SALIVARY GLAND DISEASES

A Clinical and Surgical Review for Postgraduate Students

Applied Anatomy • Sialolithiasis • Sialadenitis
Autoimmune & Granulomatous Disease • Cystic Lesions
Investigations & Milan Cytology • Benign & Malignant Neoplasms
Surgical Management • Current Guidelines

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A concise, examination-oriented text for postgraduate trainees in ENT-Head & Neck surgery, general surgery, oral & maxillofacial surgery and pathology

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Contents

Learning Objectives

LEARNING OBJECTIVES

Describe the surgical anatomy of the major and minor salivary glands, with emphasis on the facial nerve and its relations to the parotid gland.

Classify salivary gland disease by aetiology — obstructive, infective/inflammatory, autoimmune/granulomatous, cystic and neoplastic.

Diagnose and manage sialolithiasis, including the modern role of sialendoscopy.

Differentiate acute, chronic and recurrent sialadenitis, including juvenile recurrent parotitis and viral parotitis (mumps).

Recognise autoimmune and granulomatous salivary gland disease — Sjögren's syndrome, sarcoidosis, tuberculosis, IgG4-related disease and HIV-associated salivary disease.

Select appropriate imaging and cytological work-up, including the Milan System for Reporting Salivary Gland Cytopathology.

Apply the “80% rule” and current WHO classification to benign and malignant salivary neoplasms, and outline first-line management.

Describe parotidectomy and submandibular gland excision, and anticipate and manage their complications, including Frey's syndrome and facial nerve injury.

1. Introduction and Applied Surgical Anatomy

Salivary gland disease encompasses a broad spectrum of obstructive, infective, autoimmune, cystic and neoplastic conditions affecting the three paired major salivary glands (parotid, submandibular and sublingual) and several hundred minor salivary glands scattered throughout the oral cavity, oropharynx, and upper aerodigestive tract mucosa. A sound grasp of applied anatomy is the foundation for safe diagnosis and surgery in this region.

1.1 The Parotid Gland

The largest salivary gland, the parotid lies in the preauricular region, wrapping around the posterior border of the mandibular ramus. It is enclosed by the parotid (investing) fascia and is conventionally, though artificially, divided by the plane of the facial nerve into superficial and deep lobes — a distinction of great surgical importance since roughly 80% of the gland's volume, and most parotid tumours, lie in the superficial lobe. Its secretomotor innervation is parasympathetic, via the glossopharyngeal nerve (otic ganglion, auriculotemporal nerve). Saliva drains via **Stensen's duct**, which runs across the masseter, turns medially at its anterior border to pierce the buccinator, and opens into the oral cavity opposite the second upper molar. The **facial nerve (CN VII)** exits the stylomastoid foramen and enters the posteromedial parotid, dividing at the pes anserinus into temporofacial and cervicofacial divisions, then branching further into temporal, zygomatic, buccal, marginal mandibular and cervical branches; its identification and preservation is the single most important technical objective of parotid surgery. The **retromandibular vein** lies deep to the nerve and serves as a useful intraoperative landmark, and the external carotid artery lies deeper still, embedded within the gland substance.

Facial nerve branch	Principal muscles supplied	Clinical effect if injured
Temporal	Frontalis, orbicularis oculi (upper)	Loss of forehead wrinkling, brow ptosis
Zygomatic	Orbicularis oculi (lower), zygomaticus	Impaired eye closure — risk of exposure keratopathy
Buccal	Buccinator, orbicularis oris, upper lip elevators	Impaired cheek/upper lip movement, oral incompetence
Marginal mandibular	Depressor anguli oris, lower lip depressors	Asymmetric smile, lower lip droop — most vulnerable branch in neck/submandibular surgery

Facial nerve branch	Principal muscles supplied	Clinical effect if injured
Cervical	Platysma	Usually of little functional consequence if injured

Landmarks used to locate the facial nerve trunk at parotidectomy include the tragal pointer (nerve lies ~1 cm deep and inferior to it), the tympanomastoid suture line (nerve lies ~6–8 mm deep to its inferior end), and the posterior belly of digastric (nerve lies at the same depth, just superior to the muscle's insertion).

1.2 The Submandibular Gland

Located in the submandibular triangle, this gland wraps around the posterior free border of mylohyoid, with superficial and deep parts continuous with one another. **Wharton's duct** runs forward along the floor of the mouth, crossing the lingual nerve twice (lateral to medial, then medial to lateral, as the nerve loops beneath it) before opening at the sublingual papilla beside the lingual frenulum. The **marginal mandibular branch** of the facial nerve runs close to the gland, typically superficial to the facial vessels, and is at particular risk during submandibular gland excision unless the vessels are ligated and retracted appropriately.

1.3 The Sublingual Gland and Minor Salivary Glands

The sublingual gland lies in the floor of the mouth, draining via multiple small ducts (ducts of Rivinus), some joining Wharton's duct, others opening independently. Minor salivary glands are distributed throughout the palate, lips, buccal mucosa, tongue base and elsewhere; though individually small, they are the commonest site of origin for some malignant salivary neoplasms, particularly at the junction of the hard and soft palate.

2. Physiology of Salivation

Approximately 0.5–1.5 litres of saliva are produced daily, roughly two-thirds from the submandibular glands (mixed seromucinous secretion), with the parotid contributing a predominantly serous secretion that increases markedly with stimulation (e.g., mastication), and the sublingual and minor glands contributing a mucous-rich secretion important for lubrication. Saliva subserves digestion (salivary amylase), lubrication and speech, antimicrobial defence (lysozyme, lactoferrin, secretory IgA), buffering of oral pH, and dental remineralisation. Reduced salivary flow (xerostomia) — from autoimmune disease, radiotherapy, anticholinergic medication or dehydration — predisposes to dental caries, oral candidiasis and ascending sialadenitis, and is a recurring theme across several of the conditions in this review.

3. Classification of Salivary Gland Disease

A simple aetiological framework helps organise the differential diagnosis of a salivary gland swelling and guides the initial work-up:

Category	Representative conditions
Obstructive	Sialolithiasis, ductal stricture, mucus plugging
Infective / inflammatory	Acute bacterial sialadenitis, viral sialadenitis (mumps), chronic sialadenitis, juvenile recurrent parotitis, tuberculosis
Autoimmune / granulomatous	Sjögren's syndrome, sarcoidosis, IgG4-related disease, HIV-associated salivary gland disease
Cystic	Mucocele, ranula, lymphoepithelial cyst, first branchial cleft cyst (parotid region)
Neoplastic — benign	Pleomorphic adenoma, Warthin tumour, oncocytoma, basal cell adenoma, and other rarer benign entities
Neoplastic — malignant	Mucoepidermoid carcinoma, adenoid cystic carcinoma, acinic cell carcinoma, carcinoma ex pleomorphic adenoma, salivary duct carcinoma, and others
Developmental	Aplasia/agenesis, accessory salivary tissue, ductal atresia
Traumatic / iatrogenic	Sialocele, fistula, radioiodine-induced sialadenitis

A useful clinical rule of thumb is that **bilateral, diffuse** gland swelling more often reflects a systemic, infective or autoimmune process, whereas a **unilateral, discrete** mass is more likely to be neoplastic — though there is overlap, and this should prompt, not replace, appropriate investigation.

4. Sialolithiasis

Sialolithiasis is the commonest disease of the salivary glands, accounting for the majority of obstructive salivary symptoms. Approximately 80–85% of salivary calculi occur in the submandibular gland or Wharton's duct, reflecting its more viscous, alkaline, mucin- and calcium-rich secretion, its longer and more tortuous duct with an uphill course against gravity, and the wider calibre of the duct at its orifice relative to its origin. Around 10–20% occur in the parotid gland and a small minority in the sublingual or minor glands.

4.1 Pathogenesis

Stone formation is thought to begin with a nidus (desquamated cells, mucus, foreign material or bacteria) around which calcium salts — predominantly hydroxyapatite and calcium phosphate — are deposited in concentric lamellae, promoted by ductal stasis, dehydration and local inflammation.

4.2 Clinical Features

Patients typically report **meal-time pain and swelling** of the affected gland, reflecting salivary stasis as secretion is stimulated against an obstructed duct, with gradual resolution between meals as the gland empties partially around the stone. A palpable stone may be felt bimanually along the course of Wharton's duct in the floor of the mouth. Secondary infection produces a tender, erythematous, purulent-discharging gland (acute suppurative sialadenitis) and warrants prompt antibiotic therapy.

4.3 Investigations

Plain radiographs (occlusal views for the floor of mouth) detect only radio-opaque stones (≈80% of submandibular but only ≈40% of parotid calculi are radio-opaque, reflecting differing mineral content). **Ultrasound** is the first-line investigation, sensitive for stones >2 mm and free of radiation, and can additionally demonstrate ductal dilatation. **Non-contrast CT** is highly sensitive for calcified stones and is useful when ultrasound is equivocal. **Sialography** (contrast injection into the duct) and **MR sialography** delineate ductal anatomy and strictures, particularly useful pre-operatively for sialendoscopy planning, though conventional sialography is contraindicated in acute infection.

4.4 Management

Small, distal stones may be managed conservatively (hydration, sialogogues, gland massage, warm compresses, analgesia and antibiotics if infected) or removed transorally if palpable near the duct orifice. **Sialendoscopy** — endoscopic visualisation and instrumentation of the ductal system using fine semi-rigid endoscopes — has transformed management over the past two decades: it allows direct visualisation, basket or forceps retrieval of stones (typically up to 4–5 mm), intracorporeal or combined with extracorporeal lithotripsy for larger stones, and balloon dilatation of strictures, achieving gland preservation in the great majority of cases and avoiding gland excision. A combined (endoscopic-assisted transoral) approach is used for larger, more proximal submandibular stones. Gland excision (submandibular gland excision or superficial parotidectomy) is now reserved for cases where minimally invasive techniques fail, for multiple intraparenchymal stones, or an irreversibly damaged, non-functioning gland.

5. Sialadenitis

5.1 Acute Bacterial (Suppurative) Sialadenitis

Most often affects the parotid gland in dehydrated, debilitated or post-operative patients with reduced salivary flow, permitting retrograde bacterial ascent from the oral cavity (typically *Staphylococcus aureus*, streptococci and oral anaerobes). Presents with acute painful, tender, erythematous swelling, trismus and purulent discharge expressible from the duct orifice on gentle massage; fever and leucocytosis are common. Management is with rehydration, sialogogues, gland massage, oral hygiene and empirical antibiotics (covering *S. aureus* and anaerobes); abscess formation, suggested by fluctuance or on ultrasound/CT, requires incision and drainage or image-guided aspiration.

5.2 Viral Sialadenitis (Mumps)

Caused by the paramyxovirus mumps virus, this remains the commonest cause of painful bilateral parotid swelling worldwide, particularly in unvaccinated populations, with peak incidence in school-age children but rising recognition in incompletely immunised young adults. Prodromal fever, malaise and myalgia precede parotid swelling with earlobe displacement; the diagnosis is largely clinical, supported by exposure history, and management is supportive (analgesia, hydration). Complications include orchitis (a notable cause of subfertility if bilateral), oophoritis, pancreatitis, meningoencephalitis and sensorineural hearing loss. MMR vaccination remains the mainstay of prevention.

5.3 Chronic and Recurrent Sialadenitis

Repeated episodes of gland swelling and pain, often related to low-grade ductal obstruction, stricture or sialectasis, lead to progressive parenchymal fibrosis and reduced function. **Juvenile recurrent parotitis** is the commonest cause of recurrent parotid swelling in children after mumps-preventable disease, characteristically unilateral or asymmetrically bilateral, non-obstructive (no stone), with a presumed congenital ductal ectasia aetiology; it typically remits around puberty, and sialendoscopy (both diagnostic — demonstrating characteristic sialectasis — and therapeutic, via ductal irrigation and steroid instillation) has become a key tool in reducing recurrence.

5.4 Radioiodine-Induced Sialadenitis

Salivary glands concentrate iodine and are frequently affected by radioiodine (I-131) therapy for thyroid cancer, producing acute and sometimes chronic sialadenitis, xerostomia and an increased risk of secondary obstruction; sialendoscopy with ductal irrigation has an emerging role in symptomatic patients, alongside preventive measures such as sialogogues and hydration peri-therapy.

6. Autoimmune and Granulomatous Salivary Gland Disease

6.1 Sjögren's Syndrome

A chronic autoimmune exocrinopathy causing lymphocytic infiltration of salivary and lacrimal glands, producing the classic sicca symptoms of xerostomia and keratoconjunctivitis sicca. It may be primary or secondary (associated with rheumatoid arthritis, systemic lupus erythematosus or other connective tissue disease), and predominantly affects women in the fourth to sixth decades. Diagnosis uses the **2016 ACR/EULAR classification criteria**, which combine objective ocular staining, Schirmer's test, unstimulated salivary flow rate, minor salivary gland (labial) biopsy focus score, and serology (anti-Ro/SSA, anti-La/SSB). Bilateral, diffuse, non-tender parotid enlargement is a recognised feature, and patients carry a significantly increased lifetime risk (estimated 5–10%, i.e., 15–20-fold general-population risk) of developing **mucosa-associated lymphoid tissue (MALT) lymphoma** of the salivary gland, which should be suspected with a new, persistent, firm or asymmetric mass in a patient with known Sjögren's syndrome. Management is symptomatic (saliva and tear substitutes, sialogogues, meticulous dental care) with systemic immunomodulation reserved for extraglandular disease.

6.2 Granulomatous Disease — Sarcoidosis and Tuberculosis

Sarcoidosis may involve the parotid gland, producing painless swelling; the combination of parotid enlargement, uveitis, facial nerve palsy and fever constitutes **Heerfordt's syndrome (uveoparotid fever)**, a recognised though uncommon presentation. **Tuberculosis** may affect intraparotid or periparotid lymph nodes or, less commonly, the gland parenchyma itself, mimicking a neoplasm clinically; diagnosis rests on FNAC/biopsy with acid-fast staining, culture or nucleic acid amplification testing, and treatment is standard anti-tuberculous chemotherapy.

6.3 IgG4-Related Disease

A fibroinflammatory systemic disease characterised by IgG4-positive plasma cell infiltration, storiform fibrosis and obliterative phlebitis, classically producing chronic, painless, firm enlargement of the submandibular glands (historically described as “Küttner tumour”) and/or parotid and lacrimal glands (Mikulicz's disease). Elevated serum IgG4 supports the diagnosis but is neither fully sensitive nor specific; tissue biopsy remains the diagnostic standard. It typically responds well to corticosteroids, distinguishing it therapeutically from Sjögren's syndrome and neoplasia, both of which are important differentials.

6.4 HIV-Associated Salivary Gland Disease

HIV infection may produce bilateral parotid swelling from benign lymphoepithelial cysts (multiple, often bilateral, cystic parotid lesions associated with diffuse infiltrative lymphocytosis syndrome) or diffuse CD8+ lymphocytic infiltration; this should be considered in the differential of bilateral parotid cystic swelling, particularly in patients with unknown or untreated HIV status, and antiretroviral therapy often improves the swelling.

7. Cystic Lesions of the Salivary Glands

7.1 Mucocele and Ranula

A **mucocele** results from extravasation of mucus following minor salivary duct trauma (most often the lower lip, from lip-biting) or, less commonly, true retention from ductal obstruction; it presents as a soft, fluctuant, bluish submucosal swelling. A **ranula** is a mucus extravasation pseudocyst arising from the sublingual gland in the floor of the mouth, termed “simple” when confined to the floor of the mouth, and **“plunging” (cervical)** when it dissects through or around mylohyoid into the neck. Management of small mucoceles may be expectant, but recurrent or larger lesions, and ranulas, generally require excision of the mucocele/ranula with the associated sublingual gland (marsupialisation alone carries a high recurrence rate, especially for plunging ranulas).

7.2 Lymphoepithelial and First Branchial Cleft Cysts

Benign lymphoepithelial cysts of the parotid, discussed above in the context of HIV, may also occur sporadically. A **first branchial cleft anomaly** may present as a periauricular or periparotid cyst, sinus or fistula with a close, sometimes intimate, relationship to the facial nerve (Work classification types I and II); recognition is important since excision requires facial nerve identification and dissection analogous to parotid surgery.

8. Differential Diagnosis of a Salivary Gland Swelling

A structured approach to a patient presenting with salivary gland swelling — combining laterality, tempo and associated features — narrows the differential rapidly before investigation is even ordered:

Pattern of presentation	Leading differential
Bilateral, diffuse, chronic	Sjögren's syndrome, sarcoidosis, HIV-associated lymphoepithelial disease, bulimia/alcohol-related sialosis, diabetes mellitus, drug-induced sialosis (e.g., iodine-containing agents)
Bilateral, acute, painful	Viral sialadenitis (mumps and other viruses), acute bilateral bacterial sialadenitis in a debilitated patient
Unilateral, related to meals, fluctuant	Sialolithiasis, ductal stricture
Unilateral, acute, painful, febrile	Acute bacterial sialadenitis / abscess
Unilateral, chronic, painless, slow-growing, mobile	Benign neoplasm (pleomorphic adenoma, Warthin tumour) — first-line work-up with ultrasound and FNAC
Unilateral, rapidly enlarging, painful, fixed, ± facial weakness	Malignant neoplasm until proven otherwise — urgent triple assessment (clinical, imaging, cytology)
Cystic, fluctuant, floor of mouth / lip	Ranula or mucocele

9. Xerostomia and Sialorrhoea

9.1 Xerostomia (Dry Mouth)

Xerostomia — the subjective sensation of dry mouth, usually but not always reflecting reduced salivary flow (hyposalivation) — is common and under-recognised, and predisposes to dental caries, oral candidiasis, dysphagia and ascending sialadenitis. Common causes include medication (anticholinergics, antihistamines, antidepressants, diuretics — by far the commonest cause in older adults, and often multifactorial from polypharmacy), head and neck radiotherapy (dose-dependent, often irreversible above certain cumulative doses to the major glands), Sjögren's syndrome and other autoimmune disease, dehydration, uncontrolled diabetes mellitus, and, less commonly, salivary gland aplasia. Assessment includes a careful drug history, unstimulated and stimulated salivary flow rate measurement, and — where an autoimmune cause is suspected — the investigations outlined in Section 6.1. Management is largely supportive: saliva substitutes, frequent sips of water, sugar-free sialogogues (e.g., xylitol chewing gum), meticulous dental care and fluoride supplementation, and cholinergic agonists (pilocarpine, cevimeline) in patients with residual salivary tissue and no contraindication (e.g., uncontrolled asthma, narrow-angle glaucoma). Amifostine may be used as a radioprotective agent during head and neck radiotherapy in selected patients.

9.2 Sialorrhoea (Drooling)

Excessive drooling most often reflects impaired oral or pharyngeal swallowing coordination rather than true salivary hypersecretion, and is common in cerebral palsy, Parkinson's disease, motor neurone disease and post-stroke dysphagia; it may also occur acutely with local irritation (e.g., teething, oral infection) or as a medication side-effect (e.g., clozapine). Assessment focuses on the severity and impact (skin breakdown, social embarrassment, aspiration risk) and the underlying neurological cause. A stepwise management ladder is generally followed: behavioural and postural techniques and oromotor therapy first line; anticholinergic medication (glycopyrrolate, hyoscine patches) or **botulinum toxin injection into the salivary glands** (parotid ± submandibular, typically ultrasound-guided) as a well-established, repeatable second-line option; and, for severe, refractory cases, surgery — submandibular duct relocation (rerouting the ducts posteriorly toward the tongue base to reduce anterior drooling while preserving some lubrication), submandibular gland excision, or, less commonly, tympanic neurectomy or Wharton's and Stensen's duct ligation.

10. Salivary Gland and Duct Trauma

Penetrating or blunt facial trauma overlying the parotid region — lacerations from assault, road traffic collisions or iatrogenic injury during facial surgery — may injure the parotid gland parenchyma, Stensen's duct, or branches of the facial nerve, and all three must be actively assessed in any deep facial laceration crossing the line from the tragus to the midpoint of the upper lip (the surface projection of Stensen's duct).

10.1 Assessment

Facial nerve function should be examined and documented before any local anaesthetic is given, since paralysis identified afterward cannot be reliably attributed to injury versus anaesthetic effect. Duct injury is suspected with a laceration overlying its course, salivary leakage from the wound, or later development of a sialocele or cutaneous fistula; it can be confirmed by cannulating the duct orifice intraorally and gently irrigating with saline or dilute methylene blue, watching for extravasation at the wound, or by sialography/MR sialography in delayed presentations.

10.2 Management

Parenchymal gland injury alone is managed by meticulous wound closure, a pressure dressing, and anticholinergic medication or gland-output suppression to reduce sialocele formation. Ductal injury proximal to the masseter is best managed by primary microsurgical repair over a fine stent (e.g., an intravenous cannula or silastic tubing) passed via the intraoral orifice; injuries distal to the masseter, or where the proximal duct stump cannot be identified, may be managed by ligation of the duct (accepting subsequent gland atrophy) or, where facilities allow, duct re-routing into the oral cavity. Facial nerve branches identified as transected anterior to a vertical line at the lateral canthus are repaired primarily where possible (fine branches distal to this line have such extensive arborisation and cross-innervation that individual repair is often unnecessary); more proximal or trunk injuries generally warrant exploration and epineural repair or cable grafting.

11. Investigations

11.1 Imaging

Modality	Role / key features
Ultrasound	First-line for most salivary complaints — distinguishes cystic from solid, intra- from extraglandular, guides FNAC, and detects sialolithiasis and ductal dilatation. Operator-dependent and limited for deep-lobe parotid or deeply seated lesions.
MRI	Best soft-tissue definition for salivary neoplasms — characterises benign versus concerning features (well- vs ill-defined margins, T2 signal, diffusion restriction), assesses deep-lobe extension and parapharyngeal space involvement, and detects perineural spread (important in adenoid cystic carcinoma).
CT	Excellent for cortical bone involvement, calcified sialoliths, and staging nodal/distant disease in malignancy; first-line in acute infective/inflammatory complications (abscess, fascial space spread).
Sialography / MR sialography	Delineates ductal architecture — strictures, sialectasis, filling defects — particularly useful in planning sialendoscopy for chronic obstructive or recurrent disease.
Sialendoscopy	Both diagnostic (direct visualisation of the ductal lumen) and therapeutic (stone retrieval, stricture dilatation, steroid irrigation) for non-neoplastic obstructive and inflammatory disease.

11.2 Fine-Needle Aspiration Cytology and the Milan System

FNAC (ideally ultrasound-guided) is the first-line tissue diagnostic test for a salivary mass, with core needle biopsy as an increasingly used adjunct where cytology is indeterminate. The **Milan System for Reporting Salivary Gland Cytopathology** — now in its second edition — standardises reporting into six categories, each carrying an implied risk of malignancy (ROM) that guides management:

Category	Diagnostic category	Typical management
I	Non-diagnostic	Repeat FNA / image-guided sampling
II	Non-neoplastic	Clinical correlation ± follow-up
III	Atypia of undetermined significance (AUS)	Repeat FNA or clinical/imaging follow-up
IVa	Benign neoplasm	Surgery (per lesion type) or observation as appropriate
IVb	Salivary gland neoplasm of uncertain malignant potential (SUMP)	Surgical excision generally recommended
V	Suspicious for malignancy	Surgical planning as for malignancy; consider frozen section
VI	Malignant	Definitive oncological surgical planning ± neck staging

12. Salivary Gland Neoplasms

Salivary neoplasms are relatively uncommon but strikingly histologically diverse — the WHO classification (5th edition, 2022) recognises over 20 distinct entities. The traditional **"80% rule"** remains a useful (if imperfect) teaching aid: approximately 80% of salivary tumours arise in the parotid gland; of parotid tumours, approximately 80% are benign; and of benign parotid tumours, approximately 80% are pleomorphic adenomas. Critically, this relationship inverts as gland size decreases — tumours of the submandibular gland are malignant in roughly 40–50% of cases, of the sublingual gland in the majority, and of minor salivary glands in over half, making the site of origin itself a clinically important prognostic clue.

12.1 Benign Neoplasms

Tumour	Key features
Pleomorphic adenoma	The commonest salivary tumour overall; a slow-growing, painless, mobile mass with a mixed epithelial and myoepithelial/stromal (chondromyxoid) architecture. Has an irregular, often incomplete capsule with microscopic pseudopodial extensions, predisposing to recurrence if enucleated rather than excised with a margin of normal tissue; carries a small but real long-term risk of malignant transformation (carcinoma ex pleomorphic adenoma), which rises with disease duration and multiple recurrences.
Warthin tumour (papillary cystadenoma lymphomatosum)	The second commonest benign parotid tumour; strongly associated with smoking, more common in older men, bilateral or multifocal in a meaningful minority, and classically avid on technetium-99m pertechnetate scanning (unlike most other salivary tumours).
Oncocytoma and other rarer benign tumours	Basal cell adenoma, myoepithelioma, canalicular adenoma and others — individually uncommon, generally managed as for pleomorphic adenoma with complete surgical excision.

12.2 Malignant Neoplasms

Tumour	Key features
Mucoepidermoid carcinoma	The commonest primary salivary malignancy overall, and the commonest salivary malignancy in children. Graded low, intermediate or high; low-grade tumours behave indolently while high-grade tumours are aggressive with nodal and distant metastatic potential.
Adenoid cystic carcinoma	Notorious for perineural invasion and spread — often presenting with pain or facial nerve dysfunction disproportionate to tumour size — with a relentless natural history of slow growth, late (sometimes very late, >10-year) distant recurrence (particularly pulmonary), and a tendency to spread along nerves well beyond the visible tumour margin, making margin status and adjuvant radiotherapy particularly important.
Acinic cell carcinoma	Typically low-grade and slow-growing, most often in the parotid; generally favourable prognosis with complete excision.
Carcinoma ex pleomorphic adenoma	Malignant transformation within a long-standing or recurrent pleomorphic adenoma — a sudden increase in growth rate, new pain, or facial nerve weakness in a patient with a known longstanding parotid lump should raise this suspicion.
Salivary duct carcinoma	An aggressive, high-grade adenocarcinoma histologically resembling high-grade breast ductal carcinoma, often androgen-receptor and/or HER2 positive (informing emerging targeted systemic therapy), with a high rate of nodal and distant metastasis.

12.3 Staging and Prognostic Factors

Malignant salivary tumours are staged using the AJCC/UICC TNM system, incorporating tumour size and extraparenchymal extension (T), nodal involvement (N) and distant metastasis (M). Key adverse prognostic factors, independent of stage, include high histological grade, perineural and lymphovascular invasion, positive margins, facial nerve involvement at presentation, and deep-lobe or extraparotid extension. Facial nerve weakness at presentation is a particularly important clinical red flag — it is uncommon with benign disease and should be assumed to indicate malignancy with nerve involvement until proven otherwise.

12.4 Clinical Assessment of a Salivary Mass

History should establish duration, rate of growth, pain, and cranial nerve symptoms (especially facial weakness); examination should assess mobility versus fixation to skin or deep structures, facial nerve function (all branches), cervical lymphadenopathy, and intraoral/oropharyngeal bulge (suggesting deep-lobe parotid extension into the parapharyngeal space). Any parotid mass with facial nerve palsy, rapid growth, pain, skin fixation or cervical lymphadenopathy should be treated as malignant until proven otherwise.

RED FLAGS SUGGESTING MALIGNANCY IN A SALIVARY MASS

Facial nerve weakness (parotid) or lingual/hypoglossal nerve involvement (submandibular).

Rapid increase in size, or new pain in a previously stable long-standing lump.

Fixation to skin or deep structures; skin ulceration.

Cervical lymphadenopathy.

Site of origin in the submandibular, sublingual or minor salivary glands (higher baseline malignancy rate than the parotid).

DIAGNOSTIC AND MANAGEMENT PATHWAY — A NEW PAROTID MASS

Step 1 — History and examination: Duration, growth rate, pain, facial nerve function (all branches), skin fixation, cervical nodes.

Step 2 — First-line imaging: Ultrasound to confirm intraglandular origin, define cystic vs solid, and guide biopsy.

Step 3 — Tissue diagnosis: Ultrasound-guided FNAC, reported per the Milan System (± core needle biopsy if cytology is indeterminate).

Step 4 — Cross-sectional imaging: MRI (± CT for bone/nodal detail) for any lesion that is large, deep-lobe, clinically suspicious, or cytologically indeterminate/malignant, to define extent and plan surgery.

Step 5 — Multidisciplinary planning: Benign, superficial, Milan IVa lesion → superficial parotidectomy/extracapsular dissection. Suspicious or malignant (Milan V/VI) → oncological parotidectomy ± neck dissection, discussed at a head and neck tumour board, with adjuvant therapy planned according to final histology.

13. Surgical Management

13.1 Parotidectomy

Superficial (extracapsular) parotidectomy, removing the superficial lobe with formal identification and preservation of the facial nerve trunk and its branches, is the standard operation for most benign superficial-lobe tumours; a more limited **extracapsular dissection/partial superficial parotidectomy** with a cuff of normal tissue is increasingly favoured for small, clearly benign-appearing tumours to reduce morbidity, provided oncological principles are respected. **Total (or near-total) parotidectomy**, removing both lobes, is required for deep-lobe tumours or malignant disease, and nerve sacrifice is reserved for cases of gross nerve invasion, with immediate reconstruction (nerve grafting, typically using the great auricular or sural nerve) where the nerve is sacrificed in a curable patient. Intraoperative facial nerve monitoring is widely used, particularly in revision surgery or when anatomy is distorted by inflammation or tumour.

13.2 Submandibular Gland Excision

Performed through a transcervical incision, with careful ligation and elevation of the facial vessels to protect the marginal mandibular nerve, and identification of the lingual and hypoglossal nerves as the gland is mobilised from the floor of the mouth.

13.3 Management of Malignant Disease

Surgery (parotidectomy or gland excision with clear margins) remains the primary treatment for resectable malignant salivary tumours. Elective neck dissection is considered for high-grade tumours, large (T3/T4) tumours, or clinically/radiologically involved nodes, given the meaningful risk of occult nodal metastasis in these groups. **Adjuvant radiotherapy** is recommended for high-grade histology, close or positive margins, perineural or lymphovascular invasion, advanced T-stage, or nodal metastasis — and is particularly emphasised for **adenoid cystic carcinoma** given its propensity for perineural spread beyond clear margins. Systemic therapy has a limited but growing role, reserved chiefly for unresectable, recurrent or metastatic disease, informed increasingly by molecular profiling (e.g., androgen receptor and HER2 status in salivary duct carcinoma, and NTRK fusion status in secretory carcinoma) — reflecting current NCCN Head and Neck Cancer guidance.

13.4 Complications of Salivary Gland Surgery

- **Facial nerve palsy** — temporary (from traction/oedema) in up to 20–30% of parotidectomies, usually resolving over weeks to months; permanent injury is uncommon (<5%) in benign disease when the nerve is not directly involved by tumour.
- **Frey's syndrome (gustatory sweating)** — aberrant parasympathetic reinnervation of sweat glands following division of the auriculotemporal nerve's secretomotor fibres during parotidectomy, producing sweating and flushing over the cheek with eating; managed with topical antiperspirants or botulinum toxin injection in symptomatic cases, and its incidence is reduced by interposing a barrier (e.g., SMAS flap, acellular dermal matrix) at the time of primary surgery.
- **Sialocele and salivary fistula** — accumulation or leakage of saliva from residual gland tissue, usually managed conservatively with pressure dressings, aspiration and anticholinergic medication or botulinum toxin in persistent cases.
- **First-bite syndrome** — severe pain in the parotid region with the first bite of each meal, thought to reflect sympathetic denervation supersensitivity after deep-lobe or parapharyngeal space surgery; typically improves gradually over time.
- **Greater auricular nerve injury** — numbness of the ear lobe and lower auricle, common when the nerve is sacrificed for access, usually well tolerated by patients.
- **Cosmetic contour deformity** — from loss of parotid volume, increasingly mitigated by reconstruction with a local flap (e.g., SMAS or sternocleidomastoid flap) at the time of surgery.

14. Special Clinical Situations

Salivary Gland Tumours in Children

Salivary neoplasms are rare in childhood, but when they occur, mucoepidermoid carcinoma is disproportionately common relative to adults, and haemangioma (in infants) is the commonest parotid mass overall in this age group, typically involuting spontaneously and managed expectantly or with propranolol rather than surgery. Any solid, growing parotid mass in a child warrants the same diagnostic rigour (imaging, FNAC) as in an adult.

Recurrent Pleomorphic Adenoma

Recurrence after inadequate initial surgery (enucleation, or capsule violation with tumour spillage) is characteristically multifocal, technically challenging to clear given scarring and distorted anatomy, and carries a cumulative risk of malignant transformation with each further recurrence — reinforcing the importance of adequate margins at the index operation.

Salivary Gland Disease in Pregnancy

Physiological changes are minor, but pre-existing sialolithiasis or chronic sialadenitis may flare due to altered fluid balance and dietary changes; imaging (ultrasound is first-line and safe) and conservative management are preferred, with surgery deferred where possible to the second trimester or postpartum unless urgent drainage of infection is required.

15. Summary of Current Guidelines and Evidence

- **WHO Classification of Head and Neck Tumours, 5th edition (2022)** — current reference classification for salivary gland neoplasms, refining several entities and diagnostic criteria since the 4th edition.
- **Milan System for Reporting Salivary Gland Cytopathology, 2nd edition** — standardises FNAC reporting into six risk-stratified categories, guiding surgical decision-making.
- **NCCN Clinical Practice Guidelines in Oncology — Head and Neck Cancers** — current, regularly updated (version 2.2025 at time of writing) guidance on surgical margins, indications for neck dissection, and adjuvant radiotherapy/systemic therapy for malignant salivary tumours.
- **2016 ACR/EULAR Classification Criteria for Primary Sjögren's Syndrome** — the current validated, data-driven classification standard, incorporating objective sicca testing, serology and minor salivary gland biopsy.
- Contemporary narrative reviews and systematic reviews (2024–2025) continue to support sialendoscopy as first-line, gland-preserving management for uncomplicated sialolithiasis and juvenile recurrent parotitis, reserving gland excision for refractory cases.

Key Points for Revision

HIGH-YIELD SUMMARY

80% of salivary tumours are parotid, 80% of parotid tumours are benign, and 80% of benign parotid tumours are pleomorphic adenomas — but malignancy risk rises sharply as gland size falls (submandibular > sublingual > minor salivary glands).

80–85% of salivary calculi occur in the submandibular gland, owing to its viscous, alkaline secretion and long, uphill duct course.

Meal-time pain and swelling = obstruction (sialolithiasis/stricture); bilateral, diffuse, non-tender swelling suggests a systemic, infective or autoimmune cause; a discrete, unilateral mass should be treated as neoplastic until proven otherwise.

Facial nerve weakness with a parotid mass is malignancy until proven otherwise.

Sialendoscopy has become first-line, gland-preserving management for uncomplicated sialolithiasis and juvenile recurrent parotitis.

Ultrasound is the first-line imaging test for most salivary complaints; MRI best characterises a neoplasm and its deep extent; the Milan System standardises FNAC reporting and risk-stratifies management.

Preserve the facial nerve as the central technical objective of parotid surgery; know Frey's syndrome, sialocele, first-bite syndrome and facial nerve palsy as the key complications to counsel patients about pre-operatively.

Adenoid cystic carcinoma is defined by perineural spread and late distant recurrence — margin status and adjuvant radiotherapy are central to its management.

This review is intended as a structured revision resource for postgraduate trainees preparing for surgical, ENT-Head & Neck and pathology postgraduate examinations (e.g., FCPS, MS, MRCS, DOHNS and equivalent), and as a bedside/clinic reference. It should be read alongside primary guideline documents and institutional protocols, which take precedence in individual clinical decision-making.

Further Reading and Key Sources

The following primary sources informed this review and are recommended for deeper study:

1. Skálová A, Stenman G, Simpson RHW, et al. Update from the 5th Edition of the World Health Organization Classification of Head and Neck Tumors: Salivary Glands. *Head Neck Pathol.* 2022;16(1):40-53.

2. Rossi ED, Faquin WC, Baloch Z, et al. Second edition of the Milan System for Reporting Salivary Gland Cytopathology: refining the role of salivary gland FNA. *Cancer Cytopathol.* 2024.

3. Shiboski CH, Shiboski SC, Seror R, et al. 2016 American College of Rheumatology/European League Against Rheumatism classification criteria for primary Sjögren's syndrome. *Ann Rheum Dis.* 2017;76(1):9-16.

4. National Comprehensive Cancer Network (NCCN). Clinical Practice Guidelines in Oncology: Head and Neck Cancers, Version 2.2025.

5. Thirty years of experience and current trends in the management of sialolithiasis: a narrative review. 2025.

6. Hood BR, et al. Technique and efficacy of sialendoscopy in the management of recurrent sialadenitis. *Australian Journal of Otolaryngology.*

7. Williams N, O'Connell PR, McCaskie AW, eds. *Bailey & Love's Short Practice of Surgery*, 28th ed. Boca Raton: CRC Press.
