SALIVARY GLAND NEOPLASMS

VOLUME I

Foundations and the Diagnostic Approach

A first-principles, resident-level guide to localizing a salivary-region mass, estimating malignant risk, selecting imaging, interpreting tissue sampling, and preparing for definitive treatment.

THE CENTRAL QUESTION

Before naming a tumor, determine what the first operation—or the decision not to operate—must accomplish. Diagnosis is useful because it changes the map, the nerve plan, the neck plan, and the margin plan.

Prepared July 2026 • Educational use

How to Use This Volume

Salivary tumors are difficult for a specific reason: many lesions are rare, many share the same outward presentation, and cytology often provides a risk category rather than a complete name. The safest way to learn the subject is to preserve the clinical sequence. Start with location and behavior, use imaging to map anatomy, use tissue to narrow risk, and only then build the treatment plan.

SCOPE

This volume focuses on foundations and preoperative diagnosis. Benign entities are developed in Volume II, malignant histology and biology in Volume III, surgery in Volume IV, and adjuvant therapy, recurrence, surveillance, and integrated cases in Volume V.

Learning Objectives

- Localize a mass to the parotid, submandibular, sublingual, minor-gland, nodal, or non-salivary compartment.
- Explain why the fraction of malignant tumors rises as gland size decreases, while avoiding misuse of this rule in individual patients.
- Identify clinical findings that should trigger expedited MRI, skull-base evaluation, and oncologic planning.
- Select ultrasound, contrast CT, MRI, and FDG PET/CT according to the management question each modality can answer.
- Interpret FNA and core-biopsy results using the Milan System, including discordant or indeterminate results.
- Recognize the modern role of molecular classification and the key conceptual changes in AJCC Version 9 staging.
- Construct an efficient, defensible diagnostic plan for common clinical scenarios.

A Note on Evidence and Staging

The diagnostic recommendations in this guide are anchored to the ASCO salivary-gland malignancy guideline, the ESMO–EURACAN guideline, the second edition of the Milan System, the fifth edition WHO framework, and the AJCC Version 9 salivary-gland carcinoma staging framework effective in 2026. Salivary cancer evidence is often retrospective because individual histologies are uncommon; therefore, the guide distinguishes strong principles from institution-dependent details.

Contents

1. The Clinical Story and the “Triple Test”

A patient rarely presents with “a mucoepidermoid carcinoma.” The patient presents with a lump. The diagnostician’s first task is not to recite a differential; it is to convert an imprecise lump into a structured problem. Every useful data point should answer one of three questions: Where is it? How does it behave? What can tissue prove?

Figure 1. The diagnostic sequence used throughout this series. The sequence is deliberately management-centered.

The triple test

Domain	What it contributes	Common failure mode
Clinical examination	Tempo, cranial-nerve function, fixation, mucosal/skin origin, nodal disease	Calling a painless, mobile lesion “benign”
Imaging	Compartment of origin, deep extent, nerve pathways, bone/skin/muscle involvement, nodal map	Treating morphology as histology
Tissue sampling	Risk of malignancy, grade-related features, lineage, and sometimes a specific molecularly defined entity	Treating an indeterminate category as either reassuring or diagnostic

FIRST PRINCIPLE

The three domains are not votes. When they disagree, do not average them. A benign-appearing FNA does not cancel progressive facial weakness; an infiltrative MRI does not become harmless because the lesion has grown slowly. Discordance is a diagnostic problem that must be resolved.

What the first operation must accomplish

For a likely benign superficial parotid tumor, the key goals may be complete tumor removal without capsular violation and preservation of the facial nerve. For a suspected high-grade carcinoma, the first operation may need to account for total gland resection, possible nerve sacrifice and reconstruction, neck treatment, skin or mandible involvement, and reconstruction. The preoperative workup is therefore not merely descriptive—it protects the patient from an inadequately planned first operation.

OPERATING-ROOM RELEVANCE

A diagnostic pathway succeeds when consent, equipment, reconstructive options, and the team match the disease encountered. Intraoperative uncertainty is sometimes unavoidable; incomplete preoperative planning should not be the reason.

2. Anatomic Localization

Localization comes before pathology because the same histology behaves differently when it arises in the parotid, submandibular gland, palate, sinonasal tract, or parapharyngeal space. The gland of origin also changes the prior probability of malignancy and determines which nerves, vessels, mucosal surfaces, and skull-base corridors must be evaluated.

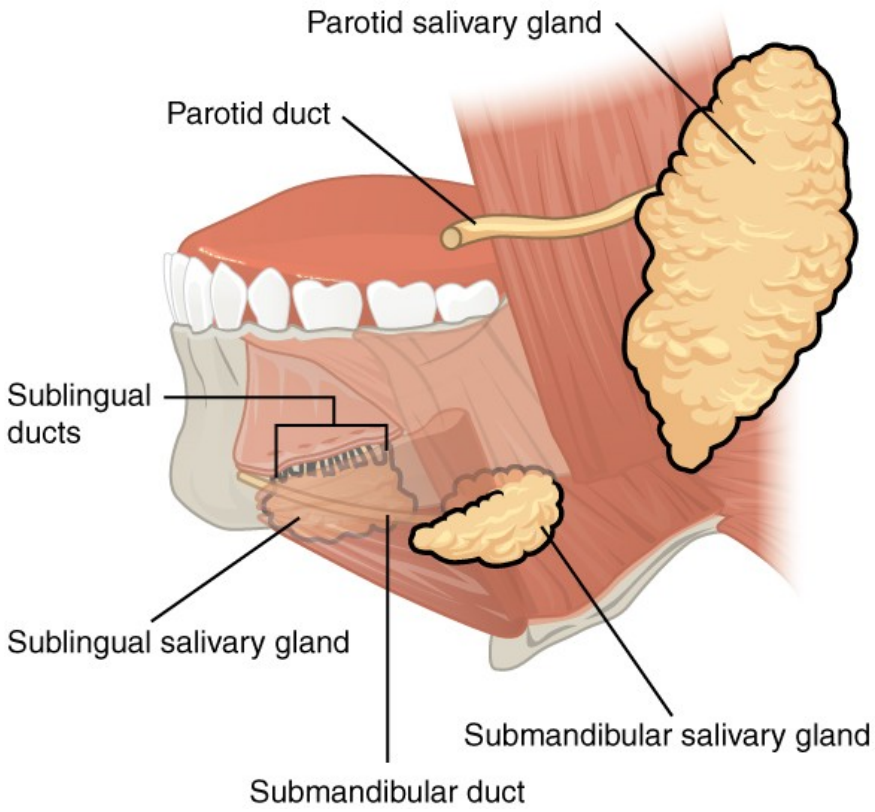


Figure 2. Major salivary glands and ducts. OpenStax College, "2408 Salivary Glands," CC BY 3.0, via Wikimedia Commons.

2.1 Parotid gland: a surgical partition built around CN VII

The parotid occupies the preauricular and retromandibular region, wrapping around the mandibular ramus. The facial nerve exits the stylomastoid foramen, enters the gland, and divides into a branching plexus. "Superficial" and "deep" lobes are surgical concepts defined by the nerve plane; they are not separated by a complete fascial septum. This matters because imaging can estimate—but cannot perfectly display—the course of the nerve through a distorted gland.

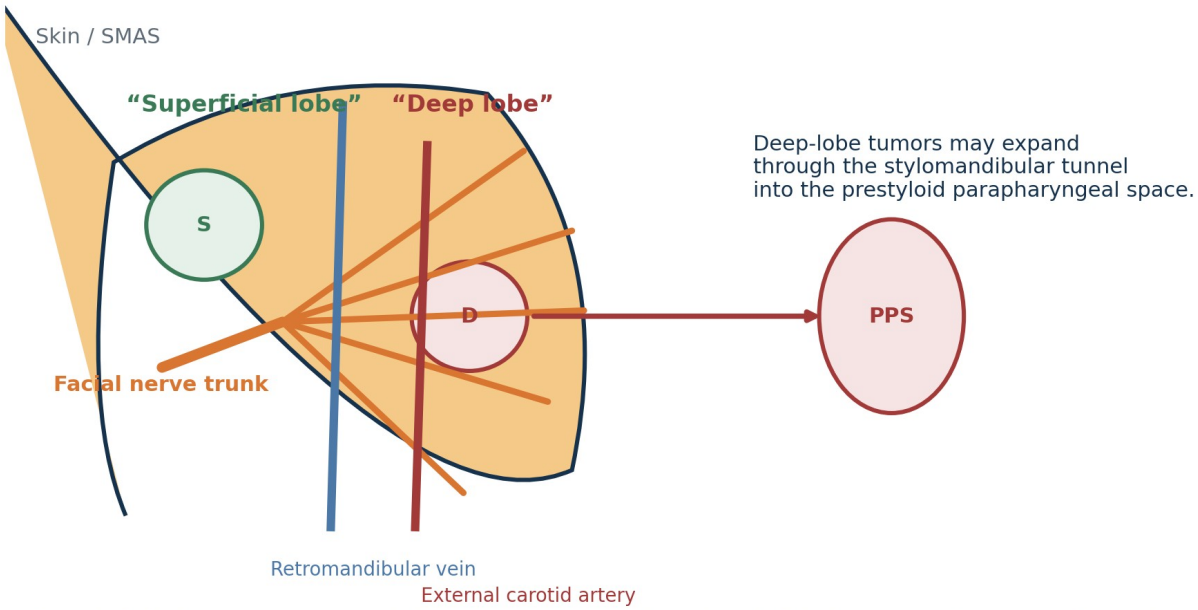


Figure 3. Simplified parotid organization. S = superficial-lobe lesion; D = deep-lobe lesion; PPS = prestyloid parapharyngeal space. Original schematic.

What to localize on imaging

- Superficial versus deep to the expected facial-nerve plane; the retromandibular vein is a useful but imperfect surrogate.
- Parotid tail versus level II lymph node. Remember that the parotid contains intraglandular lymph nodes.
- Relationship to skin, masseter, mandible, external auditory canal, mastoid, and sternocleidomastoid.
- Extension through the stylomandibular tunnel into the prestyloid parapharyngeal space.
- Facial-nerve and trigeminal pathways toward the stylomastoid foramen, facial canal, foramen ovale, foramen rotundum, and cavernous sinus.

BOARD TRAP

A parotid-region squamous cell carcinoma is more often metastatic cutaneous SCC in an intraparotid node than a primary salivary SCC. The skin examination—scalp, temple, auricle, and face—is part of the salivary mass examination.

2.2 Submandibular gland

The submandibular gland spans the posterior free edge of the mylohyoid, with a larger superficial component in the submandibular triangle and a smaller deep component in the floor of mouth. The marginal mandibular, lingual, and hypoglossal nerves are clinically and surgically relevant. A firm “submandibular mass” may instead be a level Ib node, chronic sialadenitis, a stone-associated gland, or a floor-of-mouth process.

Structure	Why it matters in neoplasia
Marginal mandibular nerve	Preoperative asymmetry may indicate tumor involvement or prior injury; postoperative weakness must not be mislabeled as facial-trunk dysfunction.
Lingual nerve	Deep extension and floor-of-mouth disease may produce numbness or tongue sensory symptoms.

Structure	Why it matters in neoplasia
Hypoglossal nerve	Weakness implies advanced local disease or a non-glandular skull-base/neck process.
Facial artery and vein	Define surgical relationships; vessel encasement or invasion alters the resection plan.
Level Ib nodes	Nodal disease may mimic a primary gland mass; metastatic oral cavity or cutaneous disease remains in the differential.

2.3 Sublingual and minor salivary glands

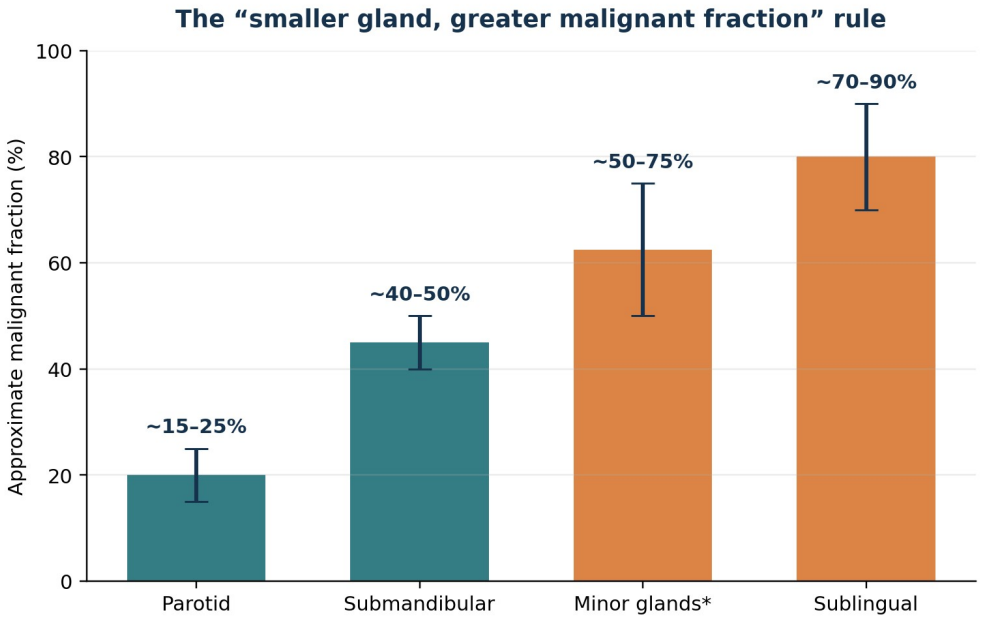
Sublingual tumors are uncommon but carry a high malignant fraction. The sublingual gland lies above the mylohyoid, lateral to Wharton duct and near the lingual nerve. Minor glands are distributed throughout the upper aerodigestive tract, especially the palate, lips, buccal mucosa, retromolar region, tongue base, sinonasal tract, and larynx. A “salivary” diagnosis in a minor-gland site must be integrated with site-specific staging and surgical anatomy.

CLINICAL PEARL

Palatal numbness, persistent deep pain, or a submucosal mass with intact mucosa should raise concern for perineural involvement even when the lesion is small and slow-growing.

3. Epidemiology and Pretest Probability

Most salivary neoplasms arise in the parotid, and most adult parotid tumors are benign. As tumors become less common by gland, the fraction that are malignant rises. This produces the classic teaching rule: the smaller the gland, the greater the probability that a neoplasm is malignant. The rule is useful as a prior—but it is not a diagnosis.



*Minor-gland risk varies substantially by anatomic site and referral population. Ranges are teaching approximations, not patient-specific probabilities.

Figure 4. Approximate malignant fractions by site. Values vary across series, subsites, age groups, and referral populations; use the direction of the relationship more confidently than any single percentage.

How to use the rule correctly

Correct use	Incorrect use
A submandibular neoplasm deserves a higher index of suspicion than an otherwise similar parotid neoplasm.	“Parotid means benign” or “sublingual means malignant.”
Site changes how aggressively discordant findings are pursued.	Replacing imaging and tissue sampling with an epidemiologic shortcut.
The rule helps set the threshold for oncologic planning.	Using broad population percentages as a patient-specific post-test probability.
Minor-gland risk is modified by anatomic subsite.	Treating all minor-gland sites as biologically equivalent.

Important modifiers of the prior

- Age: both benign and malignant salivary tumors occur across a wide range; pediatric masses require a broader developmental and vascular differential.
- Prior radiation: increases risk for subsequent salivary neoplasia.
- Cutaneous cancer history: raises the probability that a parotid mass represents nodal metastasis.
- Immunosuppression: increases concern for lymphoma and aggressive cutaneous malignancy.
- Smoking: strongly associated with Warthin tumor but does not make every tail lesion benign.
- Autoimmune disease or HIV: may create lymphoepithelial or lymphoid lesions that mimic neoplasia.

PATTERN, NOT PROOF

A slow-growing painless mass is the dominant presentation of many benign tumors—but also of low-grade malignancies, acinic cell carcinoma, secretory carcinoma, and adenoid cystic carcinoma. Clinical tempo changes risk; it rarely establishes histology.

4. History and Physical Examination

The examination should establish a baseline that can later be compared with postoperative function. The most important missed data point is often not the mass itself but an incompletely documented cranial-nerve examination, skin survey, or oral cavity examination.

4.1 History: questions that change the workup

Question	Interpretation
How long has it been present, and has the rate of growth changed?	Acceleration after years of stability raises concern for malignant transformation, hemorrhage, infection, or cystic change.
Pain, paresthesia, otalgia, trismus, or dental symptoms?	Persistent neuropathic pain or numbness raises concern for invasion/perineural spread; trismus suggests masticator-space involvement.
Facial twitching, weakness, dry eye, oral incompetence?	Any new CN VII dysfunction with a parotid mass is malignant until convincingly proven otherwise.
Skin cancers of scalp, temple, auricle, or face?	Raises concern for intraparotid nodal metastasis.
Prior salivary surgery or “lump removal”?	Recurrent pleomorphic adenoma can be multifocal; scarred planes complicate nerve preservation.
Radiation, autoimmune disease, HIV, immunosuppression?	Modifies the differential and sometimes the biopsy strategy.

4.2 Examination sequence

1. Inspect and palpate the mass: exact location, size, mobility, tenderness, skin tethering, and relationship to the mandible or mastoid.
2. Document facial function by division at rest and with movement. Note synkinesis or prior baseline asymmetry.
3. Perform a focused trigeminal examination, particularly V2 and V3 sensation when pain or numbness is reported.
4. Inspect the scalp, auricle, temple, face, and neck skin for current or prior cutaneous malignancy.
5. Inspect and palpate the oral cavity and oropharynx. Look for palatal bulge, floor-of-mouth extension, ductal abnormalities, mucosal ulceration, and tongue weakness.
6. Palpate the neck systematically, including level Ib, II, and parotid nodal basins; perform flexible endoscopy when symptoms or site warrant it.

4.3 Red flags

Finding	Why it matters	Immediate implication
Facial weakness	Strongly associated with invasive malignancy or perineural spread	Contrast MRI including skull base; tissue diagnosis; reconstructive counseling
Trigeminal numbness / neuropathic pain	Possible PNS along V2/V3 or local nerve invasion	High-resolution fat-suppressed postcontrast MRI
Fixation, skin ulceration, or rapid growth	Suggests extraglandular invasion or high-grade biology	Cross-sectional imaging and oncologic planning
Trismus	Masticator-space or deep-lobe extension	MRI; evaluate skull base and PPS
Cervical or intraparotid nodes	Regional metastasis, lymphoma, or metastatic skin cancer	Map nodal basins; sample the most informative target
Bilateral/multifocal masses	Warthin, lymphoepithelial disease, lymphoma, metastases	Broaden differential; avoid reflexively treating as two unrelated tumors

DO NOT DELAY

Facial paralysis plus a parotid mass should not be observed simply because the mass is small, painless, or previously labeled benign. Clinical perineural dysfunction can precede or exceed radiographic findings.

5. Imaging: Choose the Study That Changes Management

Imaging is best viewed as an anatomic problem-solving tool. It can establish the compartment of origin, reveal deep extension, map nerves and nodes, and identify bone or soft-tissue invasion. It cannot reliably name every salivary tumor, because benign and low-grade malignant lesions often overlap in appearance.

Clinical question	US	Contrast CT	MRI + contrast	FDG PET/CT
Superficial palpable mass	Excellent first look	Useful	Useful	Usually no
Need image-guided biopsy	Best platform	Occasional	Rare	No
Deep lobe / PPS origin	Limited	Good	Best	No
Bone invasion	Limited	Best	Complementary	No
Perineural / skull base	Poor	May suggest	Best	No
Advanced high-grade staging	Nodes only	Neck/chest anatomy	Local map	Selected cases

Modality choice should answer the next management question; no single study reliably assigns histology.

Figure 5. Practical modality selection. ASCO supports ultrasound, contrast CT, and/or MRI for suspected salivary cancer; modality is tailored to the anatomic question.

5.1 Ultrasound

Ultrasound is fast, radiation-free, and particularly effective for a palpable superficial parotid or submandibular lesion. It distinguishes solid from cystic components, assesses vascularity, surveys accessible nodes, and provides the best platform for real-time FNA or core biopsy. Its limitations are the skull base, deep parotid, retromandibular region, and lesions hidden by bone or air.

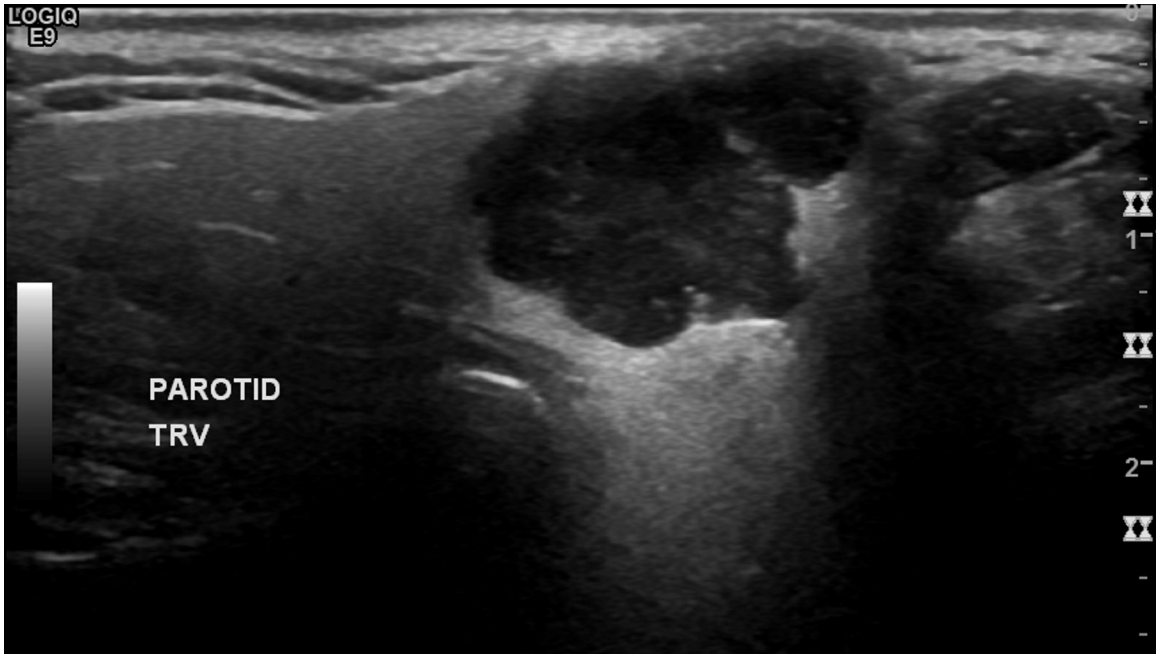


Figure 6. Transverse parotid ultrasound showing a lobulated hypoechoic pleomorphic adenoma. Schomynv, CC BY-SA 3.0, via Wikimedia Commons. Imaging appearance alone is not histologic proof.

ULTRASOUND PEARL

The most useful ultrasound question is often not “benign or malignant?” but “Is this intraparotid, nodal, cystic, vascular, and safely targetable?”

5.2 Contrast-enhanced CT

CT provides a rapid overview of the gland, neck, and airway; it is particularly useful when bone erosion, calcification, acute inflammation, or extensive cervical nodal disease is a concern. It is also practical when MRI is contraindicated or unavailable. Dental artifact and limited soft-tissue contrast can obscure the floor of mouth, deep lobe, and subtle perineural disease.

5.3 MRI

MRI is preferred when the lesion is deep, the site of origin is uncertain, or there is concern for perineural spread, skull-base involvement, parapharyngeal extension, marrow invasion, or complex soft-tissue relationships. A useful examination includes high-resolution pre- and postcontrast T1-weighted imaging with fat suppression, T2-weighted sequences, and diffusion-weighted imaging. The neck and skull base should be covered when perineural spread is suspected.

Feature	Raises concern for malignancy—but is not specific
Margins	Ill-defined, infiltrative, or trans-spatial extension
T2 signal	Low or intermediate signal in a solid component, especially with aggressive morphology
Diffusion	Restricted diffusion in a solid component; interpret with morphology and technique
Enhancement	Heterogeneous enhancement, necrosis, or infiltrative enhancement
Adjacent structures	Skin, muscle, mandible, external auditory canal, skull base, or vascular involvement
Nerves	Asymmetric thickening/enhancement, loss of perineural fat, foraminal widening, or denervation change
Nodes	Abnormal morphology, necrosis, clustering, or extranodal extension features

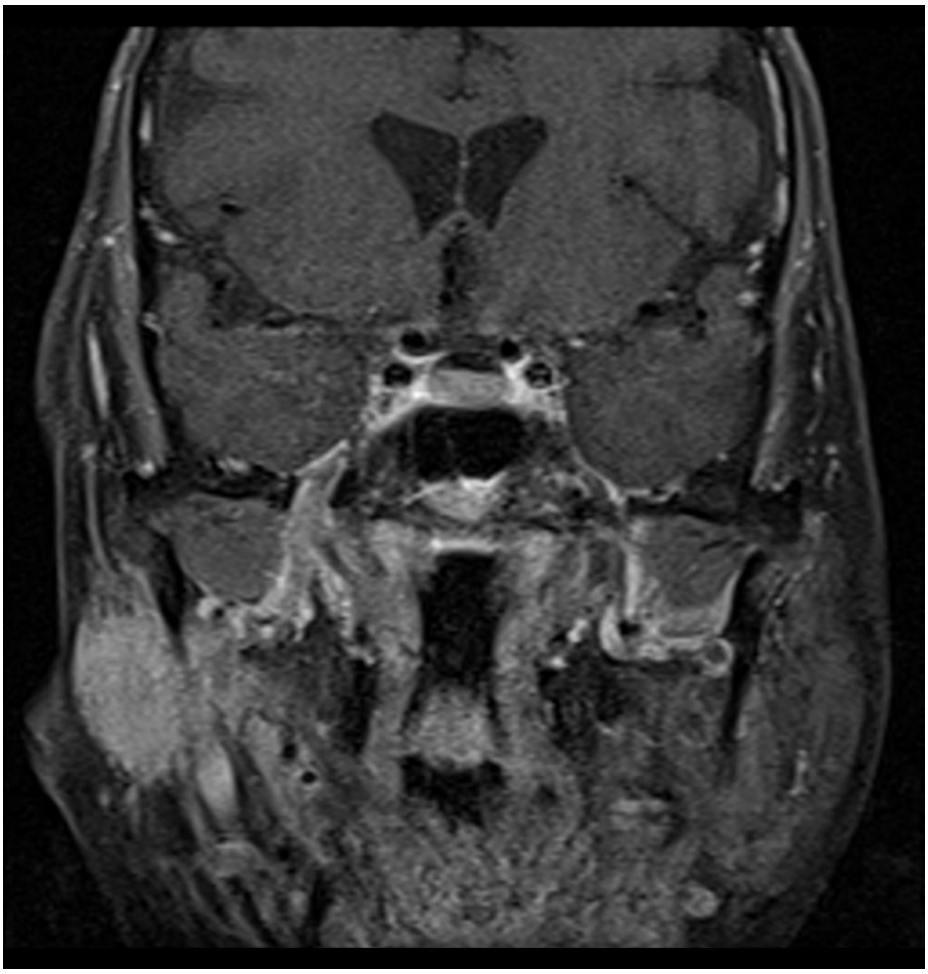


Figure 7. Coronal fat-suppressed postcontrast T1 MRI demonstrating a right parotid adenoid cystic carcinoma with perineural spread along a trigeminal pathway. Jto410, CC BY-SA 3.0, via Wikimedia Commons.

PERINEURAL SPREAD ≠ MICROSCOPIC PNI

Pathologic perineural invasion may be microscopic and invisible on imaging. Radiographic perineural spread is macroscopic disease along a named nerve pathway and changes surgical extent, skull-base assessment, and radiation volumes. A negative MRI does not override convincing cranial neuropathy.

5.4 FDG PET/CT

FDG PET/CT is not a routine discriminator of benign from malignant salivary lesions: Warthin tumors and some benign neoplasms can be intensely avid, while some low-grade malignancies are not. ASCO allows PET/CT in selected advanced-stage, high-grade cancers for regional and distant staging. Its greatest value is problem solving and whole-body staging—not assigning salivary histology.

5.5 A radiology request that answers the real question

SAMPLE MRI INDICATION

Progressive left parotid mass with new lower-division facial weakness and V3 numbness. Evaluate superficial/deep-lobe extent, facial nerve from stylomastoid foramen through intratemporal course, auriculotemporal/V3 pathway to foramen ovale and cavernous sinus, masticator space, skull base, and cervical nodes.

5.6 Imaging Pattern Gallery: Useful Clues, Not Histology

Imaging can shift probability and identify the anatomy that must be addressed, but it rarely supplies a definitive salivary histology. The most useful exercise is to ask which features are reassuring, which are concerning, and which merely overlap. Pleomorphic adenoma, Warthin tumor, low-grade carcinoma, lymphoma, and metastatic disease can each violate a simple "benign-looking versus malignant-looking" rule.

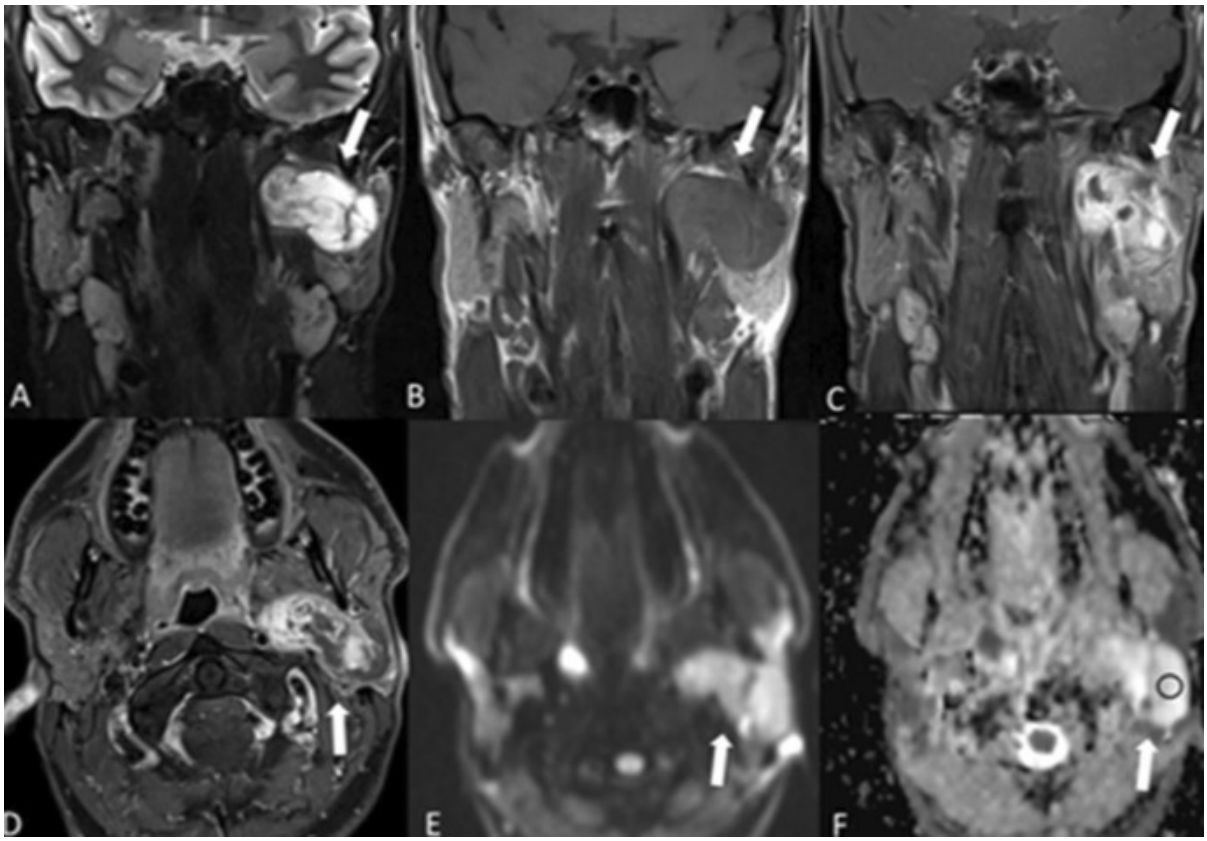


Figure 7A. Pleomorphic adenoma involving the superficial and deep parotid with an hourglass configuration. The lesion is very T2 hyperintense, enhances heterogeneously, and remains bright on the ADC map, reflecting relatively facilitated diffusion. Kim et al., BMC Medical Imaging 2022; open-access CC BY 4.0.

The image illustrates two practical points. First, the stylomandibular tunnel can permit a lesion to bridge the superficial and deep compartments without implying destructive invasion. Second, a high T2 signal and high ADC value favor pleomorphic adenoma, but neither finding is sufficiently specific to replace tissue sampling or to override discordant clinical findings.

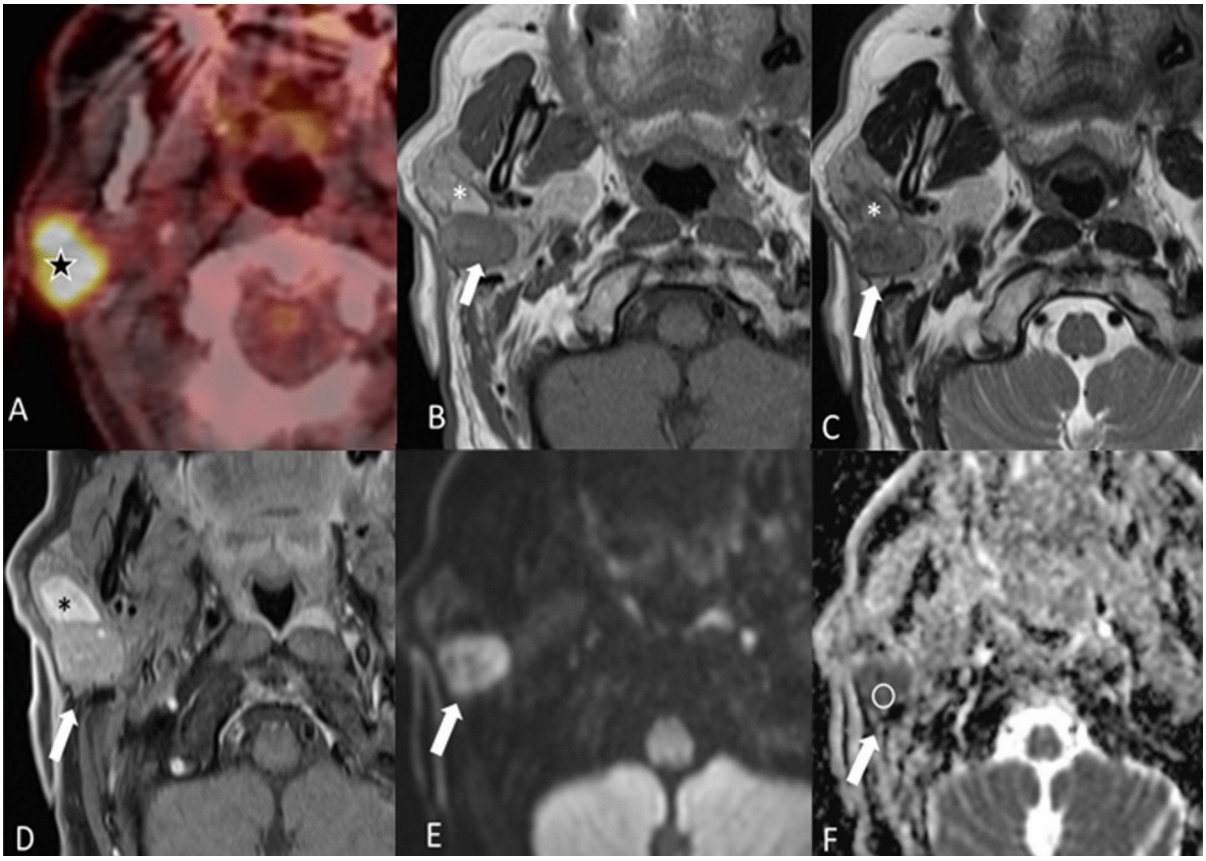


Figure 7B. Warthin tumor with intense FDG uptake and diffusion restriction. The panel demonstrates why PET avidity and a low ADC value cannot be interpreted as synonymous with malignancy. Kim et al., BMC Medical Imaging 2022; open-access CC BY 4.0.

Warthin tumor is a classic imaging trap. A well-circumscribed tail lesion in an older smoker may be strongly FDG avid, heterogeneous, partly cystic, and diffusion restricting. These features can be entirely compatible with benign disease, but the pattern should trigger targeted ultrasound and adequate tissue sampling rather than reflex observation—especially when the lesion is solitary, enlarging, or clinically discordant.

RESIDENT TAKEAWAY | Imaging is a map and a probability modifier. It is not a substitute for pathology, and pathology is not a substitute for the clinical map.

6. Tissue Diagnosis: FNA, Core Biopsy, and the Milan System

Preoperative tissue sampling should answer a management question. At minimum, it should distinguish non-neoplastic from neoplastic disease and estimate malignant risk. Ideally, it also indicates grade and lineage, permits ancillary testing, and identifies a tumor whose molecular alteration is diagnostically defining.

6.1 FNA versus core needle biopsy

Feature	Fine-needle aspiration	Core needle biopsy
Strength	Low morbidity; rapid; excellent for risk stratification; can sample multiple targets	Preserves architecture; more tissue for IHC/molecular studies; may improve subtyping

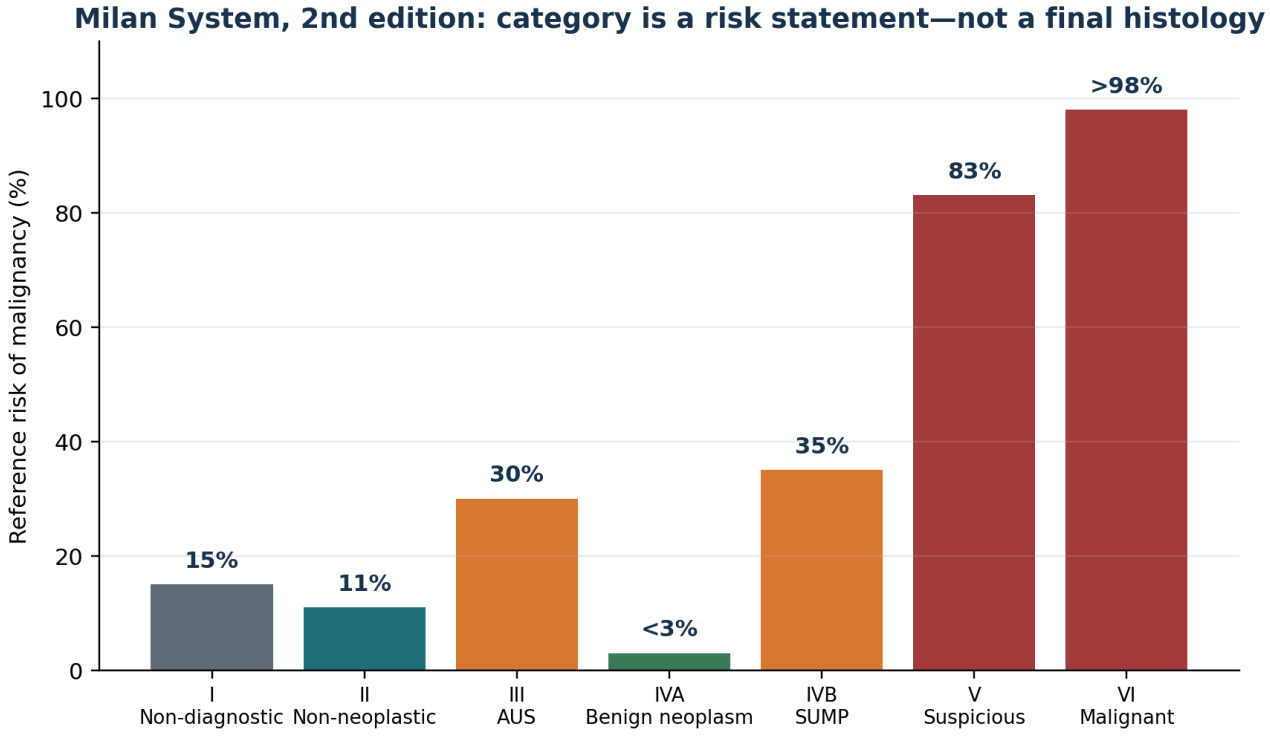
Feature	Fine-needle aspiration	Core needle biopsy
Weakness	Sampling error; limited architecture; low-grade tumors overlap; nondiagnostic cyst contents	More invasive; requires careful trajectory; hematoma/nerve concerns; pathology experience needed
Best use	Most accessible salivary masses and nodes; first-line in many centers	Inadequate/indeterminate FNA, suspected lymphoma, deep or diagnostically difficult lesion, need for architecture
Technique	Ultrasound guidance and on-site adequacy when available	Ultrasound guidance; plan path that does not compromise definitive surgery
Guideline position	ASCO supports FNA or core	ASCO specifically supports core when FNA is inadequate or the subsite limits FNA

AVOID OPEN BIOPSY OF A MAJOR-GLAND MASS

Open or incisional biopsy may violate a tumor, contaminate surgical planes, injure the facial nerve, and compromise the definitive operation. Exceptions are context-specific—for example, an unresectable lesion or a mucosal minor-gland tumor in which an incisional biopsy is the appropriate route.

6.2 The Milan System: cytology as calibrated risk

The second edition Milan System standardizes salivary cytology into seven risk categories. It acknowledges a central truth: salivary FNA often cannot assess invasion or the tumor–stromal interface, and many benign and low-grade malignant tumors share cytomorphology. The report should therefore communicate both category and risk—not force false precision.



AUS = atypia of undetermined significance; SUMP = salivary gland neoplasm of uncertain malignant potential.

Figure 8. Reference risks of malignancy in the Milan System, 2nd edition. Local institutional ROM may differ because of case mix and verification bias.

Milan category	Meaning	Typical next step
I. Non-diagnostic	Insufficient or nonrepresentative material	Repeat image-guided FNA with adequacy assessment; consider core if repeated failure
II. Non-neoplastic	Inflammatory, reactive, or other non-neoplastic process	Clinical–imaging correlation; treat cause or resample if discordant
III. AUS	Atypia exceeds benign change but is insufficient for neoplasm/malignancy category	Repeat FNA, core, ancillary testing, or excision based on clinical risk
IVA. Neoplasm—benign	Cytomorphology supports a specific benign neoplasm	Plan definitive management; do not ignore discordant nerve or imaging findings
IVB. SUMP	Neoplasm is certain, but benign versus malignant cannot be resolved	Usually surgical excision with operation planned for possible malignancy
V. Suspicious for malignancy	Features strongly favor malignancy but are not definitive	Oncologic imaging and operative planning; consider additional tissue if it changes treatment
VI. Malignant	Diagnostic of malignancy, with grade/lineage when possible	Complete staging and multidisciplinary treatment planning

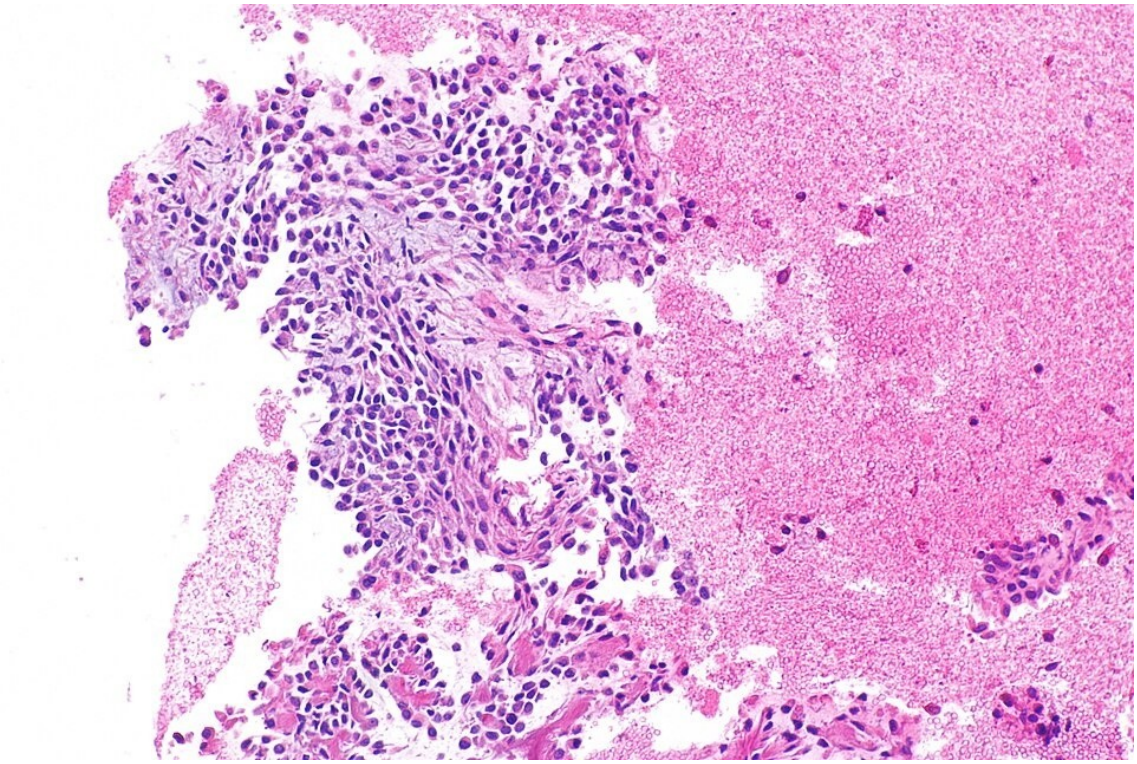


Figure 9. Cell-block preparation from FNA of a parotid lesion consistent with pleomorphic adenoma. Librepath, CC BY-SA 3.0, via Wikimedia Commons. The larger lesson is that cytology samples cells and matrix—not the entire invasive interface.

6.3 Why salivary cytology is intrinsically difficult

- Marked intratumoral heterogeneity means the needle may sample only one component.
- Low-grade malignancies may be bland and well circumscribed; cytologic atypia is not synonymous with malignant behavior.

- Basaloid, oncocytoid, clear-cell, and myoepithelial-rich tumors have broad differentials.
- Cystic lesions may yield macrophages and debris with few diagnostic cells.
- Invasion, capsular status, and the tumor–stroma interface usually cannot be assessed on FNA.
- Metastatic tumors and lymphoma may occur within intraparotid nodes and require a different ancillary workup.

THE TRIPLE-TEST RULE

A Milan IVA result is reassuring only when the clinical and imaging findings are concordant. Progressive facial weakness, fixation, or named-nerve enhancement converts an apparently benign cytology result into a likely sampling or classification problem.

6.4 Ancillary testing

Immunocytochemistry, flow cytometry, and targeted molecular testing are selected according to the differential. Flow cytometry is useful when lymphoma is suspected. Molecular alterations can confirm entities such as secretory carcinoma or help distinguish tumors with overlapping morphology. Testing should be hypothesis-driven: a broad panel on scant material may consume the sample without resolving the key question.

7. The Modern Pathology Framework

The fifth edition WHO classification moved salivary pathology further away from a purely pattern-based catalog and toward integrated diagnosis using morphology, immunophenotype, and recurrent molecular alterations. The practical resident-level implication is not that every patient needs sequencing. It is that some tumors are now best understood as molecularly defined families, and a vague morphology label may conceal a clinically meaningful diagnosis.

7.1 A first-principles cell model

Normal salivary units contain luminal ductal/acinar cells and abluminal myoepithelial or basal cells embedded in specialized stroma. Neoplasms may reproduce one or both compartments and may generate mucinous, oncocytic, clear-cell, basaloid, or myxochondroid patterns. This is why salivary pathology repeatedly returns to biphasic architecture and why a small cytology sample can be ambiguous.

Pattern family	Representative diagnostic problem	What resolves it
Mucinous / squamoid	Low-grade mucoepidermoid carcinoma vs cystic benign lesion	Adequate cellular sampling, mucous/intermediate cells, molecular support when needed
Basaloid	Adenoid cystic carcinoma vs basal cell adenoma/adenocarcinoma vs other basaloid neoplasm	Architecture, matrix, invasion, immunophenotype, molecular findings
Oncocytoid	Warthin tumor vs oncocytoma vs oncocytic carcinoma/metastasis	Lymphoid background, architecture, imaging distribution, ancillary studies
Clear cell	Hyalinizing clear cell carcinoma vs epithelial-myoepithelial tumor vs metastasis	Mucicarmine/IHC, EWSR1-related testing, clinical history

Pattern family	Representative diagnostic problem	What resolves it
Myoepithelial-rich	Pleomorphic adenoma vs myoepithelioma/carcinoma	Matrix, infiltrative growth, PLAG1/HMGA2 context, resection pathology

7.2 Grade is a biologic variable, not merely a descriptor

Tumor type and grade jointly predict nodal risk, distant metastatic pattern, and the need for elective neck treatment or postoperative radiation. A low-grade mucoepidermoid carcinoma and a high-grade salivary duct carcinoma are both "salivary carcinomas," but their diagnostic urgency, systemic targets, and expected spread are fundamentally different. When tissue allows, the pathology report should communicate high-grade features even if a precise subtype remains uncertain.

> **REPORTING PEARL**
>
> Ask the pathologist not only "What is it?" but also: Is it convincingly malignant? Is there a high-grade component? Is lymphoma or metastasis plausible? Would additional tissue or a molecular test change the operation?

8. Staging and Risk: AJCC Version 9

Staging answers a different question than histology. Histology describes what the tumor is; stage describes its anatomic burden. In 2026, AJCC Version 9 introduced a unified salivary-gland carcinoma framework applicable to major and minor salivary carcinomas, with simplified pathologic nodal groups driven by positive-node count and extranodal extension.

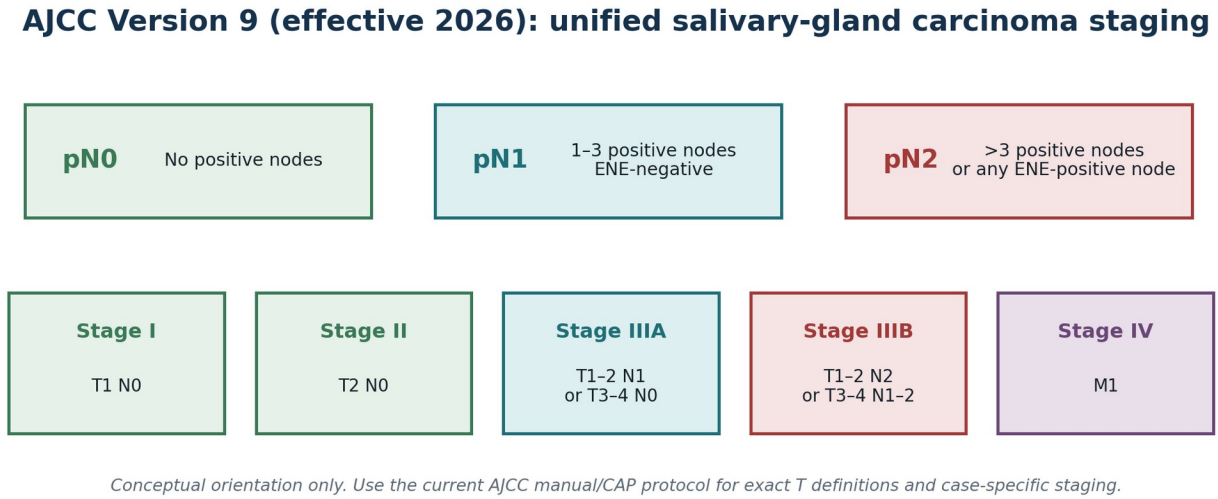


Figure 10. Conceptual AJCC Version 9 orientation based on the published development and validation study. Consult the current AJCC manual and CAP protocol for complete T definitions and final staging.

8.1 What changed conceptually

- A single salivary-gland carcinoma staging framework can be applied across major and minor salivary sites.
- Pathologic nodal risk is simplified to pN0, pN1 (1–3 positive nodes without ENE), and pN2 (>3 positive nodes or any ENE-positive node).

- Nonmetastatic advanced disease is divided into stages IIIA and IIIB; stage IV is reserved for distant metastatic disease.
- The framework aligns anatomic stage more closely with WHO histologic risk groups—but stage does not replace histologic type or grade.

8.2 Staging data to capture before treatment

Domain	Data needed
Primary tumor	Maximum dimension; gland/subsite; gross extraparenchymal or adjacent-structure invasion; skin, bone, nerve, skull-base, or mucosal involvement
Cranial nerves	Clinical dysfunction and radiographic extent along named pathways
Nodes	Laterality, levels, number suspected, size, intraparotid involvement, and ENE features; final pN requires pathology
Distant disease	Chest imaging and selected whole-body staging according to grade/stage and symptoms
Histologic risk	Subtype, grade, high-grade transformation, lymphovascular invasion, perineural invasion, margins

TRANSITION NOTE

Older notes, tumor boards, and studies may still use AJCC 8th-edition terminology. In 2026, explicitly state the staging version when communicating a case; do not assume a stage label means the same thing across editions.

9. Integrated Diagnostic Algorithms

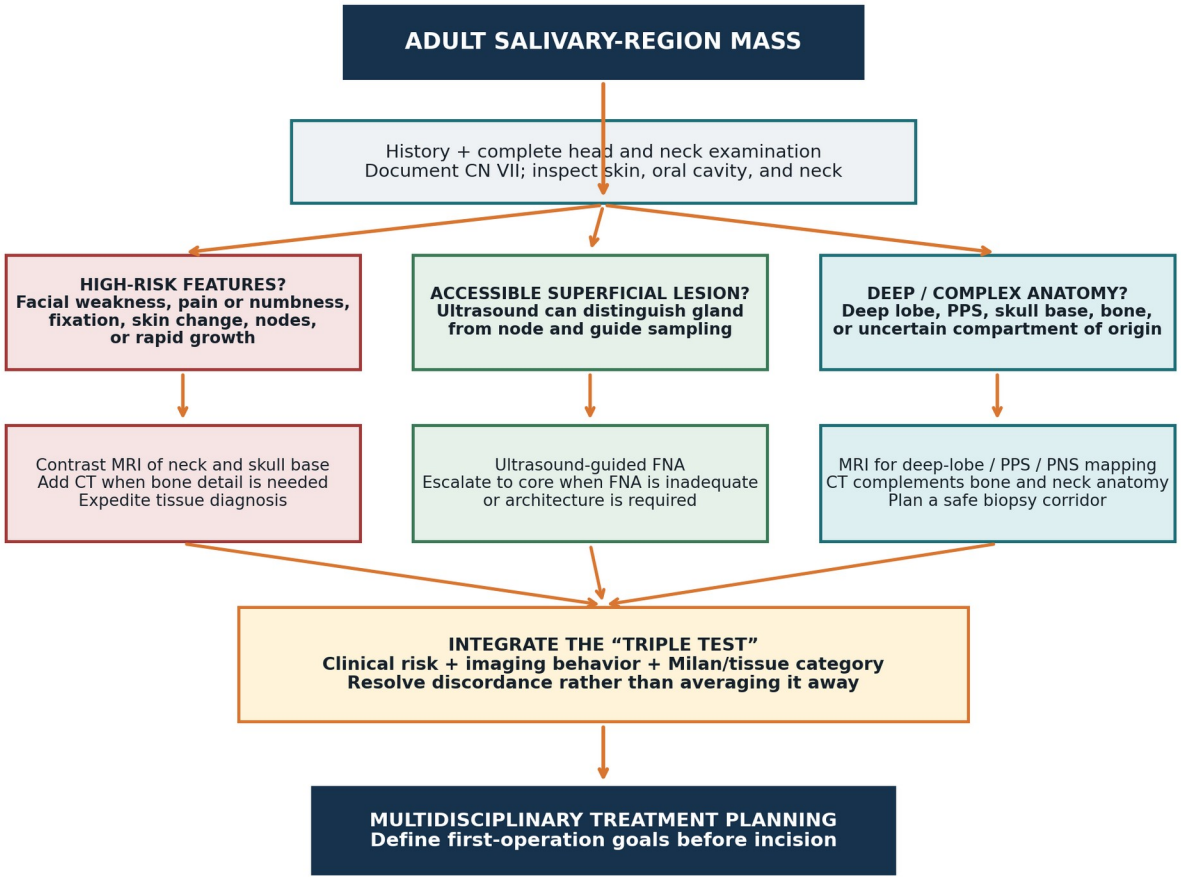


Figure 11. Integrated adult salivary-region mass algorithm. The key branch point is not "benign or malignant?" but whether high-risk features require immediate cross-sectional mapping and oncologic planning.

9.1 Adult superficial parotid mass

7. Document full facial function and examine skin and neck.
8. Use ultrasound to confirm intraparotid location, assess nodes, and guide FNA when the lesion is accessible.
9. Obtain MRI or CT when the lesion is large, deep, fixed, recurrent, clinically suspicious, or incompletely characterized by ultrasound.
10. Interpret the Milan category in the context of the examination and imaging.
11. Before surgery, decide whether the likely operation is limited benign-tumor surgery or an oncologic parotidectomy requiring neck and reconstructive planning.

9.2 Submandibular mass

12. Differentiate gland enlargement from a level Ib node and from obstructive/inflammatory disease.
13. Perform oral cavity examination and bimanual palpation of the floor of mouth; document lingual and hypoglossal function.
14. Use ultrasound for superficial characterization and guided biopsy; add contrast CT or MRI for deep extension, fixation, nerve symptoms, or malignancy concern.
15. Because the malignant fraction is higher than in the parotid, maintain a lower threshold for oncologic planning and neck evaluation.

9.3 Deep-lobe or parapharyngeal lesion

16. Use MRI to identify prestyloid versus poststyloid compartment and assess continuity with the deep parotid through the stylomandibular tunnel.
17. Assess displacement of parapharyngeal fat, carotid space, and masticator structures; evaluate skull-base foramina.
18. Choose a biopsy route that avoids neurovascular injury and does not contaminate a future surgical corridor; some lesions may be sampled transorally, transcervically, or by image-guided percutaneous approach depending on anatomy and expertise.
19. Plan the approach only after the compartment of origin and vascularity are established.

9.4 Facial weakness with a parotid mass

20. Treat as malignant/perineural disease until proven otherwise.
21. Document severity and division-specific function; examine V2/V3 and other cranial nerves.
22. Order contrast MRI including neck and skull base with high-resolution fat-suppressed sequences along CN VII and communicating trigeminal pathways.
23. Obtain tissue promptly; repeat or escalate sampling if cytology is benign or nondiagnostic and discordance persists.
24. Discuss facial nerve sacrifice, cable grafting or nerve transfer, eye protection, static suspension, neck treatment, and adjuvant therapy before definitive surgery when appropriate.

9.5 Cystic parotid-tail or upper-neck mass

The differential includes Warthin tumor, branchial anomaly, cystic nodal metastasis from oropharyngeal SCC, metastatic cutaneous SCC, lymphoma, and cystic salivary carcinoma. In an adult, a cystic neck mass is not assumed congenital. Sample the solid wall or mural nodule rather than aspirating only fluid; evaluate oropharynx, skin, and nodal architecture; and use HPV-related testing when the clinical setting supports it.

HIGH-YIELD DECISION

The best biopsy target is the lesion that will most efficiently distinguish the competing treatment pathways. A suspicious cervical node may be more informative and safer to sample than a difficult deep-lobe primary.

10. Clinical Vignettes and Board-Relevant Traps

Case 1 — The reassuring FNA that is not reassuring

A 58-year-old patient has a 2-cm parotid mass and progressive marginal mandibular weakness. Ultrasound-guided FNA is Milan IVA, “consistent with pleomorphic adenoma.”

REASONING

The clinical finding dominates. Progressive facial weakness is discordant with an uncomplicated pleomorphic adenoma. Obtain high-resolution contrast MRI of the parotid, neck, and skull base; review the cytology; and obtain repeat targeted sampling or core biopsy if it will change planning. Do not proceed as a routine benign superficial parotidectomy.

Case 2 — The small submandibular mass

A 45-year-old patient has a firm 1.5-cm mass apparently within the submandibular gland, normal tongue function, and no palpable nodes. FNA is Milan IVB SUMP.

REASONING

SUMP confirms a neoplasm but cannot resolve benign versus malignant. The site carries a higher malignant fraction than the parotid. Cross-sectional imaging should define local relationships and the neck. Definitive excision should be planned as a potentially malignant submandibular tumor, with the neck strategy determined by final/working histologic risk and institutional practice.

Case 3 — The intensely FDG-avid parotid tail lesion

An older smoker undergoes PET/CT for a lung nodule and has a highly avid, well-circumscribed parotid-tail lesion.

REASONING

Warthin tumor is a classic cause of intense FDG uptake and may be bilateral or multifocal. PET avidity alone does not establish malignancy. Confirm the lesion's sonographic features and obtain tissue when the result changes management, particularly in a patient with another suspected cancer where metastasis is possible.

Case 4 — Palatal mass and numbness

A patient has a 1.2-cm firm submucosal hard-palate mass with ipsilateral palatal numbness and no ulceration.

REASONING

Minor-gland malignancy and perineural disease are major concerns despite the small size and intact mucosa. MRI should map the greater palatine canal, pterygopalatine fossa, V2/foramen rotundum pathway, and skull base. Incisional biopsy of a mucosal minor-gland lesion may be appropriate when planned with the definitive resection in mind.

Board-relevant statements: true or false?

Statement	Answer	Why
A painless, mobile parotid mass is benign until proven otherwise.	False	Low-grade malignancies may be painless, mobile, and slow-growing.
MRI is preferred when perineural spread is suspected.	True	High-resolution fat-suppressed postcontrast imaging best maps named nerve pathways.
A Milan IVA result excludes malignancy.	False	It carries low—but not zero—risk and must be concordant with clinical/imaging findings.
Open biopsy is the preferred next step after nondiagnostic FNA.	False	Repeat image-guided FNA or core is generally preferred for major-gland masses.
Intense FDG uptake makes a parotid lesion malignant.	False	Warthin and other benign lesions may be strongly FDG-avid.

Statement	Answer	Why
Facial weakness can be ignored if MRI shows no perineural spread.	False	Clinical neuropathy may precede or exceed imaging findings.
A parotid SCC should prompt a careful skin examination.	True	Intraparotid nodal metastasis from cutaneous SCC is common relative to true primary salivary SCC.

11. One-Page Consolidation

THE FIVE-QUESTION FRAMEWORK

1) Where is it? 2) What is the clinical malignant risk? 3) What anatomic question must imaging answer? 4) What does tissue prove—and what can it not prove? 5) What must the first operation and multidisciplinary plan accomplish?

Situation	Best next diagnostic move
Superficial palpable parotid mass, normal CN VII	Ultrasound + image-guided FNA; cross-sectional imaging as size/depth/risk dictates
Facial weakness, numbness, trismus, or fixation	Contrast MRI of neck/skull base; prompt tissue diagnosis; plan for oncologic/reconstructive needs
Possible bone invasion	Contrast CT complements MRI
Deep-lobe/PPS lesion	MRI for compartment of origin and skull-base/neurovascular relationships
Nondiagnostic FNA	Repeat image-guided FNA with adequacy or proceed to core based on context
Milan SUMP	Treat as proven neoplasm with unresolved malignant status; surgery usually planned accordingly
Cystic adult upper-neck/parotid-tail mass	Target solid component; assess oropharynx, skin, and nodal metastasis
Advanced high-grade malignancy	Complete regional/distant staging; PET/CT may be selected; multidisciplinary planning

Do not miss

- Document facial function before biopsy or surgery.
- Examine the skin and oral cavity—not only the gland.
- Distinguish a parotid mass from an intraparotid node.
- Use MRI when a named nerve or skull-base pathway is clinically implicated.
- Resolve clinical–radiologic–cytologic discordance.
- Do not let an open biopsy compromise the definitive major-gland operation.
- State the AJCC edition when communicating stage during the 2026 transition.

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