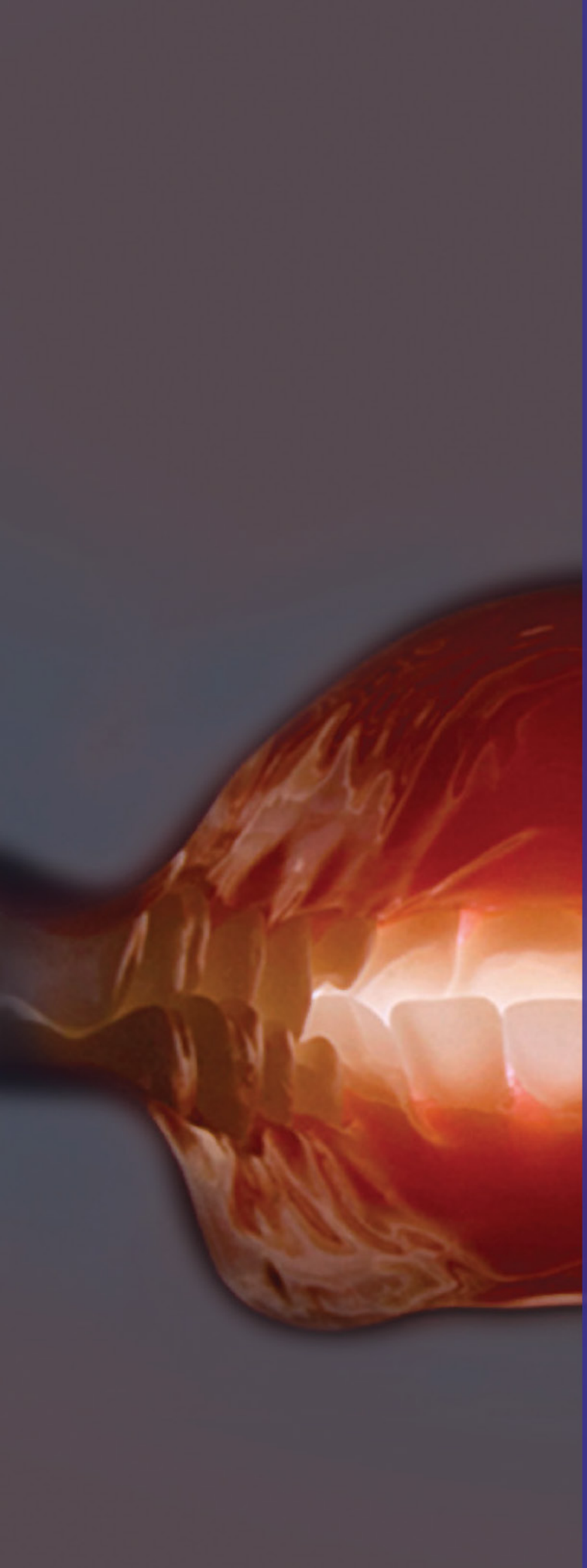

Periodontology at a Glance

Second Edition

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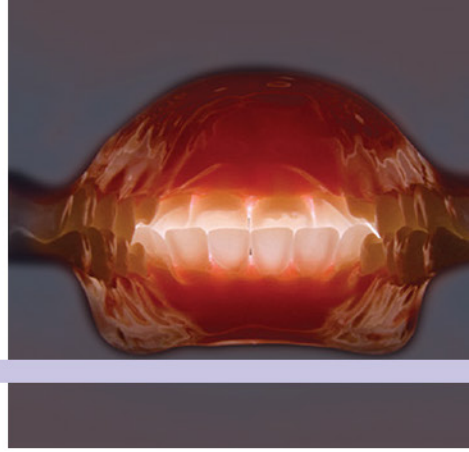

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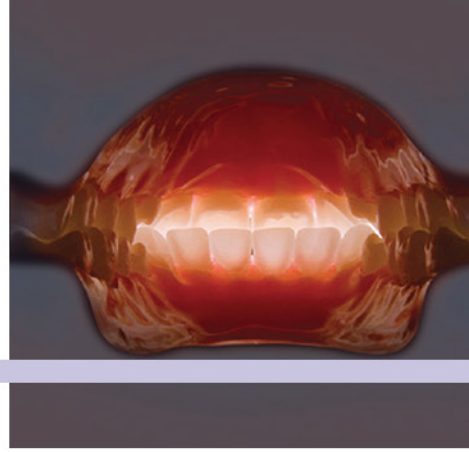
Preface to the second edition



Periodontology, as the specialty of dentistry concerned with diseases of the supporting tissues of the teeth, is an exciting and constantly developing field. Periodontal diseases are widespread. From 1990 to 2019, there was an 8.44% increase in the prevalence rate of severe periodontitis worldwide. In 2022, the World Health Organisation estimated the prevalence of severe periodontitis globally was 19%, with 1.1 billion people affected. As such, any practitioner of dentistry or dental hygiene and therapy will be confronted with patients presenting with periodontal problems on a daily basis. Current research suggests that periodontal diseases are also linked to other general health problems including diabetes mellitus, atherosclerotic cardiovascular diseases, stroke, rheumatoid arthritis, chronic kidney disease, nosocomial respiratory infections, adverse pregnancy outcomes, Alzheimer disease and certain cancers. Thus, periodontal diseases and their management may have effects beyond that of the oral cavity.

In the UK and US, as in other countries, periodontal care is delivered in general dental practice, specialist periodontal practice and the dental hospital setting. Perhaps more than any other area of dentistry at the time of writing, the management of periodontal patients is often achieved by an integrated dental team. The continuing development of the roles of professions complementary to dentistry can only enhance the scope for delivery of effective patient care.

Periodontology at a Glance, 2nd edition, is the latest title in the widely known and popular 'At a Glance' series. After the success of the first edition, we were thrilled and honoured to be approached by Wiley to write this second edition. It is designed to provide a concise and current review of the field of periodontology and peri-implant diseases and conditions and includes the underpinning principles of these subjects and their clinical periodontal applications. It is designed as a study aid and revision guide for students of dentistry, hygiene and therapy. It is also a useful tool for dental practitioners, hygienists and therapists to update their knowledge of this continually developing subject.

While preparing this second edition, we were deeply saddened by the untimely death of our dear friend, colleague and co-editor Professor Robert Genco who was a true inspiration and an international icon in the field of periodontology. However, it is with huge pleasure and delight that we welcome Professor Iain Chapple and Professor Michael Milward as co-editors of this second edition alongside Professor Valerie Clerehugh and Dr Aradhna Tugnait.

In the typical visual 'At a Glance' style, this book uses a two page spread/short chapter format for each topic. Salient information has been distilled from the literature and presented in easy-to-read notes, tables, diagrams and figures. Where teeth are referred to in the text and figures, the following notation is used: UR, upper right quadrant; UL, upper left quadrant; LR, lower right quadrant; and LL, lower left quadrant. The permanent teeth are referred to as '1' (indicating central incisor) to '8' (indicating third molar), to give UR1 as the upper right permanent central incisor and UR8 as the upper right permanent third molar.

The chapters are self-contained and can therefore be read in any order. Cross-referencing will direct the reader to additional relevant chapters in the book. Each chapter ends with a box of key points to present the reader with the essential take-home messages for a particular topic. References and further reading for each chapter are provided in Appendix 5 at the end of the book.

Periodontology at a Glance has been thoroughly updated since the first edition and incorporates the latest 2018 classification findings following the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions, which was convened jointly by the European Federation of Periodontology (EFP) and the American Academy of Periodontology (AAP) to align and update the classification with current understanding of periodontal and peri-implant diseases and conditions. The book also includes the EFP S3-level treatment guideline and the British Society of Periodontology and Implant Dentistry (BSP) implementation of this for Stages I–III periodontitis.

We have divided our second edition into six parts, each starting with a short overview of content and with its own unique colour code, designed to enhance the visual appeal of the book and facilitate the reader's journey through the different topics: Part 1 provides the core *Foundations*; Part 2 covers *Risk and periodontal diseases* – the four chapters on risk originally authored by Bob Genco have kindly been further updated for this edition by Iain Chapple while retaining the essence of Bob's original texts, for which our heartfelt thanks; Part 3 addresses the steps in *Reaching a periodontal diagnosis and treatment plan*; Part 4 focuses on the *Fundamentals of periodontal patient care* relevant to all patients; Part 5 features *Advanced periodontal patient care: periodontal surgery; dental implants; periodontal-orthodontic interface*; and finally, Part 6 includes a diverse range of pertinent periodontal clinical topics covering *Periodontal diseases and periodontal management*.

We have loved writing the second edition of *Periodontology at a Glance* and we truly hope you enjoy using this book.

From the Editors: Professor Valerie Clerehugh, Dr Aradhna Tugnait, Professor Michael R. Milward and Professor Iain L. C. Chapple.

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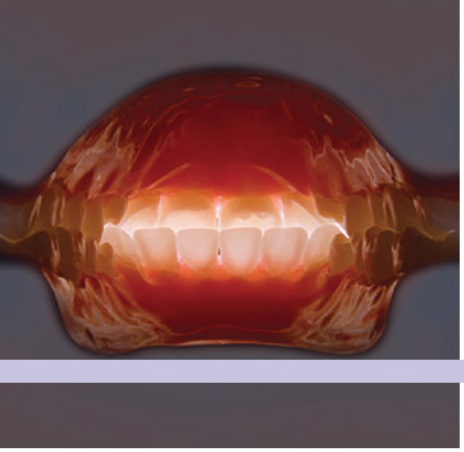

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- Mr Jaymit Patel: Fig. 29.3.
- Dr Bob Turner: Fig. 5.1.
- Dr Pip White: Fig 7.3b.
- Dr Simon Wood: Fig. 4.4.
- Ms Victoria Yorke: Fig. 18.6.

We extend our thanks to the Photography Department at Leeds Dental Institute for their expertise. We thank Quintessence for permission to publish the following images in *Periodontology at a Glance*: Fig. 32.2b, Fig. 32.3, Fig. 32.4, Fig. 32.6, Fig. 32.7, Fig. 32.8 and Fig. 38.6b. We acknowledge with thanks the British Society of Periodontology and Implant Dentistry for permission to use the figures in Appendix 3 and 4.

This book was originally inspired in collaboration with our original dear Senior Commissioning Editor, Caroline Connelly. We are very grateful to our current Managing Editor, Bhavya Boopathi, for her help, support and enthusiasm in bringing the book to fruition, to Monica Rogers, before her, for encouraging us to keep our momentum going with our beloved book, and to the wider Wiley team for their help in getting us to our publication goal. We wish to thank Devipriya Somasundaram for taking care of the Permissions for us and Holly Regan-Jones for her meticulous copyediting skills. It has been a joy to work with Adalfin Jayasingh, our Content Refinement Specialist and to see the printed magic he has created from the materials we have supplied him with. Susan Engelken and her team did a splendid job producing our front cover, and the ongoing support and advice from Fraser Dart in his role as Associate Editor has been very much appreciated. A huge amount of hard work has gone into the preparation and production of the second edition of *Periodontology at a Glance* and we acknowledge with gratitude the dedication and craftsmanship of all involved.

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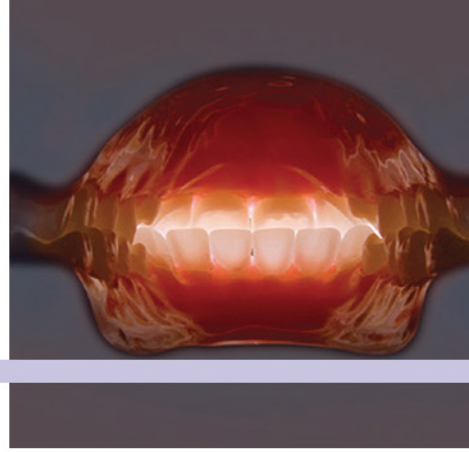
Dedications



This book is dedicated to the memory of the late Bob Genco, who was our dear friend, colleague and co-editor of the first edition of *Periodontology at a Glance*. He was a true global icon who was revered in the world of periodontology and inspired us all. It was an honour to have known him and work with him. We are immensely proud of his contribution to our book and we look back fondly and with gratitude at his words of encouragement, wisdom and guidance throughout our publication journey together. We salute you, Bob Genco.

Professor Val Clerehugh wishes to thank her daughter Mary and son-in-law Adam, her sister and brother Carolyn and John, stepsons Jonathan and Antony, and all her family, for their love and support in the preparation of this book and for their encouragement, humour and cups of tea along the way. And always remembering Tony, Mum and Dad.

Dr Aradhna Tugnait would like to thank her husband Keith, daughter Adella and son Torrin for the magic of family: its support, delight and safe harbour. She would like to dedicate this book to them and to Mum, Dad and Anuja.

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Foundations

Part 1

Chapters

Overview

Part 1 lays the foundations for the book and covers the basic sciences relevant to periodontology. The chapters begin with the anatomy of the periodontium, then a concise schematic update of the 2018 classification of periodontal diseases, based on the 2017 World Workshop Classification of Periodontal and Peri-implant Diseases and Conditions (Chapter 2), followed by key aspects of periodontal epidemiology (Chapter 3). The next two chapters incorporate updates in our understanding of the role of plaque in the aetiology of periodontal diseases and plaque biofilm microbiology, including the principles of symbiosis and dysbiosis and the latest theories underpinning these (Chapters 4, 5), followed by core aspects of calculus (Chapter 6). The final three chapters in Part 1 present thoroughly updated but concise accounts of host defences, the development of periodontal disease and progression of periodontitis (Chapters 7–9).

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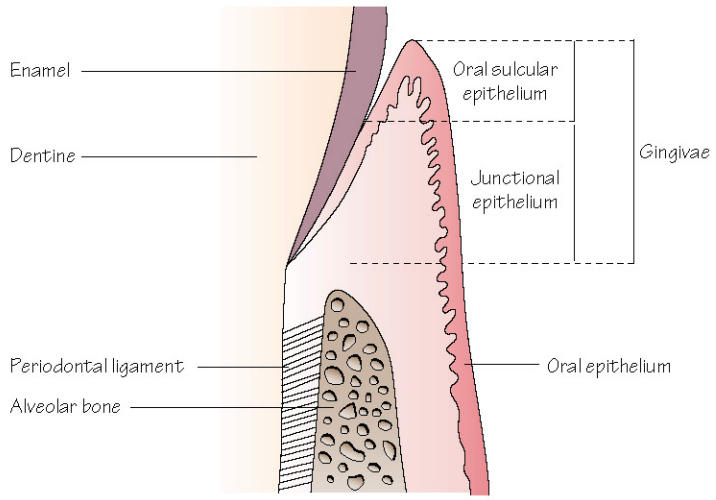
1 Anatomy of the periodontium

Figure 1.1 Longitudinal section through part of a tooth showing healthy periodontal tissues.



The gingiva is made up of the gingival epithelium and connective tissue. The gingival epithelium comprises oral epithelium, oral sulcular epithelium and junctional epithelium.

Figure 1.2 Dentogingival fibres, alveolar crest fibres and circular fibres in the gingival connective tissue.

The four principal gingival fibre groups are:
- Dentogingival fibres
- Alveologingival fibres
- Circular fibres (shown in cross section)
- Transeptal fibres

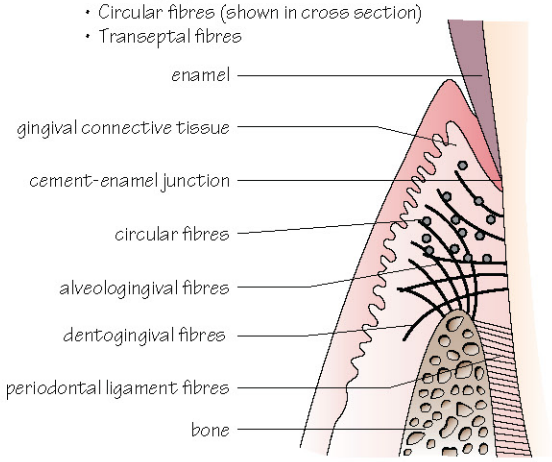


Figure 1.3 Interdental area showing transeptal and circular fibre groups in the gingival connective tissue.

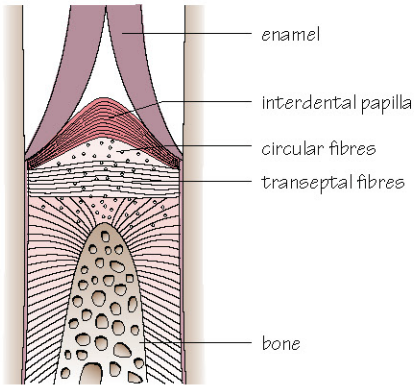
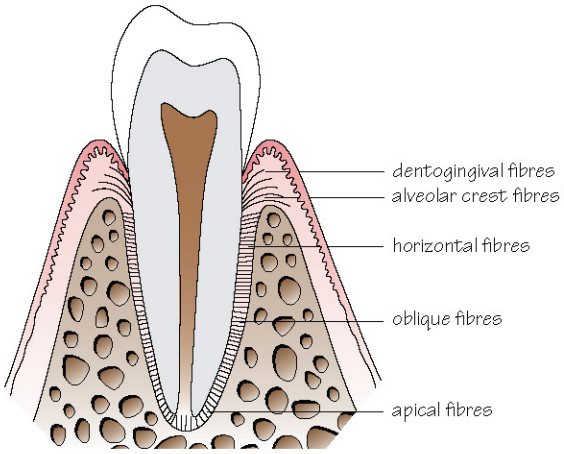
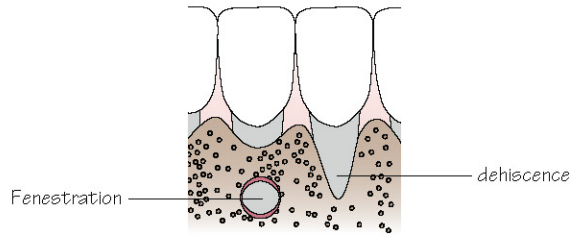


Figure 1.4 The periodontal ligament.



The four periodontal ligament fibre groups are:
- Alveolar crest fibres
- Horizontal fibres
- Oblique fibres
- Apical fibres

Figure 1.5 Bony fenestration and dehiscence.



In most situations these areas of missing bone remain undetected as they are covered with soft tissue. They may be clinically significant if associated with loss of soft tissue resulting in gingival recession (Chapter 32).

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The periodontal tissues form the supporting structures of the teeth. The principal components of the periodontium are shown in Fig. 1.1:

- Gingivae (including epithelium and connective tissue).
- Periodontal ligament.
- Cementum.
- Alveolar bone.

Gingivae

The gingivae in health are pink and firm with a knife-edge appearance, scalloped around the teeth. In certain ethnic groups the gingivae may be pigmented. In health, the gingival margin is a few millimetres coronal to the cement–enamel junction. The gingival sulcus (or crevice) is a shallow groove which may be between 0.5 and 3 mm in depth around a fully erupted tooth. The gingival tissues are keratinised and appear paler pink than sites of non-keratinised oral epithelium.

Gingival epithelium

The gingival epithelium comprises (Fig. 1.1):

- Oral epithelium (OE).
- Oral sulcular epithelium (SE).
- Junctional epithelium (JE).

The gingival sulcus is lined by SE and JE.

Oral epithelium

- The OE is an orthokeratinised, stratified, squamous epithelium.
- Surface cells lose their nuclei and are packed with the protein keratin.
- It presents an impermeable physical barrier to oral bacteria.

The basal layer of epithelial cells is thrown up into folds overlying the supporting connective tissue. These folds increase the surface area of contact between the epithelium and connective tissue and are known as rete ridges or rete pegs.

Oral sulcular epithelium

- There are no rete ridges.
- Cells are keratinised but still have nuclei (parakeratinised).

Junctional epithelium

- The JE forms a specialised attachment to the tooth via:
  - a hemidesmosomal layer within the JE cells;
  - a basal lamina produced by the epithelial cells.
- The JE is non-keratinised and has a very fast turnover of cells (2–6 days compared to 1 month for OE).
- The most apical part of the JE lies at the cement-enamel junction in health.
- The JE at its widest point is 20–30 cells thick coronally.
- The JE tapers until it is only one cell in width apically.
- The JE is permeable with wide intercellular spaces through which cells and substances can migrate (such as bacterial toxins or host defence cells).
- Migration of the JE from its position in health apically onto the root cementum indicates a loss of periodontal attachment and progression to the disease state of periodontitis.

Gingival connective tissue

The gingival connective tissue (or lamina propria) is made up of collagen fibre bundles called gingival fibres, around which lie ground substance, fibroblasts, blood and lymph vessels and neural tissues. The four fibre groups are shown in Figs 1.2 and 1.3.

Periodontal ligament

The periodontal ligament forms the attachment between the cementum and alveolar bone. It is a richly vascular connective tissue within which lie bundles of collagen fibres; these are divided into four groups based on their position (Fig. 1.4).

Within the ligament are mechanoreceptors that provide sensory input for jaw reflexes. Cells from the periodontal ligament are involved in the formation and remodelling of alveolar bone and cementum. The periodontal ligament acts to dissipate masticatory forces to the supporting alveolar bone and its width, height and quality determine a tooth's mobility.

Cementum

Cementum is a mineralised tissue overlying the root dentine. It does not undergo physiological remodelling but is continuously deposited throughout life. Cementum is classified into two types:

- Acellular.
- Cellular.

Acellular cementum

Acellular cementum forms on root dentine during root formation and tooth eruption. Fibres inserted from the periodontal ligament are mineralized within the cementum and are known as Sharpey's fibres and are abundant in acellular cementum.

Cellular cementum

Cellular cementum lies over the acellular cementum. It contains cells called cementocytes which lie in lacunae. The cellular cementum layer is thicker in the apical region of the root where it is between 0.2 and 1 mm thick.

Alveolar bone

- The walls of the sockets are lined with a layer of dense bone called compact bone, which also forms the buccal and lingual/palatal plates of the jaw bones.
- In between the sockets and the compact jaw bone walls lies cancellous bone that is made up of bony trabeculae.
- The compact bone plates of the jaws are thicker on the buccal aspect of the mandibular molars and thinnest on the labial surface of the mandibular incisors.

The thickness of the compact bone layer is relevant to the choice of local analgesia techniques as the anaesthetic solution passes through bone to reach the nerve supply. The thin bone, particularly in the lower incisor region, can manifest as incomplete bony coverage in the form of fenestrations and dehiscences (Fig. 1.5).

The tooth sockets are lined with compact bone within which the principal fibres of the periodontal ligament are inserted. This area of bone can appear as a dense white line called the lamina dura on a radiograph.

Key points

- Gingivae
  - JE forms the specialised attachment to the tooth
  - The most apical part of JE lies at the cement–enamel junction in health
  - Supported by connective tissue containing collagen fibre bundles
- Periodontal ligament
  - Forms attachment between the cementum and bone
- Cementum
  - Mineralised and deposited continuously
- Alveolar bone
  - Compact and cancellous bone
  -

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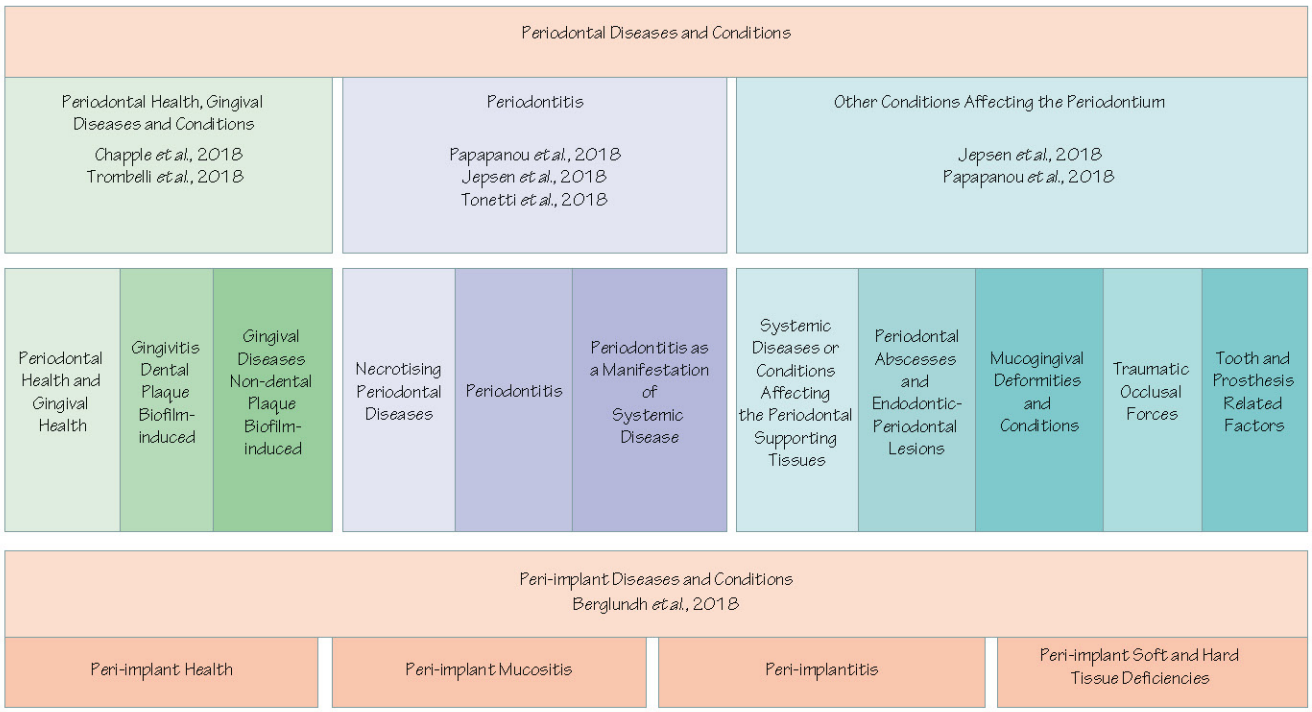
2 Classification of periodontal diseases

Box 2.1 World Workshop 2018 Classification of Periodontal and Peri-implant Diseases and Conditions

An up-to-date classification:
- allows the clinician to be aware of the full range of periodontal and peri-implant diseases and conditions that can affect the patient.
- provides a basis for the diagnosis and subsequent management of the patient.

The 2018 classification[1] in Figures 2.1–2.8
- derives from the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions (Caton *et al.*, 2018), which was convened jointly by the European Federation of Periodontology (EFP) and American Academy of Periodontology (AAP)
- replaces the previous 1999 classification scheme (Armitage, 1999).
- was intended to align and update the classification with current understanding of periodontal and peri-implant diseases and conditions.
- comprises three main categories of periodontal diseases and conditions, and includes four categories of peri-implant diseases and conditions that were introduced for the first time (Fig. 2.1).

Figure 2.1 2018 Classification of periodontal and peri-implant diseases and conditions. Source: Adapted from Caton *et al.* (2018).



1 Foot note: Although the World Workshop took place in 2017, the proceedings were published in 2018. To avoid confusion, the official decision was made to call it 'the 2018 classification' henceforth whilst acknowledging it emanated from 'the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions'. This convention of nomenclature will be adopted throughout Periodontology At A Glance, edition 2.

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Figure 2.2 (a) 2018 Classification: periodontal health and gingivitis – dental plaque biofilm induced. Source: Adapted from Chapple *et al*. (2018), Table 2. (b) Abridged 2018 classification: gingival diseases: non-dental plaque induced. Source: Adapted from Chapple *et al*. (2018),Table 2. For unabridged version, see Appendix 1.

(a)

Periodontal health:

A. Clinical health on an intact periodontium

B. Clinical gingival health on a reduced periodontium

- I. Stable periodontitis patient
- II. Non-periodontitis patient

Gingivitis - dental plaque biofilm-induced:

Intact periodontium
Reduced periodontium in non-periodontitis patient
Reduced periodontium in successfully treated periodontitis patient

A. Associated with plaque biofilm alone

B. Mediated by systemic or local risk factors

- I. Systemic risk factors (modifying factors)
  - (a) Smoking
  - (b) Hyperglycaemia
  - (c) Nutritional factors
  - (d) Pharmacological agents (prescription, non-prescription and recreational)
  - (e) Sex steroid hormones
    - Puberty
    - Menstrual cycle
    - Pregnancy
    - Oral contraceptives
  - (f) Haematological conditions
- II. Local risk factors (predisposing factors)
  - (a) Dental plaque biofilm retention factors (eg prominent restoration margins)
  - (b) Oral dryness

C. Drug-influenced gingival enlargement

(b)

Gingival diseases: non-dental plaque-induced:

A. Genetic/developmental disorders
- I. Hereditary gingival fibromatosis

B. Specific infections
- I. Bacterial origin
- II. Viral origin
- III. Fungal origin

C. Inflammatory and immune conditions
- I. Hypersensitivity reactions
- II. Autoimmune diseases of skin and mucous membranes
- III. Granulomatous inflammatory lesions (orofacial granulomatoses)

D. Reactive processes
- I. Epulides

E. Neoplasms
- I. Premalignancy
- II. Malignancy

F. Endocrine, nutritional & metabolic diseases
- I. Vitamin deficiencies

G. Traumatic lesions
- I. Physical/mechanical trauma
- II. Chemical (toxic) burn
- III. Thermal insults

H. Gingival pigmentation
- I. Melanoplakia
- II. Smoker's melanosis
- III. Drug-induced pigmentation (antimalarials, minocycline)
- Iv. Amalgam tattoo

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Figure 2.3 (a) 2018 Classification of periodontitis. (b) 2018 Classification of necrotising periodontal diseases (NPDs). Source: Adapted from Papapanou *et al.* (2018), Table 2.

(a)

- **Necrotising periodontal diseases**
- **Periodontitis**
- Staging and Grading required
- **Periodontitis as a manifestation of systemic disease**
- Rare systemic disorders like Papillon-Lefèvre Syndrome with early presentation of severe periodontitis are grouped as 'Periodontitis as a Manifestation of Systemic Disease' and classified by primary systemic disease
- Other systemic conditions eg neoplasms affecting the periodontium independent of plaque-induced periodontitis are grouped as 'Systemic Diseases or Conditions that Affect the Periodontal Supporting Tissues' (see Fig 2.4, also Appendix 2)
- All should follow the primary disease classification according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes

Note: Clinical cases of periodontitis without characteristics of necrotising periodontitis or that are not a manifestation of systemic disease should be diagnosed as 'periodontitis' and further characterised with Staging and Grading

(b)

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

Key: Necrotising Gingivitis (NG); Necrotising Periodontitis (NP), Necrotising Stomatitis (NS)

Figure 2.4 2018 Classification of systemic diseases and conditions that affect the periodontal supporting tissues. Source: Abridged from Jepsen *et al.* (2018), Table 1, which was adapted from Albandar *et al.* (2018). See Appendix 2 for unabridged version and ICD-10 codes.

1. Systemic disorders that have a major impact on loss of periodontal tissues by influencing periodontal inflammation	2. Other systemic disorders that influence the pathogenesis of periodontal diseases	3. Systemic disorders that can result in loss of periodontal tissues independent of periodontitis
1.1 Genetic disorders	Diabetes mellitus Obesity Osteoporosis Rheumatoid arthritis or osteoarthritis Emotional stress and depression Smoking (nicotine dependence) Medications	**3.1 Neoplasms**
1.1.1 Diseases associated with immunologic disorders Eg Down Syndrome; Papillon-Lefèvre Syndrome; Chediak-Higashi Syndrome; severe neutropenia (congenital or cyclic)		Primary neoplastic diseases of the periodontal tissues Eg Oral squamous cell carcinoma; odontogenic tumours; or other primary neoplasms of the periodontal tissues Secondary metastatic neoplasms of the periodontal tissues
1.1.2 Diseases affecting the oral mucosa and gingival tissue		
1.1.3 Diseases affecting the connective tissues Eg Ehlers-Danlos Syndromes (types IV, VIII)		
1.1.4 Metabolic and endocrine disorders Eg Hypophosphatasia		
1.2 Acquired immunodeficiency diseases Eg Acquired neutropenia, HIV infection		**3.2 Other disorders that may affect the periodontal tissues**
1.3 Inflammatory diseases		

Figure 2.5 2018 Classification of other conditions affecting the periodontium: periodontal manifestations of systemic diseases and developmental and acquired conditions. Source: Adapted from Caton *et al.* (2018), Table 4.

Systemic diseases or conditions that affect the periodontal supporting tissues (Albandar *et al.*, 2018; Jepsen *et al.*, 2018)	See Fig 2.4 and Appendix 2
Other periodontal conditions (Papapanou *et al.*, 2018; Herrera *et al.*, 2018)	Periodontal abscesses (Fig 2.6)
	Endodontic-periodontal lesions (Fig 2.7)
Mucogingival deformities and conditions around teeth (Cortellini and Bissada 2018)	Gingival phenotype
	Gingival/soft tissue recession
	Lack of gingiva
	Decreased vestibular width
	Aberrant frenum/muscle position
	Gingival excess
	Abnormal colour
	Condition of exposed root surface
Traumatic occlusal forces (Fan and Caton 2018)	Primary occlusal trauma
	Secondary occlusal trauma
	Orthodontic forces
Prostheses and tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis (Ercoli and Caton 2018)	Localised tooth-related factors (Fig 2.8)
	Localised dental prostheses-related factors (Fig 2.8)

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Figure 2.6 2018 Classification of periodontal abscesses based on aetiological factors involved. Source: Adapted from Papapanou *et al.* (2018), Table 4.

Periodontal abscess in periodontitis patients (in a pre-existing periodontal pocket)	Acute exacerbations	Untreated periodontitis	
		Non-responsive to therapy periodontitis	
		Supportive periodontal therapy (SPT)	
	After treatment	Post-Professional Mechanical Plaque Removal (PMPR)	
		Post-surgery	
		Post-medications	Systemic antimicrobials Other drugs: nifedipine
Periodontal abscess in non-periodontitis patients (not mandatory to have a pre-existing periodontal pocket)	Impaction		Dental floss, orthodontic elastic, toothpick, rubber dam, popcorn hulls
	Harmful habits		Wire or nail biting and clenching
	Orthodontic factors		Orthodontic forces or cross-bite
	Gingival overgrowth		
	Alteration of root surface	Severe anatomic alterations	Invaginated tooth, dens evaginatus or odontodysplasia
		Minor anatomic alterations	Cemental tears, enamel pearls or developmental grooves
		Iatrogenic conditions	Perforations
		Severe root damage	Fissure or fracture, cracked tooth syndrome
		External root resorption	

Figure 2.7 2018 Classification of endodontic-periodontal lesions. Source: Adapted from Papapanou *et al.* (2018), Table 3.

Endo-periodontal lesions with root damage	Root fracture or cracking	
	Root canal / pulp chamber perforation	
	External root resorption	
Endo-periodontal lesions without root damage	Endo-periodontal lesions in periodontitis patients	Grade 1 - narrow deep periodontal pocket in 1 tooth surface
		Grade 2 - wide deep periodontal pocket in 1 tooth surface
		Grade 3 - deep periodontal pockets in >1 tooth surface
	Endo-periodontal lesions in non-periodontitis patients	Grade 1 - narrow deep periodontal pocket in 1 tooth surface
		Grade 2 - wide deep periodontal pocket in 1 tooth surface
		Grade 3 - deep periodontal pockets in >1 tooth surface

Figure 2.8 2018 Classification of factors related to teeth and to dental prostheses that can affect the periodontium. Source: Adapted from Jepsen *et al.* (2018), Table 4.

Factors related to teeth/dental prostheses that can affect the periodontium	
Localised tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis	Tooth anatomic factors
	Root fractures
	Cervical root resorption, cemental tears
	Root proximity
	Altered passive eruption
Localised dental prosthesis-related factors	Restoration margins placed within the supracrestal attached tissues
	Clinical procedures related to the fabrication of indirect restorations
	Hypersensitivity/toxicity reactions to dental materials

Key points

- The current 2018 classification derives from the 2017 World Workshop on Classification of Periodontal and Peri-implant Diseases and Conditions and replaces the previous 1999 International Classification
- The 2018 classification:
  - defines periodontal health for the first time, as well as plaque-biofilm induced gingivitis and non-dental-plaque-induced gingival conditions
  - provides a new classification for periodontitis, including Staging and Grading
  - includes categories for periodontitis as a manifestation of systemic diseases and systemic diseases and conditions that affect the periodontal supporting tissues
  - updates other conditions affecting the periodontium
  - classifies peri-implant health, peri-implant mucositis, peri-implantitis and peri-implant soft and hard tissue deficiencies for the first time
- Knowledge of the current classification provides a basis for subsequent diagnosis and management of periodontal and peri-implant diseases and conditions

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3 Periodontal epidemiology

Figure 3.1 (a) Definition of epidemiology. (b) Definitions of epidemiological terms. Source: Last (2001)/Oxford University Press.

(a)

Epidemiology is the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems

(b)

Study	*includes*	Surveillance, observation, hypothesis testing, analytical research and experiments
Distribution	*refers to*	Analysis by time, place and classes of persons affected
Determinants	*are the*	Physical, biological, social, cultural and behavioural factors that influence (periodontal) health
Health-related states or events	*include*	(Periodontal) diseases, causes of morbidity, behaviour such as tobacco use, reactions to preventive regimes, and provision and use of health services
Specified populations	*are*	Those with identifiable characteristics
Application of this study to the control of health problems	*makes explicit*	The aim of epidemiology is to promote, protect, and restore (periodontal) health

Figure 3.2 (a) Types of periodontal epidemiology. (b) Descriptive, analytical and pragmatic studies. (c) Cross-sectional and longitudinal studies. (d) Case–control and case–cohort studies. (e) Clinical trial phases. (f) Types of trial. (g) Hierarchy of research evidence.

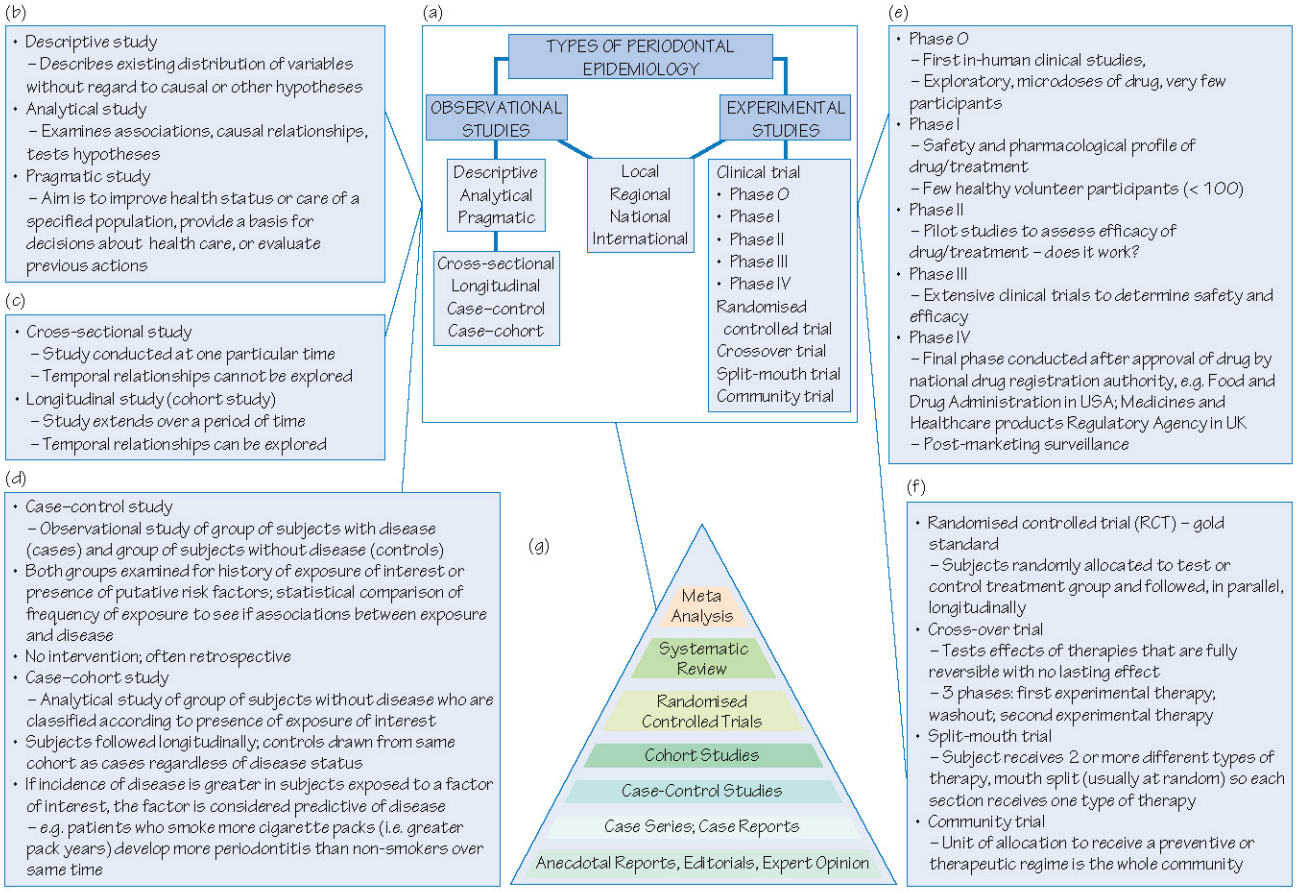

Figure 3.3 Common terms in epidemiology.

Prevalence	Number or % of affected subjects in population with disease at defined threshold
Extent	Number or % of affected teeth or sites with disease at defined threshold
Severity	How advanced disease is. May be bands of severity, *e.g.* 1–2 mm clinical attachment loss (CAL) is mild; 3–4 mm is moderate; 5+ mm is severe. May be mean mouth data, *e.g.* mean severity of CAL = 3.4 mm
Incidence	Number of new cases of disease at a defined threshold that appear in a population over a predetermined period of time
Threshold of disease	Level of disease being studied, *e.g.* clinical attachment loss of 2 mm; *e.g.* probing depths of 6 mm or more

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Figure 3.4 Attributes of a good periodontal index.

Attributes of a good index are that it should be:
- Valid (i.e. measures what it purports to measure)
- Reliable (i.e. can be reproduced if re-measured)
- Quick
- Simple
- Acceptable to the examiner and subject and use minimum equipment
- Amenable to statistical analysis

Figure 3.5 The Gingival Index. Source: Löe & Silness (1967)/ John Wiley & Sons.

Code 0	Normal gingiva
Code 1	Mild inflammation. Slight change in colour, slight oedema. No bleeding on probing
Code 2	Moderate inflammation. Redness, oedema and glazing. Bleeding on probing
Code 3	Severe inflammation. Marked redness and oedema, ulceration. Spontaneous bleeding

Figure 3.7 Factors influencing probing accuracy.

- Probing force
- Probe angulation
- Thickness of probe (thick tip diameter of probe will underestimate compared with thin probe)
- Accuracy of probe markings
- Examiner experience
- Degree of inflammation of tissues (tendency to overestimate if inflamed)
- Presence of subgingival calculus or anatomical feature (may impede probing)
- Location of probing (anterior easier than posterior, buccal more reproducible than palatal/lingual sites)

Figure 3.8 UK Adult Dental Health Survey 2009, showing the prevalence of pockets and CAL.

UK Adult Dental Health Survey 2009 6,469 dentate adults examined (representing population) using CPITN methodology:
- 37% had shallow pockets 4 mm – 5 mm
- 7% had deep pockets 6 mm – 8.5 mm
- 1% had very deep pockets ≥ 9 mm

Of adults aged ≥ 55 years examined for CAL:
- 66% had CAL ≥ 4 mm
- 21% had CAL ≥ 6 mm
- 4% had CAL ≥ 9 mm

Figure 3.6 (a) Clinical attachment loss (CAL) and probing depth (PD). (b) CAL and recession.

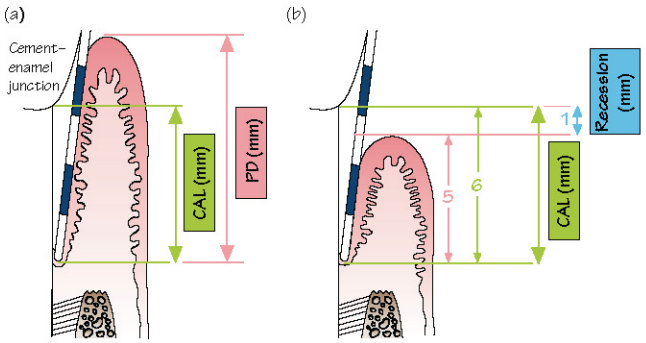
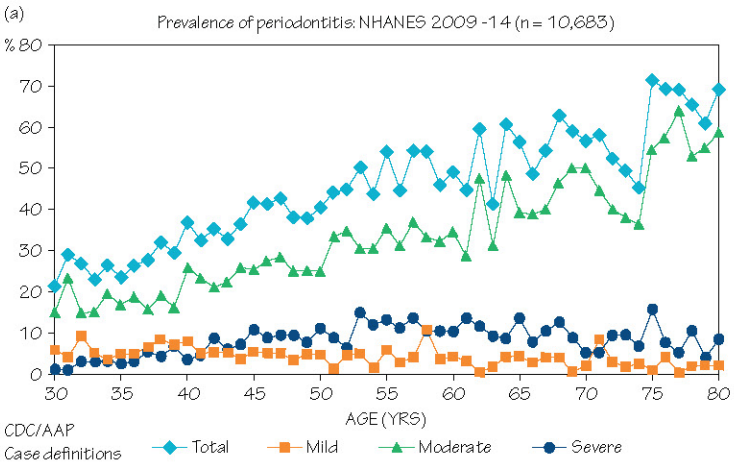
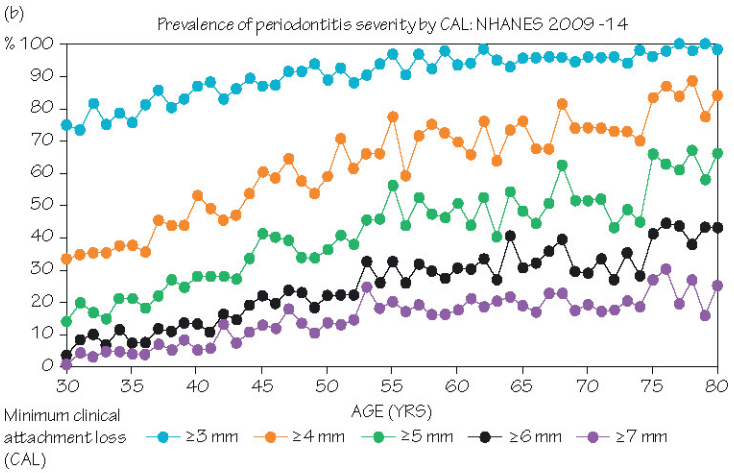
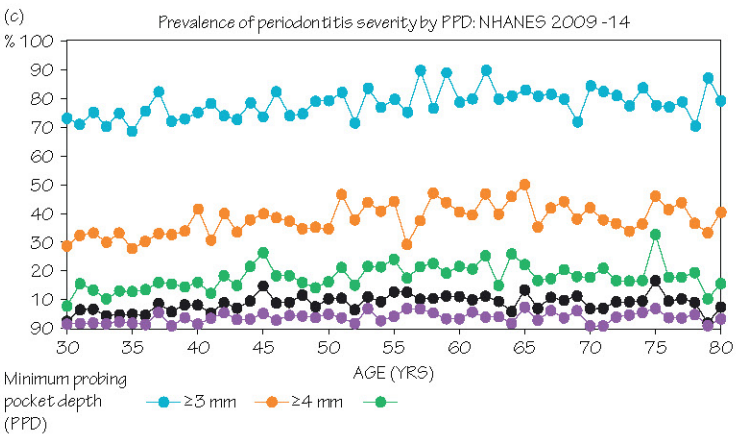


Figure 3.9 US NHANES 2009–14 prevalence of periodontitis by (a) CDC/AAP case definitions, (b) CAL and (c) PPD. Source: Modified from Eke *et al.* (2018,) Figs 1–3, with permission of Elsevier.







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Epidemiology is the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems (Fig. 3.1).

Types of periodontal epidemiology

There are different types of periodontal epidemiology (Fig. 3.2). There is a hierarchy of research evidence that can be gleaned from different study types and statistical analyses (Fig 3.2g). Periodontal epidemiological studies seek to understand the natural course of the different periodontal diseases and the factors that influence their distribution (Fig. 3.3). Causative and risk factors (Chapters 10, 11 and 14) need to be established in order to determine the aetiology and determinants of disease development. Evidence-based research is important to establish the effectiveness of treatment methods and products and preventive regimes for periodontal diseases at a population level.

Ultimately, the particular research question and the aims and objectives of the study determine the type, design, size and duration of the epidemiological study.

Methodology

Periodontal indices

Measuring periodontal disease involves the use of a periodontal index (Fig. 3.4). There is no single periodontal index that satisfies all the desirable requirements in every type of study and many exist (Barnes *et al*., 1986).

Gingival indices

In the 1960s, the Gingival Index was introduced in which the codes used mixtures of signs of inflammation: colour change, oedema, bleeding on probing and ulcerations (Fig. 3.5) – it is a compound index. Although widely used, assigning a code is difficult if not all signs are present or if signs from two codes occur. Dichotomous indices (presence or absence of the condition) are alternatives, e.g. bleeding on probing.

Plaque indices

Plaque indices have faced similar problems. The Plaque Index (Löe & Silness, 1967) assesses plaque thickness at the gingival margin. Other indices use disclosing solutions and measure plaque area (e.g. the Turesky modification of the Quigley–Hein Index), while yet others, like the O'Leary Plaque Index, simply record presence or absence but count the per cent of sites affected (Barnes *et al*., 1986).

Periodontal indices

Russell's Periodontal Index was reported in 1956 and was the first index to be used widely. This was followed by Ramfjord's Periodontal Disease Index in 1959, which introduced the method for measuring clinical attachment loss (CAL). This has been the gold standard and basis of epidemiological clinical recording ever since (Fig. 3.6).

Other recordings may involve periodontal probing pocket depths (PPD) and recession (Fig.3.6). Ethical issues around limiting radiation doses can restrict the use of radiographic measurements. Technological advances enable digital manipulation of images; subtraction radiography allows detection and measurement of small bone changes.

Recording

Full mouth recording of data provides the most information, but some partial recording systems – although generally underestimating disease levels – have been incorporated into large-scale epidemiological studies in order to increase the sample size whilst retaining key information. UK and US national surveys have used this approach.

Other recording issues relate to operator measurement errors – many factors influence probing accuracy (Fig. 3.7). Also, it is important to remember that the periodontal tissues themselves are biologically active and therefore subject to change.

Statistical management

It is essential to distinguish between association and causation. Confounding variables also need to be taken into account, i.e. when the variable is not of primary interest but may affect the study results anyway. Due to measurements of multiple sites within the mouth and repeated recordings over time in some types of study, careful appraisal of data management options is necessary. In addition to the more conventional tests, multilevel modelling and structural equation modelling offer useful approaches for periodontal epidemiology (Tu *et al*., 2008).

WHO Global Oral Data Bank

The World Health Organization (WHO) has a long tradition of epidemiological survey methodology and has encouraged countries to conduct surveys in a standardised way via its manual 'Oral Health Surveys – Basic Methods'. The WHO Global Oral Data Bank collates the epidemiological data gathered from such surveys (Nazir *et al*., 2020). The Community Periodontal Index of Treatment Needs (CPITN) originally proposed by the WHO as an index to evaluate treatment needs in populations was renamed the Community Periodontal Index (CPI) to denote its use as an epidemiological tool although it does have limitations (Leroy *et al*., 2010). In the fifth edition of the manual in 2013, the CPI was modified so that instead of being sextant based, assessment of gingival bleeding and pockets was for all teeth present using the WHO CPI probe; presence of calculus was not recorded as it is not a disease *per se*; CAL was recorded on index teeth 17, 16, 11, 26, 27, 36, 37, 31, 46, 47. For epidemiological studies, children under 15 years of age continued to be excluded from PPD/CAL probing measurements.

Global epidemiology

Population studies confirm the link between plaque and gingivitis. Adults worldwide exhibit gingival bleeding and inflammation. Gingivitis precedes periodontitis and there are no data to suggest that periodontitis develops in the absence of gingival inflammation.

- Incipient (Stage 1) periodontitis can begin in adolescents (Chapter 41).
- Mild to moderate periodontitis (Stage 1 or 2) is widespread in adults based on representative population samples from national studies but severe disease (Stage III) or very severe periodontitis (Stage IV) is less prevalent (Figs 3.8, 3.9).
- The 2009 UK Adult Dental Health Survey showed that since 1998 there has been an overall reduction in the prevalence of pocketing ≥4 mm from 55% to 45%, possibly linked to improved plaque control, but pocketing ≥6 mm had

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increased from 6% to 9%, perhaps due to retaining teeth for longer (White *et al*., 2011, 2012).

- The 2009–14 US National Health and Nutrition Examination Surveys (NHANES) showed that 42.2% of adults ≥30 years had periodontitis, comprising 34.4% with mild/moderate periodontitis and 7.8% with severe periodontitis. The prevalence was highest among current smokers, adults who did not use dental floss regularly and those who had not visited the dentist in the previous six months; it co-occurred with diabetes and increased numbers of missing teeth but not with obesity. These data provided the best estimates of periodontitis prevalence in the US but costs may be prohibitive for future surveillance (Eke *et al*., 2018).
- There is variation in the prevalence of severe periodontitis reported globally, ranging from: 11.2% (Kassebaum *et al*., 2014); then 10% (Frencken *et al*., 2017), in a comprehensive systematic review, and more recently, 19%, representing more than 1 billion cases worldwide (Chen *et al*., 2021; WHO, 2022). The review concluded that study heterogeneity and methodological issues hamper comparisons across studies over time and that geographic variation and time trends of the incidence and prevalence of periodontitis cannot be drawn from the available evidence.
- Consensus on a definition of what constitutes a periodontitis case is key (Borrell & Papapanou, 2005).
- The AAP/CDC case definition for epidemiological surveillance and the EFP case definition for risk factors research have both been used widely. Both definitions were found to be complementary for surveillance of severe diseases but not for mild/moderate periodontitis (Eke *et al*., 2012).
- A consensus definition was adopted at the 2017 World Workshop on Classification of Periodontal and Peri-implant Diseases and Conditions (see Fig. 9.6): a patient is a periodontitis case in the context of clinical care if: (1) interdental CAL is detectable at ≥2 non-adjacent teeth, or (2) buccal or lingual CAL ≥3 mm with pocketing >3 mm is detectable at ≥2 teeth but the observed CAL cannot be ascribed to non-periodontitis-related causes (Tonetti *et al*., 2018; Papapanou *et al*., 2018). Inherent in the consensus definition is the understanding that there would be bone loss at ≥2 non-adjacent teeth in a periodontitis case. While an individual case should be further characterised by staging and grading (Chapter 36), it was acknowledged that specific considerations are needed for epidemiological surveys where threshold definition is likely to be based on measurement errors (Tonetti *et al*., 2018).

Key points

- There are different types of epidemiological study
- There is no single ideal periodontal index
- The type of study and index depend on the study aims and objectives
- CPI and other epidemiological data have highlighted differences in global disease prevalence and severity

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4 Role of plaque in the aetiology of periodontal diseases

Figure 4.1 (a) Stages and (b) design of classic experimental gingivitis study in humans. Source: Adapted from Loe *et al.* (1965).

(a)

9 dental students, 2 technicians, 1 teacher with minimal periodontal disease
• Plaque Index (PII) and Gingival Index (GI) recorded at baseline examination • Plaque sampled and microbial analysis undertaken at baseline
Oral hygiene withdrawn
PII, GI and microbial analysis repeated at varying intervals
Signs of appearance of gingivitis checked for
Oral hygiene resumed when gingival inflammation observed
Experiment continued until PII and GI approached zero and clinical health regained
All subjects developed gingivitis: 3 within 10 days, 9 between 15 and 21 days
Increase in volume of plaque noted when oral hygiene withdrawn
Concomitant shift in microbial composition over study time: (i) initially predominance of Gram-positive cocci and short rods, usually > 80%; (ii) with gingivitis, Gram-positive cocci and short rods reduced to 45–60%; the rest comprised Gram-negative cocci and short rods (22%), Gram-positive filaments (10%), fusobacteria (10%), vibrios(6%) and spirochaetes (1%)
Evidence that plaque causes gingival inflammation and plaque removal leads to resolution
Demonstration of differences in patient susceptibility to gingival inflammation

(b)

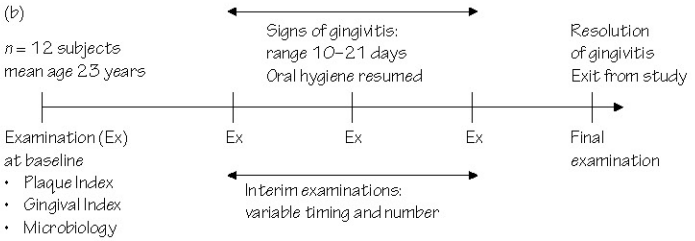

Figure 4.2 Plaque hypotheses.

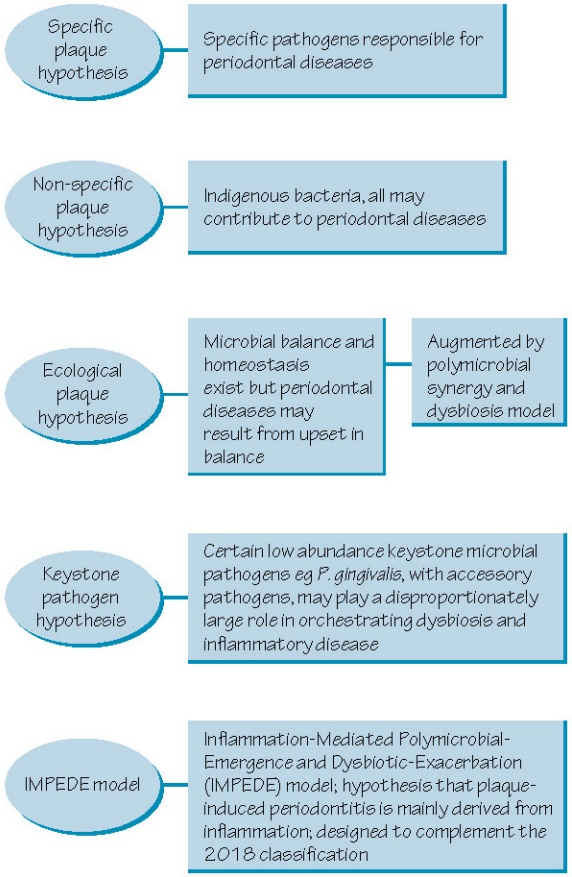

Table 4.1 Stages of plaque biofilm formation.

Stage	Plaque biofilm formation
1 Acquired pellicle formation	Host and bacterial molecules, salivary proteins, glycoproteins, lipds, bacterial glycans and enzymes are adsorbed onto the tooth surface, leading to formation of a surface-conditioning film, the acquired pellicle
2 Transport	Transport of bacteria to the pellicle occurs via natural salivary flow, Brownian movement or chemotaxis. Adsorption of coccal bacteria onto the pellicle occurs within 2 hours – pioneer species include *Neisseria*, *Streptococcus sanguis*, *S. oralis* and *S. mitis*, also Gram-positive rods, mainly *Actinomyces*
3 Reversible attachment	Long-range physicochemical interactions lead to reversible adhesion between the microbial cell surface and the pellicle involving van der Waals attractive forces and electrostatic repulsion
4 Irreversible attachment	Adherence of reversibly attached cells can become irreversible if adhesins on early bacterial colonisers bind to complementary receptors in the acquired pellicle. Once attached, these early colonisers divide and form microcolonies whose metabolism begins to modify the local environment
5 Secondary colonisation	Co-aggregation/co-adhesion of late colonisers to the already attached bacteria via adhesin–receptor interactions results in an increasingly diverse microflora, i.e. microbial succession
6 Maturation	Muliplication of attached organisms leads to confluent growth and biofilm maturation, facilitating interbacterial interactions (both synergistic and antagonistic) and formation of a biofilm matrix of extracellular polymers
7 Detachment	Detachment of bacteria if they sense adverse environmental changes allows colonisation at new sites

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Figure 4.3 Climax microbial community in plaque biofilm showing the diverse range of microbial forms. Source: Courtesy of Professor P. Marsh.

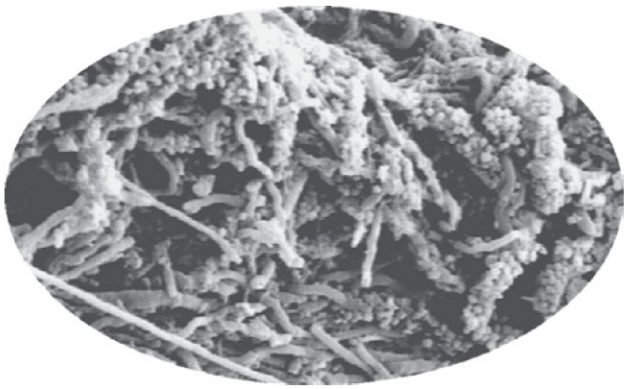


Figure 4.4 Noran Odyssey confocal laser scanning microscope image of intact one-week-old plaque biofilm formed *in vivo* using the Leeds *in situ* device. The image shows the three-dimensional structure and variations in biofilm density; biomasses of bacteria are pink; voids, channels and spaces are dark areas. Image achieved by volume rendering of a series of x-y (horizontal) sections. Scale bar = 50 μm. Source: Courtesy of Dr S. Wood (Wood *et al.*, 2002).

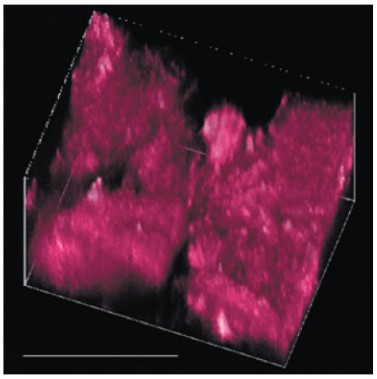


Figure 4.5 Schematic of ecological plaque hypothesis. Source: Adapted from Marsh (2022), Figure 1.5.4.

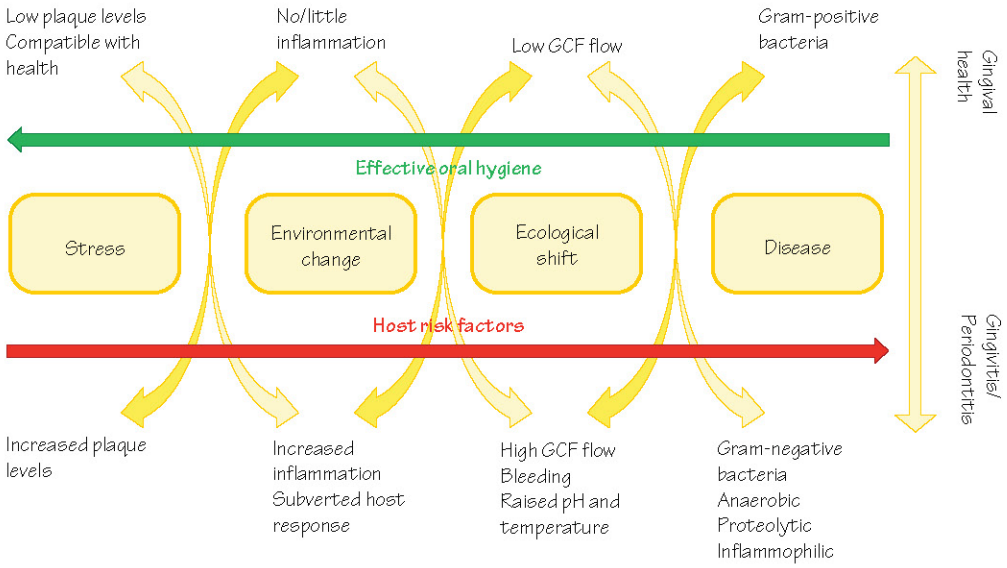
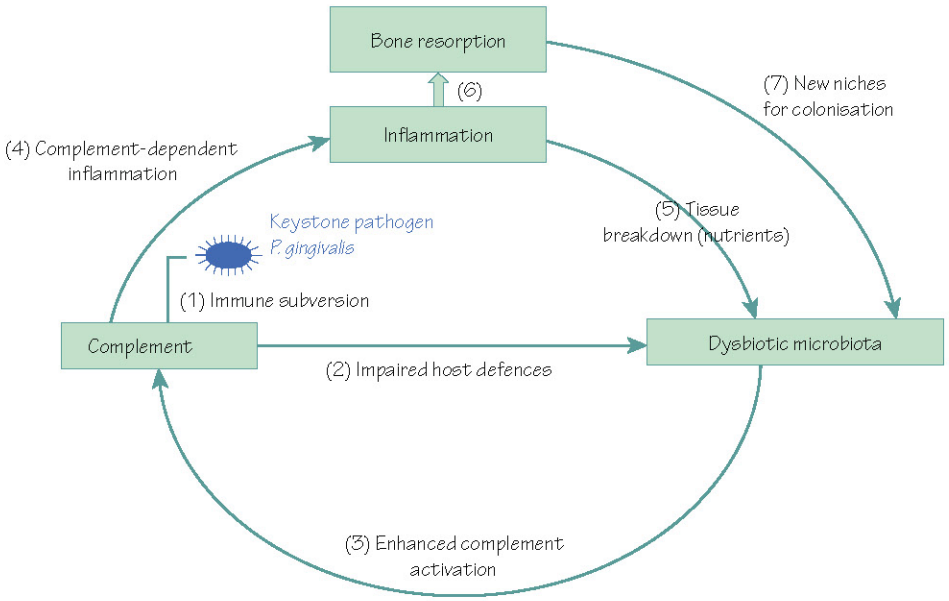


Figure 4.6 Keystone pathogen-induced dysbiosis and periodontal disease. Source: Adapted from Hajishengallis *et al.* (2012), Figure 2.



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