**Table 1a.** Classification of periodontitis based on **Stages** defined by Severity (according to the level of interdental clinical attachment loss, radiographic bone loss and tooth loss), Complexity and Extent and Distribution.

Periodontitis stage		Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss	1-2 mm	3-4 mm	≥ 5 mm	≥ 8 mm
	Radiographic bone loss	Coronal third (< 15%)	Coronal third (15-33%)	Extending to middle third of the root	Extending to apical third of the root
	Tooth loss	No tooth loss due to periodontitis		Tooth loss due to periodontitis	Tooth loss due to periodontitis
				of ≤ 4 teeth	of ≥ 5 teeth
Complexity	Local	Maximum	Maximum	In addition to Stage II Complexity:	In addition to Stage III Complexity:
		probing depth ≤4 mm	probing depth ≤5 mm	Probing depth 6-7 mm	Probing depth ≥8 mm
		Mostly horizontal bone loss	Mostly horizontal bone loss	Vertical bone loss $\geq$ 3 mm	Need for complex rehabilitation due to: Masticatory dysfunction; Secondary
				Furcation involvement Class II or III	occlusal trauma; (Tooth mobility degree $\geq$ 2); Bite collapse, drifting,
				Moderate ridge defect	flaring; Less than 20 remaining teeth
					(10 opposing pairs); Severe ridge defect
Extent & distribution	Add to Stage as	For each Stage, describe extent as localized (<30% of teeth involved), generalized or molar incisor pattern			

descriptor	

The initial Stage should be determined using CAL; if not available then RBL should be used. Information on tooth loss that can be attributed primarily to periodontitis – if available – may modify stage definition. This is the case even in the absence of complexity factors. Complexity factors may shift the Stage to a higher level, for example furcation II or III would shift to either Stage III or IV irrespective of the CAL. The distinction between Stage III and Stage IV is primarily based on complexity factors. For example, a high level of tooth mobility and/or posterior bite collapse would indicate a Stage IV diagnosis. For any given case only some, not all, complexity factors may be present, however, in general it only takes 1 complexity factor to shift the diagnosis to a higher Stage. It should be emphasized that these case definitions are guidelines that should be applied using sound clinical judgment to arrive at the most appropriate clinical diagnosis.

For post-treatment patients CAL and RBL are still the primary stage determinants. If a stage shifting complexity factor(s) were eliminated by treatment, the stage should not retrogress to a lower stage since the original stage complexity factor should always be considered in maintenance phase management. Abbreviations: CAL – clinical attachment loss; RBL = radiographic bone loss.

**Table 1b**. Classification of periodontitis based on **Grades** that reflect biologic features of the disease including evidence of, or risk for, rapid progression, anticipated treatment response, and effects on systemic health.

Periodontitis Grade			Grade A	Grade B	Grade C
			Slow rate of progression	Moderate rate of progression	Rapid rate of progression
Primary Criteria	Direct evidence of progression	Longitudinal data (PA radiographs or CAL loss)	Evidence of no loss over 5 years	2 mm over 5 years	≥ 2 mm over 5 years
	Indirect evidence of progression	Bone loss/age	< 0.25	0.25-1.0	> 1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; Specific clinical patterns suggestive of periods of rapid progression and/or Early onset disease e.g. Molar incisor pattern; Lack of expected response to standard bacterial control therapies
Grade modifiers	Risk Factors	Smoking	Non-Smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	Normoglycaemic with or without prior diagnosis of diabetes	HbA1c < 7.0 in diabetes patient	HbA1c ≥ 7.0 in diabetes patient

Grade should be used as an indicator of the rate of periodontitis progression. The primary criteria are either direct or indirect evidence of progression. Whenever available, direct evidence is used; in its absence indirect estimation is made using bone loss as a function of age at the most affected tooth or case presentation (radiographic bone loss expressed as percentage of root length divided by the age of the subject, RBL/age). Clinicians should initially assume Grade B disease and seek specific evidence to shift towards grade A or C, if available. Once grade is established based on evidence of progression, it can be modified based on the presence of risk factors.

## Table 2. Classification of Necrotizing Periodontal Diseases (NPD).

Category	Patients	Predisposing conditions	Clinical condition
		HIV+/AIDS with CD4 counts <200 and detectable viral load	
	In adults	Other severe systemic conditions	NG, NP, NS, Noma. Possible progression
Necrotizing periodontal diseases in		(immunosuppresion)	
chronically, severely compromised			
patients		Severe malnourishments*	
	In children	Extreme living conditions**	
		Severe (viral) infections***	
		Uncontrolled factors: stress, nutrition, smoking, habits	Generalized NG. Possible
		Previous NP: residual craters	progression to NP
	In gingivitic nationts	Local factors: root proximity, tooth malposition	Localized NG. Possible
Necrotizing periodontal diseases in temporarily and/or moderately	In gingivitis patients		progression to NP
compromised patients			NG. Infrequent
		Common predisposing factors for NPD (see paper)	progression
	In periodontitis	NP. Infred	
	patients		progression

\*Mean plasma/serum concentrations of retinol, total ascorbic acid, zinc and albumin markedly reduced, or very marked depletion of plasma retinol, zinc, and ascorbate; and saliva levels of albumin and cortisol, as well as plasma cortisol concentrations, significantly increased.

\*\*Living in substandard accommodation, exposure to debilitating childhood diseases, living in close proximity to livestock, poor oral hygiene, limited access to potable water and poor sanitary disposal of human and animal faecal waste.

\*\*\*Measles, herpes viruses (cytomegalovirus, Epstein-Barr virus-1, herpes simplex virus), chicken pox, malaria, febrile illness.

NG, necrotizing gingivitis; NP, necrotizing periodontitis; NS, necrotizing stomatitis.

 Table 3. Classification of endo-periodontal lesions.

ENDO-PERIODONTAL	Root fracture or cracking
LESION WITH ROOT DAMAGE	Root canal or pulp chamber perforation
DAMAGE	External root resorption

ENDO-PERIODONTAL LESION WITHOUT ROOT DAMAGE	Endo-periodontal lesion in periodontitis patients	Grade 1 – narrow deep periodontal pocket in 1 tooth surfaceGrade 2 – wide deep periodontal pocket in 1 tooth surfaceGrade 3- deep periodontal pockets in more than 1 tooth surfaces	
	Endo-periodontal lesion in non- periodontitis patients	Grade 1 – narrow deep periodontal pocket in 1 tooth surfaceGrade 2 – wide deep periodontal pocket in 1 tooth surfaceGrade 3- deep periodontal pockets in more than 1 tooth surfaces	

**Table 4.** Classification of periodontal abscesses based on the etiologic factors involved.

		•	Untreated periodontitis	
	In periodontitis (at a pre-existing periodontal pocket)		Non-responsive to therapy periodontitis	
			Supportive periodontal therapy	
		After treatment	Post-scaling	
			Post-surgery	
			Post-medication	systemic antimicrobials
De de de stat				other drugs: nifedipine
Periodontal	Non-periodontitis (no pre-existing periodontal pocket)	Impaction		dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls
abscess		Harmful habits		wire or nail biting, clenching
		Orthodontic factors		orthodontic forces or a cross-bite
		Gingival overgrowth		
		Alteration of root surface	Severe anatomic alterations	invaginated tooth, dens evaginatus or odontodysplasia
þ			Minor anatomic alterations	cemental tears, enamel pearls or developmental grooves
			latrogenic conditions	perforations
			Severe root damage	fissure or fracture, cracked tooth syndrome
			External root resorption	