

# The Dental Probe

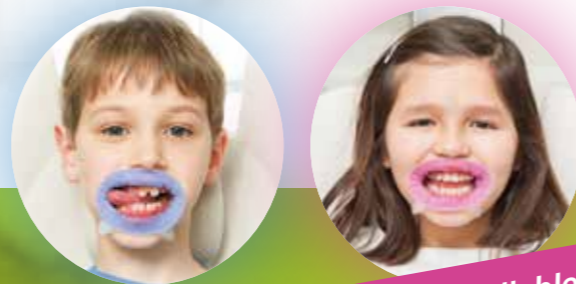
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# Editorial

**By Dr David Muscat**

Dear colleagues,

2018 has drawn to a close and soon we will be making those resolutions. One resolution many should make is to attend local dental conferences. The ones that I have been involved in locally and others that I have attended have always been of a high standard. These conferences entail a lot of hard work and planning and it is fitting that dentists take time out to attend and garner CPD. It is also helpful if conferences do not overlap and are held apart from one another so as to ensure maximum attendance.

The DAM is organising a series of Basic Life Support courses. These will be organised every three months or so and a call for applications for these courses will be made and applicants will be selected on a first come first served basis. The course will usually be a full day course and held at the Hilton. Dr Adam Bartolo runs the courses. These are co-ordinated by Dr Noel Manche and Dr Ann Meli Attard. A lecture on ceramics is envisaged as well as a lecture on infectious diseases. We are also planning lectures on Carpal Tunnel syndrome in relation to dentistry and also Fibromyalgia. I would like to feature some presentations from this year's 'Smile For Health' Conference in this issue and I would like to thank the authors for their co-operation.

We mourn the loss of Dr Herbert Messina Ferrante who passed away on New Years Eve. May the Lord grant him Eternal Rest. He was one who always fought for the rights of the dentist. The lion roars no more but his spirit is still with us.

The cover picture is by Dr Josef Awad and it is of the Church of Saint John The Baptist in Xewkija Gozo. The Dental Association Christmas Party was held on the 12th December at the Hilton.

Best regards,

*David*

Dr David Muscat B.D.S. (LON)  
Editor / Secretary, P.R.O. D.A.M.



Above: The DAM Committee

## Smile for Health Conference



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# Your dentures gave them confidence. We'll keep it going.

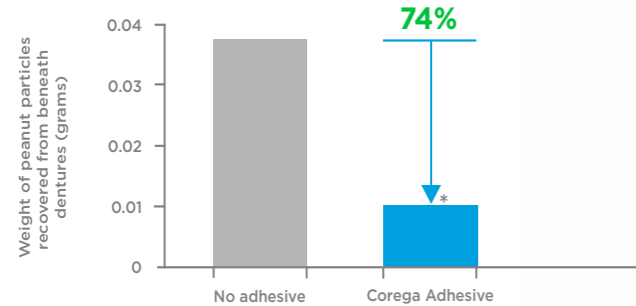
You can be confident in the knowledge that you've given your patients specially made and well-fitting dentures. However, your denture-wearing patients can have concerns around denture retention and trapped food, making it difficult for them to emotionally adjust to living with dentures. They may not tell you, but more than 1 in 3 denture wearers admit to skipping social activities because they are conscious of their dentures.<sup>1</sup>

Up to **29%** skip eating out in public,<sup>1</sup> **86%** experience food trapping under their dentures and **55%** experience denture movement.<sup>2</sup>

## These everyday challenges can hold your patients back from living life to the fullest.

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Corega Ultra Fresh denture adhesive can support your patients' throughout their denture-wearing journey.

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- Corega adhesive is proven to increase the bite force by 38% in well-fitting dentures,<sup>3</sup> **increasing patients' confidence to bite into varied foods**
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## Corega Ultra Fresh denture adhesive - Offering your patients reassurance for everyday life



Help your patients eat, speak and smile with confidence with Corega Ultra Fresh denture adhesive cream and Corega 3 Minutes denture cleansing tablets.

<sup>1</sup>vs. no adhesive

**References:** 1. P&G News. Denture Wearers Embrace New Smile Yet Avoid Popular Foods. <http://news.pg.com/press-release/pg-corporate-announcements/denture-wearers-embrace-new-smile-yet-avoid-popular-foods>. Accessed September 2013; 2. GSK Data on File; Canadian Quality of Life Study. 2005; 3. Munoz CA *et al.* *J Prosthodont.* 2011;21(2):123-129; 4. Fernandez P *et al.* Poster presented at the IADR 2011, Poster 1052.

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CHMLT/CHPLD/0003/15b (1)

# STAGING AND GRADING OF PERIODONTITIS

Edward Sammut  
BChD MSc MClintDent MFDS MRD RCSEd  
Specialist in Periodontics (UK)

## Aims and Objectives

- Give an overview of the 2017 AAP-EFP classification
- Explain the rationale behind the changes in the classification system
- Focus on the section regarding periodontitis and the key findings of the research groups
- Introduce the staging and grading framework and familiarise with different diagnostic criteria which have been suggested

## Why Classify Disease?

- Share a common international language
- Health surveys and population surveys
- Diagnosis, prognosis, patient communication
- Implications for treatment
- Enable research



## Background

- 1989 Classification
  - Adult Periodontitis
  - Early Onset Periodontitis
  - Periodontitis Associated with Systemic Disease
  - Necrotizing Ulcerative Periodontitis
  - Refractory Periodontitis



## Background

- 1989 Criticisms
  - Overlap in disease categories
  - No gingival diseases
  - Heavy emphasis on age
  - Inadequate / poorly defined classification criteria

## Background

- 1999 Classification "Armitage"
  - Gingival diseases
    - Chronic Periodontitis
    - Aggressive Periodontitis
    - Periodontitis as a manifestation of systemic diseases
    - Necrotising Periodontitis
  - Abscesses
  - Perio-endo lesions
  - Developmental or Acquired Deformities and conditions



## GINGIVAL DISEASES

Armitage 1999

1. Gingival diseases of specific local origin
  - a. leukoerythroblastic lesions
  - b. pyogenic granuloma
  - c. leukoplakia
  - d. other
2. Gingival diseases modified by malocclusion
  - a. occlusal trauma associated gingivitis
  - b. other
3. Gingival diseases modified by medications
  - a. drug-induced gingival enlargements
  - b. other
4. Gingival diseases modified by malnutrition
  - a. scurvy
  - b. other
5. Gingival manifestations of systemic disease
  - a. mucocutaneous disorders
    - i. lichen planus
    - ii. pemphigus vulgaris
    - iii. erythema multiforme
    - iv. lupus erythematosus
    - v. drug-induced
    - vi. other
  - b. allergic reactions
    - i. dental restorative materials
    - ii. mercury
    - iii. nickel
    - iv. acrylic
    - v. other
  - c. reactions attributable to
    - i. toothpaste/dentifrices
    - ii. mouthwashes
    - iii. chewing gum
    - iv. toothbrushes
    - v. other
6. Traumatic lesions (faciotoxic, iatrogenic, accidental)
  - a. chemical injury
  - b. physical injury

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Continues on page 6.

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 5.

### THE REST Armitage 1999

- I. Class: "Healthy"
  - A. Good
  - B. Generalized
- II. Aggressive Periodontitis
  - A. Localized
  - B. Generalized
- III. Periodontitis: A The status of severe disease
  - A. Associated with hereditary disorders
    - 1. Acquired neutropenia
    - 2. Leukemias
    - 3. Sider
  - B. Associated with genetic disorders
    - 1. Hanks and Ellis osteopetrosis
    - 2. Dentinogenesis imperfecta
    - 3. Tarabochia adhesion deficiency syndrome
    - 4. Tjaderhane syndrome
    - 5. Chondrolytic syndrome
    - 6. Hurler's syndrome
    - 7. Hunter's syndrome
    - 8. Hunter's growth retardation
    - 9. Chondrolytic syndrome
    - 10. Hunter's syndrome (type II and III)
    - 11. Hurler's syndrome
    - 12. Other
  - C. Not otherwise specified (NOS)
- IV. Hereditary Periodontal Disease
  - A. Hereditary fibrosing gingivitis (HFG)
  - B. Hereditary ulcerative periodontitis (HUP)
- V. Absence of the Periodontium
  - A. Congenital absence
  - B. Periodontal atrophy
  - C. Resected alveoli
- VI. Periodontitis: Periodontitis: Periodontitis: Periodontitis
  - A. Central periodontitis: severe
  - B. Developmental or Acquired: Cystic, fibrous and Dissecting
- VII. Localized tooth-related factors that modify or predispose to periodontitis: gingivitis/periodontitis
  - 1. Tooth fracture
  - 2. Dental restorations/prostheses
  - 3. Root fracture
  - 4. Caries root exposure: an abutment tooth
  - 5. Parodontal infection and conditions around teeth
    - A. Local or regional infection
    - B. Interproximal gingivitis
    - 2. Lack of functional papilla
    - 3. Occlusal malocclusion
    - 4. Abnormal keratinized tissue
    - 5. Gingival excess
      - a. gingivitis
      - b. increased gingival margin
      - c. excessive gingival display
      - d. gingival enlargement (GIA) (GIA-1 and GIA-2)
    - 6. Abnormal color
  - C. Plaque-induced gingivitis and conditions or conditions: gingivitis
    - 1. Altered occlusal contact: ridge deficiency
    - 2. Lack of gingival contact tissue
    - 3. Gingival recession
    - 4. Abnormal keratinized tissue
    - 5. Occlusal malocclusion
    - 6. Abnormal color
  - D. Occlusal trauma
    - 1. Primary occlusal trauma
    - 2. Secondary occlusal trauma

### 1999 CLASSIFICATION Criticisms

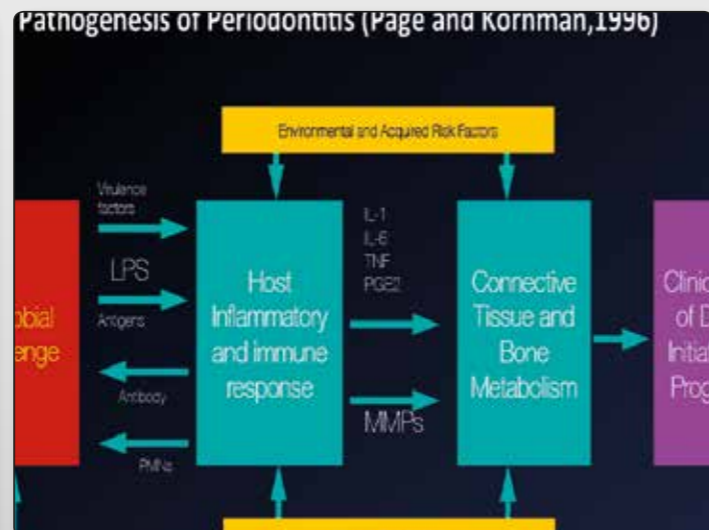
The diagnosis based on the clinical and radiographic examination is that of **generalised severe chronic periodontitis**. I have made arrangements to see this patient for a course of initial non-surgical periodontal treatment and will endeavour to report back to you after this has been completed and reviewed. Given this patient's relatively young age it may be necessary to revise the diagnosis to that of **aggressive periodontitis** and indeed we will seek to treat her comprehensively to eliminate pocketing as much as is possible.

### CRITICISMS OF 1999 CLASSIFICATION

- Classification had too many items and poorly rationalised
- Aggressive disease was well defined and chronic was defined almost by exclusion
- Many of the criteria for aggressive disease were not validated in studies
- Overlap and lack of precision of disease definitions meant many clinicians not always comfortable with their diagnosis

### CRITICISMS OF 1999 CLASSIFICATION

- No definition of health, or what constitutes the 'normal patient'
- No way to classify a treated patient – once diseased always diseased
- No mention of implants
- Developmental/acquired category mixed up mucogingival defects with tooth defects (eg enamel pearls) with tooth pathology (eg root fracture)



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# STAGING AND GRADING OF PERIODONTITIS

Continues from page 6.

### A change in the fundamental ideas of periodontology

- We are holobionts

### Fundamental shift in thinking

- Plaque causes reversible gingival inflammation – still true
- Low levels of plaque commensurate with health *in spite of* generating measurable responses
- Plaque does not cause periodontitis
- Bugs drive the immune system AND the immune system drives the bugs
- Patients are individuals with risk profiles – “precision medicine”

i woz 'ere ☺

### HOW could we classify disease?

- Pathogenesis?
  - Symbiosis of plaque is the healthy state
  - Undisturbed plaque in some patients does not result in disease
- Name it after the bugs?
  - Disease is far more complex
  - Research into what are the bugs doing suggests that they are doing the same thing everywhere!
- Name it after the host response?
  - Probably correct but too complicated and information not sufficient

### Contemporary model of host-microbe interactions in the pathogenesis of periodontitis - Chapple 2015

### Workshop on Classification, November 2017, Chicago

### HOW could we classify disease

- Aetiology
- Pathogenesis
- Clinical description of pathology
- Severity
- Extent
- Biomarkers
- Patient outcomes

### Workgroups

- 1: Periodontal Health and Gingival Diseases and Conditions
- 2: Periodontitis
- 3: Periodontal manifestations of systemic diseases and developmental and acquired conditions
- 4: Peri-implant diseases and conditions

Total of 23 papers which collectively redefine periodontology

### Group 1 - Periodontal Health and Gingival Diseases and Conditions

- Pristine health defined histologically
- Almost never seen clinically – not “normal”
- Gingival health: *An absence of clinically detectable inflammation – There is a biological level of immune surveillance consistent with clinical gingival health and homeostasis.*
- Case versus Site of health/inflammation (gingivitis)
- Clinical health can be restored following treatment of gingivitis or periodontitis

### Classification of health

- Clinical Gingival Health on Intact Periodontium
- Clinical Gingival Health on Reduced Periodontium
  - Stable periodontitis patient (successful treatment)
  - Non-periodontitis patient (crown lengthening, toothbrush trauma...)
- INTACT means absence of detectable attachment or bone loss

Continues on page 10.

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 9.

## Classification of Plaque Induced Gingivitis

- On intact and reduced periodontium
- Local and systemic risk factors
- Case defined by bleeding alone - <10%, 30% rule

## Classification of non-Plaque Induced Gingivitis

- Rationalised as a surgical sieve – Genetic/Developmental, Specific Infections, Inflammatory & Immune, Reactive, Neoplastic, Endocrine, nutritional and metabolic disorders, Traumatic, Pigment

Intact Periodontium	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets)	≤ 3mm	≤ 3mm
Bleeding on probing	<10%	Yes
Radiological bone loss	No	No

## Findings of position papers of Group II

- 3. On a population basis, the mean rates of periodontitis progression are consistent across all observed populations throughout the world.

## Findings of position papers of Group II

- 4. There is evidence, however, that specific segments of the population exhibit different levels of disease progression, as indicated by greater severity of clinical attachment loss (CAL) in subsets of each age cohort relative to the majority of individuals in the age cohort.

## OK what about the main bit?

2017 W 2017 W 2017 WORLD WORKSHOP WILEY *Journal of Clinical Periodontology*

Age-related Findings of a 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions

Maurizio Tonetti

Panos N. Papanou<sup>1</sup> | Mariano Sanz<sup>2</sup> | Nurcan Buduneli<sup>3</sup> | Thomas Dietrich<sup>4</sup> | Magda Feres<sup>5</sup> | Daniel H. Fine<sup>6</sup> | Thomas F. Flemmig<sup>7</sup> | Raul Garcia<sup>8</sup> | William V. Giannobile<sup>9</sup> | Filippo Graziani<sup>10</sup> | Henry Greenwell<sup>11</sup> | David Herrera<sup>2</sup> | Richard T. Kao<sup>12</sup> | Moritz Ketschull<sup>13</sup> | Denis F. Kinane<sup>14</sup> | Keith L. Kirkwood<sup>15</sup> | Thomas Koche<sup>16</sup> | Kenneth S. Korman<sup>17</sup> | Purnima S. Kumar<sup>17</sup> | Bruno G. Loos<sup>18</sup> | Eli Machtei<sup>19</sup> | Huanxin Meng<sup>20</sup> | Andrea Mombelli<sup>21</sup> | Ian Needleman<sup>22</sup> | Steven Offenbacher<sup>23</sup> | Gregory J. Seymour<sup>24</sup> | Ricardo Teles<sup>14</sup> | Maurizio S. Tonetti<sup>25</sup>

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## Conclusions from position papers of Group II

- 5. A classification system based only on disease severity fails to capture important dimensions of an individual's disease, including the complexity that influences approach to therapy, the risk factors that influence likely outcomes, and level of knowledge and training required for managing the individual case.

## Conclusion 1 - Lets keep ANUG and ANUP

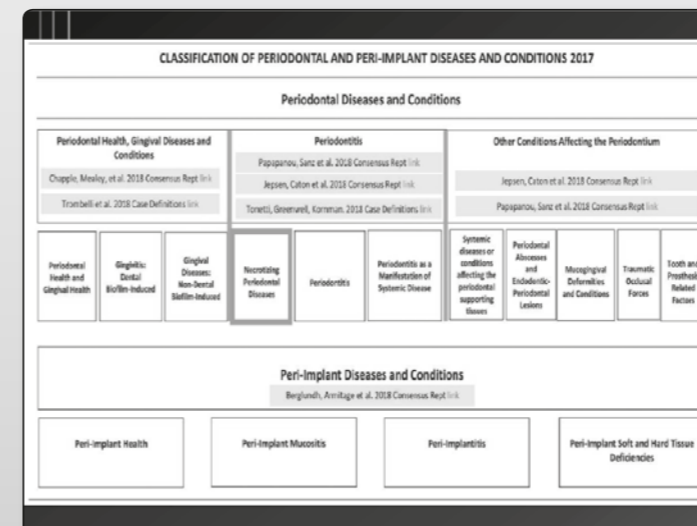
- There is sufficient evidence to consider necrotizing periodontitis a separate disease entity.
- Evidence
  1. a distinct pathophysiology characterized by prominent bacterial invasion and ulceration of epithelium
  2. rapid and full thickness destruction of the marginal soft tissue resulting characteristic soft and hard tissue defects
  3. prominent symptoms
  4. rapid resolution in response to specific antimicrobial treatment.

## Findings of position papers of Group II

- 1. There is no evidence of specific pathophysiology that enables differentiation of cases that would currently be classified as aggressive and chronic periodontitis or provides guidance for different interventions.

## Findings of position papers of Group II

- 2. There is little consistent evidence that aggressive and chronic periodontitis are different diseases, but there is evidence of multiple factors, and interactions among them, that influence clinically observable disease outcomes (phenotypes) at the individual level. This seems to be true for both aggressive and chronic phenotypes.



## Conclusion 2 – Lets keep Perio as a Manifestation of Systemic disease

- Systemic diseases which severely impair host response
- Primary diagnosis should be the systemic disease according to ICD
- For the time being, periodontitis observed in poorly controlled diabetes is a co-morbidity (two primary diagnoses)

Continues on page 12.

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 11.

CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017											
Periodontal Diseases and Conditions											
Periodontal Health, Gingival Diseases and Conditions <small>Chapple, Mesley, et al. 2018 Consensus Report <a href="#">link</a> Trenbath et al. 2018 Case Definitions <a href="#">link</a></small>			Periodontitis <small>Papapanou, Sanz et al. 2018 Consensus Report <a href="#">link</a> Jepson, Caton et al. 2018 Consensus Report <a href="#">link</a> Tonetti, Greenwell, Kornman. 2018 Case Definitions <a href="#">link</a></small>			Other Conditions Affecting the Periodontium <small>Jepson, Caton et al. 2018 Consensus Report <a href="#">link</a> Papapanou, Sanz et al. 2018 Consensus Report <a href="#">link</a></small>					
Periodontal Health and Gingival Health	Gingivitis: Dental Biofilm Induced	Gingival Diseases: Non-Dental Biofilm Induced	Necrotizing Periodontal Diseases	Periodontitis	Periodontitis as a Manifestation of Systemic Disease	Systemic diseases or conditions affecting the periodontal supporting tissues	Periodontal Abscesses and Endodontic-Periodontal Lesions	Mucogingival Deformities and Conditions	Traumatic Occlusal Forces	Teeth and Prosthesis Related Factors	
Peri-Implant Diseases and Conditions											
<small>Berglundh, Armitage et al. 2018 Consensus Report <a href="#">link</a></small>											
Peri-implant Health	Peri-implant Mucositis	Peri-implantitis	Peri-implant Soft and Hard Tissue Deficiencies								

## Conclusion 3 – throw away Aggressive v Chronic!

- There is insufficient evidence (despite heavy research since 1999) to say Chronic and Aggressive Periodontitis are different diseases.
- Arguably the most controversial change in the classification

CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017											
Periodontal Diseases and Conditions											
Periodontal Health, Gingival Diseases and Conditions <small>Chapple, Mesley, et al. 2018 Consensus Report <a href="#">link</a> Trenbath et al. 2018 Case Definitions <a href="#">link</a></small>			Periodontitis <small>Papapanou, Sanz et al. 2018 Consensus Report <a href="#">link</a> Jepson, Caton et al. 2018 Consensus Report <a href="#">link</a> Tonetti, Greenwell, Kornman. 2018 Case Definitions <a href="#">link</a></small>			Other Conditions Affecting the Periodontium <small>Jepson, Caton et al. 2018 Consensus Report <a href="#">link</a> Papapanou, Sanz et al. 2018 Consensus Report <a href="#">link</a></small>					
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Peri-Implant Diseases and Conditions											
<small>Berglundh, Armitage et al. 2018 Consensus Report <a href="#">link</a></small>											
Peri-implant Health	Peri-implant Mucositis	Peri-implantitis	Peri-implant Soft and Hard Tissue Deficiencies								

## Conclusion 4 – Let make it more personal!

- Current multifactorial models of disease applied to periodontitis appear to account for a substantial part of the phenotypic variation observed across cases as defined by clinical parameters.
- Small segment has severe disease while most have mild/moderate disease
- Twin studies suggest large portion of variance is genetic
- Future research may well increase knowledge of disease specific mechanisms and the multifactorial interactions leading to specific phenotypes
- Multi-dimensional profiles combining biological and clinical parameters are emerging - that better define phenotypes – helping us create individual diagnoses and care plans

## Conclusion 4 – Let make it more personal!

- Current multifactorial models of disease applied to periodontitis appear to account for a substantial part of the phenotypic variation observed across cases as defined by clinical parameters.
- Possible future personalisation of medicine by knowledge of treatment responses in particular disease phenotypes.

## Clinical Definition

- AAP/CDC and EFP case definitions were examined and a single definition suggested:
- Periodontitis is defined by clinical attachment loss as measured with a standardized probe referenced to the CEJ of erupted teeth
- Keeping in mind
  - Some conditions other than periodontitis can be responsible for attachment loss
  - definitions based on bone loss will miss mild/moderate disease and should be limited only to erupted teeth in mixed dentitions as reference to CEJ is not possible

Continues on page 15.



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# STAGING AND GRADING OF PERIODONTITIS

Continues from page 12.

## What constitutes a case of clinical periodontitis?

- Interdental CAL is detectable at two or more non-adjacent teeth
- OR
- Buccal/oral CAL of 3mm or more with pocketing of more than 3mm at two or more teeth
- AND
- The observed CAL cannot be ascribed to non-periodontal causes such as trauma/caries/tooth malposition/endo lesion/root fracture

## "detectable"

- Keeps a consistency of histological and clinical definitions
- Recognises that clinical experience (operator training and skill) and conditions (restorative margins/calculus/tissue tightness) may affect the ability to detect CAL
- In the very early stages of disease error in the probe and the operator will lead to misclassification
- Cannot be used epidemiologically where a specific threshold based on measurement error will need to be set.
- Thresholds chosen will affect sensitivity and specificity

## What else should a classification capture?

- Severity of disease
- Take account of tooth losses (attributable to periodontitis)
- Complexity of management
- Extent of disease
- Rate of progression
- Risk factors
- Interrelationship with general health

## Staging

- Approach used for many years in oncology
- Relies on severity and extent at presentation but also introduces the dimension of complexity of management
- Allows us to define the disease state at various points in time
- Easily communicated
- May be a factor in assessing prognosis
- A step towards personalized precision medicine

## Grading

- Rate of periodontitis progression
- Recognised risk factors for progression
- Risk assessment of the individual case affecting systemic health

TABLE 1 Primary goals in staging and grading a patient with periodontitis

Staging a Periodontitis Patient
<ul style="list-style-type: none"> <li>• Goals</li> <li>• Classify Severity and Extent of an individual based on currently measurable extent of destroyed and damaged tissue attributable to periodontitis</li> <li>• Assess Complexity: Assess specific factors that may determine complexity of controlling current disease and managing long-term function and esthetics of the patient's dentition</li> </ul>
Grading a Periodontitis Patient
<ul style="list-style-type: none"> <li>• Goals</li> <li>• Estimate Future Risk of periodontitis progression and responsiveness to standard therapeutic principles, to guide intensity of therapy and monitoring</li> <li>• Estimate Potential Health Impact of Periodontitis on systemic disease and the reverse, to guide systemic monitoring and co-therapy with medical colleagues</li> </ul>

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Simplantology, in Everything We Do



Continues on page 16.



# STAGING AND GRADING OF PERIODONTITIS

Continues from page 15.

TABLE 2 Framework for staging and grading of periodontitis

		Disease Severity and Complexity of Management			
		Stage I: Initial periodontitis	Stage II: Moderate periodontitis	Stage III: Severe periodontitis with potential for additional tooth loss	Stage IV: Advanced periodontitis with extensive tooth loss and potential for loss of dentition
Evidence or risk of rapid progression, anticipated treatment response, and effects on systemic health	Grade A	Individual Stage and Grade Assignment			
	Grade B				
	Grade C				

## Stage I periodontitis

- The borderland between gingivitis and periodontitis
- Earliest attachment loss in response to persistence of gingival inflammation
- Not just an early diagnosis – at an early age this would represent increased susceptibility to disease initiation
- Population level – maybe a cost-effective point for simple intervention
- May be a good target area for biomarkers or new imaging technologies to supersede limitation of CAL detection with a probe

## Stage II periodontitis

- Established periodontitis
- Clearly identifiable from probing examination
- Straightforward management with SPPC and debridement
- Expect disease arrest – evaluation of this response to standard treatment is essential as it may guide us to alter the grade and intensify treatment for non-responders

## Stage III periodontitis

- Significant damage has occurred
- Tooth loss may occur in the absence of treatment
- Deep lesions extending to the middle of the roots
- Intrabony defects, furcation involvement, history of periodontal tooth loss/exfoliation and ridge defects may complicate the management
- However the overall picture is of a functional dentition which does not require rehabilitation beyond the management of what we would previously call severe periodontitis

## Stage IV periodontitis

- Disease has caused considerable damage including possible tooth loss leading to loss of masticatory or aesthetic function
- Dentition is at risk of being lost
- Deep periodontal lesions extending to the apical portions of the roots
- History of tooth losses
- Mobility due to secondary occlusal trauma
- Posterior bite collapse, drifting, splaying
- Dentition requires rehabilitation over and above periodontal treatment

TABLE 3 Periodontitis stage – Please see text and appendix A (in online Journal of Clinical Periodontology) for explanation

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

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✓ CHEEKS

✓ TONGUE

✓ TEETH

- Regular toothpastes<sup>†</sup> only protect the hard tissue, which is 20% of the mouth<sup>2</sup>
- The remaining 80% of the mouth is the tongue, cheeks, and gums, which can provide a bacteria reservoir for plaque biofilm recolonization

WHY SETTLE FOR 20% WHEN YOU CAN OFFER PATIENTS PROTECTION TO 100% OF THE MOUTH'S SURFACES?



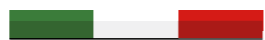
\*In addition to fluoride for cavity protection, Colgate Total® provides 12-hour antibacterial protection for teeth, tongue, cheeks, and gums.

<sup>†</sup>Defined as non-antibacterial fluoride toothpaste.

References: 1. Fine DH, Sreenivasan PK, McKiernan M, et al. *J Clin Periodontol.* 2012;39:1056-1064. 2. Collins LMC, Dawes C. *J Dent Res.* 1987;66:1300-1302.

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Continues on page 19.



## DENTAL INSTRUMENTS

It is a fact: women's hands differ from men's hands. And women involved in dentistry require specific instruments. Until now these instruments were designed for male hands—larger, with longer fingers and stronger muscle build. Asa Dental decided to change things, giving women the ideal instruments for their hands. So it created AsaLady, the new line of dental instruments designed and tailored specifically for female hands. With different handles and weights. Tailor-made for women.

### The starting point

Hands smaller by 10 to 12%. Fingers smaller by 13 to 15%.

### The differences

Smaller handles. Gripping points designed based on the average size of female hands. Smaller and lighter handles.

### The result

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- More comfort for tendons and muscles.

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# STAGING AND GRADING OF PERIODONTITIS

Continues from page 16.

Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	Mild	Mod	Sev	Adv
Grade	A	B	C	D
Complexity	Low	Mod	Sev	Adv
Diagnosis and Risk Factors	...	...	...	...

### Grading

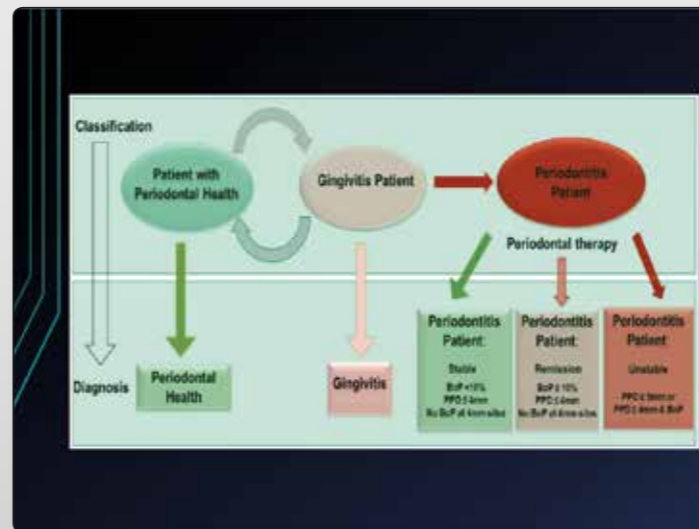
- Validated risk assessment tools can estimate risk of progression and tooth loss
- Previous classifications had the concept of grade embedded as separate specific forms (aggressive, early onset, rapidly progressive) which placed focus on the identification of a "separate" disease rather than the factors leading to the progression

### What do we look at to choose a grade?

- Recognised risk factors (smoking / diabetic control / fam hist)
- Disease severity at presentation as a function of age
  - Bone loss % divided by age of patient already used in the PRA
  - CAL percentiles for populations
  - Such calculations need to account for tooth losses otherwise are worthless
- Biomarkers may be introduced into the system when validated
- Responsiveness to treatment

TABLE 4. Periodontitis grade. Please see text and appendix 1 for the same of (class of Periodontitis) for categorisation

Periodontitis grade	Grade A	Grade B	Grade C
Primary criteria	...	...	...
Grade modifiers	...	...	...
Risk of systemic impact of periodontitis	...	...	...
Biomarkers	...	...	...



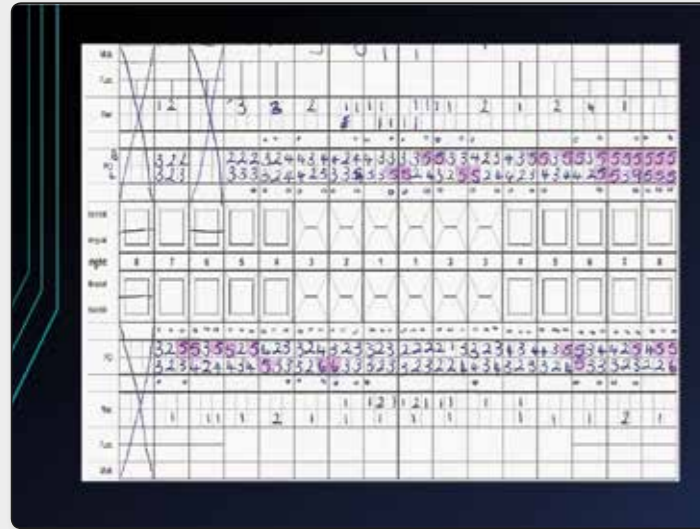
### CASE 1 - 2013

- M
- 37 years
- Clear MH
- Recession

Continues on page 20.

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 19.



**TABLE 3** Periodontitis stage - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis stage	Stage I	Stage II	Stage III	Stage IV
Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
Severity	Coronal third (<15%)	Coronal third (15% to 33%)	Extending to middle or apical third of the root	Extending to enamel or apical third of the root
Tooth loss	No tooth loss due to periodontitis	No tooth loss due to periodontitis	Tooth loss due to periodontitis of ≥4 teeth	Tooth loss due to periodontitis of ≥5 teeth
Complexity	Local	Local	In addition to stage II complexity: Probing depth ≥6 mm Furcation involvement Class II or III Mucosae ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defect Bite collapse, drifting, flaring Less than 20 remaining teeth (10 opposing pairs)
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or multifactor patient		

**TABLE 4** Periodontitis grade - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis grade	Grade A: Slow rate of progression	Grade B: Moderate rate of progression	Grade C: Rapid rate of progression
Direct evidence of progression	Longitudinal data (including the bone level or CBIL) showing a rate of progression of ≤0.25 mm over 5 years	Longitudinal data showing a rate of progression of >0.25 to 1.0 mm over 5 years	Longitudinal data showing a rate of progression of ≥1.0 mm over 5 years
Primary criteria	Stable	Stable	Stable
Secondary criteria	Stable	Stable	Stable
Grade modifiers	Stable	Stable	Stable
Risk of systemic impact of periodontitis <sup>a</sup>	Low	Low	Low
Modifiers	1	2	3

**TABLE 3** Periodontitis stage - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis stage	Stage I	Stage II	Stage III	Stage IV
Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
Severity	Coronal third (<15%)	Coronal third (15% to 33%)	Extending to middle or apical third of the root	Extending to enamel or apical third of the root
Tooth loss	No tooth loss due to periodontitis	No tooth loss due to periodontitis	Tooth loss due to periodontitis of ≥4 teeth	Tooth loss due to periodontitis of ≥5 teeth
Complexity	Local	Local	In addition to stage II complexity: Probing depth ≥6 mm Furcation involvement Class II or III Mucosae ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defect Bite collapse, drifting, flaring Less than 20 remaining teeth (10 opposing pairs)
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or multifactor patient		

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Direct evidence of progression	Longitudinal data (including the bone level or CBIL) showing a rate of progression of ≤0.25 mm over 5 years	Longitudinal data showing a rate of progression of >0.25 to 1.0 mm over 5 years	Longitudinal data showing a rate of progression of ≥1.0 mm over 5 years
Primary criteria	Stable	Stable	Stable
Secondary criteria	Stable	Stable	Stable
Grade modifiers	Stable	Stable	Stable
Risk of systemic impact of periodontitis <sup>a</sup>	Low	Low	Low
Modifiers	1	2	3

**CASE 1 - 2018**

- Stable Periodontitis Stage I Grade A
- Gingival health with reduced attachment

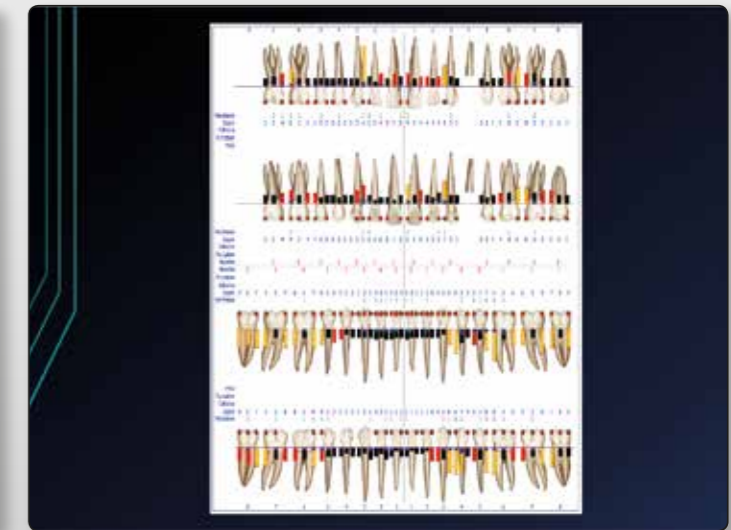
**CASE 2**

- M
- 37 years
- Clear MH
- Ex-Smoker – quit 2 years ago – low exposure
- Family history of perio

**CASE 1 - 2013**

- Periodontitis Stage II Generalised Grade B

**CASE 1 - 2018**



Continues on page 24.

# How to Reverse Type 2 Diabetes Mellitus in the Obese Patient

Comments by Dr Charles E. Corney,  
Medical Researcher

We have known about diabetes for 3000 years and also how it can be controlled. However, a simple, permanent cure or reversal has not been obtained until very recently.

In 2014, Roy Taylor, Professor of Medicine at Newcastle University, UK, publicly announced an amazing message from his research on many patients (published in the Lancet medical journal) that diabetes type 2 (T2DM) in the obese patient can be reversed permanently by using a specially designed very low (600) calorie diet daily for just 8 weeks, inducing an ideal weight loss of at least 15kg down to a normal Body Mass Index of around 27. If the history of this type of diabetes is longer than 10 years, this technique may not work.

Prof Taylor's explanation is very simple. Such a patient commonly has a history of eating too much junk food. These excess carbohydrate calories are converted by insulin to fat. Consequently, the patient is obese with accumulation of fat in and around the liver which damages insulin, preventing glucose entering the cells of the body (known as insulin resistance).

As a result, there is pooling of both insulin and glucose. This excess of insulin deposits more liver fat which now starts to secrete excessively concentrated immune chemicals (known as autoimmunity cytokines) which cause further liver fat deposition, increased cholesterol and triglycerides, raised blood pressure, and areas of chronic inflammation in many parts of the body. These reactions are collectively known as the Metabolic Syndrome, which, with the ever-rising blood glucose levels, slowly changes into T2DM.

Once this stage is reached, it cannot be reversed by conventional glucose lowering drugs such as metformin or insulin, or by eating a lowish calorie weight reducing diet of 1200 calories daily. Consequently, the diabetes becomes a relentlessly progressive disease.

However, Prof Taylor's scheme of drastically starving (600 calorie diet daily) the fat from the liver which, by lowering the insulin resistance, does reverse and switch off the T2DM and its side effects permanently.

Next, the excess abdominal fat accumulates also in the pancreas, killing the insulin secreting cells, so there is no insulin available. The patient then requires permanent insulin injections.

Continues on page 36.

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On the other hand if your policy is already insured under the scheme, you need to ensure that:

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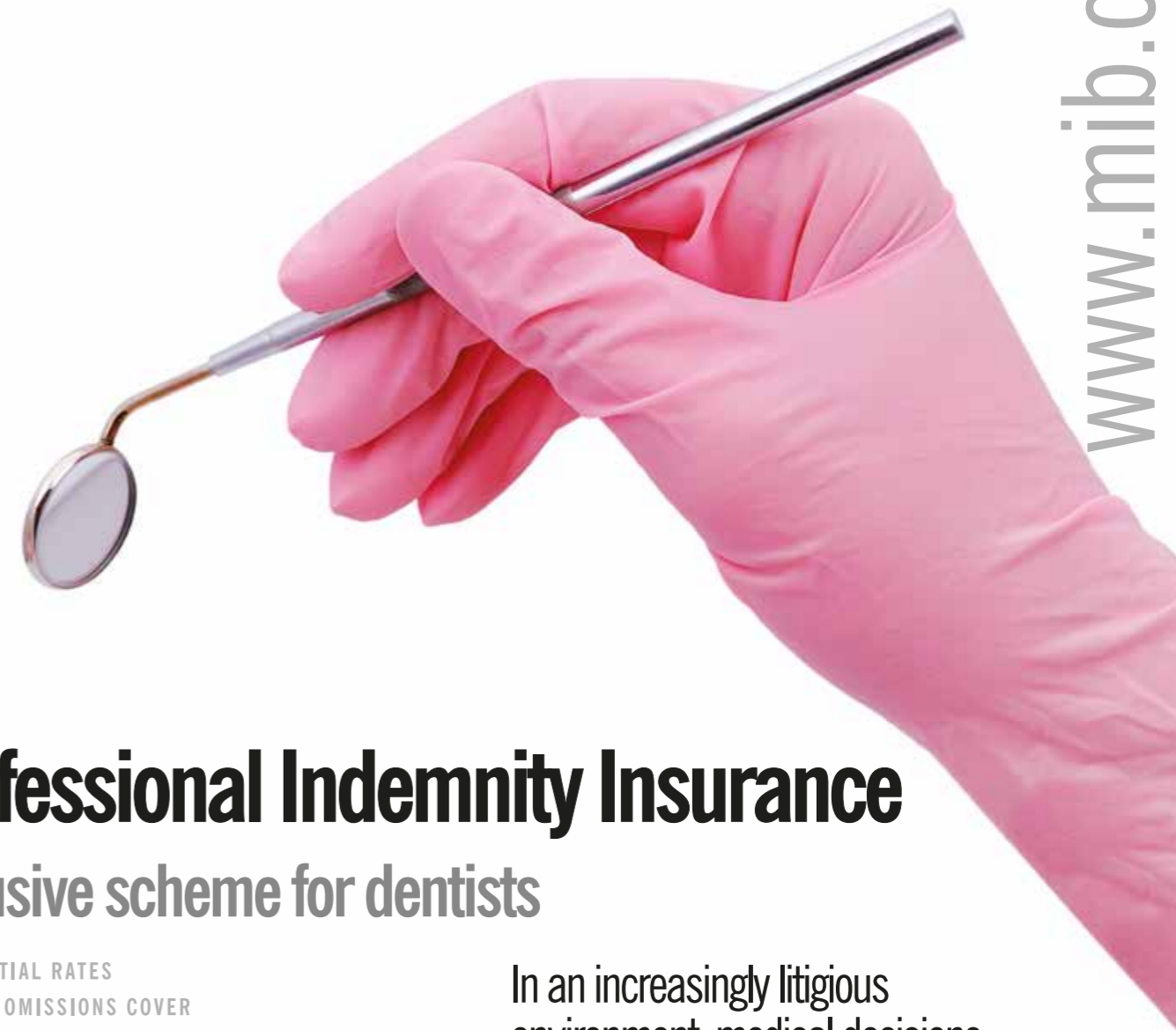
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# Help keep your patients on a journey to healthy gums



Your professional advice

At least **50%** of adults suffer from gingivitis globally,<sup>1</sup> but **2 out of 3** take no action<sup>2</sup>

## Periodontal disease impacts daily life

Patient insight research shows that gingivitis can have a negative impact on daily life causing anxiety, embarrassment and affecting social life, especially when symptoms become noticeable to others.<sup>3</sup>

**parodontax® toothpaste helps to free patients from the wider effects of gingivitis.<sup>3</sup>**

After 30 days, patients reported:



**Less anxiety**

2 out of 3 patients no longer worried about their gum health<sup>4</sup>



**Better social life**

2 out of 3 patients no longer avoided social situations<sup>4</sup>



**Greater confidence**

2 out of 3 patients were more confident<sup>4</sup>

## Treat and Maintain

In addition to good oral hygiene and professional advice, patients with, or susceptible to gingivitis may benefit from the addition of **parodontax®** for their optimum gum health.<sup>5,6</sup>

**4X** greater plaque removal\*  
**48%** greater reduction in bleeding gums\*<sup>7</sup>

**Recommend parodontax® toothpaste to help patients maintain their optimal gum health between dental visits**



Helps stop and prevent bleeding gums

Healthy gums

\*Compared to a regular toothpaste following a professional clean and 24 weeks' twice-daily brushing.  
References: 1. CDC Perio 2016; Half of American Adults have Periodontal disease. 2. Data on file, GSK, parodontax® Segmentation, August 2015. 3. Data on file, GSK, Firefish: Putting the patient first. Life impact of gum disease, March 2016. 4. Data on file, GSK, Taste Adoption study (n=600), Italy 2016. 5. Kakar A, et al. Evaluate the Efficacy of Different Concentrations of Sodium Bicarbonate Toothpastes. IADR General Session and Exhibition, Cape Town, South Africa, 2014. Abstract No: 754. 6. Data on file, GSK, RH01530, January 2013. 7. Data on file, GSK, RH02434, January 2015.  
Prepared November 2017. CHMLT/CHPDX/0002/17

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 21.



TABLE 3 Periodontitis stage - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

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

## Appendix A

- CAL of ≤ 2 is considered initial disease
- CAL of 3-4 mm is established disease
- CAL of ≥ 5 mm designates severe destruction
- CAL of ≥ 8 mm represents very severe disease
- Corresponding values for RBL are
  - < 15%
  - 15-33%
  - 33-66%
  - > 66%

TABLE 3 Periodontitis stage - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis stage	Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss: 1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
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Complexity	Local: Maximum probing depth ≤4 mm; Mostly horizontal bone loss	Maximum probing depth ≤5 mm; Mostly horizontal bone loss	In addition to stage II complexity: Probing depth ≥6 mm; Vertical bone loss ≥3 mm; Furcation involvement Class II or III; Moderate ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction; Secondary occlusal trauma (tooth mobility degree ≥2); Severe ridge defect; Bite collapse, drifting, flaring; Less than 20 remaining teeth (10 opposing pairs)
	Extent and distribution: Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern		

TABLE 4 Periodontitis grade - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis grade	Grade A: Slow rate of progression	Grade B: Moderate rate of progression	Grade C: Rapid rate of progression	
Primary criteria	Direct evidence of progression: Longitudinal data (radiographic bone loss or CAL): Evidence of no loss over 5 years	<1.5	0.25 to 1.0	>1.0
	Indirect evidence of progression: 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	Smoking: Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day	
	Risk factors: Diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes	
Risk of systemic impact of periodontitis	Inflammatory markers: High sensitivity CRP (hsCRP): <1 mg/L	1 to 3 mg/L	>3 mg/L	
Biomarkers	Indicators of CAL/bone loss: Saliva, gingival crevicular fluid, serum	?	?	

## CASE 2

- Periodontitis, Stage IV Generalised, Grade C

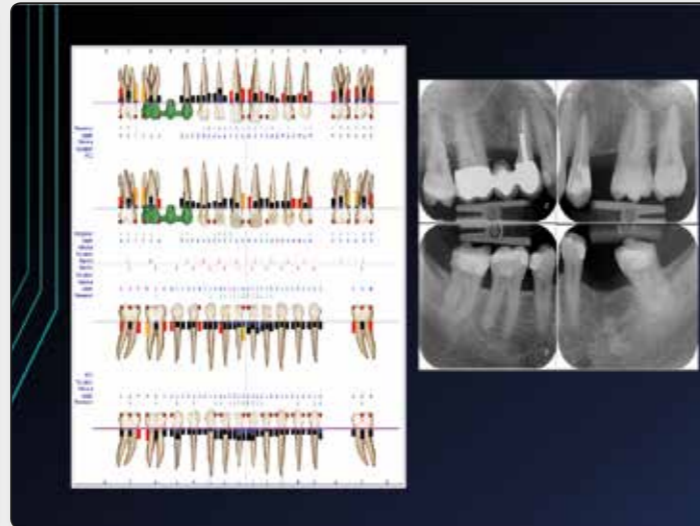
Continues on page 26.

# STAGING AND GRADING OF PERIODONTITIS

Continues from page 25.

## Case 3

- F
- 55 years
- Type I Diabetes HbA1C 6.8%
- Non-smoker
- No Family History



**TABLE 3** Periodontitis stage - Please see text and appendix A in online Journal of Clinical Periodontology for explanation

Periodontitis stage	Stage I	Stage II	Stage III	Stage IV
Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
Severity	Coronal third (<15%)	Coronal third (15 to 30%)	Extending to middle or apical third of the root	Extending to incisal or apical third of the root
Teeth lost	No teeth lost due to periodontitis	No teeth lost due to periodontitis	Teeth lost due to periodontitis of ≥4 teeth	Teeth lost due to periodontitis of ≥5 teeth
Complexity	Local	Local	Local	Generalized
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or mixed/severe pattern		

**TABLE 4** Periodontitis grade - Please see text and appendix B in online Journal of Clinical Periodontology for explanation

Periodontitis grade	Grade A - Slow rate of progression	Grade B - Moderate rate of progression	Grade C - Rapid rate of progression
Primary criteria	Interdental CAL at site of greatest loss (mm)	Interdental CAL at site of greatest loss (mm)	Interdental CAL at site of greatest loss (mm)
Grade modifiers	Low	Medium	High
Risk of systemic impact of periodontitis*	Low	Medium	High
Secondary criteria	Low	Medium	High

## CASE 3

- Periodontitis, Stage II Generalised, Grade B

## What next?

- BSP publishing a series of webinars on the subject
- Flowchart for clinical use to integrate BPE examination into new classification – BDJ Jan 2019
- Roll-out of new classification in clinical practice

**NEW**

HELPS

# REJUVENATE GUMS & REPAIR ENAMEL

IN 2 WEEKS\*



Did you know that most oral care problems originate from gums or enamel?

It provides antibacterial action on **gums**



It defends teeth against acid erosion and helps repair the **enamel**

\*In laboratory study on weakened enamel

# MULTIDISCIPLINARY TREATMENT OF DEVELOPMENTALLY MISSING TEETH

Simon Camilleri PhD MOrth FDS

definition of congenital

congenital  
/kən'dʒenɪt(ə)l/

adjective

Of a disease or physical abnormality present from birth.

synonyms: inborn, inherited, hereditary, in (the blood), in the flesh, innate, inbred, constitutional, built-in, robust, vigorous, natural, native, original, inherent, unlearned, instinctual, down-to-earth, down-to-earth, innate

• (of a person) having a particular trait from birth or by being established habit.

synonyms: invariable, compulsive, persistent, chronic, regular, pathological, established, long-established, long-standing, habitual, confirmed, consistent, sustained, habitual, abcessive, obsessive, Man

Translations, word origin, and more definitions

## Developmentally missing teeth

A tooth is defined as developmentally missing if

- It has not erupted into the oral cavity
- It is not visible on a radiograph

## Permanent dentition Prevalence

	Males %	Females%	Total%
Europe (White)	4.6 (4.5-4.8)	6.3 (6.1-6.5)	5.5 (5.3-5.6)
North America (White)	3.2 (2.9-3.5)	4.6 (4.2-4.9)	3.9 (3.7-4.1)
North America (African American)	3.2 (2.2-4.1)	4.6 (3.5-5.8)	3.9 (3.1-4.6)
Australia	5.5 (4.4-6.6)	7.6 (6.0-9.2)	6.3 (5.4-7.2)
Saudi Arabia (White)	2.7 (2.0-3.4)	2.2 (1.2-3.1)	2.5 (1.9-3.1)
Chinese Mongoloid	6.1 (4.0-8.1)	7.7 (5.4-10.0)	6.9 (5.3-8.4)

(Polder 2004)

## Epidemiology Caucasian data

- Commonest missing tooth is 3rd molar - 20-30%
- Lower second premolar - 3.4%
- Upper lateral incisor - 2.2%

However if only 1 or 2 missing teeth then laterals most frequently absent

## Developmentally missing teeth

Definition of

- Hypodontia - 6 missing teeth or less
- Oligodontia - more than 6 missing teeth
- Anodontia - no teeth at all

These definitions do NOT include 3rd molar agenesis

## Psychosocial

Hypodontia affects quality of life

Measure is Oral-Health Related Quality of Life (OHRQoL)

- Impact of hypodontia is considerable
- Gender related (girls>boys)
- Not related to number/location of missing teeth
- Provision of prostheses improves score

## Epidemiology Local data

Survey of 530 schoolchildren in 2003

Results of published studies show:

- High prevalence of lateral incisor hypodontia (>3%)
- High prevalence of ectopic teeth, especially maxillary canines (>5.5%)

## Epidemiology Local data

In a hospital-based survey, the prevalence of hypodontia and other dental anomalies was found to be significantly higher than that in the published literature

### Features of hypodontia patients

Microdontia	May be localised or generalised; may affect the crowns and roots of teeth; is a contributor to spacing
Conical teeth	May be localised (for example, peg lateral incisors) or generalised
Ectopic eruption	Ectopic eruption into the lateral incisor space; impaction and transposition may affect the maxillary canine
Retained primary teeth	Teeth maybe infra-occluded
Reduced alveolar development	Can complicate prosthodontic management and orthodontic tooth movement
Delayed eruption of permanent teeth	Average delay in eruption is 1.5 years. Can delay onset of orthodontic treatment
Altered craniofacial morphology	Tendency towards Class III malocclusion and reduced lower anterior facial height

## Deciduous dentition Prevalence

- Less common in primary dentition – 0.4 to 0.9% (Grahnen and Granath 1961)
- Generally one or two teeth missing, no sex bias (Arte 2001)
- Strong correlation between hypodontia in primary and permanent dentitions

## Epidemiology Origins of Population

- Origins of population uncertain, possibly Phoenician/Carthaginian
- Islands invaded and left uninhabited by Tunisians about 800 AD, repopulated primarily from Sicily 100 years later
- Disease and slave - gathering raids suppressed population growth
- Military security from Knights of St John and hygiene through British Forces allowed exponential population growth from <20,000 to >450,000 in a space of 20 generations


## Epidemiology Origins of Population

- National Geographic Study in 2004- similarities to Lebanese people
- Cassar et al (2008)- Maltese most similar to Western Sicilians
- DiGaetano et al (2008)
  - Eastern Sicilians bear some similarities to Greeks
  - Western Sicilians carry Phoenician/African genetic markers

# MULTIDISCIPLINARY TREATMENT OF DEVELOPMENTALLY MISSING TEETH

Continues from page 29.

### Epidemiology Origins of Population

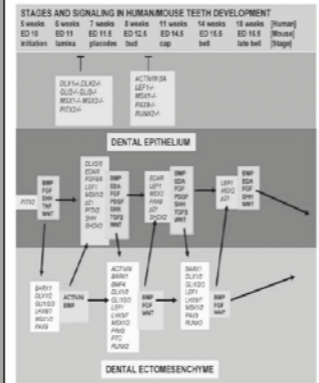


- Genes present in early population may be over or under-represented in present population through a process known as 'genetic drift'
- Purely random, these traits do not offer any selective advantage
- Phenomenon known as 'Founder Effect'. Several examples in Maltese medical literature

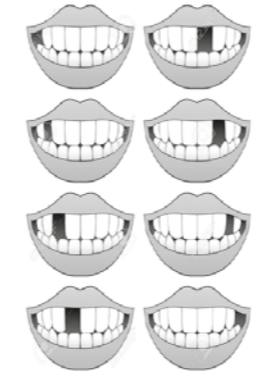
### Genetics

Dozens if not hundreds of genes directly or indirectly involved in tooth development

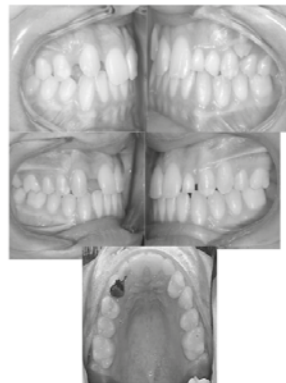
Dysfunction of any one may result in agenesis



### Management



### Interceptive




- Periodontal ligament maintains alveolar bone
- Deciduous teeth should be left in situ as long as possible in order to preserve the ridge until adulthood

### Genetics

#### Oligodontia - Syndromic

Generally inherited in a recessive pattern  
Hypohydrotic ectodermal dysplasias

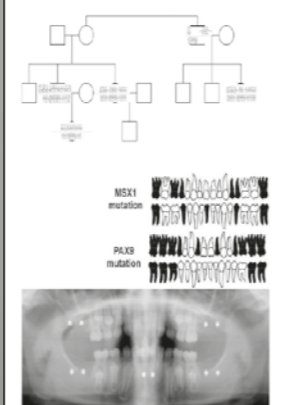
- EDA EDAR EDARADD genes
- Abnormal development of ectodermal structures including skin hair, nails, teeth, and sweat glands
- Multiple missing unerupted and and conical teeth
- Severe forms require multidisciplinary specialist treatment



### Genetics

#### Oligodontia - Nonsyndromic

- Autosomal dominant
  - MSX1
  - PAX9
  - AXIN2 - linked to colorectal cancer
  - WNT10A mutations found in 30-50% of cases of nonsyndromic oligodontia (Van den Boogaard 2017)
- Variable expression
- Variable penetrance

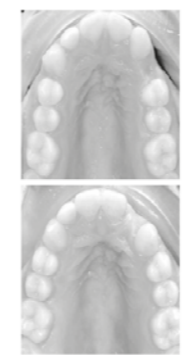


### Interceptive

#### Missing incisors

#### Site Development

- May extract Bs to allow mesial movement of canine
  - Will preserve ridge thickness
  - May then be distalised if required, **ridge will resorb by 1% in 4 years**
  - Recent extraction sites resorb at 35% in 4 years



Kishin YG. Maxillary lateral incisor implants: planning with the aid of orthodontics. Int J Oral Maxillofac Surg. 2004;62:48-56.  
Diller MS, Kishin YG. Alveolar ridge changes in patients congenitally missing mandibular second premolars. J Prosthet Dent. 1996;71:544-546.

### Interceptive

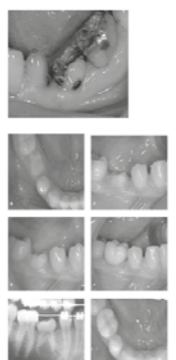
#### Retained deciduous molars

If no crowding and the crown/root in good condition may leave in situ as these last a long time.

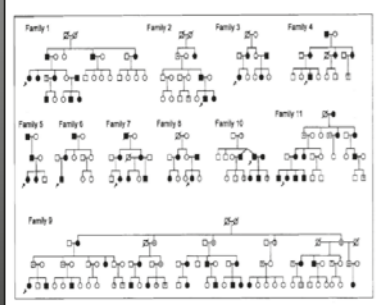
Ejkelin K, Bennett J. The long-term survival of lower second primary molars in subjects with agenesis of the premolars. Eur J Orthod. 2000;23(3):245-55.

#### Submerging deciduous molars may be built up with composite and eventually ceramic onlays

Keeping the occlusion high may break the ankylosis




### Genetics



- Grahen (1956) showed that incisor-premolar hypodontia transmission was most likely genetic and transmitted in autosomal dominant fashion
- Arte (2001) Failed to find causative gene
- No gene(s) identified as yet

### Environmental




- Disease (rickets, nutritional disturbance)
- Irradiation
- Chemotherapy
- Prevalence of hypodontia reported higher in twins – possibly due to higher nutritional demands (Keene 1971)
  - Discordance of identical twins may be due to different position in uterus leading to different blood supply – nutrition?

### Definitive treatment



### Open or close spaces Orthodontic considerations

- Space available (or can be created) for prosthesis
  - Number of missing teeth
- Incisor relation
  - Class 2 – easier to close upper spaces
  - Class 3- easier to open upper spaces
  - Opposite applies to lower arch



Continues on page 32.




# MULTIDISCIPLINARY TREATMENT OF DEVELOPMENTALLY MISSING TEETH

Continues from page 31.


### Open or close spaces Orthodontic considerations

- Symmetry and centreline in unilateral cases
- Smile line
- Gingival margin levels
- Buccal segment occlusion – can this be altered?



### Open or close spaces Orthodontic considerations

- Steep cusps/low FMA - hinders tooth movement
- Reduced number of teeth - anchorage problems
- Anterior space closure may be problematic, particularly in Class 1 or Class 3 cases
  - Intermaxillary elastics
  - Headgear
  - TADS (miniscrews)




### Orthodontic Management (What can orthodontics do to help?)

- Overbite reduction
- Space closure/opening
  - Aesthetic considerations – Golden % and gingival margins
  - Redistribute space in the arch
  - Upright teeth to aid preparation
- Extrusion/intrusion of teeth

### Overbite reduction

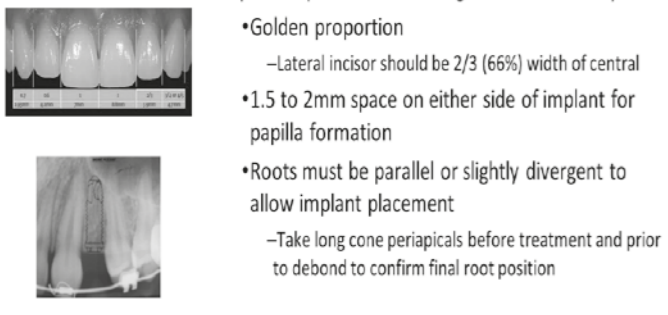
- Hypodontia cases often have reduced vertical dimensions
- Forward growth rotation makes OB reduction difficult – and makes prosthesis placement difficult
  - Biteplanes
    - During treatment
      - Ant. Biteplanes/Turboobites – Must have lower incisors
    - Retention
  - Curves of Spee
  - Intermaxillary elastics
  - Intrusion arches
  - TADS (miniscrews)



### Open or close spaces Orthodontic considerations

Space required in incisor region determined by:

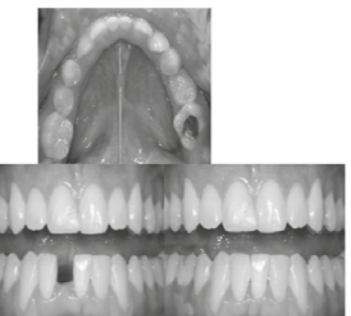
- Golden proportion
  - Lateral incisor should be 2/3 (66%) width of central
- 1.5 to 2mm space on either side of implant for papilla formation
- Roots must be parallel or slightly divergent to allow implant placement
  - Take long cone periapicals before treatment and prior to debond to confirm final root position



### Open or close spaces Orthodontic considerations

Similar considerations for the lower arch

- Close space
  - Lower centreline?
- Open space
  - 1 2 or more teeth?




### Space closure Aesthetic considerations



Several factors affecting smile aesthetics:

- Smile arc
- Symmetry
- Gingival exposure and gingival margins

### Space closure Symmetry



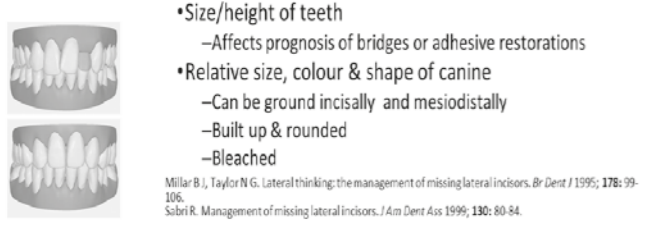
Symmetry is always a problem in unilateral cases  
Extractions may be unconventional

### Open or close spaces Restorative factors

- Size/height of teeth
  - Affects prognosis of bridges or adhesive restorations
- Relative size, colour & shape of canine
  - Can be ground incisally and mesiodistally
  - Built up & rounded
  - Bleached

Miller B. J., Taylor N. G. Lateral thinking: the management of missing lateral incisors. Br Dent J 1995; 178: 99-106.  
Sabri R. Management of missing lateral incisors. J Am Dent Ass 1999; 130: 80-84.

Diagnostic (Kesling) setup very useful to visualize results  
–patient consent



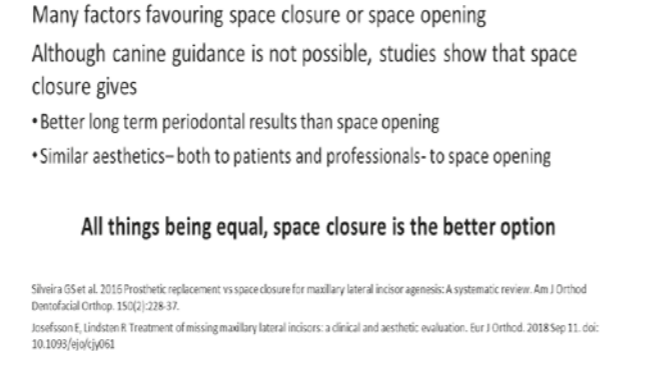
### Open or close spaces

Many factors favouring space closure or space opening  
Although canine guidance is not possible, studies show that space closure gives

- Better long term periodontal results than space opening
- Similar aesthetics – both to patients and professionals - to space opening

**All things being equal, space closure is the better option**

Silveira GS et al. 2015 Prosthodontic replacement vs space closure for maxillary lateral incisor agenesis: A systematic review. Am J Orthod Dentofacial Orthop. 150(2):228-37.  
Josefsson E, Lindsten R. Treatment of missing maxillary lateral incisors: a clinical and aesthetic evaluation. Eur J Orthod. 2018 Sep 11. doi: 10.1093/ejo/cjy061



### Space closure Gingival margins

If smile line is low, gingival aesthetics are not so important. Here:

- Canines light colour
- Reduced in size
- Contoured
- Gingival margin lower than central  
(Though the premolars still look like premolars)



### Space closure Gingival margins

Higher smile line

Missing UR central incisor

- Space closure
  - Good occlusion
  - Imperfect result
    - Central
    - Canine
    - Premolar



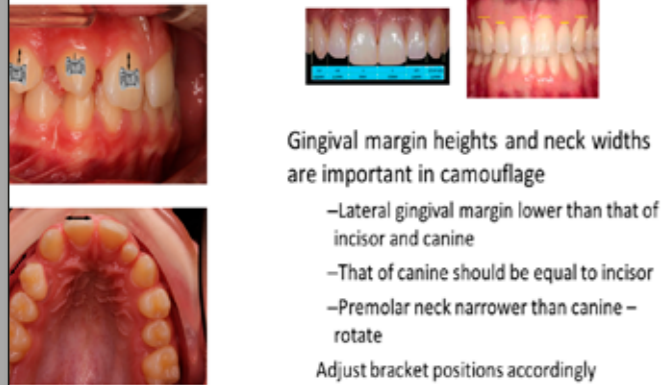
Continues on page 34.

# MULTIDISCIPLINARY TREATMENT OF DEVELOPMENTALLY MISSING TEETH

Continues from page 33.

## Space closure

### Gingival margins



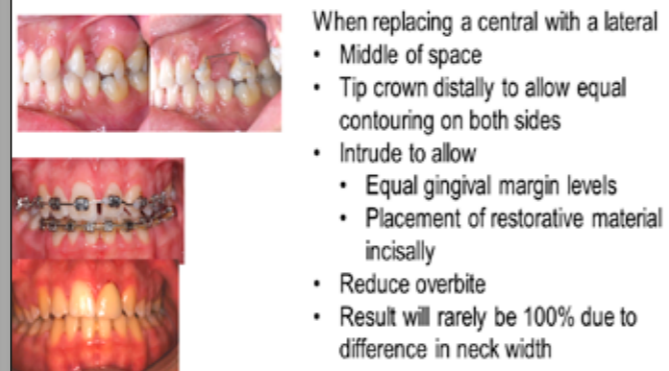
Gingival margin heights and neck widths are important in camouflage

- Lateral gingival margin lower than that of incisor and canine
- That of canine should be equal to incisor
- Premolar neck narrower than canine - rotate

Adjust bracket positions accordingly

## Space closure

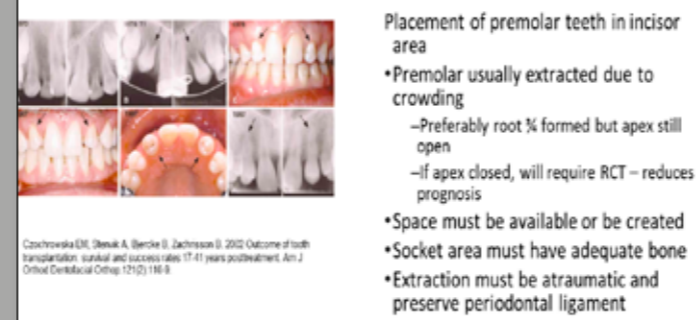
### Uprighting to aid correct preparation



When replacing a central with a lateral

- Middle of space
- Tip crown distally to allow equal contouring on both sides
- Intrude to allow
  - Equal gingival margin levels
  - Placement of restorative material incisally
- Reduce overbite
- Result will rarely be 100% due to difference in neck width

## Autotransplantation



Placement of premolar teeth in incisor area

- Premolar usually extracted due to crowding
  - Preferably root 1/4 formed but apex still open
  - If apex closed, will require RCT - reduces prognosis
- Space must be available or be created
- Socket area must have adequate bone
- Extraction must be atraumatic and preserve periodontal ligament

Czechowska E.H., Slemak A., Berke S., Zachmann G. 2002 Outcome of tooth transplantation: survival and success rates 17-41 years posttreatment. Am J Orthod Craniofac Orthop 123(2):116-9.

## Retention

### Space closure

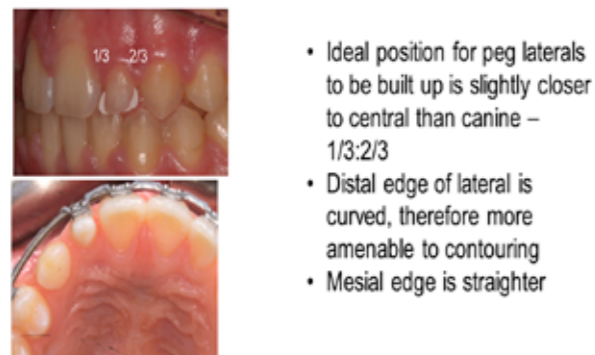
The less crowding originally, the greater tendency to re-opening of spaces

- Removable retainer
- Fixed retainer - beware of detachment\*, particularly upper arch - 58% within 4 years\*\*
- \*inadvertent movement of teeth
- Combination



\*Taner T, Aitsu M. A prospective clinical evaluation of mandibular lingual retainer survival. Eur J Orthod 2012; 34: 470-474.  
\*\*Schneider E, Ruf S. Upper bonded retainers. Angle Orthod 2011; 81: 1050-1058.

## Redistribution of space



- Ideal position for peg laterals to be built up is slightly closer to central than canine - 1/3:2/3
- Distal edge of lateral is curved, therefore more amenable to contouring
- Mesial edge is straighter

## Space opening



- Generally easier than space closure
- May need to extract/strip enamel to provide sufficient space
- May be preferable if smile line high
- 'Push/pull' mechanics
- Roots must be parallel to allow implant placement

## Retention

### Space opening

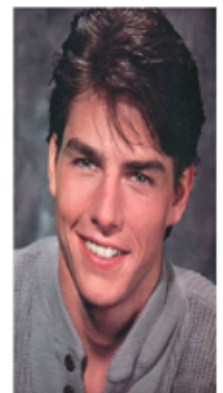
- Place prosthesis
  - Removable
    - Short term - VFR with prosthetic tooth
    - Longer term - URA with wire stops
      - Can incorporate biteplane to control OB
  - Immediate Maryland
    - Beware of single abutment cantilever



## Teamworking

### Treatment priorities

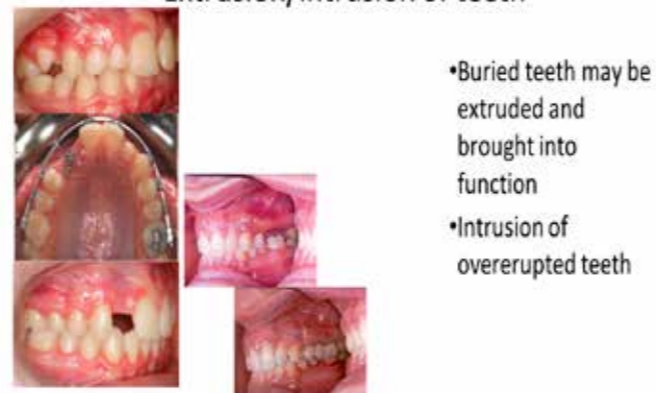
- Stabilise oral condition:
  - Good periodontal condition
  - Endodontics carried out
  - Provisional restorations
  - good idea to send to orthodontist at this stage
  - Removal of poor prognosis teeth
- Orthodontics
- Final prostheses



## Space opening



## Extrusion/intrusion of teeth



- Buried teeth may be extruded and brought into function
- Intrusion of overerupted teeth

## Teamworking

### Communication is essential

Discussion will be required, face to face or electronic

### Orthodontist needs to know:

- Spacing requirements
- Position of restorations
- Type of restorations
  - Temporary
  - Final

Orthodontist will then advise what is feasible - some things can be done - others can't



And of course



DISCUSSIONS SHOULD INVOLVE THE PATIENT

# How to Reverse Type 2 Diabetes Mellitus in the Obese Patient

Continues from page 22.

Another common cause of obesity is excessive alcohol intake. One of the features of alcohol is that the vast majority of calories in it are of the alcohol type (usually 100–200 or more calories per tin) and not of the carbohydrate type which is often low. The advertisers laud it as a low carb drink to keep down weight but, omit to say that large numbers of alcohol calories are present, so fat is deposited and the weight rises! Furthermore, the liver breaks down the alcohol into an intermediate known as acetic acid which takes much liver time to be completely broken down, resulting in the breakdown of fats being slowed. Hence fat accumulates yet again.

The high blood glucose of uncontrolled diabetes mellitus damages and narrows the arteries, producing stroke, heart attack, renal failure, blindness and limb artery blocks—sometimes requiring amputation. The same happens with nicotine when the patient smokes. The effect of both together can be devastating, so the diabetic patient should never smoke. Also, nicotine slows weight loss when attempts are made to do this.

## PROF TAYLOR'S VERY LOW CALORIE DIET (600 CALORIES/DAY)

All daily meals are replaced by sachets containing high amounts of protein

and vitamins and low amounts of carbohydrates. The protein curbs hunger and varying tastes relieve monotony. Each sachet contains 200 calories, so by using three of these per day supplies 600 calories/day. In his research, Prof Taylor used Optifast sachets, but he informs me that Exante is an equally good alternative. Do not eat any additional food, such as fruit. Drink 3 litres of water or calorie free beverages daily, but do not drink alcohol. Walk for 30 minutes per day. Avoid aggressive exercise as you will feel tired. Follow this diet for 8 weeks, with regular weight and abdominal circumference measurements. The latter is normally less than 100cm for men and 90cm for women. Regular estimations of fasting blood glucose should be performed, as it will slowly drop, requiring stepped reduction of all the medication associated with the diabetes.

At 8 weeks onwards, check if diabetes is reversed. Is the fasting blood glucose now normal? If yes, replace some or all of sachets with good organic, high protein and low carbohydrate meals—such as breakfast two eggs, lunch ham salad and dinner chicken and coloured vegetables.

If the fasting blood glucose rises above normal, revert to 3 sachets daily for 4 weeks, but keep retesting the fasting blood glucose until it stabilises on normal levels. Then we can hopefully

say that the diabetes has reversed, permitting a restart of the meals. However repeated checking for the next 2 years is still necessary. Prof Taylor informs me that almost all diabetic complications resolve, but the genetic risk to the offspring is reduced but not entirely eliminated.

## FACTS AND MYTHS ABOUT FOOD

- The curse of the carbohydrates – glucose – fattens
- Processed food, additives, alcohol fatten
- Fresh fruit fructose does not fatten but synthetic fructose sweetener does
- Fat does not fatten, but transfat (margarine/lard) does
- Cholesterol (e.g. eggs) doesn't cause cholesterol

## CONCLUSION

T2DM incidence in Malta is 10%, having almost doubled on the last ten years, and it is still increasing. So, the simple technique of its permanent reversal will have a profoundly beneficial effect on the nation's health.

## Note

1. A return to bad eating or alcohol lifestyles will cause the return of the T2DM and obesity.
2. Reversal of T2DM in the slim patient and in the T1DM, T3DM and T4DM patients cannot be performed by the above technique. ❏

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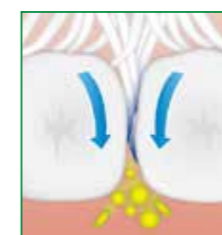
7x along the sulcus



7x beneath the sulcus



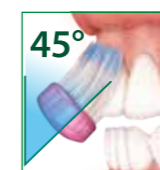
1.6x between the teeth



0.01

45°

45° angle Quad-Grip handle for optimal Bass/Modified Bass brushing technique



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\* than conventional bristles.  
1. In-Vitro Test, YRC Inc., September 2008

SUNSTAR





Dr Herbert Messina Ferrante receiving the plaque from the Dental Association of Malta in the presence of past President Dr David Muscat and Dr Adam Bartolo Vice President

## THE HON. DR HERBERT MESSINA FERRANTE

ME MOM BChD (Malta) LDS RCS (Eng) FICD (USA) FPFA (USA) FRSM (UK) FRSH

An appreciation by Dr David Muscat

As you leave a wonderfully formal black tie evening at one of the majestic Palazzos in Mdina in the cold crisp air late into the night and gently pick up speed down the Saqqajja hill under a full moon and a starry sky, you may be forgiven in failing to resist the temptation to squeeze down on your accelerator in the absence of any traffic. Approaching Notary Zarb street in Attard it is well advised to reduce your speed to that of a slow horse and carriage as there is a hawk eye speed camera opposite Messina De Ville, house of the late Dr Herbert Messina Ferrante. 'The reason I lost so many Attard Council votes' lamented Herbert once at a meeting we had attended together, 'because I was blamed for putting the camera there'.

Like him or hate him, Herbert was no ordinary person. 'Always consider yourself superior in an argument' he once told me, a fighter who tried to take no prisoners. He was brash and abrasive in his conflicts when he crossed swords, but a joy to work with when there was agreement on the goal which was required to be reached. His energy knew no bounds, even when his health started to deteriorate. His passion to do what was right and fair for the profession in general was admirable indeed. A person who always craved for the spotlight and wanted attention and importance and in a way of course he did achieve just that. Herbert's voice was the first to be heard and he was involved in a

variety of associations and political party attachments.

In local politics he was a former Partit Nazzjonalista local councillor and also President for the party's Association of Pensioners. Among the self employed, a founder and past President of the Self-Employed Union. He followed football passionately and served as President of Sliema Wanderers Football club, for a number of years during a particularly successful period. He was also in the Committee of the Malta Football Association. He also served as vice President to the Malta Football Association Council and as chairman of its disciplinary appeals Board.

He received the National Order of Merit in 2012, an honor which was very close to his heart. He received an award from The Dental Association of Malta for his contribution to dentistry as well as another award from the Medical Council of Malta for his sincere contributions during the many years of active membership on the Council's Committee. He was also awarded the French Pierre Fauchard Award for Dentistry. Dr Messina Ferrante was also awarded a Fellowship at The Murry and Leonie Guggenheim Dental Clinic in New York. He is a founder member and on the executive committee for the European Dental Society. He was honored with the

Distinguished Leadership Award. Herbert was a Knight of the Holy Order of the Sepulchur of Jerusalem, a Commander of the Order of Saint Lazarus of Jerusalem, and a Knight of the Angelic Order of Constantine The Great.

Dr Herbert Messina Ferrante passed away on New Year Eve and is survived by his wife Elizabeth, their son Edward, his wife Daniela and their two children Marcus and Elisa. Herbert may not be with us any more but his passion towards anything he dared to be involved with was to be greatly admired. His baritone voice, booming laugh and big smile will never be forgotten. I am sincerely proud to have known and worked aside such a great personality who possessed such a rich character and a sense of humour. *Au revoir* dear colleague, you are and will be missed.

Now Messina De Ville is silent. The Alsatian whines and seeks her master but he is no longer there. His portrait near the marble stairs stares down at the living and the grandfather clock chimes twelve times at midnight. Each chime is like a final heartbeat. Leaves circle round in eddies in the strong wind near the imposing ornate gate. The candle in his study flickers one last time until it is slowly extinguished. A little whisper of smoke trails to the stucco ceiling and the halo slowly dissipates into nothing as a new year unfolds and the past gently rolls into the present. 🕯

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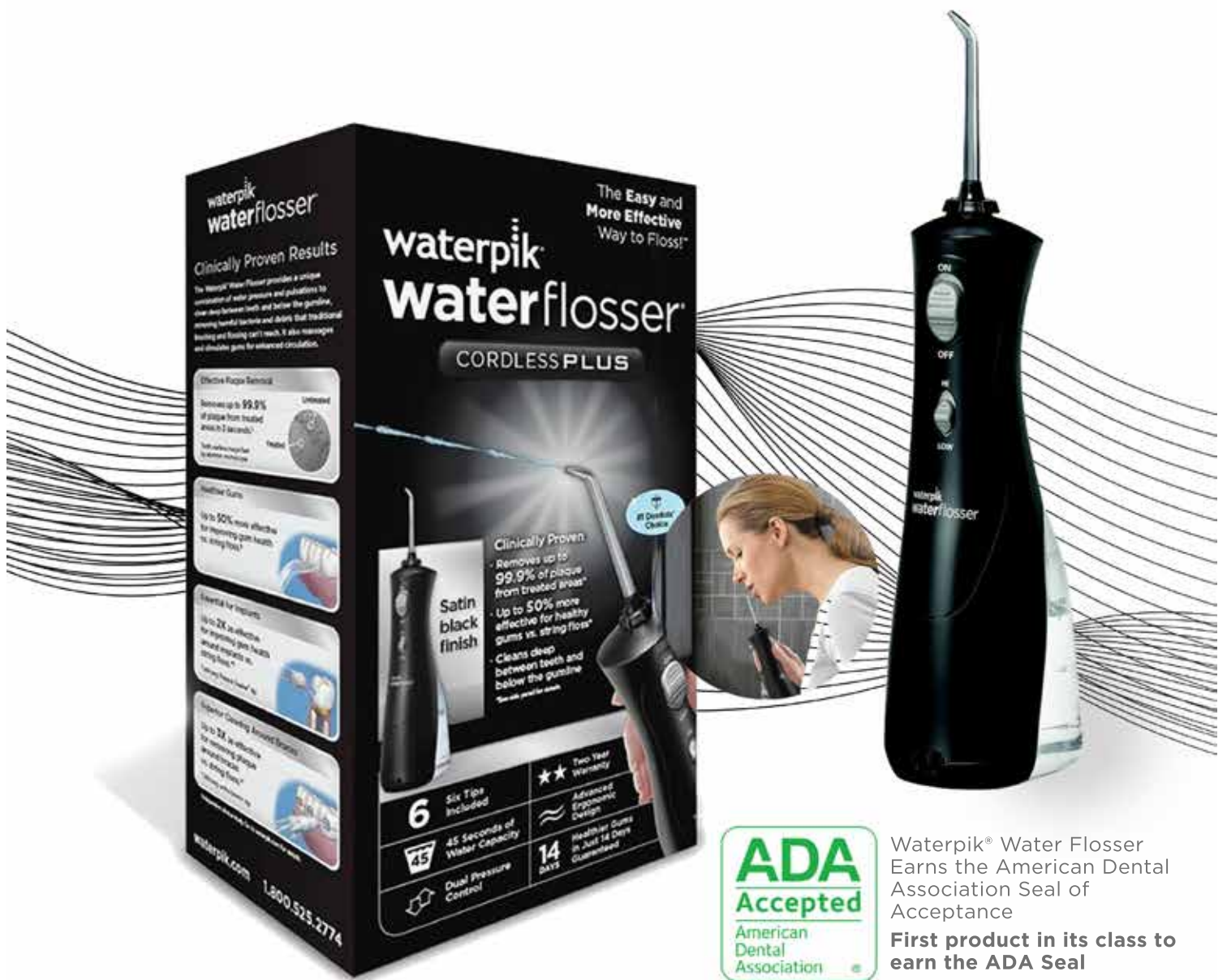
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**Clinically Proven Results**  
The Waterpik Water Flosser provides a unique combination of water pressure and pulsations to reach deep between teeth and below the gumline, removing harmful bacteria and debris that traditional brushing and flossing can't reach. It also massages and stimulates gums for enhanced circulation.

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Removes up to **99.9%** of plaque from treated areas in 2 seconds.

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**Essential for Invisalign**  
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**Superior Cleaning Around Braces**  
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**Satin black finish**

**Clinically Proven**  
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- Cleans deep between teeth and below the gumline  
\*Results provided for adults

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