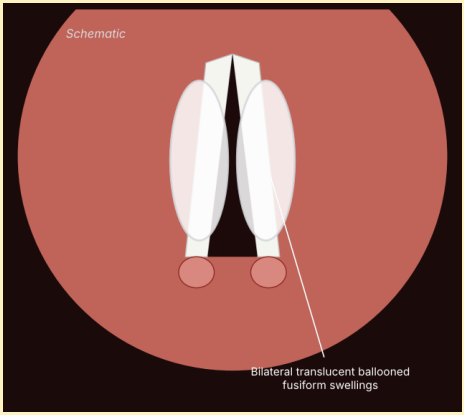
**HEADS UP**

Thumb = epiglottitis (lateral), steeple = croup (AP); don't swap them. Diphtheria complications = toxic myocarditis, palatal paralysis. Reinke's edema is NOT a diphtheria complication (it's non-infectious, from smoking / voice abuse).

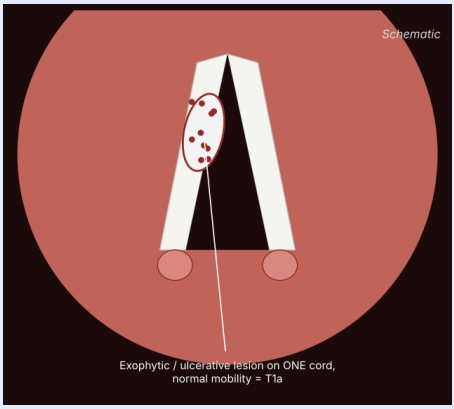
# 4. Benign Vocal Fold Pathology

## Phonotrauma and subepithelial fluid

- Repetitive mechanical trauma or chronic chemical irritation → microvascular leak, submucosal edema, fibrous remodelling.
- **Reinke's edema (polypoid corditis):**
- – Chronic fluid in Reinke's space (superficial lamina propria), diffusely ballooned with gelatinous fluid.
- – **Heavy smoking, vocal abuse, hypothyroidism.**
- – More vibrating mass → lower pitch: deep, rough "smoker's voice", most striking in females.
- Vocal nodules ("singer's nodules"): phonotrauma at the point of maximal impact, the junction of anterior 1/3 and posterior 2/3 → bilateral, symmetric nodules.




Reinke's edema




T1a glottic carcinoma

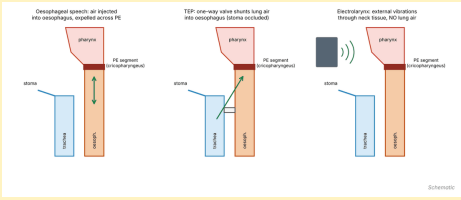
## 8. Post-Laryngectomy Voice Rehabilitation

- Total laryngectomy separates the airway from the pharynx → permanent tracheostoma, loss of the laryngeal sound source.
- Oesophageal speech: air injected / inhaled into the upper oesophagus and expelled across the pharyngo-oesophageal (PE) segment (mainly cricopharyngeus + mucosa), which vibrates.
- Tracheoesophageal prosthesis (TEP): one-way valve in a surgical TE fistula shunts lung air into the oesophagus (stoma occluded) to vibrate the PE segment.
- Electrolarynx: external battery-powered transducer pressed on the neck.

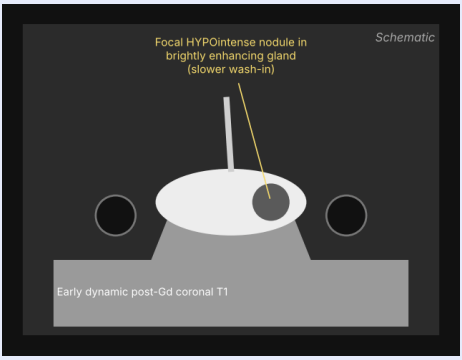
**HEADS UP**

Trouble initiating oesophageal speech but fluent once going = cricopharyngeal hypertonicity (UES fails to relax on air injection). Electrolarynx uses no lung air; it pushes mechanical sound through the neck soft tissue into the mouth.

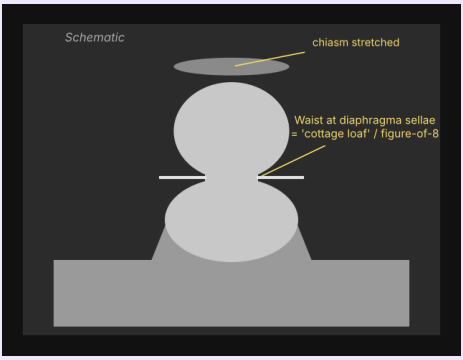
Post-laryngectomy voice rehabilitation
- Oesophageal speech
  - source: PE segment / cricopharyngeus
  - dysfunction: cricopharyngeal HYPERTONICITY
    - → difficulty STARTING voice, fluent once started
- Electrolarynx
  - vibrations transmitted EXTERNALLY through neck into mouth
- TEP
  - one-way valve sends lung air into oesophagus when stoma is occluded




Oesophageal speech, TEP, electrolarynx




Microadenoma on dynamic coronal MRI

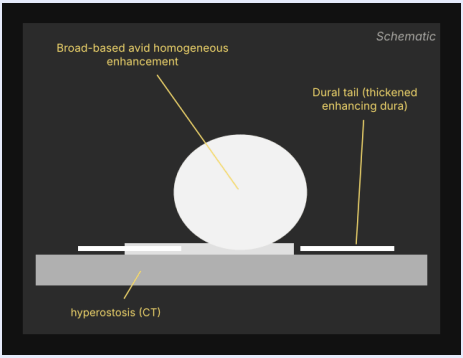



Cottage loaf macroadenoma

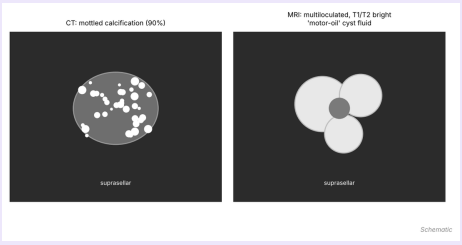
- Parasellar meningioma: contrast T1 shows a broad-based dural attachment with a linear dural tail (thickened enhancing adjacent dura); bony hyperostosis on CT.

- Craniopharyngioma: CT mottled calcification (seen in 90% of pediatric cases); MRI multiloculated suprasellar mass with T1/T2 hyperintense "motor-oil" / "machine-oil" cyst fluid rich in protein and cholesterol crystals.

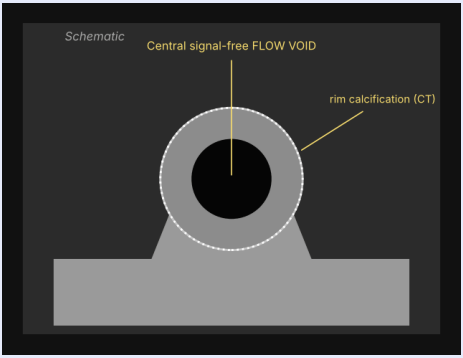
- Internal carotid aneurysm: spin-echo T1 and T2 show a well-circumscribed sellar mass with a central signal-free flow void from rapid turbulent arterial flow.




Meningioma with dural tail



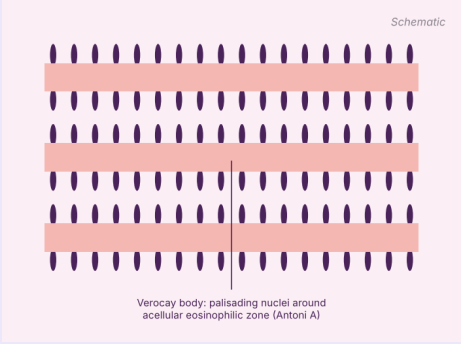

Craniopharyngioma on CT and MRI




ICA aneurysm flow void

## Schwannoma (neurilemmoma)

- Benign, slow-growing, encapsulated; exclusively Schwann cells.
- **Strongly S-100 +.**
- Antoni A: hypercellular spindle cells with nuclear palisading around acellular eosinophilic zones = Verocay bodies.
- Antoni B: hypocellular, loose, myxoid tissue with microcysts.



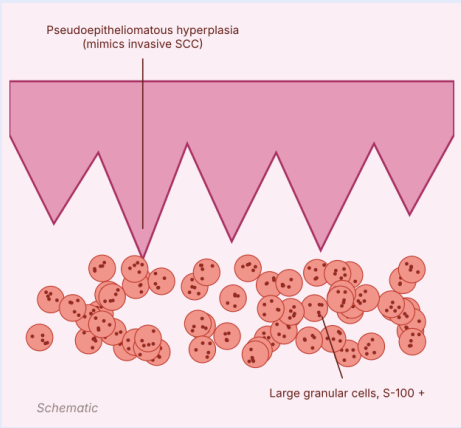

Schwannoma: Verocay bodies

## Granular cell tumour

- Benign, Schwann cell origin; > 50% on the tongue (dorsum) (dense nerve supply).
- Large polygonal cells with abundant eosinophilic cytoplasmic granules (lysosomes, PAS +); strongly S-100 +.
- Pseudoepitheliomatous hyperplasia (PEH) of the overlying epithelium in 30%.

**HEADS UP**

PEH over a granular cell tumour can mimic invasive SCC on a superficial biopsy. It's a benign reactive change over S-100+ granular cells; recognise it to avoid needless radical surgery.




Granular cell tumour with PEH

**HEADS UP**

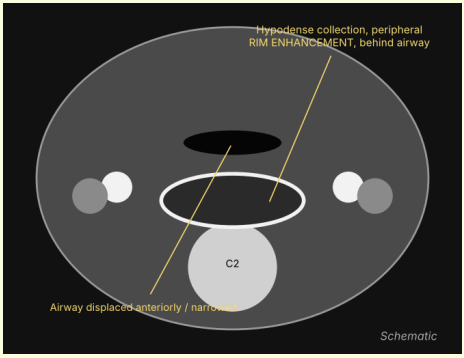
Congenital epulis (granular cell tumour of the newborn): smooth pedunculated mass on the maxillary alveolar ridge of newborns (F:M 8:1). Histologically identical granular cells, but no PEH and S-100 NEGATIVE (non-neural origin).

## Retropharyngeal abscess

- Children (< 5 years): suppurative lymphadenitis of the retropharyngeal nodes of Rouvière after URTI.
- Adults: penetrating trauma, foreign body perforation, tuberculous cervical spine (Pott's disease).
- High fever (39°C), toxic, drooling, dysphagia, neck stiffness (pseudomeningismus), posterior pharyngeal wall bulge.
- CT: hypodense collection with peripheral rim enhancement behind the pharyngeal air column, pushing the airway forward and narrowing it.
- Rx: secure the airway in theatre → transoral or external incision and drainage.

**HEADS UP**

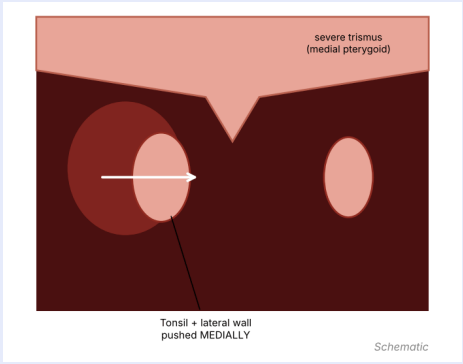
Lateral neck X-ray: prevertebral soft tissue > 7 mm at C2, or > 14 mm (children) / 22 mm (adults) at C6, suggests retropharyngeal pathology, but contrast CT is mandatory to confirm an abscess and plan surgery.




Retropharyngeal abscess on contrast CT

## Parapharyngeal abscess

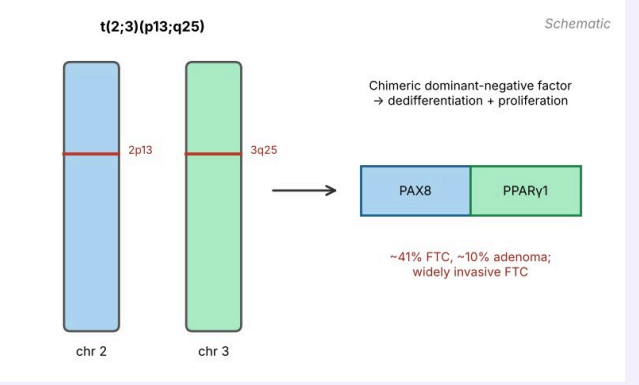
- Space = inverted pyramid, skull base to hyoid; split into prestyloid and poststyloid compartments by the tensor-stylo-vascular fascia.
- Causes: 60% acute tonsillitis / tonsillectomy, 30% dental, 10% otogenic.
- Organisms: Strep viridans (39%), Staph epidermidis (22%), Staph aureus (22%).
- Hallmark: severe trismus (medial pterygoid irritation) + medial displacement of the tonsil and lateral pharyngeal wall, fever, swelling behind the angle of the mandible.
- Rx: small cellulitic foci may settle on high-dose IV antibiotics; established collections → external drainage via a transverse neck incision medial to the carotid sheath.




Parapharyngeal abscess: tonsil pushed medially

## Follicular carcinoma: RAS and PAX8-PPARγ (cont.)

- **RAS:**
  - Point mutations at **codons 12, 13, 61** of **HRAS, KRAS, NRAS**; lock RAS in the active **GTP-bound** state.
  - **~30% to 50% of FTC**, **~50% of follicular adenomas**, and **FVPTC**.
  - Found in benign and malignant lesions → **early event**; in FTC it correlates with **vascular invasion, haematogenous metastasis, mortality**.
- **PAX8-PPARγ:**
  - **t(2;3)(p13;q25)** fuses thyroid transcription factor **PAX8** with **PPARγ1** (lipid metabolism / adipocyte differentiation receptor).
  - **~41% of FTC**, **~10% of follicular adenomas**.
  - Chimeric protein = **dominant-negative** factor → dedifferentiation + proliferation; positive FTCs are usually **widely invasive**.
- **Tumour suppressor loss: LOH at 3p and 11q13 (MEN1 locus)** pushes follicular clones toward malignancy.



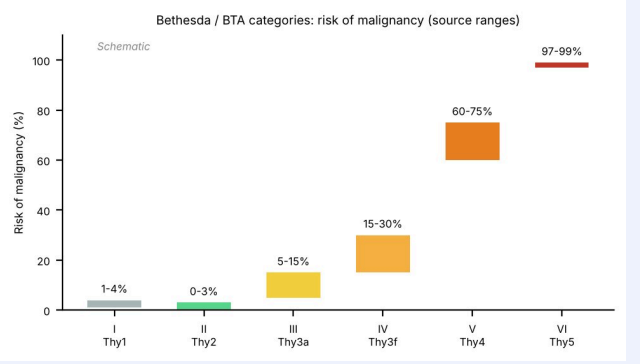

PAX8-PPARγ translocation

## Anaplastic carcinoma: stepwise dedifferentiation

- Rapidly lethal; arises from **accumulated hits in pre-existing differentiated cancer**.
- **TP53: ~48% of ATC** (vs ~14% of thyroid cancers overall); loss of the **G1/S checkpoint** and DNA repair → genetic instability, **aneuploidy**, lost apoptosis.
- **CTNNB1 (β-catenin):** impaired degradation → **nuclear β-catenin**, **Wnt hyperactivation** → dedifferentiation.
- **Shared early drivers persist: RAS (~26% to 50%)** or **BRAF V600E (~26%)** → proves **clonal evolution** from DTC.

## FNAC and cytological risk (cont.)

| BTA Category | Bethesda Category | Diagnostic Nomenclature | Risk of Malignancy | Mandatory Clinical Management |
|---|---|---|---|---|
| Thy1 / Thy1c | Bethesda I | Non-diagnostic / unsatisfactory (acellular, obscuring blood) | 1-4% (up to 10% if cystic) | Repeat FNAC under ultrasound guidance; consider surgery if solid and enlarging |
| Thy2 / Thy2c | Bethesda II | Benign (colloid nodule, Hashimoto's, granulomatous) | 0-3% | Clinical observation; repeat FNAC in 3-6 months to confirm stability |
| Thy3a | Bethesda III | Atypia of undetermined significance (AUS / FLUS) | 5-15% | Repeat FNAC, consider molecular testing, or MDT discussion |
| Thy3f | Bethesda IV | Follicular neoplasm / suspicious for follicular neoplasm | 15-30% (15-45% if Hürthle cell) | Diagnostic hemithyroidectomy / lobectomy (frozen section NOT recommended) |
| Thy4 | Bethesda V | Suspicious for malignancy (PTC, medullary, lymphoma) | 60-75% | Surgical intervention (total thyroidectomy or lobectomy based on size / risk) |
| Thy5 | Bethesda VI | Malignant (definitive PTC, anaplastic, medullary) | 97-99% | Total thyroidectomy + compartmental neck dissection if nodes involved |




Risk of malignancy across Bethesda categories

**Heads up:**

**FNAC cannot tell follicular adenoma from carcinoma**, or Hürthle adenoma from carcinoma. Malignancy needs **transcapsular invasion or angioinvasion** on **permanent paraffin sections**. **Frozen section is unhelpful** for Thy3 / Bethesda IV.

## Ultrasound risk criteria

- **High risk: solid hypoechoic**, **microcalcifications** (psammoma bodies), irregular / microlobulated margins, **taller-than-wide**, **central / intranodular** hypervascularity on Doppler.
- **Low risk / benign:** purely **cystic**, **spongiform**, **peripheral eggshell calcification**, **peripheral / rim** vascularity.

**Comprehensive H&N Exam**
↓
**& Bimanual Palpation**
↓
**Cross-Sectional Imaging**
↓
**(CECT / MRI / OPG)**
↓
**MUST be BEFORE mucosal or core biopsy**
↓
**Tissue Confirmation**
↓
**Incisional biopsy: mucosal oral lesions**
↓
**FNAC / core: neck / PPS masses]**
↓
**Distant Staging & Second Primary Screen**
↓
**Non-contrast chest CT or 18F-FDG PET-CT**
↓
**MDT Staging & Treatment Planning**
↓
**Margins, mandible management,**
↓
**neck dissection strategy]**

## Step 1: Clinical Examination & Bimanual Palpation

- Complete UADT exam including **flexible fibreoptic nasendoscopy** is mandatory.
- **Bimanual palpation** of oral tongue, FOM and submandibular triangle establishes:
  - Objective **tumour size**.
  - **Deep muscular fixation**.
  - **Tongue base extension**.

### Step 2: Imaging Before Biopsy

- **CECT or MRI MUST precede biopsy.**
- Biopsy causes **oedema, inflammation, haematoma** → degraded imaging → **false-positive overstaging** of extent and deep invasion.
- **OPG and bone-window CT: cortical erosion vs medullary invasion** of mandible or maxilla.
- **Contrast MRI (gold standard)** for:
  - Soft tissue **DOI**.
  - **Tongue muscle** infiltration.
  - **Perineural spread** (**CN V3 and VII**).
  - **PPS fat plane** integrity.

### Step 3: Tissue Biopsy & Cytology

- **Mucosal oral lesions: incisional edge biopsy** including adjacent normal mucosa.
  - **Excisional biopsy is contraindicated** for suspected primary oral cancer.

# 1. Pathophysiologic Lead-In: Epidermal Architecture, Carcinogenesis and Genetic Syndromes

## How UV makes a cancer

- Chronic **ultraviolet B (UVB)** exposure is the principal environmental carcinogen. It causes **pyrimidine dimer** formation, **reactive oxygen species** and cumulative DNA damage that impairs tumor suppressor genes, most notably **p53 and PTCH1**.
- Tumor behavior is set by the **cell of origin**, the **depth of invasion** and the **host's immune competence**.
- Clinical hallmark on sun-exposed head and neck skin: a **non-healing scaly papule**, a **pearly nodule with rolled borders**, or a **rapidly enlarging violaceous lesion** that bleeds with minor trauma.

## Which layer makes which tumor

- **Stratum spinosum:** interdigitating squamous keratinocytes joined by desmosomes. Malignant transformation → **cutaneous squamous cell carcinoma (cSCC)** and its precursors (**actinic keratosis**, **Bowen's disease**).
- **Stratum basale:** the deepest layer, with proliferating basal cells, melanocytes and mechanoreceptors.
  - Basal keratinocytes → **basal cell carcinoma (BCC)**.
  - Neural crest-derived melanocytes → **cutaneous melanoma**.
  - Neuroendocrine mechanoreceptor (Merkel) cells → **Merkel cell carcinoma (MCC)**.

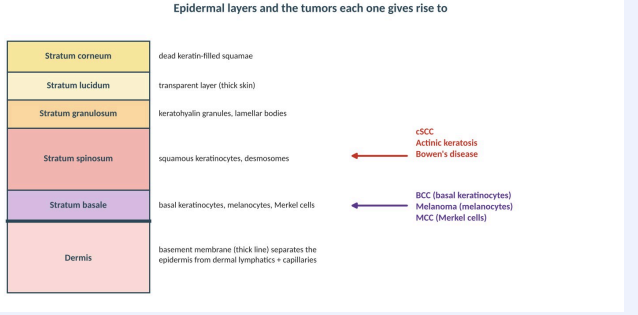



**Figure 1.** Epidermal layers, the basement membrane, and the tumors each layer gives rise to. Schematic

## Hereditary genodermatoses