# Standaardrapport mondholte

Conclusie

**Neoadjuvante therapie:** Kies een optie.

**Tumorlokalisatie:**

**Lip**: Kies een optie.

**Mondholte**: Kies een optie.

*Indien andere, specificeer:*

**Lateraliteit**: Kies een optie.

**Focaliteit:** Kies een optie.

*Indien multifocaal, specificeer aantal foci:*

*Indien niet beoordeeld kan worden, specificeer:*

**Tumorafmetingen**: Kies een optie.

Maximale tumordimensie (in mm):

*Indien niet bepaald kan worden, specificeer:*

**Histologisch tumortype[[1]](#footnote-1):** Kies een optie.

*Indien van toepassing, specificeer type:*

**Differentiatiegraad:** Kies een optie.

*Indien niet beoordeeld kan worden, specificeer:*

**Worst pattern of invasion (WPOI):** Kies een optie.

**Invasiediepte[[2]](#footnote-2):** Kies een optie.

Exacte invasiediepte (in mm):

*Indien niet beoordeeld kan worden, specificeer:*

**Patroon van invasie**: Kies een optie.

**Lokale uitbreiding**: Kies een optie.

*Indien lokale uitbreiding werd aangetoond:*

Invasie van bot[[3]](#footnote-3): Kies een optie.

Invasie in huid van aangezicht/nek: Kies een optie.

Invasie in mondbodem (lip): Kies een optie.

Invasie in sinus maxillaris (mondholte): Kies een optie.

*Indien lokale uitbreiding niet beoordeeld kan worden, specificeer:*

**Lymfovasculaire invasie**: Kies een optie.

*Indien onderscheid tussen lymfevat- en bloedvatinvasie mogelijk, specificeer:* Kies een optie.

*Indien niet met zekerheid te bepalen, specificeer:*

**Perineurale invasie[[4]](#footnote-4)**: Kies een optie.

*Diameter grootste aangetaste zenuwtak (in mm):* Kies een optie.

*Indien niet met zekerheid te bepalen, specificeer:*

**Definitieve minimale tumorvrije marge invasief carcinoom**

**(incl. eventuele naresecties en vriescoupes)[[5]](#footnote-5):** Kies een optie.

Definitieve minimale tumorvrije marge (in mm):

Specificeer dichtstbijzijnde snedevlak, indien mogelijk:

*Indien niet beoordeeld kan worden, specificeer:*

**Definitieve minimale vrije marge ernstige dysplasie/in situ carcinoom**

**(incl. eventuele naresecties en vriescoupes)[[6]](#footnote-6):** Kies een optie.

Definitieve minimale vrije marge (in mm):

Specificeer dichtstbijzijnde snedevlak, indien mogelijk:

*Indien niet beoordeeld kan worden, specificeer:*

**pTNM classificatie (UICC TNM 8e editie met errata)[[7]](#footnote-7):** **pT** Kies een optie.

Voorvoegsels:

m (multipele primaire tumoren): Kies een optie.

r (recidief): Kies een optie.

y (na neoadjuvante therapie): Kies een optie.

Scope

Resectie- en excisiebiopsiestalen van maligniteiten van de mondholte, inclusief slijmvlies van de lip en tong (mucosale carcinomen, maligniteiten van kleine speekselklieren).

Opmerkingen

* Incisiebiopsieën en andere biopsiestalen zijn niet opgenomen in deze dataset.
* Voor resecties van recidieven kan de dataset pragmatisch worden gebruikt, hoewel sommige variabelen mogelijk niet toepasbaar of te beoordelen zijn.
* Mucosaal melanoom, lymfomen en sarcomen worden in aparte datasets behandeld.
* Neuro-endocriene neoplasmen worden in een aparte dataset behandeld.
* Halsdissecties en klierexcisies worden behandeld in een aparte dataset. Deze dataset moet, indien van toepassing, in combinatie worden gebruikt.
* Voor aanvullende onafhankelijke (multicentrische) tumoren moet voor elke tumor een aparte dataset worden ingevuld.

Dataset gebaseerd op

Müller S, Day TA, Griffith CC, Magliocca KR, Mori T, Richardson MS, Sloan P, Tilakaratne WM, Zain RB, Helliwell T, Thompson LDR (2024). *Carcinomas of the Oral Cavity Histopathology Reporting Guide. 2nd edition*. International Collaboration on Cancer Reporting; Sydney, Australia. ISBN: 978-1-922324-45-0.

Bijkomende informatie[[8]](#footnote-8)

**Note 1 –Tumour site**

The anatomy and surgical interventions of the oral cavity are complex, and it is important to ensure accurate and precise communication between the pathologists and the treating and diagnostic team with respect to exact anatomic site of involvement, tumour laterality and specific operative procedures.9-11

The protocol applies to all carcinomas arising at these sites (see Figure 1). For large cancers that involve more than one site, the primary site of involvement should be recorded.

Mucosal Lip. Begins at the junction of the wet and dry mucosa (vermilion border) that comes in contact with the opposing lip. The dry vermilion lip and vermilion border are staged using the cutaneous dataset.12

Buccal Mucosa (Inner Cheek). Mucous membrane lining of the inner surface of the cheeks and lips of contact of the opposing lips to the line of attachment of mucosa of the upper and lower alveolar ridge and pterygomandibular raphe.

Lower Alveolar Ridge. Mucosa overlying the alveolar process of the mandible, which extends from the line of attachment of mucosa in the buccal vestibule to the line of free mucosa of the floor of the mouth.

Posteriorly it extends to the ascending ramus of the mandible.

Upper Alveolar Ridge. Mucosa overlying the alveolar process of the maxilla, which extends from the line of attachment of mucosa in the upper gingival buccal vestibule to the junction of the hard palate. The posterior margin is the upper end of the pterygopalatine arch.

Floor of the Mouth. Semilunar space over the mylohyoid and hyoglossus muscles, extending from the inner surface of the lower alveolar ridge to the undersurface of the tongue. The posterior boundary is the base of the anterior pillar of the tonsil. It is divided into two sides of the submaxillary and sublingual salivary glands.

Hard Palate. This is the semilunar area between the upper alveolar ridge and the mucous membrane covering the palatine process of the maxillary palatine bones. It extends from the inner surface of the superior alveolar ridge to the posterior edge of the palatine bone.

Anterior Two-Thirds of the Tongue (Oral Tongue). The freely mobile portion of the tongue that extends anteriorly from the line of circumvallate papillae to the undersurface (ventral) of the tongue at the junction of the floor of the mouth. It includes the tip of tongue, lateral borders, dorsal surface and ventral tongue. The ventral tongue is listed as a separate tumour site in the ICCR reporting guide.

Retromolar trigone. A triangular shaped region extending distal from the mandibular third molar as the base and attaches to the hamulus of the medial pterygoid process of the sphenoid bone as the apex.

‘Not specified’ should be used rarely and only after good effort has been employed to obtain the requisite information.

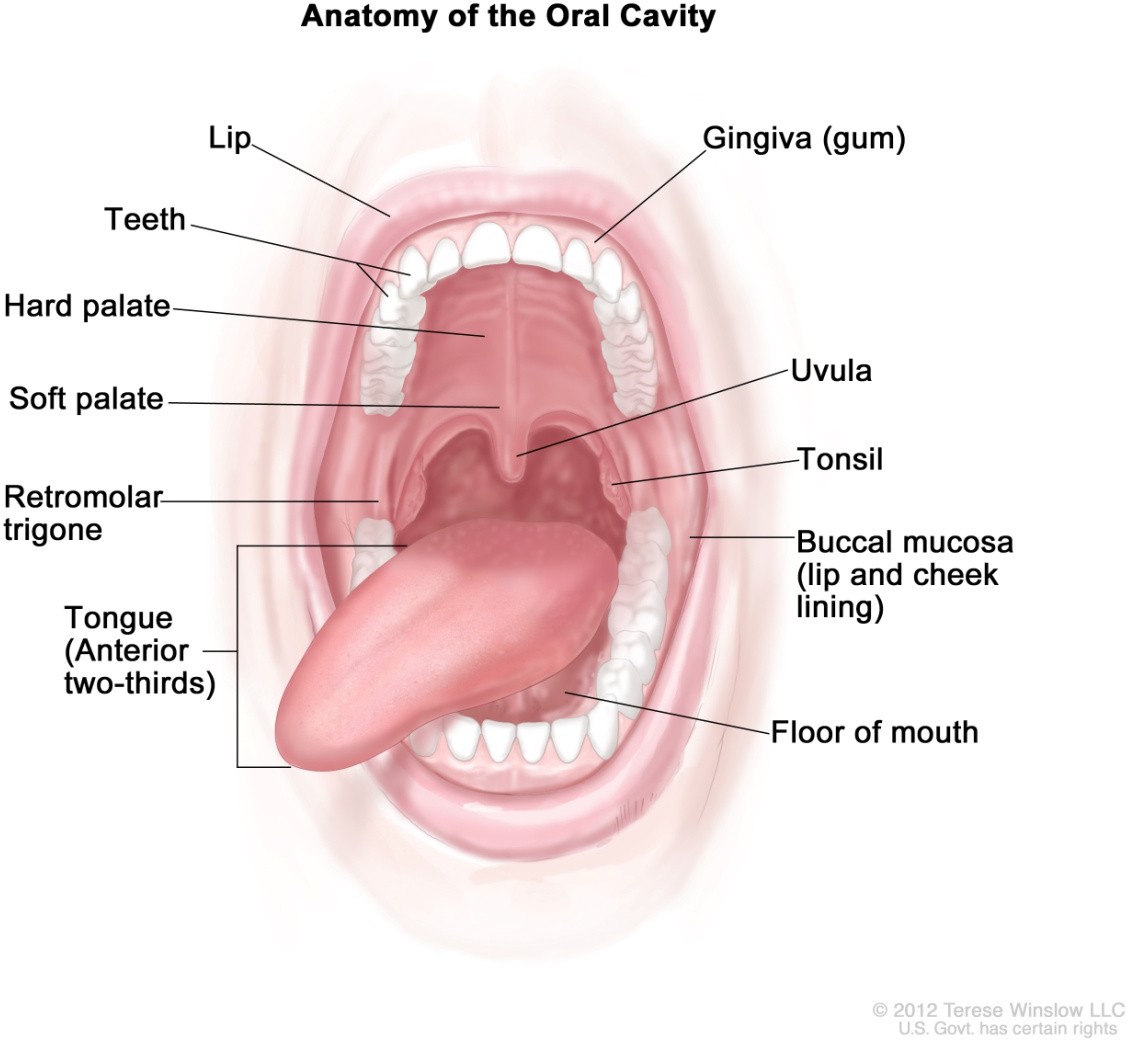


Figure 1: Anatomic sites and subsites for lip and oral cavity.

* The vermilion/dry lip is considered cutaneous.
* The uvula and soft palate and tonsil are considered oropharynx.

**Note 2 – Tumour dimensions**

Tumour dimension is an important component in pathologic staging.16,17 If available, measurements are made on fresh tissue. The macroscopic diameter (in millimetres) should be used unless the histological extent is greater than macroscopically apparent, in which case the microscopic dimension is used. At times only microscopic evaluation differentiates what clinically appears to be tumour from what is actual invasion (not dysplasia or inflammation). At least the greatest tumour dimension should be reported; preferably all three dimensions should be evaluated. Measurements are made pragmatically, acknowledging distortion of tissues by fixation and processing.18

**Note 3 – Histological tumour type**

All tumours of the oral cavity should be given a type based on the most recent edition of the WHO Classification of Head and Neck Tumours, 5th edition, 2024 (Table 1).4 The major histologic tumour types of SCC as recognised by the WHO classification are SCC, conventional type, basaloid, papillary, spindle, adenosquamous, acantholytic, lymphoepithelial, verrucous carcinoma and carcinoma cuniculatum. Hybrid lesions such as verrucous carcinoma and SCC exist and should be recognised as it may affect prognosis.

Subtypes should be assigned for both prognosis and cancer registry.19,20

Salivary gland carcinoma histologic type essentially defines its biologic behaviour and thus influences prognosis, patterns of recurrence and thus clinical management.21 Some carcinoma types (i.e., basal cell adenocarcinoma, conventional acinic cell carcinoma) are more indolent with locoregional recurrence but low nodal and distant metastatic rates.22 For guidance on histological typing of minor salivary gland carcinomas, please refer to the ICCR Carcinomas of the major salivary gland dataset.23

The classification and grading of neuroendocrine carcinomas (NEC) is discussed in **Note 4 HISTOLOGICAL TUMOUR GRADE**.

Table 1: World Health Organization classification of subtypes of squamous cell carcinoma of the oral cavity and mobile tongue.4

|  |  |
| --- | --- |
| **Descriptor** | **ICD-O codes**a |
| **Epithelial tumours and lesions** |  |
| Squamous cell carcinoma, conventional type | 8070/3 |
| Spindle cell (sarcomatoid) squamous cell carcinoma | 8074/3 |
| Basaloid squamous cell carcinoma | 8083/3 |
| Acantholytic squamous cell carcinoma | 8075/3 |
| Adenosquamous carcinoma | 8560/3 |
| Papillary squamous cell carcinoma | 8052/3 |
| Lymphoepithelial carcinoma | 8082/3 |
| Verrucous carcinoma | 8051/3 |
| Carcinoma cuniculatum | 8051/3 |
| **Epithelial neuroendocrine neoplasms** |  |
| Small cell neuroendocrine carcinoma | 8041/3 |
| Large cell neuroendocrine carcinoma | 8013/3 |
| Carcinoma mixed with small cell neuroendocrine carcinomab | 8045/3 |
| Carcinoma mixed with large cell neuroendocrine carcinomab | 8013/3 |

a These morphology codes are from the International Classification of Diseases for Oncology, third edition, second revision (ICD-0-3.2).24 Behaviour is coded /0 for benign tumours; /1 for unspecified, borderline, or uncertain behaviour:

/2 for carcinoma in situ and grade Ill intraepithelial neoplasia; /3 for malignant tumours, primary site: and /6 for malignant tumours, metastatic site. Behaviour code /6 is not generally used by cancer registries.

b This terminology is synonymous with the ICD-O terminology of combined small/large cell neuroendocrine carcinomas.

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**Note 4 – Histological tumour grade**

Based on the WHO classifications, three histologic grades of SCC, conventional type are used: well, moderately or poorly differentiated.4 The most aggressive or highest grade should be recorded if the tumour has a varied histology. Grading requires the assessment of keratinisation, mitotic activity, cellular and nuclear pleomorphism, pattern of invasion and host response.7,25-27 SCC subtypes are not graded. Still, several grading systems for each tumour type are available, with differing merits, and as such, recording which system has been applied is more clinically meaningfully (use ‘specify’ to state the system used), with the ICCR deferring to the WHO classification current edition for grading guidance and preference.4

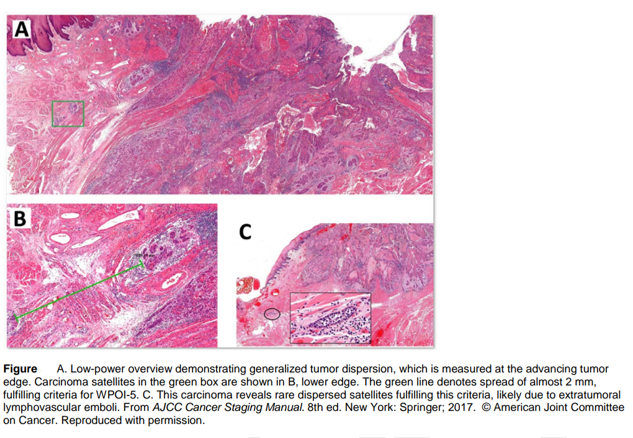
Grading of minor salivary gland tumours follows the criteria for major salivary gland tumours.22,23

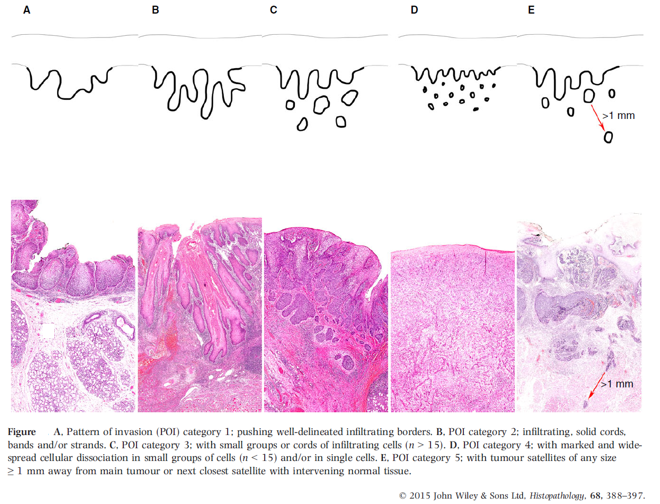
Neuroendocrine neoplasms, as newly defined,4 include paraganglioma/pheochromocytoma, neuroendocrine tumours, and NECs. Neuroendocrine tumours are separated into grades (1, 2, and 3) based on mitotic rate and Ki-67 proliferation indices, but these criteria are not yet fully developed for each of the anatomic sites in the head and neck. At present, the general cutoffs are: grade 1: <2 mitoses/2 millimetre (mm)2 and <2%

Ki-67 proliferation index; grade 2: ≥2-10 mitoses/2 mm2 and 2-20% Ki-67 proliferation index; grade 3: ≥11 mitoses/2 mm2 and >20% Ki-67 proliferation index.28,29 Further, NECs are separated into small cell and large cell categories, showing tumour necrosis, >10 mitoses/2 mm2 and >20% Ki-67 proliferation index,28,30-32 with universal Rb1 loss and common p53 overexpression.33 At present, the site, tumour category, and grade (non- core) should be reported, with additional advances in this field incorporated when validated further.

**Note 5 – Worst pattern of invasion**

Worst pattern of invasion (WPOI) has been validated as a prognosticator for oral cavity squamous carcinomas. While there are 5 patterns noted, distinction between WPOI-5 and other patterns is what is most relevant. WPOI-5 is defined by tumor dispersion ≥ 1mm between tumor satellites. Examples of pattern 5 are shown in the figures below. WPOI has been validated on multivariate analysis in oral tumors, also specifically in low stage tumors. However, WPOI can be viewed as redundant and only optional for reporting purposes as extramural perineural invasion (PNI), and angiolymphatic invasion also count as WPOI-5.





**Note 6 – Depth of invasion**

Depth of invasion (DOI) in OSCC, particularly of the tongue, has been identified as an important prognostic indicator, and is therefore a core element. The Union for International Cancer Control (UICC)/American Joint Committee on Cancer (AJCC) TNM staging systems incorporate DOI in determining the tumour stage (T).16,17,34 DOI increases T by 1 step for every 5 mm, whereby T1 is tumour ≤20 mm (≤2 centimetres (cm)) and DOI ≤5 mm, T2 is tumour ≤20 mm (≤2 cm) and DOI >5 mm and ≤10 mm, T3 tumour is >20 mm (>2 cm) and

≤40 mm (≤4 cm) and >10 mm DOI and T4a is tumour >40 mm (>4 cm) or any tumour >10 mm DOI. DOI measures the invasiveness of the carcinoma. To measure DOI, the basement membrane is identified, and an imaginary line is drawn across the tumour. A vertical or ‘plumb line’ extends to the deepest part of the tumour which represents the DOI. When the tumour is widely dispersed (see **Note 6 – PATTERN OF INVASIVE FRONT**), the measurement should be from the most distance tumour nest. It is important to note that DOI is not synonymous with tumour thickness. An exophytic tumour (Figure 2A) may be thicker than an ulcerative tumour (Figure 2B), but the DOI of the ulcerative lesion may be greater.35,36

The maximum DOI should be recorded as core and the discussion should include how/why DOI is different than tumour thickness.18,37-39

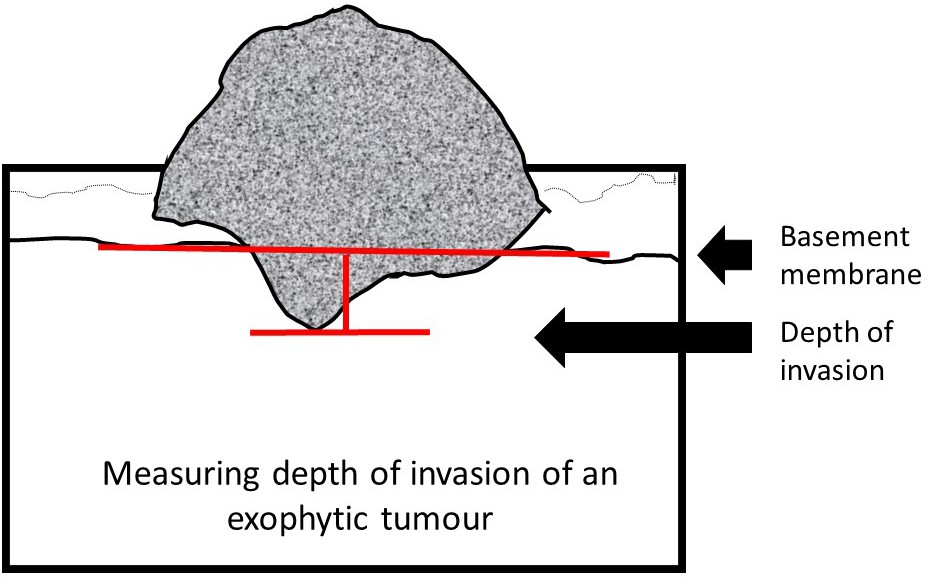


Figure 2A: Measuring depth of invasion.

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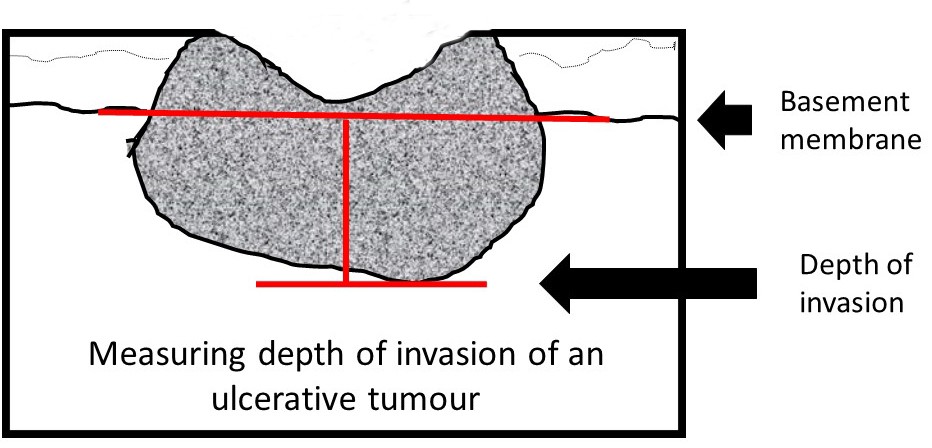


Figure 2B: Measuring depth of invasion.

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**Note 7 – Pattern of invasive front**

The pattern of invasion in OSCC has proven prognostic value and should be reported as cohesive or non- cohesive (Figure 3).25,40-43 It is important to evaluate the most complex area of tumour-stroma interface (‘worst’ area), usually at the advancing edge, and ideally assessment should only be made on resection specimens or excisional biopsies. Acknowledgement is made that at times non-surgical treatment decisions are made on incisional biopsy specimens only and consequently the best assessment of pattern of invasion should be noted. Cohesive invasion is defined as broad sheets of cancer cells and/or tumour nests of >15 tumour cells. Non-cohesive invasion shows a spectrum of appearances that includes narrow strands, small groups of ≤15 tumour cells and single infiltrating tumour cells.35,36 For stage T1/T2 OSCC, particularly those arising in the tongue, there is evidence that tumour satellites localised ≥1 mm away from the main tumour or nearest satellite (worst pattern of invasion WPOI-5) is a valid adverse prognostic factor.25,40-42,44

Additionally, tumour budding has emerged as a promising biomarker in various carcinomas, with early evidence suggesting that it is an independent adverse prognostic factor in carcinoma of the oral cavity.45-52

Tumour budding is defined as single tumour cells or clusters of up to four tumour cells at the invasive tumour front. There is no consensus yet how it should be assessed and graded in oral carcinoma. It has been recommended to count the number of buds in 2 mm2 high power field (HPF) (x40) after scanning 10 HPFs in areas showing maximal budding.53 Budding activity is graded as low if 1 to 14 buds per 2mm2 and high if ≥15 buds per 2 mm2 are counted.

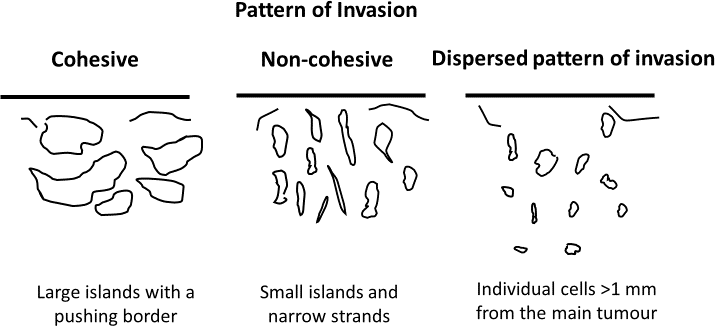


Figure 3: Pattern of Invasive front.

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**Note 8 – Extent of invasion**

Infiltrative bone involvement by SCC correlates with a worse prognosis. Bone invasion may be a macroscopic feature, however sampling through the involved bone for histologic examination should be performed to obtain histologic evidence. The presence of bone invasion affects tumour staging and patients with bone invasion often have a worse prognosis. It is important to distinguish superficial cortical bone erosion from infiltrative invasion to the medullary bone as this is critical in accurate tumour staging. If bone is resected, then bone margins should be recorded.54 Tumour involvement of the maxillary sinus, and skin of the face and neck increases the pathological stage and should be noted.

**Note 9 – Lymphovascular invasion**

There is a need to distinguish between intravascular tumour embolization and retraction artefact. Positive lymphovascular invasion is a risk factor for decreased overall survival and should be reported only when tumour emboli are identified within endothelial lined spaces. No distinction between venous channels and small lymphatics is required.27,55,56

Cases that are still equivocal after taking additional steps may be reported as ‘indeterminate’ for lymphovascular invasion, but this designation should be sparingly used, and it is useful to provide the reason in a comment in the report.

**Note 10 – Perineural invasion**

Perineural invasion is associated with a worse prognosis, regardless of nerve size and should be recorded. The presence or absence of perineural and/or endoneural/intraneural invasion may impact subsequent therapy and prognosis.7,25,27,57

**Note 11 – Margin status**

All surgical margins should be measured in millimetres histologically for both mucosal and deep margins. In the comments section, acknowledgement should be made how the surgical margin was measured. For example, if the margin was submitted from the tumour bed margin at the time of the operative procedure rather than from the surgical specimen.58-60 The presence of severe dysplasia/carcinoma in situ at the margin is associated with an increased risk of local recurrence and this should be recorded. The definition of a ‘close’ margin is not standardised but in the oral cavity from a surgical point of view >5 mm is clear, and 1-5 mm is close, while <1 mm is involved. Acknowledgement is made of fixation and processing distortion on measurements which may cause tissue shrinkage including the surgical margin.61 Acknowledgement is also made of any laser or electrocautery associated tissue distortion such as cellular and nuclear polymorphism, nuclear hyperchromatism, epithelial cell separation, collagen denaturation, etc. on measurements including the surgical margin.62 Any bone resection margins should be identified and comment on the presence or absence of carcinoma at these margins should be provided.7,54 Dysplastic changes include abnormal cellular organisation, increased mitotic activity, and nuclear enlargement with pleomorphism.7,25,63,64 Although terminology varies, using the 2024 WHO criteria for oral dysplasia,4 dysplasia limited to the lower one-third of the epithelium is generally referred to as mild dysplasia. However, this can undercall higher dysplasia grades when both the architectural and cytological features of dysplasia are confined to the lower third depending on the individual features, such as tumour budding, bulbous rete and pleomorphism.4,9,65-67 Moderate dysplasia is defined as cytological atypia extending to the middle third of the epithelium and severe dysplasia extends to the upper third of the epithelium. Carcinoma in situ is considered synonymous with severe dysplasia.

Reporting of surgical margins for carcinomas of the minor salivary glands should follow those used for SCC of oral cavity.

**Note 12 – Ancillary studies**

For neuroendocrine neoplasms core elements are neuroendocrine markers, epithelial markers, and Ki-67 proliferation index. The diagnosis of neuroendocrine neoplasms (specifically NETs and NECs) must be confirmed immunohistochemically, with positive reaction for neuroendocrine markers (synaptophysin, chromogranin, INSM1) and for epithelial markers (pancytokeratin, cytokeratin). A proliferation index as determined by KI-67 immunohistochemical analysis is recommended for grading all NETs, and helping to confirm NECs. Both p53 and Rb1 may be helpful in distinguishing between NET and NEC, especially G3 NET from NEC.29,33,70

In most cases, further studies are not required for diagnosis of other tumours. Epithelial immuno- histochemical markers may be required for poorly differentiated or spindle cell carcinoma including AE1/ AE3, CK5/6, p63 and p40.71 Lymphoepithelial SCC in the oral cavity is rare and although not all cases are Epstein-Barr virus (EBV)-positive, EBV-encoded small RNAs (EBERs) studies are indicated.72 There is currently no role for routine HPV high risk type testing in OSCC.19,71 HPV-associated epithelial dysplasia requires in-situ hybridization/PCR confirmation.19

Programmed cell death-ligand 1 (PD-L1) expression has been used as predictive biomarker for checkpoint inhibitor therapy since the anti-programmed cell death-1 receptor (PD-1) antibodies, nivolumab and pembrolizumab, have been approved for the treatment of patients with recurrent and/or unresectable metastatic head and neck SSC,73-76 with various cutoffs of expression associated with betters responses, although not in all patients.77

**Note 13 – Pathological staging**

By UICC/AJCC convention,16,17 the designation ‘T’ refers to a primary tumour that has not been previously treated. The symbol ‘p’ refers to the pathologic classification of the stage, as opposed to the clinical classification, and is based on gross and microscopic examination. pT entails a resection of the primary tumour adequate to evaluate the highest pT category, pN entails removal of nodes adequate to validate lymph node metastasis, and pM implies microscopic examination of distant lesions. There is no pathologic M0 category as this designation requires clinical evaluation and imaging. Clinical classification (cTNM) is usually carried out by the evaluating clinician before treatment during initial evaluation of the patient or when pathologic classification is not possible.

Pathological staging is usually performed after surgical resection of the primary tumour and depends on documentation of the anatomic extent of disease, whether or not the primary tumour has been completely removed. If a biopsied tumour is not resected for any reason (e.g., when technically unfeasible) and if the highest T and N categories or the M1 category of the tumour can be confirmed microscopically, the criteria for pathologic classification and staging have been satisfied even though total removal of the primary cancer was not performed.

**Primary tumour (pT)**

TX Primary tumour cannot be assessed

Tis Carcinoma in situ

T1 Tumour 2 cm or less in greatest dimension and 5 mm or less depth of invasion

T2 Tumour 2 cm or less in greatest dimension and more than 5 mm depth of invasion or tumour more than 2 cm but not more than 4 cm in greatest dimension and depth of invasion no more than 10 mm

T3 Tumour more than 2 cm but not more than 4 cm in greatest dimension and depth of invasion more than 10 mm or tumour more than 4 cm in greatest dimension and not more than 10 mm depth of invasion

T4a (Lip) Tumour more than 4 cm in greatest dimension and more than 10 mm depth of invasion or tumour invades through cortical bone, inferior alveolar nerve, floor of mouth, or skin (of the chin or the nose)

T4a (Oral cavity) Tumour more than 4 cm in greatest dimension and more than 10 mm depth of invasion or tumour invades through the cortical bone of the mandible or maxilla or involves the maxillary sinus, or invades the skin of the face

T4b (Lip and oral cavity) Tumour invades masticator space, pterygoid plates, or skull base, or encases internal carotid artery

**TNM Descriptors**

For identification of special cases of TNM or pTNM classifications, the ‘m’ suffix and ‘y’ and ‘r’ prefixes are used. Although they do not affect the stage grouping, they indicate cases needing separate analysis.

The ‘m’ suffix indicates the presence of multiple primary tumours in a single site and is recorded in parentheses: pT(m)NM.

The ‘y’ prefix indicates those cases in which classification is performed during or following initial multimodality therapy (i.e., neoadjuvant chemotherapy, radiation therapy, or both chemotherapy and radiation therapy). The cTNM or pTNM category is identified by a ‘y’ prefix. The ycTNM or ypTNM categorises the extent of tumour actually present at the time of that examination. The ‘y’ categorisation is not an estimate of tumour prior to multimodality therapy (i.e., before initiation of neoadjuvant therapy).

The ‘r’ prefix indicates a recurrent tumour when staged after a documented disease-free interval, and is identified by the ‘r’ prefix: rTNM.

For the pN classification of regional lymph nodes, see ICCR Nodal excisions and neck dissection specimens dataset.3

Reporting of pathological staging categories (pT, pN, pM) is based on the evidence available to the pathologist at the time of reporting. As indicated in UICC TNM8 and AJCC TNM8,16,17 the final stage grouping of a patient's tumour is based on a combination of pathological staging and other clinical and imaging information.

Pathological staging should not be reported if the submitted specimen is insufficient for definitive staging, especially with biopsy samples (core needle, incisional or excisional). Staging is based on the submitted resection, and even if there is grossly residual disease or there is tumour at the margin, pT staging should only be reported on findings in the resection specimen and/or at operation.16,17

The reference document TNM Supplement: A commentary on uniform use, 5th Edition (C Wittekind et al. editors) may be of assistance when staging.78

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1. Bij neoplasmata van kleine speekselklieren moet de optie “speekselkliercarcinoom (specificeer)” gekozen worden. Het histologische subtype kan vervolgens gespecificeerd worden volgens de lijst opgenomen in het standaardrapport “Grote speekselklieren”. [↑](#footnote-ref-1)
2. De invasiediepte heeft betrekking op de tumorinvasie, onafhankelijk van een exofytische component. Deze wordt gemeten door de ‘horizon’ van de basale laag van het aangrenzende plaveiselepitheel te bepalen. Een loodrechte ‘plumb line’ wordt getrokken vanaf deze ‘horizon’ tot aan het diepste punt van de tumorinvasie (invasiediepte). Voor meer informatie, zie ‘Note 6 – Depth of invasion’ onderaan dit document. [↑](#footnote-ref-2)
3. De aanwezigheid van botinvasie beïnvloedt de stadiëring van de tumor en is een negatieve prognostische factor. Het is belangrijk om onderscheid te maken tussen oppervlakkige corticale boterosie en infiltratieve invasie in het medullaire bot, boterosie zal immers geen invloed hebben op de nabehandeling. Voor meer informatie, zie ‘Note 8 – Extent of invasion’ onderaan dit document. [↑](#footnote-ref-3)
4. De aan- of afwezigheid van peritumorale perineurale invasie kan invloed hebben op de behandeling en prognose. Het gaat hierbij om peritumorale perineurale invasie langs of voorbij de tumorgrens, en dus niet om intratumorale uitbreiding. Voor meer informatie, zie ‘Note 10 – Perineural invasion’ onderaan dit document. [↑](#footnote-ref-4)
5. De definitie van ' Definitieve minimale tumorvrije marges’ is niet gestandaardiseerd, maar in de mondholte is vanuit chirurgisch oogpunt <1 mm positief, 1-5 mm ‘close’ en >5 mm ‘clear’. De aanwezigheid van ernstige dysplasie/carcinoom in situ in het snedevlak wordt geassocieerd met een verhoogd risico op lokaal recidief en dit moet ook worden geregistreerd. Voor meer informatie, zie ‘Note 11 – Margin status’ onderaan dit document. [↑](#footnote-ref-5)
6. De definitie van ' Definitieve minimale tumorvrije marges’ is niet gestandaardiseerd, maar in de mondholte is vanuit chirurgisch oogpunt <1 mm positief, 1-5 mm ‘close’ en >5 mm ‘clear’. De aanwezigheid van ernstige dysplasie/carcinoom in situ in het snedevlak wordt geassocieerd met een verhoogd risico op lokaal recidief en dit moet ook worden geregistreerd. Voor meer informatie, zie ‘Note 11 – Margin status’ onderaan dit document. [↑](#footnote-ref-6)
7. De pathologische stadiëring is gebaseerd op de ‘TNM Classification of Malignant Tumours’ (8e editie, UICC). Een beschrijving van elke pT-categorie is beschikbaar in ‘Note 13 – Pathological staging’ onderaan dit document. [↑](#footnote-ref-7)
8. Bijkomende informatie (‘notes’) overgenomen en aangepast vanuit

   Müller S, Day TA, Griffith CC, Magliocca KR, Mori T, Richardson MS, Sloan P, Tilakaratne WM, Zain RB, Helliwell T, Thompson LDR (2024). *Carcinomas of the Oral Cavity Histopathology Reporting Guide. 2nd edition*. International Collaboration on Cancer Reporting; Sydney, Australia. ISBN: 978-1-922324-45-0 [↑](#footnote-ref-8)