

Periodontal disease risk assessment and management

Purpose

Clinical Practice Guidelines (CPG's) are systematic developed statements intended to support clinicians in providing high quality, best practice evidence-based care. They are not intended to be wholly prescriptive or a legal directive for clinical decisions. While their application is an acceptable ground for client care, clinicians should carefully consider the individual circumstances and the specifics of their work environment in conjunction with these guidelines. Selection of alternative treatment modalities, based on clinical judgment and/or specialist advice, may be justified in certain clinical scenarios. In such cases, justification for the chosen treatment must be clearly documented in the client records.

This Clinical Guideline aims to provide a standardised approach to the periodontal disease risk assessment and management of periodontal diseases and condition.

Introduction

The tissues of the periodontium comprise of the tooth's periodontal ligament with attachment to the cementum and alveolar bone and the overlying gingiva. The primary function of the gingiva, and in particular the gingival attachment, is to provide a protective environment for the underlying periodontal attachment and alveolar bone support. Clinical health is described as an absence (or significantly reduced) periodontal inflammation in either intact or reduced periodontium. Periodontal health should be considered from a preventive starting point and a therapeutic endpoint.

The most common conditions of the periodontium are chronic inflammatory diseases of bacterial aetiology related to oral biofilm formation on the tooth surface, and resulting in either reversible or irreversible changes to the tissues of the periodontium. Host determinants have an important role in susceptibility to disease. The clinical presentation, prognosis and response to treatment may be influenced by local predisposing factors (periodontal pockets, restorations, root anatomy and crowding) and systemic modifying factors (host immune function, systemic health, genetics). Environmental determinants (smoking, medications, stress, nutrition) also play a role.

Periodontal health is a fundamental component of health and is essential for physical, social and psychological wellbeing. Periodontal health and stability are important components of integrated oral health care and treatment.

The overarching principles for this Clinical Practice Guideline:

- a) Most diseases and conditions of the periodontium are associated with oral biofilm formation and are therefore preventable
- b) Periodontal screening and assessment are an essential part of an oral health assessment for all dentate clients.
- c) Periodontal risk assessment, diagnosis and prognosis should be considered as fundamental components of a complete Periodontal Management Plan.
- d) The management of most diseases and conditions of the periodontium should incorporate principles of chronic disease management including; prevention; education; self-management; early intervention/proactive care; co-ordinated treatment; and, long-term care (maintenance).

This clinical guideline is divided into 3 sections

SECTION 1 - Periodontal Screening and Assessment in cases of comprehensive care

SECTION 2 – Management of Periodontal diseases

SECTION 3 - Assessment and management of Acute Periodontal Conditions

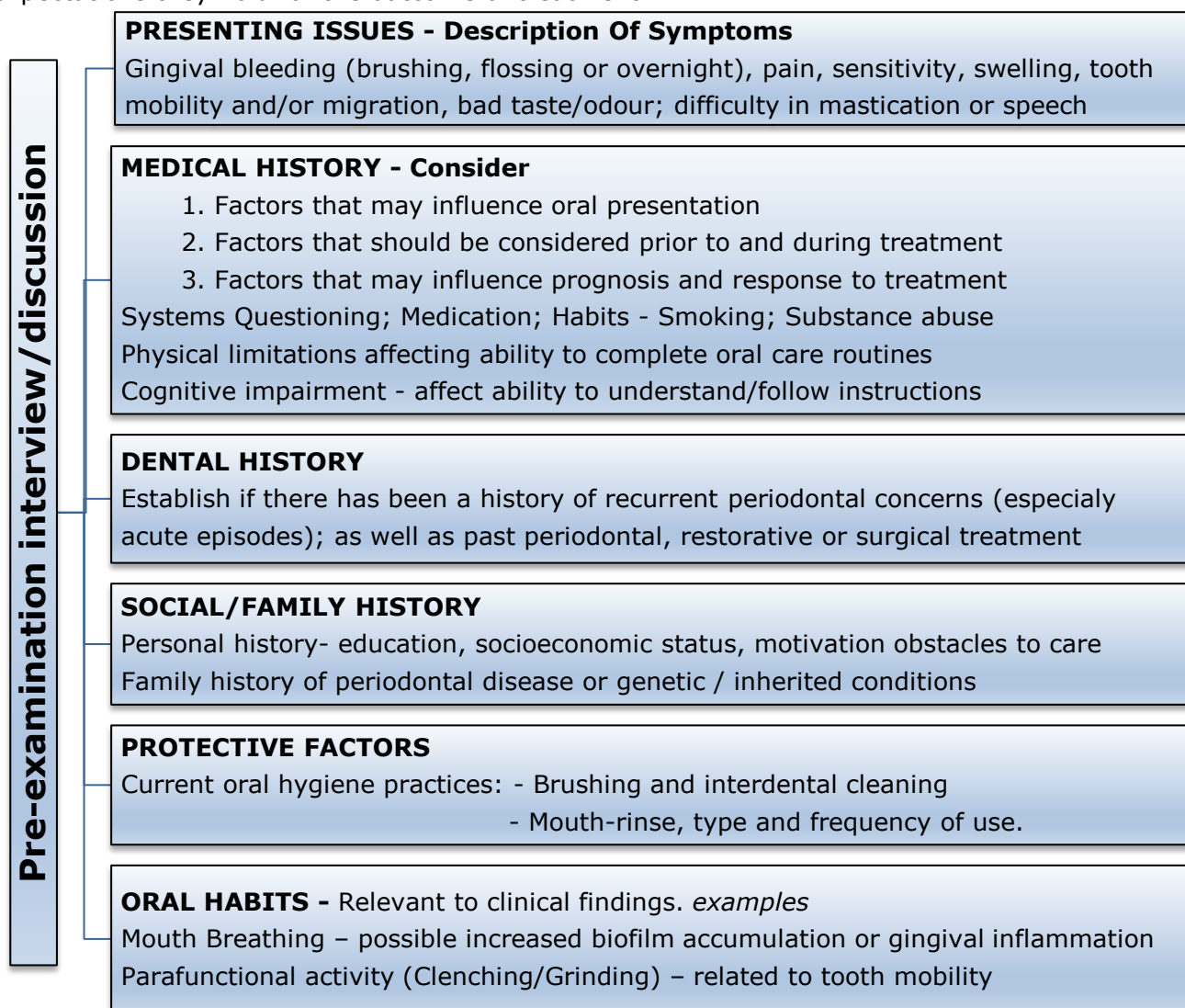
Section 1 - Periodontal Screening and Assessment in cases of comprehensive care

The initiation and progression of periodontal diseases is complex. The management will need to consider the impact of local and systemic predisposing and modifying factors as well as the clients understanding and motivation to achieve agreed outcomes.

There are a number of risk factors for periodontal diseases (both local and systemic) and the information collected during the history taking and clinical/radiographic assessment will assist to understand the impact of these risk factors in determining the relative importance, and the prognosis and outcomes to treatment.

1. Pre-examination interview/discussion (History Taking):

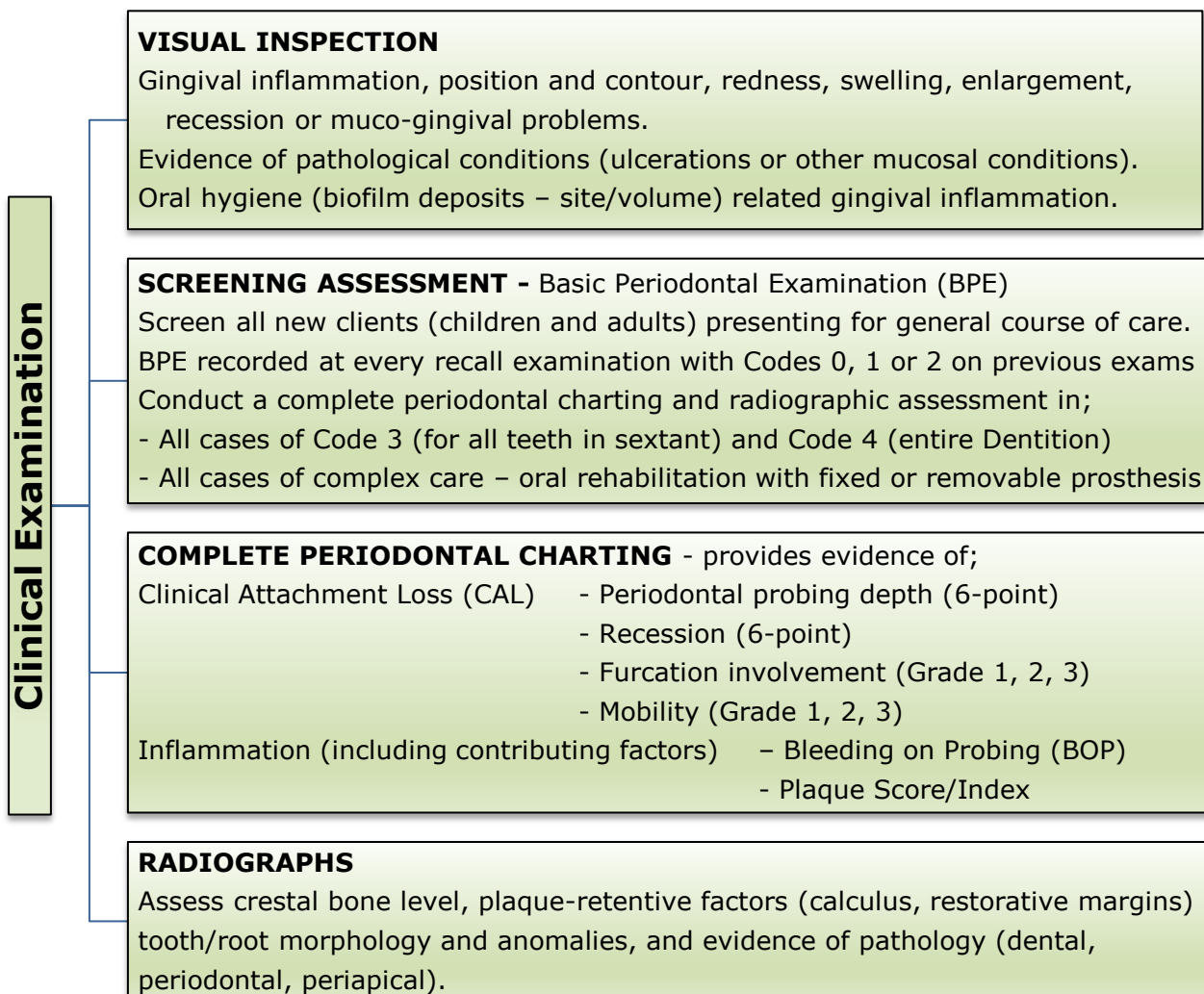
History taking allows for an opportunity for the client to provide information regarding their experience and understanding of their condition, their general health and habits, as well as the expectations they hold for the outcome of treatment.



The Clinician Team should be mindful of features that identify concerns regarding the clients understanding, behaviour, motivation and increased disease risk potential.

2. Clinical Examination

The elements of a clinical examination of the periodontal tissues include assessment of gingival appearance and periodontal attachment level (screening assessment or complete periodontal charting). A comprehensive clinical examination should also assess the local factors that contribute to biofilm accumulation, interfere with adequate oral hygiene or contribute to damage to the periodontal tissues.



Basic Periodontal Examination (BPE) Assessment¹	
Adults	Children (<18 years of age)
Assess entire dentition divided into sextants (17 to 14), (13 to 23), (24 to 27) (47 to 44), (43 to 33), (34 to 37)	Assesses index teeth (16, 11, 26, 36, 31, 46)
All teeth in each sextant (with at least 2 teeth) are examined (exception of 8's unless 6's and/or 7's missing).	<ul style="list-style-type: none"> • 7 to 11 year-olds - (mixed dentition phase) BPE codes 0 - 2 are used • 12 to 17 year-olds – (permanent dentition) BPE codes 0, 1, 2, 3, 4 and * can be used
The probe is "walked around" the sulcus/pockets in each sextant, and the highest score recorded for each sextant	The probe is "walked around" the sulcus/pockets in each index tooth, and the highest score recorded for each tooth

Note: The Titanium Periodontal Screening pop-up screen is titled *CPI Entry*. This screen can be used for BPE Assessment except the * symbol cannot be used. X should be entered in sextants where no teeth are present.

BPE Score	Descriptor	Interpretation & Recommended action
0	Probing depth <3.5mm, no calculus/overhangs, no BOP	No need for periodontal treatment
1	Probing depth <3.5mm, no calculus/overhangs, BOP	Self-care advice (oral hygiene information and risk factor control)
2	Probing depth <3.5mm, supra or subgingival calculus/overhangs	<ul style="list-style-type: none"> As for Code 1 plus removal of plaque retentive factors, including supra and subgingival calculus
3	Probing depth 3.5--5.5mm	<ul style="list-style-type: none"> Complete Periodontal Charting¹ of all teeth in Code 3 Sextant As for Code 2 plus Periodontal Debridement as required
4	Probing depth >5.5mm	<ul style="list-style-type: none"> Complete Periodontal Charting of entire dentition As for Code 3 plus Assess need for complex care; specialist referral may be indicated
*	Furcation involvement	<ul style="list-style-type: none"> As per BPE Code (0-4). Assess need for more complex care; specialist referral may be indicated

Note: - a screening assessment (BPE) should not be used for diagnosis nor replace a complete periodontal examination. It is inadequate to monitor the response to periodontal therapy or to monitor the periodontal condition for clients in Maintenance/ Supportive Periodontal Therapy.

Note: The overall Clinical Assessment may identify features that could contribute to the initiation/progression of disease or will influence the provision and outcome of treatment. These features may include:

- Tooth position and malocclusion - (rotation, tipping, supra-eruption)
- Food impaction
- Inadequate restoration
- Traumatic occlusal contact or function
- Fixed and removable prostheses or orthodontic appliances.

Additional Tests

Additional clinical investigations may be required to assist in the formulation of a definitive diagnosis and/or the clarification of restorative, endodontic and/or periodontal prognosis.

Assessment of Implants

The disease of the peri-implant hard and soft tissues can occur in a similar way to the process around natural teeth. However, the relationship between bone/soft tissue and implant are different with the soft tissue around implants is less resistant to probing. The probing depth measurements around implants may be influenced by implant position (depth and angulation) and tissue health. Screening assessment (BPE) is not appropriate for the assessment of implants. Complete probing (four or six points) and the presence of any bleeding or suppuration should be recorded around each implant. Summary of features of peri-implant health and disease².

Subcategory	Defining Features
Peri-implant health	Absence of erythema, BOP, swelling and suppuration No increase in PPD compared to previous examinations. No crestal bone loss (beyond changes resulting from initial bone remodelling)
Peri-implant mucositis	Erythema, BOP, swelling and/or suppuration No crestal bone loss (beyond changes resulting from initial bone remodelling)
Peri-implantitis	BOP, swelling and/or suppuration. Increased PPD compared to previous examinations. Bone loss (beyond changes resulting from initial bone remodelling) If no previous examination data diagnosis based on: Presence of BOP and/or suppuration. Probing depths of ≥ 6 mm. Bone levels ≥ 3 mm apical to most coronal portion of intraosseous part of implant

3. **Diagnosis and Classification**

The Pre-examination interview, clinical and radiographic assessment and additional investigations provide information to establish a periodontal diagnosis and classification.

The decision-making process should consider

1. Case categorization of the periodontal condition
 - a. Clinical Gingival Health Intact or Reduced Periodontium
 - b. Gingival Disease – Plaque or Non-dental plaque biofilm-induced
 - c. Periodontitis
2. Specific type (Subcategory) of Gingival Disease or Periodontitis, and
3. Description of the clinical presentation and other elements that affect clinical management, prognosis, and potential influences on both oral and systemic health in relation to
 - a. The underlying issues associated with
 - i. Non-dental plaque biofilm-induced gingival diseases
 - ii. Necrotizing Periodontitis^e
 - iii. Periodontitis – as a direct manifestation of systemic disease
 - b. Periodontitis Staging (disease severity and management complexity) and Grading (biological features, rate of progression; risk assessment expected outcomes)

Category		Subcategory	Defining Features
Clinical Gingival Health ^a		Intact periodontium	<u>Absence of</u> - Signs (BOP [#] , erythema, oedema); Patient symptoms; Clinical attachment & bone loss
		Reduced Periodontium	
		Non-periodontitis case ^b	<u>Absence of</u> - Signs (BOP, erythema, oedema) & Patient symptoms
		History of periodontitis - Stable	<u>Presence of</u> - reduced clinical attachment & bone levels
Gingival Diseases	Dental plaque-induced Gingivitis	Intact periodontium	<u>Presence of Signs of inflammation</u> - Redness; Swelling; BOP; Discomfort on gentle probing <i>(Extent - mild <10%; moderate 10-30%; severe >30% sites)</i> <u>Possible Symptoms</u> - Bleeding gums; metallic/alterd taste; Pain; Halitosis; Difficulty eating; red swollen appearance (gums); Reduced oral health-related quality of life May present - Associated with Biofilm alone - Mediated by systemic or local risk factors - Drug-influenced gingival enlargement
		Reduced periodontium	
		Non-periodontitis case ^b	
		Reduced periodontium	
		History of periodontitis – Stable ^c	
	Non-dental plaque biofilm-induced gingival diseases ^d	Genetic/developmental disorders	Severity of clinical manifestations often depends on plaque accumulation & subsequent gingival inflammation <i>Note - other health-care providers may be involved in diagnosis and treatment.</i>
		Specific Infections	
		Neoplasms	
		Reactive processes	
		Endocrine, nutritional & metabolic diseases	
		Inflammatory/immune conditions	
		Traumatic lesions	
		Gingival Pigmentation	
Periodontitis	Necrotizing Periodontitis ^e	1. Interdental CAL [±] detected at ≥2 non-adjacent teeth ^g , or 2. Buccal/oral CAL ≥3mm with PPD [°] ≥3 mm detected at ≥2 teeth ^f	
	Periodontitis - manifestation of systemic disease ^f		
	Periodontitis <i>All remaining clinical cases not categorized above.</i>		

[#] BOP = Bleeding on Probing [±] CAL = Clinical Attachment Loss [°] PPD = Periodontal Probing Depth

^a May be evidence of gingival inflammation BOP <10% (localized mild & delayed BOP at isolated sites)

^b Recession or following crown lengthening procedure

^c Recurrent Periodontitis cannot be ruled out

^d Refer to Appendix A

^e See Section 3

^f Rare conditions and diseases - Follow the classification of the primary systemic disease according to respective International Statistical Classification of Diseases and Related Health Problems (ICD) codes. Refer to Appendix B

^g CAL not ascribed to non-periodontitis-related causes; 1) gingival recession of traumatic origin; 2) dental caries extended to the cervical area of the tooth; 3) CAL on distal of 7's associated with an 8 malposition or extraction, 4) endodontic lesion draining through the marginal periodontium; and 5) the occurrence of a vertical root fracture.

Derived from Ref. 3, 4,5

Periodontitis Stage			Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL		1-2mm	2-3mm	≥5 mm	≥5 mm
	Bone loss		<15%	15-30%	50-65%	50-65%
	Tooth loss		None due to Periodontitis		≤ 4 teeth	≥ 5 teeth
Complexity	Local	PPD	Max. ≤ 4mm	Max. ≤ 5mm	≥ 6mm	≥ 6mm
		Bone Loss	Mostly Horz.	Mostly Horz.	Vertical ≥ 3 mm	Vertical ≥ 3 mm
		Furcation			Class II or III	Class II or III
		Ridge Defect			Moderate	Severe
		Masticatory dysfunction				Bite Collapse 2° Occlusal Trauma < 10 opposing pairs
Extent	Add Descriptor	Molar/Incisor; Localized (<30% of sites); Generalized				
Periodontitis Grade		Grade	A	B	C	
		Rate of Progression	Slow	Moderate	Rapid	
Primary Criteria	Direct evidence of Progression	Longitudinal data (bone loss or CAL)	No Loss over 5 yrs	<2mm over 5 yrs	≥2mm over 5 yrs	
	Indirect evidence of progression	% bone loss/age	< 0.25	0.25 – 1.0	>1.0	
		Case phenotype	Biofilm > level of Destruction	Biofilm = level of Destruction	Destruction > Biofilm	
Grade Modifiers	Risk Factors	Smoking cigarettes/day	Non-smoker	Smoker <10	Smoker ≥ 10	
		Patient with Diabetes	Normoglycemic	HbA1c<7.0%	HbA1c≥7.0%	

4. Risk Assessment

Establishing the prognosis, proposing management options and formulation of a management plan, requires assessment of relevant risk factors for each particular case (controllable and uncontrollable predisposing and modifying factors). The ability to remove, reduce or compensate for these factors will influence decisions for treatment and the predictability and longevity of treatment outcomes.

- Predisposing factors - any agent/condition that contributes to the accumulation of dental plaque (eg, tooth anatomy, tooth position, restorations).
- Modifying factors - any agent/condition that alters the way in which an individual responds to subgingival plaque accumulation (eg, smoking, systemic conditions, medications)

Risk Factors	Controllable		Uncontrollable	
Local	Acquired	Anatomical	Root morphology - Furcation - Root grooves & concavities	
	Plaque/Calculus Restorative defect Partial denture	Tooth position		
Systemic	Smoking Certain Medications	Diabetes Stress	Genetic factors SES status	Blood dyscrasia Hormonal changes

Both clinicians and clients should be aware of the impact of identified risk factors. Management options should consider the ability to control (eliminate or reduce), where possible, identified risk factors or compensate for where risk factors are uncontrollable.

Application of Risk Assessment

- Health or Gingivitis – In cases of minimal or reversible disease the aim of the periodontal management plan is to recognise the potential risk for disease development or progression. An increased risk may be identified from the symptoms (gingival bleeding, pain/sensitivity, mobility), medical history (diabetes, smoking and certain medications) and clinical findings (signs of inflammation, plaque score/index, plaque retentive sites).
- Periodontitis – irreversible disease has commenced. There are several Periodontal Risk Assessment Tools (PRAT) proposed in the literature. Most of these tools have been applied and evaluated longitudinally at the time of re-evaluation following initial treatment. Lang and Tonetti⁶ proposed and evaluated the PRAT for clients in maintenance (**note the PRAT is applied at re-evaluation following active treatment to assess the risk of recurrence of periodontitis**). The tool identifies 6 domains of risk and attributes criteria that are associated with Low, Medium and High risk of disease progression. It is important to recognise that there is a dynamic interplay between risk factors.

c.

DOMAINS OF RISK	RISK STRATIFICATION		
	LOW	MEDIUM	HIGH
Percentage BOP	<10% of Surfaces		>25% of surfaces
Prevalence of residual pockets > 4 mm	<4 residual pockets		>8 residual pockets
Tooth loss (excl. 8's)	4 teeth lost		8 teeth lost
Bone loss per client's age*	0.25 BL/Age [#]	0.5 BL/Age [#]	1.0 BL/Age [#]
Systemic/genetic condition	None confirmed		Confirmed
Environmental factors Cigarettes/day	Non-smokers (NS) Past smokers (FS) >5yrs	Moderate smokers (MS) 10-19/day	Heavy smokers (HS) > 1 pack/day
* Estimate alveolar bone loss in posterior region on either - periapical radiographs - worst site affected estimated % root length - bitewing radiographs - worst site affected estimated in millimetre – 1mm = 10% bone loss # BL/Age factor - Bone Loss percentage per age. Divide percentage Bone Loss by client age. (Eg: 40-year-old client with 20% bone loss at worst affected posterior site - BL/Age = 0.5)			

A client may be categorised as Risk of recurrence of Periodontitis as

- Low** - all parameters within the low-risk or only one parameter in moderate-risk category
Moderate - ≥ two parameters in moderate category, but at most one in high-risk category
High - at least two parameters in the high-risk category

5. Prognosis and Treatment Planning

Prognosis is broadly considered as the ability to control, through self-management and treatment, the inter-related disease-associated factors to achieve the agreed client and clinical-based outcomes.

The client-based outcomes may relate to comfort, function and/or aesthetics. The clinical outcomes aim to support these client-based outcomes, where possible, by achieving a level of periodontal stability and allow for maintenance. Periodontal Stability is achieved through elimination/reduction of gingival inflammation and regaining or maintaining clinical attachment level. Achieving Periodontal Stability will be influenced by General and Local Factors:

General Factors	Local Factors
Client adherence General level of Plaque Control Smoking Diabetic control Other Systemic Disease/ Condition Genetic Factors Stress	Plaque/calculus deposits Sub-gingival restorations Periodontal Pocket Depth/Clinical Attachment Loss Tooth mobility Anatomical Factors: <ul style="list-style-type: none"> - Furcation Involvement - Short tapered roots - Root proximity - Developmental grooves - Root concavities

Prognosis is not an exact science and there have been a number of prognosis categories described in the literature. Kwok and Caton⁷ have attempted to simplify these categories.

Category	Description (As pre: Kwok & Caton ⁷)
Favourable	Periodontal stability achievable with periodontal treatment & maintenance. Future attachment loss unlikely if conditions met
Questionable	Periodontal status influenced by local and/or systemic factors that may or may not be able to be controlled. Can be stabilised with periodontal treatment & maintenance if these factors are controlled; otherwise, future breakdown may occur
Unfavourable	Periodontal status influenced by local and/or systemic factors that cannot be controlled. Periodontal breakdown likely even with periodontal treatment and maintenance
Hopeless	Treatment not indicated – Extraction considered

NOTE:

- Periodontal Prognosis should be considered a dynamic process - given the history of disease and the influence of local and systemic factors. At the initial assessment the clinician may consider establishing a provisional prognosis. This may be confirmed or refined following the instigation of elements of self-management and treatment.
- The overall prognosis of the tooth or dentition should consider the restorative, endodontic occlusal, as well as the periodontal prognosis. (Samet & Jotkowitz⁸)

Section 2 – Management of Periodontal Diseases

The management of Acute periodontal conditions is addressed in Section 3. Periodontal diseases (and in particular periodontitis) involve a number of inter-related factors.

The periodontal management plan aims to address these factors through a partnership in care between the client and clinician team incorporating behaviour modification and self-management (prevention, education and health literacy); early intervention/proactive care; co-ordinated treatment; and, long-term care (maintenance).

Partnership in Care

In the periodontal management plan both the client and clinician team have a role in reducing the periodontal risk status and achieving and maintaining periodontal stability. Periodontal stability is reliant on the reduction/elimination of inflammation; regaining/maintaining clinical attachment level and reducing the impact of systemic factors that have a negative impact on host response and/or healing.

It is important for the client to understand their responsibility in reducing periodontal risk through behaviour modification and self-management. Key to positive self-management is addressing local factors such as improved oral hygiene, and systemic factors such as smoking cessation and improved medical management (eg. Diabetes management).

The clinician team include the oral health educator; Dental Hygienist/Dental Therapist/ Oral Health Therapist; Dentist and Periodontist. The broader clinician team may also include the client's medical and/or allied health practitioners.

Within the scope of practice the members of the clinician team can support the client in reducing periodontal risk through the various management stages;

i	Education and health literacy	Successful management is reliant on an explanation of the disease process; the role of bacterial biofilm; the association with local and systemic risk factors; and, the scope, impact and limitation of treatment. It is important to recognise the importance of periodontal health and stability on overall oral health and general health.
ii	Behaviour modification	Employ motivational interviewing for altering oral health behaviours. - Oral Hygiene education - Smoking cessation - Improved systemic health management – Diabetes Care
iii	Periodontal debridement	- Removal of supra- and subgingival plaque and calculus - Establish a biocompatible root/tooth surface There is little evidence to suggest that the outcome is influenced by the mode of treatment or scheduling of appointments. The clinician should be mindful of client comfort in length of appointments, the mode of instrumentation and the use of local anaesthetic. - Treatment Mode - hand (scalars/curettes) or ultra-sonic instrument - Scheduling - client-focused and may allow for quadrant; half-mouth or full-mouth instrumentation
iv	Assist in home care	Reduce/eliminate restorative and prosthodontic deficiencies and defects
v	Re-evaluation Reassessment	Review symptoms following treatment and periodontal examination - Assess achievement of client-based and clinical outcomes - Update prognosis and risk assessment Re-evaluation decisions may include - Re-instrumentation of poorly responding/unstable sites - Surgical management for increased access and/or improved gingival adaptation/healing - Maintenance care – Risk-based appointments scheduling - Low Risk - 12-24 months - Medium Risk- 6-9 monthly - High Risk - 2-3 monthly

Case Management and Referral

In most circumstances, periodontal care can be successfully provided by the clinician team in the General Practice setting. The ability to provide the appropriate level of care will be dependent on;

- i. The general practice profile, the training and experience of the members of the clinician team
- ii. Client engagement, medical history and periodontal risk factors
- iii. Periodontitis Case – Stage and Grade.

In most cases, irrespective of case Stage and Grade the initial periodontal management including oral hygiene education/instruction and periodontal debridement should be completed in the general practice setting. Smoking cessation referral (to QUIT) or counselling should also be initiated by the general practice clinician team.

Due to the findings on initial examination or the poor clinical outcomes following treatment a referral to a medical or allied health practitioner may be indicated in cases of suspected or known medical modifying factors.

Referral to a Periodontist may be indicated in cases of:

- Severe disease and/or complex treatment requirements
- Client's desire for a specialist opinion or treatment
- Other complicating factors such as a client's medical history or other co-morbidity

Following the completion of active treatment in the specialist Periodontal setting, the general practice clinician team should be able to address the maintenance care requirements at appropriate time intervals based on risk assessment.

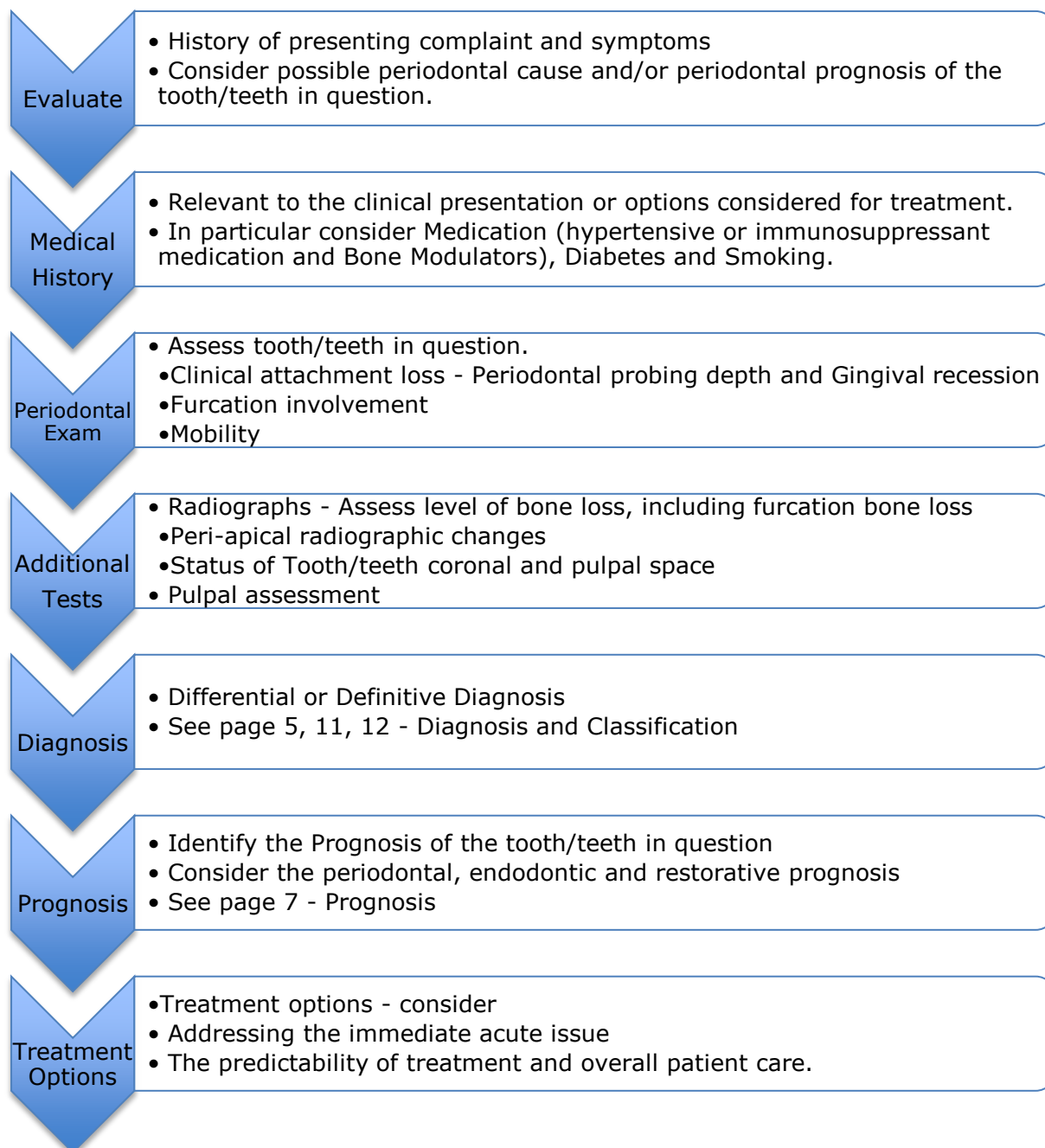
The table below provides a guide for management within General Practice of referral. Referral may be to a Periodontist, an Oral Medicine specialist, Oral and Maxillofacial Surgeon and/or a relevant medical practitioner.

Case Diagnosis and Classification	Manage by
Dental plaque-induced Gingivitis	
Associated with Biofilm alone	General Practice
Mediated by systemic or local risk factors	General Practice
Drug-influenced gingival enlargement	Referral
Non-dental plaque biofilm-induced gingival diseases	
Genetic/developmental disorders	General Practice
Specific Infections	General Practice
Neoplasms	Referral
Reactive processes	General Practice
Endocrine, nutritional & metabolic diseases	Referral
Inflammatory/immune conditions	Referral
Traumatic lesions	General Practice
Gingival Pigmentation	General Practice
Necrotizing Periodontitis	Referral
Periodontitis - manifestation of systemic disease	Referral
Periodontitis	
Stage 1, 2, 3	General Practice
Stage 3 (non-responding) & Stage 4	Referral
Grade A & B	General Practice
Grade B (non-responding) & Grade C	Referral
Peri-implantitis	Referral
Modifying Factors Relevant to Periodontal Treatment that may be considered for referral: <ul style="list-style-type: none"> - Co-ordinated medical or dental multi-disciplinary care - Medical history that significantly affects clinical management <ul style="list-style-type: none"> ▪ history of head / neck radiotherapy or intravenous bisphosphonate therapy ▪ significantly immunocompromised or immunosuppressed ▪ significant bleeding dyscrasia / disorder ▪ potential drug interaction 	

SECTION 3 - Assessment and management of Acute Periodontal Conditions

Acute periodontal conditions are rapid-onset clinical conditions that involve the periodontium or associated structures. They may be characterised by pain/discomfort, tissue destruction, swelling and tooth mobility. They may or may not be related to gingivitis or periodontitis. They may be localized or generalized, with possible systemic manifestations. However, these symptoms may also be associated with pathology involving the pulp and/or peri-apical tissues.

Alternatively, in cases where symptoms relate to dental caries, pulpal and/or peri-apical pathology, the periodontal prognosis of the tooth/teeth should be considered when outlining treatment options and formulating a treatment plan.



more common are;

1. Periodontal abscess, and
2. Necrotising periodontal diseases – Necrotising gingivitis and Necrotising periodontitis
3. Primary herpetic gingivostomatitis – Non-dental plaque-induced Gingival Disease – viral origin

The

1. Periodontal Abscess

Classification³

Periodontitis Case	Acute exacerbation	Untreated Periodontitis	
		Non-responsive to therapy	
		Supportive Periodontal Therapy	
	After Treatment	Post-scaling	
		Post-surgery	
		Post-medication	Systemic antimicrobials
			Other medication: nifedipine
Non-periodontitis Case	Impaction		Dental floss, orthodontic elastics, toothpicks
	Harmful habits		Wire/nail biting & clenching
	Orthodontic forces		Orthodontic forces or cross-bite
	Gingival overgrowth		
	Alteration of root surface	Severe anatomical alteration	<i>Dens evaginatus</i> or odontodysplasia
		Minor anatomical alteration	Developmental groove; Cemental tear
		Iatrogenic conditions	Perforations
		Severe root damage	Fissure or fracture cracked tooth syndrome
		External root resorption	

Abscesses of the Periodontium - Periodontal Abscess

Clinical Features	Predisposing Factors	Treatment
<ol style="list-style-type: none"> 1. Localised fluctuant, painful swelling of gingiva or mucosa 2. Involved/adjacent teeth are tender to percussion 3. Tooth mobile and/or high in occlusion 4. Possible lymphadenopathy and fever 5. Possible diffuse facial or neck cellulitis (Rare) 6. Possible radiographic evidence of defect. 	<p><u>Local factors</u></p> <ol style="list-style-type: none"> a) Preexisting periodontal pocket <ul style="list-style-type: none"> - drainage impaired complex pocket topography or pocket occlusion; infection extends to supporting tissue b) No pre-existing periodontal disease; <ul style="list-style-type: none"> - Root canal perforation or root fracture - Foreign body impaction - Root anomaly – <i>dens evaginatus</i> - External root resorption c) Secondary infection of lateral periodontal cyst. <p><u>Systemic Factors</u></p> <ol style="list-style-type: none"> a) Change in host response (eg. poorly controlled diabetes mellitus) b) Change in microbial virulence (eg. increased resistance with broad spectrum antibiotics) 	<p>Treatment:</p> <ol style="list-style-type: none"> a) Drainage via either: <ul style="list-style-type: none"> - root debridement/irrigation of periodontal pocket - incision and drainage - tooth extraction b) Adjunctive use of systemic antibiotics; only in cases of systemic symptoms or developing cellulitis <ol style="list-style-type: none"> 1. <i>Phenoxymethylpenicillin 500mg (child: 10mg/kg to 500mg) orally; 6-hourly for 5 days OR</i> 2. <i>Amoxycillin 500mg (child: 10mg/kg to 500mg) orally 8-hourly for 5 days OR</i> 3. <i>Clindamycin 300mg (child: 7.5mg/kg up to 300mg) orally 8-hourly for 5 days</i> <p>(refer to Therapeutic Guidelines⁹)</p>

2. Necrotising Periodontal Diseases

Classification³

Category	Client	Predisposing conditions	Clinical condition
Chronically, severely compromised clients	In adults	HIV+ - detectable viral load & CD4 count < 200	NG, NP, NS, Noma Possible progression
		Severe systemic conditions (immunosuppression)	
	In children	Severe malnourishment	
		Extreme living conditions	
Temporarily and/or moderately compromised clients	In gingivitis case	Severe (viral) infections	Generalized NG Possible progression to NP
		Uncontrolled factors: stress, nutrition, smoking, habits	
		Previous NPD: residual craters	Localized NG Possible progression to NP NG Infrequent progression
		Local factors; root proximity, tooth malposition	
	In periodontitis case	Common predisposing factors	NP Infrequent progression

NG – Necrotising Gingivitis; NP – Necrotising Periodontitis; NS – Necrotising Stomatitis

Necrotising Periodontal Diseases - Necrotising Gingivitis		
Clinical Features	Predisposing Factors	Treatment
<p>Acute, sudden onset, rapidly destructive, may be recurrent.</p> <p>a) Marginal necrosis</p> <ul style="list-style-type: none"> - often limited to gingiva, - necrotic tip of papillae, - may form necrotic area <p>b) Pseudomembrane</p> <p>c) Linear erythema</p> <p>d) Bleeding - to light touch</p> <p>e) Pain</p> <p>f) Halitosis</p> <p>g) Lymphadenitis</p> <p>h) Fever and Malaise</p>	<p>1) Local Factors</p> <p>A. Plaque accumulation</p> <ul style="list-style-type: none"> - Poor oral hygiene - Restorative overhangs - Food impaction - Malposition of teeth - Calculus <p>B. Tissue ischaemia</p> <ul style="list-style-type: none"> - Cigarette smoking <p>2) Systemic Factors:</p> <p>a) Systemic Diseases</p> <p>b) Nutritional Deficiencies</p> <p>c) Emotional Stress</p> <p>d) Hormonal Imbalance</p>	<p>a) Remove bacteria and local factors. Debridement with local anaesthetic</p> <p>b) Anti-microbial rinse to complement plaque control during healing.</p> <p>c) Adjunctive use of systemic antibiotics indicated in cases of systemic symptoms of infection</p> <p><i>1. Metronidazole 400mg (child: 10mg/kg to 400mg) orally; 12-hourly for 5 days PLUS</i></p> <p><i>2. Chlorhexidine 0.2% mouthwash, 10mL rinsed in the mouth for 1 minute, 8- to 12-hourly OR</i></p> <p><i>3. Chlorhexidine 0.12% mouthwash, 15mL rinsed in the mouth for 1 minute, 8- to 12-hourly</i></p> <p>(refer to Therapeutic Guidelines⁹)</p>

3. Primary herpetic gingivostomatitis

Primary herpetic gingivostomatitis (PHG)		
Aetiology	Herpes simplex virus types 1 and 2	
Clinical Features	Treatment	
<ul style="list-style-type: none"> • Common in children 18mths - 4 yrs (most cases present subclinically) • Incubation period 1-26 d (mean 7 d) - prodromal fever and malaise. • Rapid onset, fever, malaise, painful cervical lymphadenopathy, and severe oral pain inhibiting eating and drinking (risk of dehydration) • Formation of numerous small vesicles in mouth and sometimes on skin. Burst to form multiple yellowish grey ulcers, irregular lobulated margins. Gingiva beefy-red and swollen. • Highly contagious. Infection is self-limiting (lasts ~ 14 days). • Severe cases; stomatitis/pharyngitis, multiple oral ulcers and often fever & lymphadenopathy. 	<p>Supportive therapy</p> <ul style="list-style-type: none"> - maintain fluids, - antipyretics, - analgesics 	

APPENDIX A – Gingival Diseases²

Gingivitis – dental plaque induced	
A. Associated with biofilm alone	
B. Modified by systemic or local risk factors	
i. <u>Systemic risk factors (Modifying factors)</u> Smoking Hyperglycemia Nutritional factors Pharmacological agents - prescription, non-prescription & recreational Sex steroid hormones - Puberty - Menstrual cycle - Pregnancy - Oral contraceptives Haematological conditions	ii. <u>Local risk Factors (Predisposing Factors)</u> Dental plaque biofilm retention Oral dryness
C. Drug-influenced gingival enlargement	
Gingival Disease – non-dental plaque-induced	
A. Genetic/developmental disorders Hereditary gingival fibromatosis ^a	B. Endocrine, nutritional & metabolic diseases Vitamin deficiency ^a (Vitamin C deficiency)
C. Specific Infections Bacterial Origin - <i>Neisseria gonorrhoeae</i> ^a - <i>Treponema pallidum</i> ^a - <i>Mycobacterium tuberculosis</i> ^a - Streptococcal gingivitis Viral origin - Coxsackie virus ^a - Herpes simplex I & II ^a - Varicella Zoster ^a - Molluscum contagiosum - Human Papilloma Virus Fungal Origin - Candidosis - Other mycoses (histoplasmosis, aspergillosis)	D. Inflammatory and immune conditions Hypersensitivity - Contact Allergy ^a - Plasma cell gingivitis ^a - Erythema multiforme ^a Autoimmune diseases of skin and mucous membranes - Pemphigus vulgaris ^a - Pemphigoid ^a - Lichen Planus ^a - Lupus erythematosus ^a (Systemic & Discoid) Granulomatous inflammatory lesions (oro-facial granulomatosis) - Crohn's disease ^a - Sarcoidosis ^a
E. Neoplasms Premalignancy - Leukoplakia - Erythroplakia Malignancy - Squamous cell carcinoma ^a - Leukemic cell infiltration ^a - Lymphoma ^a (Hodgkin & Non-Hodgkin)	F. Traumatic lesions Mechanical trauma - Frictional keratosis - Mechanically induced gingival ulceration - Factitious injury (self-harm) Chemical (toxic) burn Thermal insults (burn)
G. Reactive processes Epulides - Fibrous epulis - Calcifying fibroblastic granuloma - Vascular epulis (pyogenic granuloma) - Peripheral giant cell granuloma	H. Gingival Pigmentation - Melanoplakia ^a - Smoker's melanosis - Drug-induced pigmentation (anti-malarials; minocycline) - Amalgam tattoo

^a other health-care providers may be involved in diagnosis and treatment.

APPENDIX B – Systemic diseases/conditions affecting periodontal tissues⁴

1. Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation	
1.1. Genetic disorders	ICD-10 code
1.1.1 Diseases associated with immunologic disorders	
Down syndrome	Q90.9
Leukocyte adhesion deficiency syndromes	D72.0
Papillon-Lefèvre syndrome	Q82.8
Haim-Munk syndrome	Q82.8
Chediak-Higashi syndrome	E70.3
<u>Severe neutropenia</u>	
Congenital neutropenia (Kostmann syndrome)	D70.0
Cyclic neutropenia	D70.4
<u>Primary immunodeficiency diseases</u>	
Chronic granulomatous disease	D71.0
Hyperimmunoglobulin E syndromes	D82.9
Cohen syndrome	Q87.8
1.1.2. Diseases affecting the oral mucosa and gingival tissue	
<u>Epidermolysis bullosa</u> – Dystrophic epidermolysis bullosa	Q81.2
– Kindler syndrome	Q81.8
Plasminogen deficiency	D68.2
1.1.3. Diseases affecting the connective tissues	
Ehlers-Danlos syndromes (types IV, VIII)	Q79.6
Angioedema (C1-inhibitor deficiency)	D84.1
Systemic lupus erythematosus	M32.9
1.1.4. Metabolic and endocrine disorders	
Glycogen storage disease	E74.0
Gaucher disease	E75.2
Hypophosphatasia	E83.30
Hypophosphatemic rickets	E83.31
Hajdu-Cheney syndrome	Q78.8
1.2. Acquired immunodeficiency diseases	
Acquired neutropenia	D70.9
HIV infection	B24
1.3. Inflammatory diseases	
Epidermolysis bullosa acquisita	L12.3
Inflammatory bowel disease	K50 K51.9 K52.9

2. Other systemic disorders that influence the pathogenesis of periodontal diseases	
	ICD-10 code
Diabetes mellitus (type 1)	E10
(type 2)	E11
Obesity	E66.9
Osteoporosis	M81.9
Arthritis (rheumatoid arthritis, osteoarthritis)	M05 M06 M15- M19
Emotional stress and depression	F32.9
Smoking (nicotine dependence)	F17
Medications	

3. Systemic disorders that can result in loss of periodontal tissues independent of periodontitis	
	ICD-10 code
3.1. Neoplasms	
<u>Primary neoplastic diseases of the periodontal tissues</u>	
– Oral squamous cell carcinoma	C03.0 – 1
– Odontogenic tumors	D48.0
– Other primary neoplasms of the periodontal tissues	C41.0
Secondary metastatic neoplasms of the periodontal tissues	C06.8
3.2. Other disorders that may affect the periodontal tissues	
Granulomatosis with polyangiitis	M31.3
Langerhans cell histiocytosis	C96.6
Giant cell granulomas	K10.1
Hyperparathyroidism	E21.0
Systemic sclerosis (scleroderma)	M34.9
Vanishing bone disease (Gorham-Stout syndrome)	M89.5

REFERENCES

1. British Society of Periodontology. The Good Practitioner's Guide to Periodontology. Revised March 2016, 3rd version
www.bsperio.org.uk/publications/good_practitioners_guide_2016.pdf?v=3
2. Berglundh T *et al* : Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S286–S291.
3. Chapple ILC. *et al*: Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S68–S77.
4. Papapanou PN *et al*: Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri- Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S162–S170.
5. Jepsen S *et al*: Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S219–S229.
6. Lang NP & Tonetti MS: Periodontal Risk Assessment (PRA) for Patients in Supportive Periodontal Therapy (SPT) *Oral Health & Preventive Dentistry* 1/2003, S. 7-16.
7. Kwok V & Caton JG: Prognosis Revisited: A System for Assigning Periodontal Prognosis. *J Periodontol* 2007;78:2063-2071.
8. Samet & Jotkowitz⁶, Classification and prognosis evaluation of individual teeth—A comprehensive approach. *Quintessence Int* 2009; 40:377–387
9. Therapeutic Guidelines : oral and dental. Melbourne : Therapeutic Guidelines, 2012



Clinical Guidelines

Answer the following questions about what you have just read.
1 Scientific CPD point is available on completion.

QUESTIONNAIRE

Your Name:

Email Address:

Title of Clinical Guideline:

Question 1

List 3 key issues this Clinical Guideline reinforced for you?

Question 2

Were there areas of the Clinical Guideline you were previously unaware of? If yes, please list them.

Question 3

How will you share this information with your peers?

Click the button below to submit your answer for verification or email the completed pdf to CPD.Application@dhsv.org.au