ENT Exam Supplement

Missing Points Not Covered in the High-Yield File

Based on Past Paper Analysis · Exam-Focused · Concise

How to use this document: Everything here is information *not* found in the High-Yield ENT file yet required to answer the past paper questions. Read it as a flowing study supplement, not a Q&A; list.

1 · External Ear

Referred Otalgia — Nerve Supply

The ear receives sensory supply from four cranial nerves and two cervical roots, all of which can mediate referred otalgia: CN V (trigeminal), CN VII (facial), CN IX (glossopharyngeal), and CN X (vagus — via Arnold's nerve, its auricular branch), plus C2–C3. The **hypoglossal nerve (CN XII) is the only cranial nerve that does NOT contribute** to ear sensation and therefore cannot cause referred otalgia.

- The **glossopharyngeal nerve (CN IX)** is the most clinically important source of referred ear pain from the pharynx and tonsils.
- **Arnold's nerve** (auricular branch of CN X) mediates the cough reflex triggered by ear canal manipulation — stimulating it during ear cleaning produces a cough response.

Auricular Hematoma — Management

The size of a hematoma determines management. **Small hematomas** may be aspirated and compressed. A **large auricular hematoma** requires formal **evacuation (incision and drainage) + pressure bandaging + systemic antibiotics**. Failure to evacuate allows clot organisation → fibrocartilage → *cauliflower ear* deformity. Aspiration alone is inadequate for large hematomas due to re-accumulation.

- A child with a swollen, red pinna the day after a fall: treat as auricular hematoma → **aspiration/drainage + pressure dressing**.

Otomycosis — Specific Points

• **Most common fungus:** Aspergillus niger (produces black conidia — the 'black dots' seen on otoscopy). Aspergillus fumigatus is second; Candida is third.

• Treatment of fungal otitis externa = **topical antifungal for 3 weeks** (not 1–2 weeks). Aural toileting precedes any drops.

• Aspergillus niger specifically responds to **2% sodium bicarbonate** ear drops (alkalinises the canal environment).

• The dominant symptom of otomycosis is **ear blockage/fullness**. Unlike bacterial OE, there is no severe pain, no high fever, and discharge is not profuse.

Malignant Otitis Externa — Exclusions

• The hallmark of malignant OE is granulation tissue at the bony–cartilaginous junction, NOT malignant (mitotic) cells.

• Mitotic figures / cellular atypia are NOT features of malignant OE — the name 'malignant' refers to its aggressive behaviour, not histology.

• Predominantly affects elderly diabetics; Pseudomonas aeruginosa is the causative organism; immunocompromised state is a key risk.

Furuncle (Furunculosis) of the Vibrissae

• Presents with: fever, painful swelling, pus-filled discharge, and nasal obstruction.

• Headache is NOT a typical feature of a furuncle of the vibrissae.

2 · Acute Otitis Media

Aetiology — Key Exclusions

• Pseudomonas aeruginosa is NOT a common cause of AOM. It is characteristic of malignant otitis externa and chronic suppurative otitis media with a wet perforation, not classic AOM.

• **Dental infection** is not a recognised factor in causing otitis media. The accepted factors are Eustachian tube dysfunction, immune deficiency, and ineffective mucociliary clearance.

• **Bullous myringitis** (haemorrhagic blisters on the tympanic membrane) is caused by **viral** infection (classically Mycoplasma pneumoniae or respiratory viruses), not bacteria.

Phases of AOM — Treatment by Phase

AOM progresses through defined stages. In the suppurative (second) phase, the treatment includes systemic antibiotics, local hyperthermia, and myringotomy if indicated. **Topical antibiotic ear drops are NOT indicated** in this phase — they cannot penetrate an intact tympanic membrane and are not part of the treatment algorithm at this stage.

Complications — Differentiation

Complication	Key Distinguishing Feature	Route
Epidural abscess	Severe headache + high fever, NO meningeal signs	Direct extension
Meningitis	High fever + meningeal signs (neck stiffness, Kernig)	Haematogenous
Brain abscess	Focal neuro deficits, raised ICP, may have seizures	Direct extension

Complication	Key Distinguishing Feature	Route
Lateral sinus thrombosis	Spiking fever + rigors + otorrhoea, Queckenstedt test +ve	Mastoid

- Intracranial complications of otitis media reach the intracranial space **primarily via direct extension** through bone or through pre-formed pathways.
- The **earliest symptom** of any intracranial complication developing from ear disease is **headache**.

3 · Chronic Otitis Media

Initial Workup in Suspected CSOM

A young adult presenting with longstanding, foul-smelling ear discharge and hearing loss since childhood requires **CT of the temporal bone as the first investigative step**. This characterises the extent of bony destruction, identifies cholesteatoma, and guides surgical planning — preceding culture swabs or empirical antibiotics in this clinical context.

Palatal Cleft and Otitis Media

- Children with a **cleft palate** almost universally develop **secretory (serous) otitis media**. The levator veli palatini and tensor veli palatini muscles, which open the Eustachian tube during swallowing and yawning, are abnormal in cleft palate, leading to chronic ET dysfunction and middle-ear effusion.

Most Common Persistent Symptom of COM

- The most common *persistent* symptom of chronic otitis media is **conductive hearing loss**, not otorrhoea. Discharge may resolve between infective episodes, but the hearing deficit persists.

Complications of Secretory Otitis Media (Glue Ear)

- Recognised complications: cholesteatoma, cholesterol granuloma, adhesive otitis media, acute suppurative OM.
- **Sensorineural deafness is NOT a complication of secretory OM — it is strictly a middle-ear (conductive) process.**

Traumatic Tympanic Membrane Perforation — Management

- **Recent, dry traumatic perforation** → **watchful observation**. The majority of traumatic perforations heal spontaneously within 3 months. Neither immediate surgical repair nor antibiotic drops are indicated unless there is contamination.
- **Dry traumatic rupture** (no contamination) → **protect the ear from water entry**. This is the single most important intervention.
- Antibiotic ear drops, systemic antibiotics, and systemic steroids are not indicated for a clean, dry traumatic perforation.

4 · Vertigo

Peripheral vs Central Nystagmus — Key Points

Peripheral Nystagmus	Central Nystagmus
Latency period before onset	No latency period

Peripheral Nystagmus	Central Nystagmus
Fatiguable (diminishes with repeated testing)	Non-fatiguable
Duration <1 minute	Duration >1 minute
Suppressed by visual fixation	NOT suppressed by visual fixation
Unidirectional (does not change direction)	May change direction with gaze
Fast phase AWAY from lesion (Ewald's 2nd law)	Fast phase variable

• Peripheral nystagmus does NOT change direction with different head positions — this is a cardinal feature distinguishing it from central pathology.

Vestibular Neuritis vs Labyrinthitis vs Menière

• **Vestibular neuritis:** sudden onset vertigo, *no* hearing loss, *no* tinnitus, self-limiting (3 days – 3 weeks). Typically viral.

• **Labyrinthitis:** vertigo *with* hearing loss and tinnitus — cochlea also involved.

• **Menière disease:** episodic, intermediate duration (20 min to several hours), with fluctuating SNHL, tinnitus, and aural fullness.

• Vertigo and unsteadiness are **most prevalent in the elderly (>60 years)**, mainly due to presbyvestibulopathy and BPPV.

5 · Hearing Assessment in Children

ABR Indications and Exclusions

• ABR is indicated when an infant has: congenital infections (TORCH), family history of early-onset hearing loss, hyperbilirubinemia, low birth weight, prematurity, prolonged NICU stay, or exposure to ototoxic drugs.

• Maternal severe depression is NOT an indication for neonatal ABR screening.

• The **highest-risk combination** in the NICU: prolonged stay + aminoglycoside treatment + pathological hyperbilirubinemia → synergistic cochleotoxicity.

• ABR can detect **auditory neuropathy** (dyssynchrony); OAE cannot, because the outer hair cells are intact in auditory neuropathy.

• **Caesarean section delivery is NOT a risk factor** for hearing loss in children.

Speech Delay in a 5-Year-Old

• A 5-year-old with speech delay and inattention should be investigated for **otitis media with effusion (OME)** first — it is the most common cause of acquired hearing loss in this age group and is easily missed clinically.

6 · Hearing Loss and Audiological Concepts

Otosclerosis — CHL not SNHL

• **Otosclerosis** causes stapedial fixation → **conductive hearing loss**. It is NOT a cause of sensorineural hearing loss. It may rarely progress to Schwartze sign (flamingo pink blush), but the audiological hallmark is a Carhart's notch at 2000 Hz on bone conduction, NOT true SNHL.

Otitis Externa and Hearing Loss

• Otitis externa does **not** cause significant sensorineural or conductive hearing loss sufficient to warrant hearing screening. Patients with a history of OE do **not** need hearing loss surveillance programs.

Two Most Important Risk Factors for Hearing Loss

• The two most clinically important risk factors for hearing loss are **aging (presbycusis)** and **noise exposure**.

Sensorineural Hearing Loss — Clinical Behaviour

• SNHL patients **speak in a loud voice** (cannot monitor their own voice adequately).

• They hear **worse in noise** (poor frequency resolution; unlike CHL patients who hear better in noise — paracusis Willisii).

• **Recruitment** (abnormal loudness growth — small increase in sound intensity causes disproportionate increase in perceived loudness) is a hallmark of **cochlear** (sensory) hearing loss.

Tympanometry — Important Distinctions

• Tympanometry is NOT a hearing test. It measures middle ear compliance (impedance), not hearing thresholds.

• It cannot be performed reliably if the ear canal contains wax.

• Type B tympanogram (flat trace) occurs in both middle-ear effusion and tympanic membrane perforation. Differentiation: **increased external canal volume** on tympanometry = perforation; normal canal volume with B trace = effusion.

• Absent stapedial reflex accompanies Type B regardless of cause.

Weber and Rinne Test Interpretation

Condition	Rinne	Weber
CHL (right)	Negative (BC>AC)	Lateralises to RIGHT (affected ear)
SNHL (right)	Positive (but reduced)	Lateralises to LEFT (away from lesion)
False-negative Rinne*	Negative on LEFT	Lateralises to RIGHT

False-Negative Rinne: Occurs when there is *severe SNHL on one side*. Bone conduction from the worse ear (left) crosses to the better cochlea (right), making BC appear better than AC on the left → Rinne appears negative on the left. Weber lateralises to the BETTER (right) ear. This mimics left CHL but is actually severe left SNHL. Key clue: Weber goes to the OPPOSITE side of the 'negative Rinne'.

Presbycusis — Audiogram Pattern

• Characteristic audiogram: **bilateral, symmetrical, high-frequency sensorineural hearing loss with a downward sloping pattern**. Affects the basal (high-frequency) cochlear turns first.

• Patients have greatest difficulty with consonant discrimination and hearing in noisy environments.

Mixed Hearing Loss — Audiogram

• Mixed hearing loss = both air conduction AND bone conduction are abnormal, with an air-bone gap present. Examples: otosclerosis with cochlear involvement, CSOM with inner ear fistula.

7 · Epistaxis and Nasal Trauma

Paediatric Epistaxis

• In children, epistaxis is most commonly caused by **local inflammation and digital trauma** (nose-picking). Foreign bodies are the second most common cause in this age group but are less frequent than inflammation/trauma.

• Tumours and bone destruction suggest malignancy and are associated with adult/elderly presentations, not children.

Posterior Nasal Packing — Monitoring Requirements

• Posterior packing is used for severe, uncontrolled posterior bleeding from Woodruff's plexus.

• It requires **hospital admission and close monitoring** for: apnoea, hypoxia, and cardiac arrhythmias (vagal reflex from pack pressure).

• It is used in combination with anterior packing, NOT as a standalone measure for mild cases.

Septal Haematoma — Must-Know Points

• Septal haematoma is located **between the perichondrium and the cartilage**, depriving the avascular cartilage of its nutrient supply.

• It is usually unilateral and results from nasal trauma.

• Drainage is ALWAYS required — even a small haematoma must be evacuated. Leaving it leads to: organisation → fibrosis → avascular necrosis of the cartilage → *saddle nose deformity*. There is no safe threshold for watchful waiting.

8 · Rhinosinusitis, Rhinitis and Nasal Pathology

Allergic Rhinitis — Allergen Testing

• The most common **perennial** allergen (including in Jordan) is the **house dust mite** (Dermatophagoides pteronyssinus/farinae).

• If a skin prick test is negative in a clinically allergic patient, the most likely explanation is that the **specific allergen was not included in the test panel** — not that the patient is non-allergic. The test only screens for allergens included in the panel.

Vasomotor Rhinitis

• Diagnosed by **exclusion** of all other causes of rhinitis (allergic, infectious, drug-induced, hormonal).

• Has two subtypes: **eosinophilic (NARES)** and **non-eosinophilic**.

• Triggers include cold air, strong odours, alcohol, and changes in atmospheric pressure — not allergens or infection.

Atrophic Rhinitis — Points Not in HY File

• Characterised by **enlarged nasal cavities** with atrophied turbinates, green or grey crusts, and a foul odour (ozena — paradoxical anosmia despite an open airway).

• More common in **females**; associated with **Klebsiella ozaenae**.

• **Polyps are NOT present** in atrophic rhinitis — this distinguishes it from hypertrophic/allergic conditions.

• Surgical treatment involves **narrowing or closure (Young's operation)** of the nasal cavities to reduce airflow and allow mucosa to regenerate.

Unilateral Nasal Obstruction — Workup

• In an adult with *unilateral* nasal obstruction and rhinorrhoea, the **first investigative step is CT of the sinuses**. This rules out a tumour or other structural pathology before proceeding to biopsy or surgery.

Sinus Drainage Anatomy

Drainage Site	Structure Draining Into It
Superior meatus	Posterior ethmoidal sinuses
Middle meatus (ostiomeatal complex)	Anterior ethmoidal, frontal, and maxillary sinuses
Inferior meatus	Nasolacrimal duct
Sphenoethmoidal recess	Sphenoidal sinus

Sinus Development — Birth to Maturity

• At birth, **only the ethmoid and maxillary sinuses** are present (though small).

• Frontal sinus: begins development at 6–7 years, fully pneumatised in early adulthood.

• Sphenoidal sinus: begins at 5–6 years.

• This is why **ethmoid sinusitis is the most common sinusitis in children under 8** — it is the only fully developed sinus. Ethmoidal sinuses also have a poor natural drainage system.

Complications of Sinusitis

• Recognised intracranial complications: subdural abscess (most common), intracerebral abscess, epidural abscess, meningitis, cavernous sinus thrombosis.

• Temporal lobe abscess is NOT a complication of sinusitis — the most common source of brain abscess from sinusitis is the frontal sinus, which can cause a frontal lobe abscess, not temporal.

• Orbital complications follow Chandler's classification (preseptal cellulitis → orbital cellulitis → subperiosteal abscess → orbital abscess → cavernous sinus thrombosis).

• **Fungal sinusitis** should raise suspicion of underlying **immunodeficiency** (DM, haematological malignancy, immunosuppressive therapy).

Nasal Polyps — Theory and Subtypes

• The pathogenesis of nasal polyps is **multifactorial**: involves allergic inflammation, imbalance of inflammatory mediators, and genetic predisposition.

• Chronic infection alone is NOT the underlying cause of nasal polyps.

• Nasal polyps are most commonly ethmoidal in origin and arise from the middle meatus / anterior ethmoid region.

• **Antrochoanal polyps** arise specifically from the **maxillary sinus** mucosa. They are unilateral, non-eosinophilic, and **benign** — they do NOT undergo malignant transformation and do not bleed easily.

• Best view for maxillary sinus on plain X-ray: **Water's view** (occipitofrontal with chin raised).

Chronic Rhinosinusitis — Diagnostic Criteria and Management

• Diagnostic criteria for CRS require 2 or more cardinal symptoms for ≥12 weeks: nasal blockage/congestion, nasal discharge, facial pain/pressure, and reduced sense of smell. Sneezing is NOT a diagnostic criterion for CRS.

• CRS does **NOT** have a 90% cure rate — it is a chronic, relapsing condition. Medical therapy controls rather than cures.

• **Wegener's granulomatosis (GPA): involves small and medium vessels; presents with nasal, pulmonary, and renal disease. It is fatal without treatment — aggressive immunosuppression (cyclophosphamide + steroids) is required.**

• Acute sinusitis **pain has diurnal variation**: maxillary sinus pain worsens in the morning (supine pooling) and improves as secretions drain when upright.

9 · Adeno-Tonsillar Disease

Adenoid Development and Immunoglobulin Production

• Adenoids produce IgA, IgG, IgM, and IgD. They are NOT well-developed at birth — they peak in size at approximately **age 6** and then progressively atrophy until adolescence.

Pharyngeal Anatomy — Pillars

• The **anterior pillar** of the fauces = palatoglossus muscle.

• The **posterior pillar** = **palatopharyngeus muscle**.

Velopharyngeal Function

• **Adenoid hypertrophy** → nasal obstruction → **hyponasality** (rhinolalia clausa — muffled voice).

• **Velopharyngeal insufficiency (VPI)** — failure of the soft palate to seal the nasopharynx — causes **hypernasality**. This occurs after adenoidectomy in patients with sub-mucous cleft palate or short palate.

Antibiotic Choice in Bacterial Tonsillitis

• First-line antibiotic for acute bacterial tonsillitis caused by Group A β-haemolytic streptococcus = **Penicillin** (or amoxicillin). Penicillin is superior to amoxicillin in preventing rheumatic fever and PSGN.

• For tonsillitis not responding to antibiotics with rash and hepatosplenomegaly → **infectious mononucleosis (EBV)**. Antibiotics (especially amoxicillin/ampicillin) should be avoided as they cause a maculopapular rash in EBV.

Scarlet Fever

• Caused by Group A β-haemolytic streptococcus producing **erythrogenic toxin**.

• Characteristic findings: **strawberry tongue** + **sandpaper (blanching) rash** starting in skin folds and spreading centrally.

• Tonsils appear swollen and red, often with exudate. Distinguish from EBV by absence of hepatosplenomegaly and presence of the rash.

Diphtheria

• Caused by **Corynebacterium diphtheriae** (NOT viral). Produces a grey, adherent **pseudomembrane** that bleeds when removed. Can extend to the larynx (croup-like) and produce toxin causing myocarditis and neuropathy.

• Treated with antitoxin + antibiotics (penicillin or erythromycin). Antibiotics alone are insufficient.

Pharyngitis — Antibiotic Effectiveness

• Antibiotics reduce the risk of acute glomerulonephritis (PSGN) when treating Group A strep pharyngitis. The claim that they reduce PSGN risk by *50%* is incorrect — evidence for PSGN prevention by antibiotics is not as robust as for rheumatic fever prevention.

Adenoidectomy — Contraindication

- **Cleft palate is an absolute contraindication to adenoidectomy. The adenoid mass compensates for the velopharyngeal gap; removal causes velopharyngeal insufficiency and hypernasality.**

Tonsillectomy — Timing of Complications

- Early complications (within 24 hours): primary haemorrhage, dental damage, TMJ dislocation, uvular injury.
- **Secondary haemorrhage (5–10 days post-op, due to slough separation from the tonsillar bed) is a LATE complication, not early.**

Parapharyngeal Abscess

- Treatment requires **external drainage + systemic antibiotics**. Unlike peritonsillar abscess (intraoral drainage), the parapharyngeal space is accessed externally due to proximity to the carotid sheath.

10 · Stridor and Airway Management

Congenital vs Acquired Stridor

- **Epiglottitis is an ACQUIRED cause of stridor, not congenital. It is caused by Haemophilus influenzae type b or, increasingly in vaccinated populations, Staphylococcus aureus and Group A strep.**
- Congenital causes: laryngomalacia (most common), vocal cord paralysis, subglottic stenosis, laryngeal webs, haemangiomas, cysts.

Laryngomalacia vs Vocal Cord Palsy — Differentiation

- **Laryngomalacia:** inspiratory stridor, omega-shaped (floppy) epiglottis, short aryepiglottic folds, cry is normal in quality, self-limiting (resolves by ~18 months).
- **Vocal cord palsy: weak cry + hoarseness** in a neonate — the classic presentation. May be biphasic stridor if bilateral. Often associated with CNS abnormality or birth trauma.
- Stridor laryngismus (laryngospasm from hypocalcaemia) is **NOT associated with pyrexia** — this distinguishes it from infective causes of stridor.

Vocal Cord Paralysis — Aetiology

- Most common cause of **unilateral** VC paralysis overall: **thyroid surgery** (injuring the RLN during thyroidectomy).
- Most common cause overall (including bilateral): **surgical trauma** in the neck and chest.
- Causes of **bilateral** VC paralysis: carcinoma of the upper oesophagus (compresses both RLNs in the mediastinum), thyroid carcinoma, or CNS pathology.
- Left RLN palsy alone: caused by lesions along its longer intrathoracic course — left bronchus, aortic arch, mediastinal lymphadenopathy.

Stridor Location by Type

Stridor Type	Likely Location
Inspiratory	Supraglottic or glottic lesion
Expiratory	Subglottic or intrathoracic tracheal lesion
Biphasic	Fixed obstruction at glottis or subglottis (e.g. laryngeal web, subglottic stenosis

Croup — Treatment Exclusion

• Acute laryngotracheobronchitis (croup) is caused by **parainfluenza virus** (predominantly type 1). Treatment: humidified oxygen, nebulised adrenaline, systemic corticosteroids.

• Antibiotics are NOT part of croup treatment — it is a viral disease and bacterial superinfection is rare.

Tracheostomy — Indications and Exclusions

• Tracheostomy is indicated for: bilateral VC paralysis, laryngeal tumours, prolonged ventilation (complicated thoracic/abdominal surgery, COPD with respiratory failure), trauma to upper airway.

• Unilateral VC (RLN) paralysis does NOT require tracheostomy — the contralateral cord compensates.

• Severe upper lung atelectasis is managed bronchoscopically or by chest physiotherapy, not tracheostomy.

• Ludwig's angina (bilateral submandibular space cellulitis): potentially life-threatening, may progress to airway compromise → **may require tracheostomy**.

11 · Neck Masses

Congenital Neck Mass Classification

• **Most common congenital neck mass overall = thyroglossal duct cyst** (midline, moves with tongue protrusion and swallowing, at or below the hyoid).

• Midline neck masses include: thyroglossal cyst, dermoid cyst, thyroid enlargement, submental lymph node.

• Branchial cleft cyst is a LATERAL neck mass — it is NOT a midline mass.

• Dermoid cyst: rubbery, movable from side to side (distinguishing it from thyroglossal cyst which moves with swallowing/tongue protrusion), not rapidly progressive, not tender.

Carotid Body Tumour

• Carotid body tumour (chemodectoma/paraganglioma): highly vascular, located at the carotid bifurcation, splays the carotid vessels (lyre sign on angiography).

• Biopsy is CONTRAINDICATED due to risk of massive haemorrhage. Diagnosis is by CT/MRI/angiography.

Juvenile Nasopharyngeal Angiofibroma (JNA)

• Should be suspected in an **adolescent male** with recurrent, severe, intractable epistaxis.

• Diagnosis: **CT scan** (shows enhancing mass in the nasopharynx, often with bony erosion). MRI is complementary but CT is the diagnostic standard for bony detail. Biopsy is contraindicated pre-embolisation.

• Treatment: pre-operative **embolisation** followed by **surgical resection**. Surgery is the definitive treatment; embolisation alone is not sufficient.

Malignant Parotid Tumours

• Among malignant parotid gland tumours, **mucoepidermoid carcinoma** has the best prognosis (especially the low-grade variant).

12 · Head and Neck Oncology

Nasopharyngeal Carcinoma (NPC)

• Most common cranial nerve palsy = **CN VI (abducens)**, resulting in diplopia. CN VI has the longest intracranial course and traverses Dorello's canal, making it vulnerable to compression at the skull base.

• Unilateral OME (secretory otitis media) in an adult should raise suspicion of NPC until proven otherwise — tumour blocks the Eustachian tube opening in the fossa of Rosenmüller.

• After radiation to the nasopharynx/neck, a long-term risk is the development of **radiation-induced thyroid carcinoma**.

Glottic Tumour Staging (T Classification)

Stage	Description
T1a	Tumour confined to one vocal cord, normal mobility
T1b	Tumour involves BOTH vocal cords, normal mobility
T2	Tumour extends to supraglottis or subglottis, or impaired cord mobility
T3	Fixed vocal cord (tumour confined to larynx)
T4	Tumour extends through cartilage or beyond larynx

Lymph Node Metastasis in Laryngeal Cancer

• **Glottic tumours have a very low rate of cervical lymph node metastasis** because the glottis has a sparse lymphatic network. This contributes to their early diagnosis (hoarseness) and best prognosis among laryngeal cancers.

• Diagnosis of laryngeal tumours is by **direct laryngoscopy with biopsy** — not indirect laryngoscopy alone.

Hidden Primary Tumours in H&N; SCC

• 'Hidden primary' refers to tumours that remain clinically silent until they present with a cervical lymph node metastasis.

• Typical hidden primary sites: base of tongue, tonsil, pyriform fossa, supraglottic larynx, nasopharynx.

• Vocal cord (glottis) tumours are NOT hidden primaries — they cause early, obvious hoarseness and are detected at an early stage.

• Workup for unknown primary (FNA shows SCC): **pan-endoscopy** (triple endoscopy: laryngoscopy + bronchoscopy + oesophagoscopy) to identify the primary tumour.

Clinical Behaviour of Specific H&N; Tumours

• **Pyriform fossa (hypopharyngeal) early tumour** → presents with **referred otalgia** via CN IX (Jacobson's nerve) before the patient notices dysphagia. Dysphagia is a late symptom.

• Dysphagia + referred otalgia on laryngoscopy → investigate for **hypopharyngeal (piriform fossa) tumour**.

• **Most common oral cavity tumour**: base of tongue carcinoma.

• **Metastases to cervical nodes** are most commonly sourced from **tonsil** primaries (among oropharyngeal sites).

• The **major function of the larynx** is **protection of the lower airway** (sphincteric function during swallowing), not phonation.

Supraglottic Tumours

• The **earliest symptom of supraglottic tumours** is **dysphagia** (or change in voice quality — 'hot potato voice'). Unlike glottic tumours, they do NOT cause early hoarseness because the vocal cords are not involved initially.

• Supraglottic tumours have a **rich lymphatic supply** → early bilateral neck node metastasis → worse prognosis than glottic tumours.

Lingual Thyroid — Foramen Caecum Mass

• A mass at the **foramen caecum** (base of tongue) may represent a **lingual thyroid** — ectopic thyroid tissue that may be the patient's only functioning thyroid tissue.

• Before any biopsy or surgical excision, a thyroid radionuclide scan must be obtained to determine whether functional thyroid tissue exists in the neck. Removing the only thyroid tissue causes permanent hypothyroidism.

Zenker's Diverticulum and Oesophageal Pathology

• **Zenker's diverticulum** (pharyngeal pouch): posterior herniation of pharyngeal mucosa through Killian's triangle (between inferior constrictor and cricopharyngeus), commonly in elderly patients, presents with regurgitation of *undigested* food, halitosis, and dysphagia.

• Oesophageal foreign body: **mediastinitis** is the most fatal complication (from oesophageal perforation). Fish bones are the most common FB, and the *cricopharyngeus* (upper oesophageal sphincter) is the most common site of impaction.

• LPR (laryngopharyngeal reflux / GERD) causes **posterior laryngitis** (posterior commissure erythema and oedema) — NOT anterior laryngitis.

Bell's Palsy

• Bell's palsy: idiopathic (unknown aetiology, possibly viral reactivation of HSV-1), affects the facial nerve, may be preceded by post-auricular pain, causes unilateral LMN facial palsy.

• Treatment: oral corticosteroids ± antivirals (aciclovir). Surgery is NOT the standard treatment for Bell's palsy.

• Prognosis: 70–80% recover completely without intervention.

Greater Superficial Petrosal Nerve

• The greater superficial petrosal nerve (GSPN) is a branch of CN VII carrying **preganglionic parasympathetic (secretomotor) fibres** to the pterygopalatine ganglion, ultimately innervating the lacrimal gland and minor palatal salivary glands.

Eustachian Tube — Anatomy

• The Eustachian tube is **approximately 3.5 cm** in adults and **shorter (≈2.5 cm) in children**. The shorter, more horizontal orientation in children explains the higher incidence of otitis media in this age group. The tube opens by action of the tensor veli palatini (NOT levator).

Superior Maxillary Sinus Cancer

• Tumours in the **superior compartment** of the maxillary sinus (above Ohngren's line) have a worse prognosis. Treatment of choice: combined **radiotherapy + total maxillectomy + orbital exenteration** when the orbit is involved.

Note on Ambiguous or Potentially Erroneous Past Paper Questions

- A question stated 'Tinnitus is seen in all EXCEPT: A. Loud noise' with answer A. Loud noise IS a recognised cause of tinnitus. This appears to be a typographical or answer-key error in the past paper; clinically, noise-induced tinnitus is well-established.
- The question on atrophic rhinitis (answer A = 'mainstay of surgical treatment is narrowing or closure') is internally inconsistent with standard teaching, which affirms Young's operation (closure). This may reflect an ambiguity about 'mainstay' vs 'adjunct'.
- The question on CSOM intracranial complications with answer D (local osteomyelitis) as the exception may reflect institutional variation. Standard teaching includes osteomyelitis as a route for intracranial extension.

These discrepancies are noted for awareness. When faced with such questions in the exam, choose the answer that best matches the majority teaching in your course slides.
