

# ORAL CAVITY CANCER

A Clinical and Surgical Review for Postgraduate Students

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Applied Anatomy & Staging • Potentially Malignant Disorders  
Diagnostic Workup • Surgical Management & Reconstruction  
Management of the Neck • Adjuvant Therapy  
Site-Specific Considerations • Current Guidelines

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

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## Learning Objectives

- Describe the applied surgical anatomy and lymphatic drainage of the oral cavity subsites relevant to cancer spread.
- List the risk factors and potentially malignant disorders of the oral cavity, with particular reference to tobacco and areca nut use in South Asia.
- Apply the AJCC 8th edition TNM staging system for oral cavity cancer, including depth of invasion (DOI).
- Outline a structured diagnostic workup for a patient with a suspicious oral cavity lesion.
- Describe the principles of surgical resection, neck management and reconstruction in oral cavity cancer.
- Identify indications for adjuvant radiotherapy and chemoradiotherapy.
- Recognise site-specific patterns of disease and their surgical implications.
- Outline current guideline-based approaches to prevention, screening, surveillance and follow-up.

## 1. Introduction and Epidemiology

Oral cavity cancer is a squamous cell malignancy arising from the mucosal lining of the lips, buccal mucosa, gingiva, hard palate, anterior two-thirds of the tongue, floor of the mouth and retromolar trigone. It is one of the most common head and neck malignancies worldwide and carries a disproportionately high burden in South and Southeast Asia, where it ranks among the leading cancers in men and, in many series, in women as well. This regional excess is driven largely by the widespread use of smokeless tobacco and areca nut (betel quid, gutka, naswar and paan) — a pattern distinct from the HPV-driven oropharyngeal cancers now rising in Western populations.

Globally, oral cavity cancer accounts for a substantial share of all head and neck cancers, with marked geographic variation: incidence is highest in India, Pakistan, Bangladesh, Sri Lanka and parts of Southeast Asia, intermediate in parts of Europe and South America (associated with tobacco and alcohol), and comparatively lower in North America. Late presentation is common in resource-limited settings, with a majority of patients presenting with locally advanced (stage III/IV) disease at diagnosis — underscoring the importance of early recognition of potentially malignant disorders and opportunistic screening.

### **Key Epidemiological Points**

- Male predominance, though the gap is narrowing in regions with high female smokeless-tobacco use.
- Peak incidence in the fifth to seventh decades, but younger patients (<40 years) are increasingly seen, often without classical risk factors.
- Buccal mucosa and gingivobuccal sulcus cancers are disproportionately common in South Asia (areca nut chewing habit), whereas tongue and floor-of-mouth cancers predominate in Western series (tobacco and alcohol).
- Oral cavity cancer is largely preventable — tobacco and areca nut control remain the single most effective public health interventions.

## 2. Applied Surgical Anatomy of the Oral Cavity

The oral cavity extends from the vermilion border of the lips anteriorly to the junction of the hard and soft palate superiorly and the circumvallate papillae of the tongue posteriorly — the boundary that separates it from the oropharynx. Precise knowledge of its subsites is essential, since tumour behaviour, patterns of spread and reconstructive options differ substantially between them.

| Subsite                        | Anatomical extent / notes                                                                                  |
|--------------------------------|------------------------------------------------------------------------------------------------------------|
| Lip (vermilion)                | Junction of skin and mucosa to the wet line; excludes skin and oral commissure.                            |
| Buccal mucosa                  | Inner lining of cheeks and lips from the upper to lower gingivobuccal sulcus, retromolar area.             |
| Upper & lower alveolus/gingiva | Mucosa overlying the alveolar processes; prone to early bone invasion.                                     |
| Hard palate                    | Semilunar area between the upper alveolar ridge and the mucous membrane covering the palatine bone.        |
| Anterior two-thirds of tongue  | Tip, lateral borders, dorsum and undersurface anterior to the circumvallate papillae.                      |
| Floor of mouth                 | Horseshoe-shaped area between the lower alveolar ridge and the ventral tongue, containing Wharton's ducts. |
| Retromolar trigone             | Small triangular area behind the last lower molar; early bone/pterygoid invasion is common.                |

### Lymphatic Drainage

Lymphatic drainage broadly follows predictable pathways that guide the choice of elective neck dissection levels: anterior tongue and floor of mouth drain principally to levels I–III (submental, submandibular, upper and mid jugular), with tongue lesions notable for occasional skip metastases directly to level IV. Buccal mucosa and gingival lesions drain to level I and II nodes, and lip cancer drains first to the submental and submandibular (level I) nodes, with lower lip lesions also at risk of bilateral spread near the midline.

#### Neck Levels — Quick Reference

**Level I** — submental (Ia) and submandibular (Ib) nodes.

**Level II** — upper jugular nodes, from skull base to hyoid bone (IIa anterior/medial, IIb posterior to the accessory nerve).

**Level III** — mid jugular nodes, hyoid to cricoid cartilage.

**Level IV** — lower jugular nodes, cricoid to clavicle.

**Level V** — posterior triangle nodes.

**Level VI** — central compartment (pretracheal, paratracheal, prelaryngeal) — not routinely relevant in oral cavity disease.

### 3. Aetiology and Risk Factors

Oral cavity squamous cell carcinoma is a multifactorial disease resulting from chronic mucosal exposure to carcinogens, chronic irritation and, in a minority, genetic predisposition. Unlike oropharyngeal cancer, HPV plays only a minor aetiological role in true oral cavity primaries.

#### ***Established Risk Factors***

- **Tobacco** — smoked (cigarettes, bidis, hookah) and smokeless (naswar, gutka, khaini) forms; risk is dose- and duration-dependent and synergistic with alcohol.
- **Areca (betel) nut** — chewed alone or as part of betel quid/paan/gutka; a major independent carcinogen and the principal cause of oral submucous fibrosis in South Asia.
- **Alcohol** — synergises with tobacco to multiply risk severalfold beyond either agent alone.
- **Chronic mechanical irritation** — ill-fitting dentures, sharp/broken teeth, and habitual cheek or tongue biting have been linked to lesions at the site of trauma.
- **Betel quid without tobacco** — still carcinogenic due to areca nut alone; commonly under-recognised by patients as a risk.
- **Immunosuppression** — solid organ transplant recipients and other chronically immunosuppressed patients carry increased risk.
- **Poor oral hygiene and periodontal disease** — contributory rather than primary factors.
- **HPV** — a minor contributor to true oral cavity SCC, in contrast to its dominant role in oropharyngeal cancer.
- **Genetic syndromes** — Fanconi anaemia and dyskeratosis congenita confer markedly elevated lifetime risk, often at a young age.

## 4. Potentially Malignant Disorders

A range of clinically recognisable oral mucosal conditions carry an increased risk of progression to invasive carcinoma and should prompt biopsy, risk-factor cessation counselling and structured follow-up.

| Disorder                            | Features and approximate malignant transformation risk                                                                                                                                                                                                        |
|-------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Leukoplakia                         | White patch that cannot be rubbed off or attributed to another diagnosis; overall transformation risk approx. 1–5%, higher for non-homogeneous/verrucous or proliferative forms and lesions on the floor of mouth/ventral tongue.                             |
| Erythroplakia                       | Red, velvety patch; far less common than leukoplakia but carries a substantially higher risk of severe dysplasia or carcinoma at biopsy (often >50%).                                                                                                         |
| Oral submucous fibrosis (OSMF)      | Progressive fibrosis of the submucosa causing trismus and a burning sensation, strongly linked to areca nut chewing; malignant transformation reported in a meaningful minority of long-standing cases and considered an irreversible premalignant condition. |
| Oral lichen planus                  | Chronic inflammatory condition with reticular/erosive patterns; a small but real transformation risk, higher in erosive/atrophic subtypes.                                                                                                                    |
| Proliferative verrucous leukoplakia | Multifocal, persistent, treatment-resistant leukoplakia with a very high (often >50–70%) lifetime transformation risk.                                                                                                                                        |

Any non-healing ulcer, indurated plaque, or persistent white/red lesion present for more than two to three weeks — particularly in a patient with tobacco or areca nut exposure — should be biopsied rather than observed empirically.

## 5. Pathology

More than 90% of oral cavity malignancies are squamous cell carcinomas (SCC), arising through a stepwise progression from hyperplasia through increasing grades of dysplasia to carcinoma in situ and invasive carcinoma — the concept of **field cancerisation**, in which the entire mucosal field exposed to carcinogens carries an elevated risk of synchronous and metachronous second primary tumours.

### *Histological Variants*

- **Conventional SCC** — the great majority; graded well, moderately, or poorly differentiated.
- **Verrucous carcinoma** — an exophytic, warty, well-differentiated variant with a low metastatic potential but a tendency to local recurrence if inadequately excised.
- **Basaloid SCC** — an aggressive variant with early nodal spread.
- **Spindle cell (sarcomatoid) SCC** — rare, biphasic, behaves aggressively.
- **Adenosquamous carcinoma** — mixed glandular and squamous differentiation; more aggressive than conventional SCC.

Histopathological reporting should record tumour grade, depth of invasion, pattern of invasion (cohesive vs. non-cohesive/worst pattern of invasion), lymphovascular and perineural invasion, margin status, and — where a neck dissection has been performed — the number of nodes involved and presence or absence of extranodal extension (ENE), all of which carry independent prognostic significance and directly inform adjuvant treatment decisions.

## 6. Clinical Features

Presentation depends on subsite but shares common warning features. Early lesions may be asymptomatic or produce only mild discomfort, which contributes to delayed presentation, particularly in areas where potentially malignant lesions are common and normalised (e.g., areca nut-stained mucosa).

### Red-Flag Features Warranting Urgent Referral and Biopsy

A non-healing oral ulcer persisting beyond three weeks.  
 An indurated (hard) mass or ulcer, especially with rolled or everted edges.  
 Unexplained loosening of teeth, or a non-healing tooth socket after extraction.  
 Progressive trismus (restricted mouth opening).  
 Referred otalgia (ear pain) in a patient with a tongue or oropharyngeal lesion — mediated via the lingual and glossopharyngeal nerves.  
 A persistent neck lump, particularly if firm, non-tender and enlarging.  
 Dysphagia, odynophagia, or unexplained weight loss.  
 Numbness of the lip or chin (mental nerve involvement) suggesting mandibular canal invasion.

### Site-Specific Presentations

- **Tongue** — ulcer or mass on the lateral border, often painful; may cause dysarthria or dysphagia with advanced disease; deeply infiltrative tumours may be more extensive than visible mucosal disease suggests.
- **Floor of mouth** — ulcer or indurated plaque, often close to the midline; early invasion of the sublingual space and genioglossus is common.
- **Buccal mucosa** — ulceroproliferative lesion, frequently at the site of quid placement, with a tendency for early involvement of the buccinator, skin and mandible in advanced disease.
- **Gingivobuccal sulcus** — a common South Asian site associated with quid-chewing habits, with early periosteal and bony invasion.
- **Hard palate** — often minor salivary gland in origin rather than SCC when arising centrally; SCC here should raise suspicion of extension from an antral or gingival primary.
- **Lip** — usually presents as a crusted, non-healing lesion on the lower lip vermilion; generally the best-prognosis oral cavity subsite owing to early presentation and a rich lymphatic-sparing course.

## 7. Diagnostic Workup

### **Clinical Assessment**

A complete head and neck examination is mandatory, including bimanual palpation of the tongue and floor of mouth (to assess depth and fixation, which cannot be reliably judged by inspection alone), assessment of trismus, dentition, and a systematic examination of all cervical lymph node levels. Cranial nerve examination (particularly the lingual, hypoglossal, inferior alveolar and mental nerves) should be documented, as should mouth opening in centimetres.

### **Biopsy**

An incisional or punch biopsy of the most representative area (avoiding necrotic centre and including adjacent abnormal-appearing mucosa) is the diagnostic gold standard. Fine needle aspiration cytology (FNAC) is used for a clinically suspicious neck node when the primary is already biopsy-confirmed, or to investigate a neck mass of unknown primary.

### **Imaging**

- **MRI** — the modality of choice for assessing soft-tissue extent, depth of invasion, perineural spread and marrow invasion of the mandible; superior soft-tissue contrast to CT.
- **Contrast-enhanced CT** — preferred for assessing cortical bone erosion and is often more accessible; useful for neck nodal staging and surgical planning.
- **Orthopantomogram (OPG)/dental imaging** — assesses dentition and gross mandibular involvement; a routine adjunct before treatment planning.
- **PET-CT** — useful for detecting occult regional or distant metastasis in advanced disease, for unknown primary work-up, and for post-treatment surveillance in selected cases.
- **Chest imaging** (CT chest or, at minimum, chest X-ray) — to exclude pulmonary metastasis or a synchronous second primary, particularly relevant given the shared risk-factor profile with lung cancer.
- **Examination under anaesthesia (EUA) with panendoscopy** — considered when a synchronous second primary in the aerodigestive tract is suspected, or when full assessment is precluded by trismus/pain in clinic.

## 8. Staging (AJCC 8th Edition)

The 8th edition of the AJCC/UICC TNM classification introduced **depth of invasion (DOI)** as a T-staging parameter for oral cavity cancer, in recognition of its strong independent association with occult nodal metastasis and outcome — a change from the 7th edition, which staged tumours by surface diameter alone. DOI is measured from the level of the basement membrane of adjacent normal mucosa vertically down to the deepest point of invasion, and is distinct from tumour thickness.

| T stage | Definition (AJCC 8th edition)                                                                                                                                                                                               |
|---------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Tis     | Carcinoma in situ.                                                                                                                                                                                                          |
| T1      | Tumour ≤2 cm, depth of invasion (DOI) ≤5 mm.                                                                                                                                                                                |
| T2      | Tumour ≤2 cm with DOI >5 mm and ≤10 mm; OR tumour >2 cm but ≤4 cm with DOI ≤10 mm.                                                                                                                                          |
| T3      | Tumour >4 cm, OR any tumour with DOI >10 mm.                                                                                                                                                                                |
| T4a     | Moderately advanced local disease: invades through cortical bone of mandible/maxilla, maxillary sinus, or skin of the face; (lip tumours: invades through cortical bone, inferior alveolar nerve, floor of mouth, or skin). |
| T4b     | Very advanced local disease: invades masticator space, pterygoid plates, skull base, or encases the internal carotid artery.                                                                                                |

### N Staging (Incorporating Extranodal Extension)

The 8th edition also incorporates clinically and radiologically detected extranodal extension (ENE) into N staging — its presence upstages disease independent of nodal size or number.

| N stage | Definition                                                                                             |
|---------|--------------------------------------------------------------------------------------------------------|
| N1      | Single ipsilateral node ≤3 cm, ENE negative.                                                           |
| N2a     | Single ipsilateral node >3 cm but ≤6 cm, ENE negative; OR single ipsilateral node ≤3 cm, ENE positive. |
| N2b     | Multiple ipsilateral nodes, none >6 cm, ENE negative.                                                  |
| N2c     | Bilateral or contralateral nodes, none >6 cm, ENE negative.                                            |
| N3a     | Node >6 cm, ENE negative.                                                                              |
| N3b     | Any node with clinically overt ENE.                                                                    |

### Stage Grouping (Summary)

| Stage | T / N / M                     |
|-------|-------------------------------|
| I     | T1 N0 M0                      |
| II    | T2 N0 M0                      |
| III   | T3 N0 M0, or T1–T3 N1 M0      |
| IVA   | T4a any N M0, or T1–T4a N2 M0 |
| IVB   | Any T N3 M0, or T4b any N M0  |
| IVC   | Any T any N M1                |

## 9. Principles of Surgical Management

Surgery remains the primary modality for resectable oral cavity cancer, offering the best chance of cure and the advantage of complete pathological staging to guide adjuvant treatment. The central surgical principle is complete tumour excision with adequate three-dimensional margins, balanced against preservation of function (speech, swallowing, mastication) wherever oncologically safe.

### Margins

A clear margin is generally defined as  $\geq 5$  mm of normal tissue on histopathology; a close margin as 1–5 mm; and a positive margin as tumour present at the inked resection edge (0 mm) — the convention now favoured by most contemporary series and guidelines over the older  $< 5$  mm definition of "positive". Intraoperative frozen section of the resection bed is used liberally to confirm adequate clearance and permit immediate re-resection.

### Resection by Subsite

- **Glossectomy** — partial (wedge/hemiglossectomy) for smaller lesions; subtotal or total glossectomy for extensive disease, with corresponding impact on speech and swallowing that must be discussed with the patient pre-operatively.
- **Mandibulectomy** — *marginal (rim)* mandibulectomy preserves mandibular continuity when there is mucosal/periosteal involvement without deep bone invasion and sufficient mandibular height remains; *segmental* mandibulectomy (with reconstruction) is required when there is gross cortical or medullary invasion, a previously irradiated mandible, or an edentulous atrophic mandible.
- **Maxillectomy** — for hard palate/upper alveolar tumours with bone involvement; ranges from infrastructure maxillectomy to more extensive resections depending on the extent of antral, orbital or skull-base involvement.
- **Composite resection** — combined resection of the primary tumour, involved mandible/maxilla and the regional lymphatics in continuity, historically termed "commando" resection, still relevant for locally advanced disease with bone and nodal involvement.
- **Buccal mucosa/gingivobuccal sulcus** — wide local excision with cheek reconstruction; through-and-through defects involving skin require composite flap reconstruction.

## 10. Management of the Neck

Because occult (clinically undetectable) nodal metastasis is common even in early oral cavity cancer, a management decision for the clinically node-negative (cN0) neck is required in almost every case, in addition to therapeutic dissection for clinically or radiologically node-positive disease.

### *Elective Neck Dissection in the cN0 Neck*

Depth of invasion is the key determinant. Landmark randomised evidence (including the POI/D'Cruz trial) demonstrated a significant survival benefit for elective neck dissection over a watch-and-wait ("observe and salvage") policy in early (T1–T2) cN0 oral cancer. Current practice, reflected in NCCN and other major guidelines, recommends elective neck dissection (typically selective dissection of levels I–III, extending to level IV for tongue primaries) when DOI exceeds approximately 4 mm; below this threshold, the risk of occult metastasis is low enough that observation with imaging surveillance is a reasonable alternative in a compliant patient.

### *Sentinel Lymph Node Biopsy*

Sentinel node biopsy is an accepted, less morbid alternative to elective neck dissection in carefully selected early (T1–T2) cN0 oral cavity cancers at experienced centres, sparing the majority of node-negative patients a full neck dissection while maintaining comparable regional control when performed to protocol.

### *Neck Dissection Classification*

| Type                             | Description                                                                                                                                                          |
|----------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Radical neck dissection          | Removal of levels I–V plus the internal jugular vein, sternocleidomastoid muscle and spinal accessory nerve.                                                         |
| Modified radical neck dissection | Levels I–V with preservation of one or more of the internal jugular vein, sternocleidomastoid, and accessory nerve.                                                  |
| Selective neck dissection        | Preservation of one or more nodal levels, tailored to the primary's known drainage pattern (e.g., levels I–III for anterior oral cavity, extended to IV for tongue). |
| Extended neck dissection         | Radical dissection plus removal of additional nodal groups or non-lymphatic structures (e.g., carotid artery, hypoglossal nerve) as required by disease extent.      |

## 11. Reconstruction

Reconstructive choice follows the principle of the "reconstructive ladder/elevator" — matching the simplest technique capable of restoring form and function to the size and composition of the defect, while accounting for planned adjuvant radiotherapy, patient comorbidity and donor site morbidity.

- **Primary closure/healing by secondary intention** — suitable only for small, superficial defects.
- **Local flaps** (e.g., tongue flap, buccal fat pad flap, nasolabial flap) — for small-to-moderate defects with adjacent tissue available.
- **Regional flaps** — the pectoralis major myocutaneous flap remains a reliable workhorse, particularly where free tissue transfer is unavailable or the patient is unfit for prolonged microsurgery.
- **Free flaps** — the current standard for most moderate-to-large composite defects: the *radial forearm free flap* for thin, pliable soft-tissue defects (e.g., tongue, floor of mouth); the *fibula free flap* for segmental mandibular defects, given its long, reliably vascularised bone stock suitable for osseointegrated dental implants; and the *anterolateral thigh (ALT) flap* for large soft-tissue or composite defects requiring bulk.
- **Dental rehabilitation** — osseointegrated implants (often into fibula grafts) and prosthodontic planning should be considered early, ideally as part of a multidisciplinary reconstructive plan.

## 12. Adjuvant Therapy

Adjuvant treatment decisions are based on the pathological risk features identified in the resected specimen and neck dissection, integrated at a multidisciplinary tumour board.

### Indications for Postoperative Radiotherapy (PORT)

Close margins (1–5 mm), or positive margins where re-resection is not feasible.  
pT3 or pT4 primary tumour.  
Nodal involvement — particularly multiple positive nodes, or bulky/pN2–pN3 disease.  
Perineural invasion.  
Lymphovascular invasion.  
Depth of invasion >10 mm as a contributing high-risk feature in some protocols.  
Advanced pathological T or N stage even in the absence of other individual high-risk features.

### Indications for Postoperative Concurrent Chemoradiotherapy

Positive surgical margins (tumour at ink) that cannot be surgically revised.  
Extranodal extension (ENE) — the strongest indication, supported by EORTC 22931 and RTOG 9501 evidence for a survival benefit with the addition of concurrent platinum-based chemotherapy to radiotherapy in these high-risk patients.

Radiotherapy is typically delivered to the primary tumour bed and at-risk neck levels over five to six weeks, with concurrent cisplatin-based chemotherapy in ENE-positive or margin-positive disease where the patient's performance status and renal function permit. Altered fractionation and intensity-modulated radiotherapy (IMRT) are used to optimise disease control while limiting toxicity to salivary and swallowing structures.

## 13. Complications of Treatment

### ***Surgical Complications***

- Flap failure (partial or total) requiring salvage reconstruction or return to theatre.
- Orocutaneous or salivary fistula.
- Wound infection and dehiscence, particularly in previously irradiated or malnourished patients.
- Haemorrhage, including delayed carotid blowout in heavily pretreated/irradiated necks.
- Chyle leak following lower neck dissection (thoracic duct injury, typically left-sided).
- Shoulder dysfunction from accessory nerve injury or sacrifice.

### ***Functional and Late Complications***

- Speech and swallowing dysfunction, proportional to the extent of tongue/floor-of-mouth resection.
- Trismus, from surgical scarring, radiation fibrosis of the masticatory muscles, or both.
- Xerostomia and dental caries from radiotherapy-induced salivary gland damage.
- Osteoradionecrosis of the mandible — a serious late complication of radiotherapy, particularly after dental extraction in a previously irradiated field; pre-treatment dental clearance of unsalvageable teeth is a standard preventive measure.
- Lymphoedema of the face/neck and psychosocial impact related to disfigurement and altered function.

## 14. Site-Specific Considerations

- **Tongue** — the most common oral cavity subsite in many series; deeply infiltrative growth pattern and a relatively high rate of occult nodal metastasis even in small tumours make DOI assessment particularly important here.
- **Buccal mucosa** — strongly associated with areca nut/quid-chewing habits and oral submucous fibrosis; prone to early skin and mandibular involvement, and trismus from underlying OSMF can mask or delay diagnosis.
- **Gingivobuccal sulcus** — a distinctly common presentation in the Indian subcontinent, with early cortical bone invasion; often requires marginal or segmental mandibulectomy even at presentation.
- **Floor of mouth** — midline lesions carry a risk of bilateral nodal spread; proximity to Wharton's ducts and the sublingual space demands careful assessment of deep extension.
- **Hard palate/upper alveolus** — SCC here is less common than minor salivary gland malignancy; maxillary sinus involvement should be actively excluded.
- **Lip** — generally the most favourable prognosis of all oral cavity subsites owing to early detection, a well-defined anatomical plane and a lower propensity for early nodal spread; upper lip and commissure lesions behave more aggressively than lower lip lesions.
- **Retromolar trigone** — a small, deceptively deep subsite adjacent to the pterygomandibular raphe and ascending ramus; early invasion of the mandible, masticator space and pterygoid muscles is characteristic, and disease here is often understaged clinically.

## 15. Recurrence, Surveillance and Prognosis

Local recurrence is most common within the first two years after treatment, and structured surveillance aims to detect recurrence and second primary tumours (which occur in the "condemned mucosa" of the aerodigestive tract at a rate of several percent per year) while they remain salvageable.

### ***Follow-up Schedule (Typical)***

- Every 1–3 months for the first year.
- Every 2–4 months in the second year.
- Every 4–6 months in years three to five.
- Annually thereafter, with lifelong review given ongoing second primary risk, particularly if risk-factor exposure continues.

### ***Adverse Prognostic Factors***

- Advanced T stage and depth of invasion.
- Nodal involvement, especially multiple nodes and extranodal extension.
- Positive or close surgical margins.
- Perineural and lymphovascular invasion.
- Poor histological differentiation and non-cohesive/worst pattern of invasion.
- Continued tobacco/areca nut use after treatment.

Overall five-year survival ranges broadly from over 80% for early stage I disease to well under 40% for stage IV disease, reinforcing the central importance of early detection.

## 16. Prevention and Early Detection

Given the well-established, largely avoidable risk-factor profile of oral cavity cancer, prevention and early detection offer the greatest potential to reduce the disease burden in high-incidence regions.

- Public health measures to reduce tobacco and areca nut/gutka consumption, including taxation, packaging regulation, advertising restriction and community cessation programmes.
- Opportunistic oral visual screening by dentists, primary care physicians and community health workers in high-risk populations, given the accessibility of the oral cavity to direct examination.
- Patient and public education on the significance of a non-healing oral ulcer or persistent white/red patch.
- Structured surveillance and biopsy-based follow-up of patients with known potentially malignant disorders (leukoplakia, erythroplakia, OSMF, proliferative verrucous leukoplakia).
- Integration of oral cancer screening into existing community and primary care platforms, which has shown mortality benefit in high-incidence, high-risk population studies.

## 17. Summary of Current Guidelines and Evidence

Management recommendations in this review are aligned with the AJCC 8th edition staging manual, the NCCN Clinical Practice Guidelines in Oncology for Head and Neck Cancers (most recently updated 2025), and the ASCO Clinical Practice Guideline on management of the neck in squamous cell carcinoma of the oral cavity and oropharynx. Key evidence-based practice points include the incorporation of depth of invasion into T staging and elective neck dissection decision-making, the survival benefit of elective neck dissection over observation in early cN0 disease (POI trial), and the addition of concurrent chemotherapy to postoperative radiotherapy in patients with extranodal extension or positive margins (EORTC 22931, RTOG 9501).

As with all oncological guidance, recommendations continue to evolve; trainees should confirm current protocol details against the latest published guideline versions and their institutional multidisciplinary tumour board policies before applying them in clinical decision-making.

## Key Points for Revision

### High-Yield Summary

Oral cavity cancer is largely a preventable, tobacco/areca-nut-driven disease with a disproportionate burden in South Asia; buccal mucosa and gingivobuccal sulcus involvement reflects local quid-chewing patterns.

Leukoplakia, erythroplakia, oral submucous fibrosis and proliferative verrucous leukoplakia are the key potentially malignant disorders — biopsy any persistent or suspicious lesion.

AJCC 8th edition staging incorporates depth of invasion (DOI) into T stage and extranodal extension (ENE) into N stage — both are strong independent prognostic factors.

Bimanual palpation is essential to assess tumour depth and fixation; imaging (MRI for soft tissue/perineural spread, CT for bone) complements clinical staging.

DOI >4 mm is the practical threshold favouring elective neck dissection in the cN0 neck; sentinel node biopsy is an accepted alternative at experienced centres for early disease.

Clear margins are  $\geq 5$  mm; positive margins are now defined as tumour at ink.

Extranodal extension and positive margins are the key indications for adding concurrent chemotherapy to postoperative radiotherapy.

Free flap reconstruction (radial forearm, fibula, ALT) is the current standard for most moderate-to-large defects; fibula grafts additionally allow osseointegrated dental rehabilitation.

Second primary tumours of the aerodigestive tract remain a lifelong risk — surveillance never truly ends, especially with continued risk-factor exposure.

## Further Reading and Key Sources

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

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