Periodontal prognosis.

A comprehensive approach

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Chapter 1

Introduction

This book introduces the LTOP system for assigning comprehensive periodontal prognosis by integrating overall prognosis and individual tooth prognosis. This approach is based on data gathered during more than 25 years of research on periodontal prognosis (Martinez-Canut 2015, Martinez-Canut, Llobell & Romero 2017, Martinez-Canut, Alcaraz, Alcaraz Jr. et al 2018 and Martinez-Canut & Llobell 2018) and includes the following contributions:

- Development of Perioproject, a prediction model of tooth loss due to periodontitis (hereinafter, tooth loss refers to tooth loss due to periodontitis, and excludes other reasons for tooth loss).
- Development of the LTO index (Long-term outcome index) to identify, at baseline, the reduced percentage of patients with the highest risk of tooth loss, as well as identifying the two different profiles of patients (Type 1 and Type 2) associated with bruxism and at risk of losing more teeth.
- Development of the LTOP system to assign periodontal prognosis by integrating the LTO index and the estimated survival time assigned to periodontally involved teeth with Perioproject.

Figure 1.1 depicts the assignment of periodontal prognosis utilising the LTOP system. Overall prognosis is assigned with the LTO index (category 4 in this particular case), identifying a patient with a high risk of tooth loss. Individual tooth prognosis is assigned with the Perioproject prediction model, estimating survival times for periodontally compromised teeth (black). The actual tooth loss in time (yellow) of these teeth represents the accuracy of the tooth loss prediction. This accuracy is much higher in the reduced percentage of patients with a higher risk

of tooth loss (LTO index categories 4 and 5).

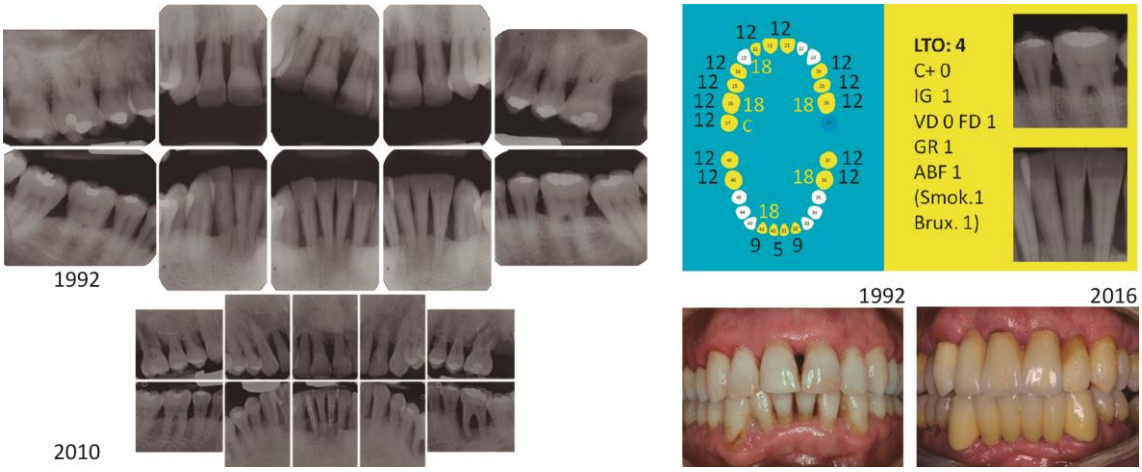

Figure 1.1 Assignment of periodontal prognosis implementing the LTOP system.

The results of the research project presented in this book offer a different perspective on periodontal prognosis, which applies to the following aspects:

1. Several generally assumed conventional concepts might need to be re-evaluated and perhaps reinterpreted (i.e., the actual meaning of conventional questionable or hopeless prognostic categories or the prognostic meaning of baseline gingival inflammation and subgingival calculus deposits, etc.).
2. Bruxism might be the second most prevalent condition that affects human mouths, after caries. However, there is a paucity of knowledge about its diagnosis and the actual impact on healthy and reduced periodontium. The research to develop the LTO index has contributed to a better understanding of this condition. Bruxism will be assessed in depth in this book, introducing a practical classification of tooth wear from a prognostic perspective.
3. Routine clinical and radiological periodontal parameters, which are mainly utilised to define the diagnosis, can be interpreted from a prognostic perspective. A classification of osseous and furcation defects from this perspective will also be

introduced. With this classification we not only try to understand tooth loss due to periodontitis, but also the predictability of periodontal regeneration.

The radiological and clinical parameters of a 39-year-old female patient (Figure 1.2) are interpreted to diagnose stage III periodontitis based on probing pocket depth, attachment loss, periodontal osseous defects, furcation involvement, and tooth mobility. However, from a prognostic perspective, periodontal prognosis can be assigned based on certain clinical and radiological parameters that in turn are associated with the risk factors smoking and bruxism, as well as the type of periodontium.

So when assigning periodontal prognosis, these risk factors are not only interpreted according to the self-reported information given by the patient, dichotomous and sometimes imprecise, but through the clinical and radiographical impact of these risk factors, objective and measurable, on the periodontium.

These clinical and radiographical parameters are the type and number of periodontal osseous defects and furcation lesions, radio-opaque subgingival calculus, mean gingival inflammation, mean gingival recession and abfraction, as well as other signs of occlusal trauma plus certain characteristic features of two types of patients (type 1 and type 2) at risk of losing more teeth, as will be addressed throughout this book.

4. The LTO index enables the baseline identification of the reduced target group of patients that will concentrate the highest tooth loss rates, whose percentage ranges from 3% to 8.9% according to studies exclusively analysing tooth loss due to periodontal disease (Checchi et al. 2002, Fardal et al. 2004, Chambrone & Chambrone, 2006, Muzzi et al. 2006, Lü et al. 2013, Martinez-Canut 2015). This has been a major goal of research on periodontal prognosis.

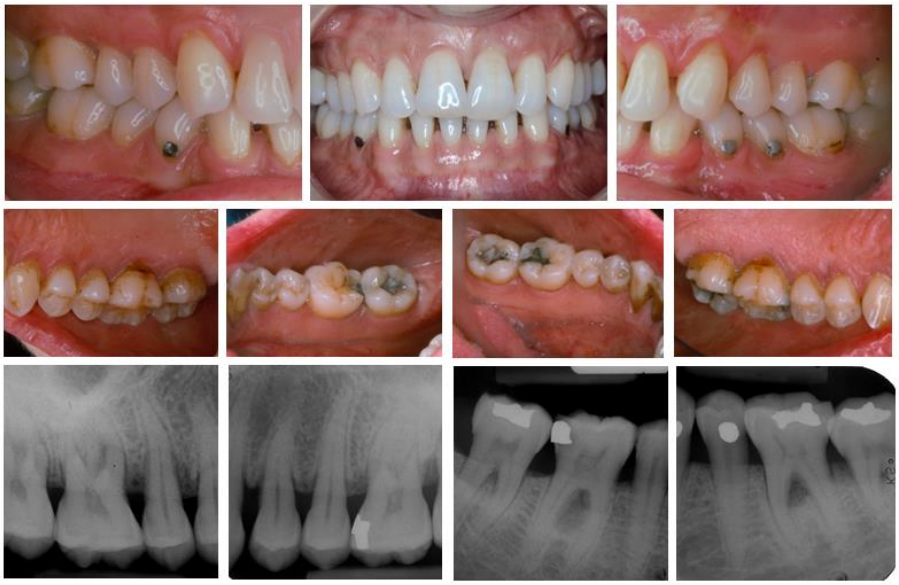

Figure 1.2 Several clinical and radiological parameters of this particular patient, together with data on patient-related prognostic factors, can be used to predict that she is at a high risk of experiencing a worse long-term outcome, losing many teeth. More details of this particular patient will be addressed in the following chapters.

5. The LTOP system makes the most stringent goal of simultaneously assigning overall and individual tooth prognosis possible. Utilising this approach, the conventional interpretation of periodontal prognosis from the dichotomous perspective of tooth loss + or tooth loss – faces the more realistic interpretation of periodontal prognosis in terms of mere probability: the probability of tooth loss of periodontally compromised teeth, depending on the extent of periodontal involvement, the estimated survival time and the particular risk of the patient experiencing tooth loss.

The probabilistic interpretation of periodontal prognosis seems to address the final outcome of periodontal patients more suitably. It certainly does not seem possible to define a good or a bad long-term outcome due to the relativeness of

factors involved in such a definition. For instance, a stage III periodontitis patient with grade III furcation involvement and a mean 60% attachment loss loses the most severely involved teeth after 20 to 25 years following periodontal maintenance. Is this a good or a bad outcome?

A more realistic and objective interpretation would be to define the probability of losing a certain number of teeth over a period of time depending on the above-mentioned variables.

Besides the scientific content presented in this book, the reader will be able to perform his or her own clinical research by carefully observing the clinical and radiological images of almost one hundred long-term follow-ups of periodontal patients. We believe this could be an exciting and enriching exercise aimed at increasing clinical experience and providing a more solid basis for assigning periodontal prognosis.

Utilising the available information on periodontal prognosis along with the information contained in this book, it will be possible to assign overall, individual and comprehensive periodontal prognosis to varied and numerous periodontal cases. This is presented as a practical seminar in the final chapter (Figure 1-3).

Figure 1.3 Assignment of periodontal prognosis implementing the LTOP system to different patients.

Chapter 3

Current knowledge and new insights

1. Current knowledge

Despite the limitations of research on periodontal prognosis as already addressed, and in contrast with the most pessimistic perspective of the paucity of knowledge on the subject, we have gathered quite relevant information, which has resulted from the following contributions:

1. Disease progression and tooth loss risk assessment (Lang & Tonetti 2003, Page et al. 2002, Fors & Sandberg 2001, Lindskog et al. 2010, Teich 2013), despite the limitation of equating risk with prognosis, have improved knowledge of certain predictors and some of these tools have been validated in different populations (Lang et al. 2015).

2. More recent studies have reported an increase in the risk of tooth loss according to each category of several tooth-related factors in the presence or absence of certain patient-related factors (Miller et al. 2014, Graetz et al. 2015, Martinez-Canut 2015, Dannewitz et al. 2016). BY taking together the most consistent findings on the impact of the most relevant patient- and tooth-related factors, we have accumulated quite broad knowledge of the predictors of tooth loss. The key issue might be how to interpret and apply this knowledge.

3. The identification, at baseline, of patients with a higher risk of tooth loss is fortunately possible nowadays. This was no easy task, due to the difficulties to gather and analyse these types of samples (Wasserman & Hirschfeld 1988, Tonetti et al. 1998, Martinez-Canut 2015, Fardal et al. 2016, Martinez-Canut, Llobell & Romero 2017).

2. New insights

Most recent research on periodontal prognosis has introduced a new perspective, thereby broadening the scope of the subject. This implies reinterpreting the assignation of periodontal prognosis and interpreting certain routine clinical and radiological parameters from a prognostic perspective.

2.1. Reinterpreting the assignation of periodontal prognosis

Insofar as research on periodontal prognosis enlarges the list of regression coefficients and relative risks (O.R and R.R) of predictors of tooth loss, clinicians should interpret the data as practically as possible. However, there are no clearly defined guidelines for using this data in a practical and useful manner, in order to assign a meaningful prognosis in terms of treatment decisions.

The results of the multilevel analysis of predictors of tooth loss performed in our research (Martinez-Canut 2015, Martinez-Canut et al. 2017) provided almost 15 predictors for molars and another 15 predictors for non-molars, with different impacts depending on the type of molar and non-molar. Several interactions between certain patient-related factors were also identified. The increase in the risk of tooth loss of all predictors represents an enormous amount of data. How can we mentally process this information to assign a meaningful periodontal prognosis?

This does not seem to be an easy task. This is the rationale to develop a prediction model to calculate the probability of tooth loss and assign the associated survival time, in other words, the rationale of the Perioproject prediction model. It might appear to be a convoluted approach, but it is based on objective and measurable parameters, as well as accepting the fact that tooth loss mostly takes place in the long-term. To our understanding, this approach is simpler than the subjective interpretation of prognostic factors without clearly defined guidelines. This might explain the very existence of the meaningless questionable prognostic category.

A prediction model is no more than the result of a statistical analysis that helps to make a decision. If we could open up the bowels of this prediction model, we would be able to reveal an enormous amount of data that is perfectly arranged in the form of a multilevel analysis, containing the relative risk of tooth loss corresponding to each category of the predictors of tooth loss included in the analysis, each playing their role simultaneously.

As shown in Figure 3.1, individual tooth prognosis of this 59-year-old male chronic periodontitis patient has been assigned according to the probability of tooth loss (p. value). This probability results from calculating the increase in the risk depending on the following predictors: the patient is a heavy smoker and presents heavy bruxism. The age and number of baseline teeth are also considered. At the tooth level, the extent of attachment loss (probing pocket depth, bone loss and furcation involvement), tooth mobility and the particular type of molar and non-molar teeth are considered.

Teeth with a higher probability of tooth loss were lost earlier: the actual survival time in years is depicted in yellow. By analysing different tooth loss samples, it is possible to associate the probability of tooth loss with the survival time, and associate thresholds of probability of tooth loss with survival times.

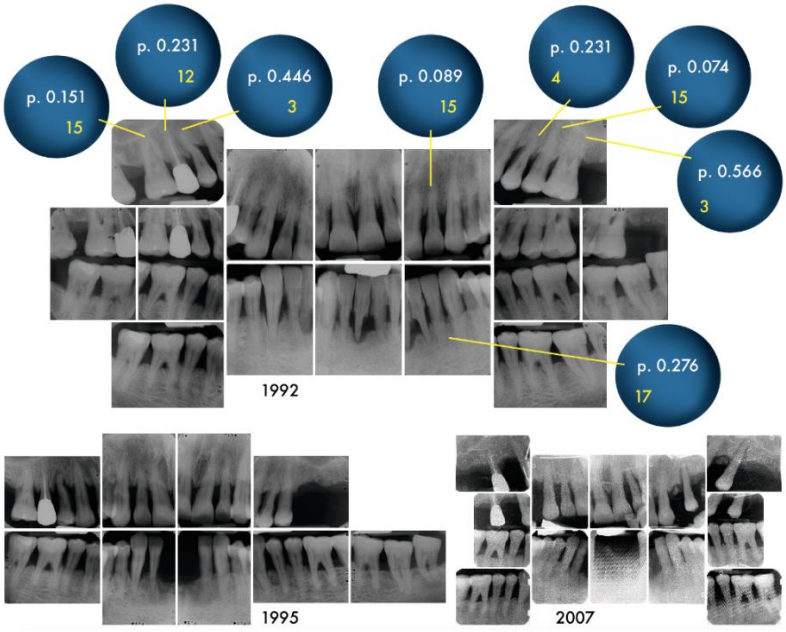

Figure 3.1 Individual tooth prognosis of a chronic periodontitis patient assigned according to the probability of tooth loss (p. value).

It has generally been assumed that the mean probability of accurately predicting tooth loss, excluding good prognosis, is a question of luck comparable to tossing a coin (McGuire & Nunn 1996). However, individual tooth prognosis represents one side of the coin, and requires the other side, i.e., overall prognosis, to increase the usefulness and accuracy of periodontal prognosis. Overall periodontal prognosis consists of the assessment of the particular risk of the patient experiencing tooth loss, and this might increase the accuracy of the tooth loss prediction. This is the rationale behind implementing the LTO index to estimate the particular risk of the patient experiencing tooth loss.

Figure 3.2 shows several clinical and radiological features that enable identification of a patient with a high risk of tooth loss. These peculiarities will be addressed throughout this book.

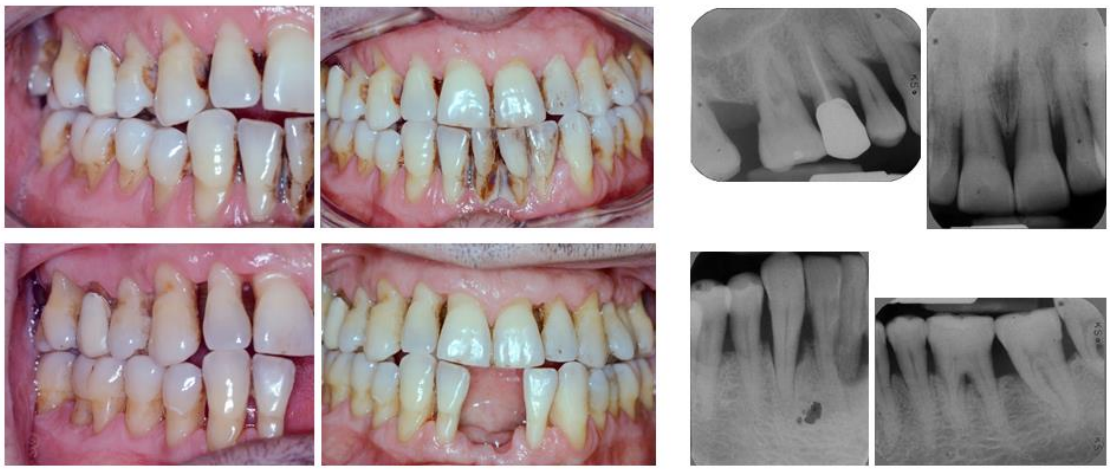

Figure 3.2 Clinical and radiological features that enable identification of a patient with a high risk of tooth loss.

Integrating individual tooth prognosis and overall prognosis may be the most stringent goal of research on periodontal prognosis. To date, there have been no unanimously accepted comprehensive and standardised approaches to assigning overall and individual tooth prognosis. This book introduces two alternative approaches to assigning individual and overall prognoses. It also introduces a system to assign both prognoses simultaneously. This is the LTOP system, which might be helpful for a better understanding of periodontal prognosis, enabling the assignation of more accurate and useful prognosis.

Neither the assignation of survival time to periodontally compromised teeth nor the LTO index are definitive tools. They basically represent a language to communicate and develop further research on periodontal prognosis.

Figure 3-3 depicts the simultaneous assignation of overall and individual tooth prognosis. In this particular case, the patient presents an LTO index value of 5, with the highest risk of tooth loss. This results from assessing the presence of five variables defining the LTO index. Additionally, the presence and severity of smoking and bruxism would reinforce the identification of a patient at risk of losing teeth.

The odontogram shows several periodontally compromised teeth (in yellow) with certain probability of tooth loss. This probability is associated with a certain survival time. There are several survival times (in black): from 12 to 22 years, from 9 to 20 years, etc. The actual survival time is also shown, making it possible to assess the accuracy of the tooth loss prediction.

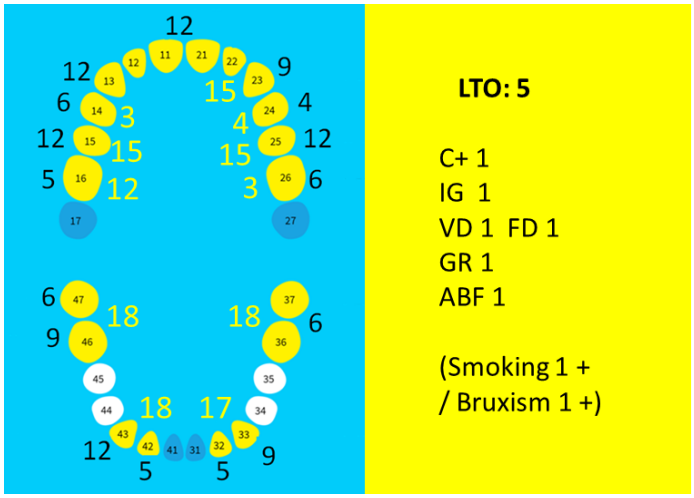

Figure 3.3 Simultaneous assignation of overall and individual tooth prognosis.

Routine clinical and radiological parameters from a prognostic perspective.

Overall and individual periodontal prognosis has traditionally been assigned based on incomplete knowledge of prognostic factors and lacking homogeneous criteria to interpret them. For instance, whether being older or younger is the actual prognostic factor or not for tooth loss remains unclear.

This paucity of knowledge also applies to treatment, since certain techniques have been performed without a complete understanding of the etiological and predisposing factors involved. This applies, for instance, to vertical osseous defects and gingival recession.

Our research has provided more profound knowledge of the predictors of tooth loss and this has brought new insight into the following four routine clinical and

radiological parameters. Chapter 7 addresses the prognostic meaning of these parameters in more detail and from a practical perspective.

1. Gingival inflammation and subgingival calculus

2. Vertical, circumferential and furcation defects

3. Gingival recession

4. Bruxism

5. Early diagnosis of bruxism and prognostic meaning of abfractions

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2. Vertical, circumferential and furcation defects

Analysing the way vertical, circumferential and furcation defects associate with bruxism, smoking, loss of attachment (stage III and IV periodontitis) and tooth loss rate provided valuable information about the development and prognostic meaning of these lesions. Our research analysed comparable sub-samples according to these prognostic factors featuring 230 grade II and III furcation defects, 234 vertical and circumferential defects and 516 abfractions, which increase up to 1,070 at the end of the follow-up. One hundred patients out of 174 (57.4%) lost 289 teeth due to periodontal disease.

The observational approach utilised consisted of a close inspection of the baseline and final clinical and radiological records, with special attention to the radiographs of the teeth that experienced the worst outcomes. These images were discretionally magnified on the computer screen to display any details and enable comparison between the follow-up images. The relevance was confirmed of having more than one radiograph per tooth to properly identify the morphological characteristics of the defects, as well as clinical images of several periodontal regeneration techniques. The following are the most relevant findings:

The conventional distinction between vertical osseous defects and furcation lesions might need to be re-evaluated since there are several types of these, each with a different prognostic meaning depending on the involvement of smoking and/or bruxism. The following Figure 3.5 introduces certain relevant differences in the way these lesions behave depending on smoking and bruxism.

As a general rule, vertical bone defects are more clearly associated with bruxism while furcation lesions are more clearly associated with smoking. By incorporating the final outcome of these lesions and the tooth loss rate, it seems possible to understand their actual prognostic meaning.

Based on all this information, a comprehensive, descriptive classification of periodontal osseous defects and furcation defects was developed and will be introduced in Chapter 7, section 3. Further research allowed us to classify these defects according to their prognostic meaning (Chapter 7, section 6).

Figure 3.4 shows the way furcation lesions and vertical defects presented in the subsamples of patients were analysed, according to smoking and bruxism. It details the mean grade II and III furcation lesions (FL II, FL III) and vertical defects (VD) per patient and the percentage of patients with more than 2 vertical defects. (p., number of patients analysed). Figure 3.5 depicts different types of defects with different prognostic meanings. It presents ten vertical, circumferential and furcation defects with quite different prognostic meanings depending on the characteristics of the defect and the patient.

SMOKING – & BRUXISM – (41 p.)
FL II 0.4, FL III 0.4 & VD 0.4, >2 VDs 5%
SMOKING – & BRUXISM + (70 p.)
FL II 0.5, FL III 0.4 & VD 1.1, >2 VDs 20%
SMOKING + & BRUXISM – (16 p.)
FL II 1.3, FL III 1.1, & VD 0.9, >2VDs 12%
SMOKING + & BRUXISM + (47 p.)
FL II 2, FL III 2.5 & VD 2.6, >2 VDs 51%

Figure 3.4 Furcation lesions and vertical defects presented in the subsamples according to smoking and bruxism.

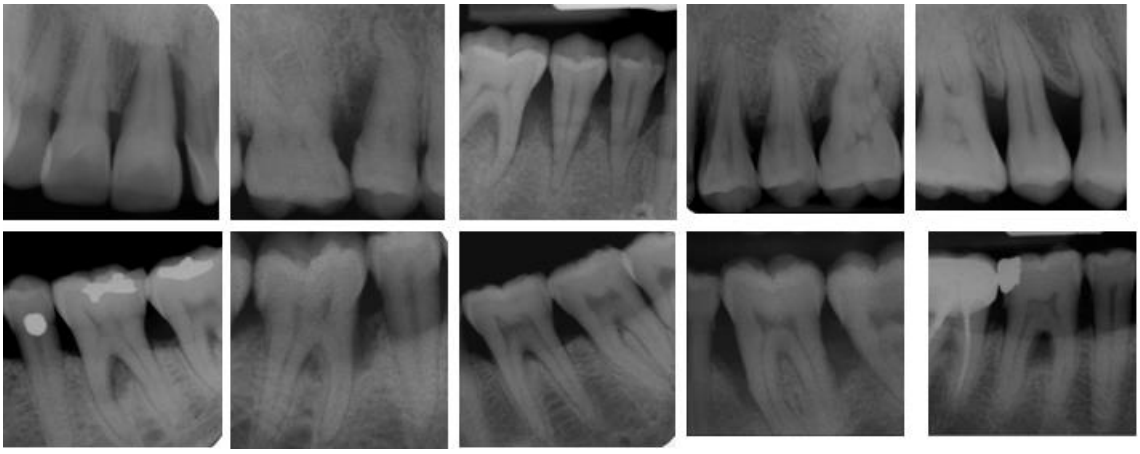

Figure 3.5 Different types of defects with different prognostic meanings.

3. Gingival recession

Research on gingival recession has mainly focused, in a similar fashion to that of periodontal defects, on the design, efficacy and predictability of the available treatment techniques. Research efforts aimed at increasing our knowledge of the development of these lesions and assessing the actual impact of the suspected etiological and predisposing factors has been rather scarce. Among these factors, the anatomical characteristics of the marginal periodontium and plaque-associated inflammation have capitalised attention.

Our research on predictors of long-term outcomes included a close inspection of the behaviour of the marginal periodontium during active periodontal treatment as well as during follow-up. Four main factors were associated with gingival recession: the extent of attachment loss and gingival inflammation, smoking and bruxism.

Figure 3.6 shows two follow-ups corresponding to non-smoking patients. The two central images show gingival recession taking place during active periodontal treatment. The images on the right show gingival recession at the end of the follow-up. The upper case corresponds to a non-bruxist patient, with minimal gingival changes. The lower case corresponds to a bruxist patient with a pattern of clenching. In this case, the extent of gingival recession during active periodontal treatment was comparable to the extent of gingival recession occurring during the follow-up period.

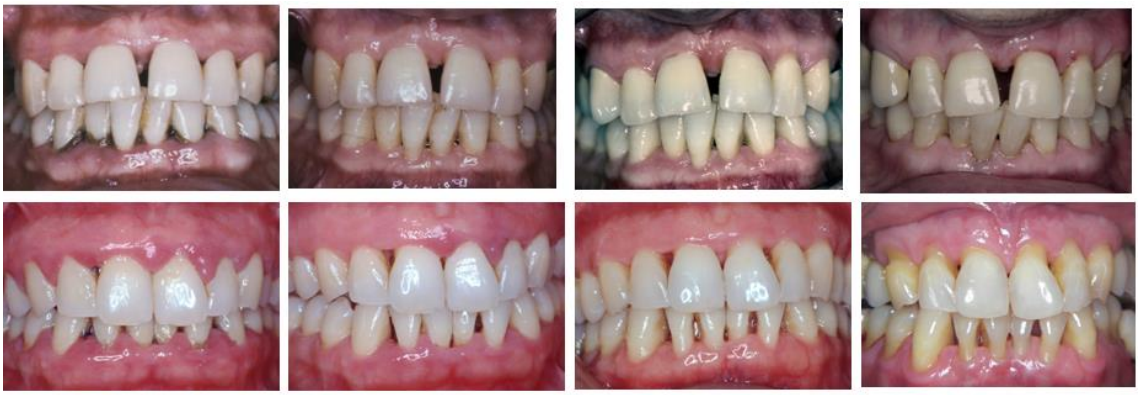

Figure 3.6 Two follow-ups corresponding to non-smoking patients.

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4. Bruxism

The role of bruxism in periodontal patients remains an open question. It is poorly understood and represents one of the most controversial issues in dentistry. Consequently, it is a matter that warrants serious scientific discussion (Perlistsh, 2016, Manfredini et al. 2015, Manfredini et al. 2016), besides the literature on

bruxism is characterised by the scarce quantity and quality of the studies (Manfredini et al. 2015).

Data supporting the association of occlusal contacts and bruxism with periodontal disease is contradictory (Yuodelis & Mann 1965, Jin & Cao 1992, Shefter & McFall 1984, Pihltrom et al. 1986, Hanamura et al. 1987). A recent review by experts concluded that bruxism, -per se-, might not cause damage in a healthy periodontium (primary occlusal trauma), while the hypothesis that periodontal disease could cause the tooth to suffer from occlusal trauma could be more plausible (Manfredini et al. 2015).

Bruxism is one of the most prevalent features that has an impact on the mouth of human beings. As it was reviewed (Manfredini et al. 2013), the prevalence of bruxism in the general population is approximately 25%, whereas a two-fold rate of bruxism (264 patients out of 500, 53%) has been reported in periodontal patients (Martinez-Canut 2015). When only moderate and severe periodontitis was considered, in 174 patients, this prevalence rose to 67% (Martinez-Canut, Llobell & Romero 2017). Therefore, the role of bruxism merits further research.

Only two studies have addressed the association between bruxism and tooth loss in patients following supportive periodontal therapying the long-term (McGuire & Nunn 1996, Martinez-Canut 2015) and the results were quite consistent. Bruxism doubled the risk of tooth loss and this impact was comparable to smoking. An almost four-fold increase in the risk of losing more teeth was reported when bruxism was associated with smoking (Martinez-Canut 2015). Our research on 174 moderate and severe chronic periodontitis patients found bruxism to be clearly associated with a worse outcome, being possible to characterise these patients according to several clinical and radiological features. Our results seem to indicate that vertical defects and furcation defects are characteristic features of bruxism and smoking respectively (Martinez-Canut, Llobell & Romero 2017).

It should be kept in mind that tooth loss occurs invariably associated with a periodontal defect. So the characterisation and prognostic meaning of these defects is one of the objectives of this book.

5. Early diagnosis of bruxism and prognostic meaning of abfractions

The diagnosis of bruxism is a key issue and has been a main limitation of research on the subject. To begin with, occlusal attrition resulting from grinding is considered the pathognomonic sign of bruxism. However, eccentric bruxism is much less prevalent than centric bruxism, whose extent of occlusal wear is limited to wear facets while the relevant tooth wear locates in the cervical area as abfractions. In our sample of bruxist patients, only 10% presented eccentric bruxism.

Interestingly, abfraction and gingival recession are two signs that would help to identify and interpret bruxism. They have been theoretically associated with bruxism and/or occlusal overload in the absence of clinical studies supporting this association. Our research, following the long-term outcome of 174 patients with and without bruxism enabled a better understanding of the actual meaning of abfraction and gingival recession.

We pointed out that the most reliable predictor of an unfavorable outcome was baseline vertical and circumferential defects and/or furcation lesions associated with increased attrition and/or abfractions, especially in smoking patients. Therefore the identification of emerging abfractions and periodontal defects, at early stages of disease might help to make a more precise diagnosis and institute the most appropriate prophylactic and therapeutic measurements for a patient at risk of losing more teeth.

Figure 3.7 depicts baseline clinical images of fifteen chronic periodontitis patients with a high risk of tooth loss. All of them experienced higher tooth loss rates in the long-term. Based on attachment loss, gingival inflammation, gingival recession and

tooth wear (attrition and abfraction), as well as the involvement of smoking and bruxism, three profiles of patients can be differentiated, and these are shown in the upper, central and bottom images respectively.

The first profile consists of centric or eccentric bruxism in the absence of smoking. The second is centric bruxism in the presence of smoking. In these ten clinical cases, the marginal periodontium is rather thick and abfraction develops to a varied extent; more consistently in the clenching pattern of bruxism as opposed to the grinding pattern. These ten patients would correspond to the type 1 patients that lose most teeth. An interesting feature of bruxism combined with smoking is the development of hairline cracks on the facial aspect of central incisors (Figure 3.8).

The third profile of patients shown in the bottom images consists of centric bruxism in the presence of thinner marginal periodontium. These patients are also characterised by increased loss of attachment and increasing tooth mobility, with lower prevalence of abfraction. This profile would correspond to the type 2 patients with a higher risk of tooth loss. Chapter 6, section 6, offers detailed information on these two types of patients. Chapter 7 addresses the clinical manifestations of bruxism in depth. In the final Chapter 9, a classification of physiologic and pathologic tooth wear will be introduced. This may appear to be somewhat hidden among the twenty follow-ups presented to assign periodontal prognosis, but there is a particular purpose for doing this, so we beg the reader not to read this section before.

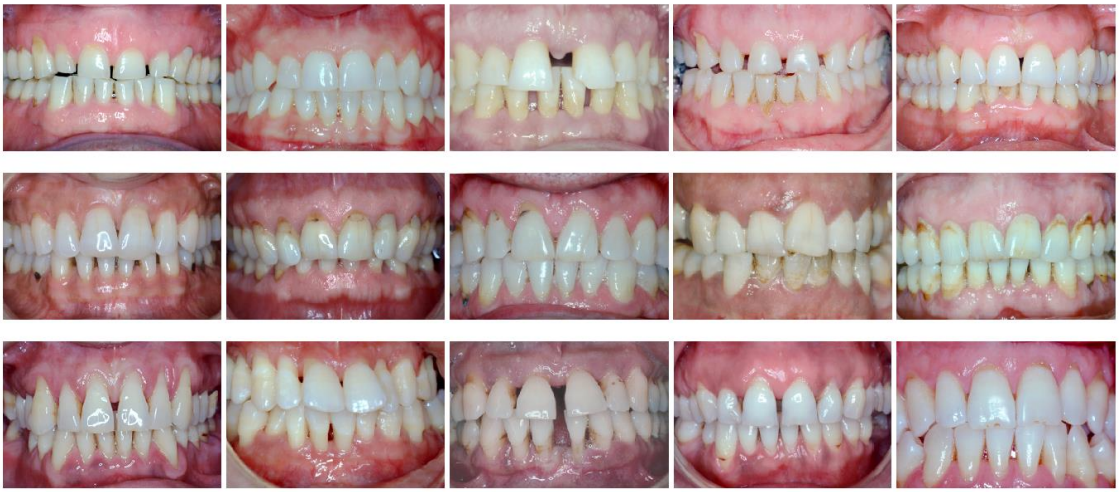

Figure 3.7 Baseline clinical images of fifteen chronic periodontitis patients with a high risk of tooth loss.

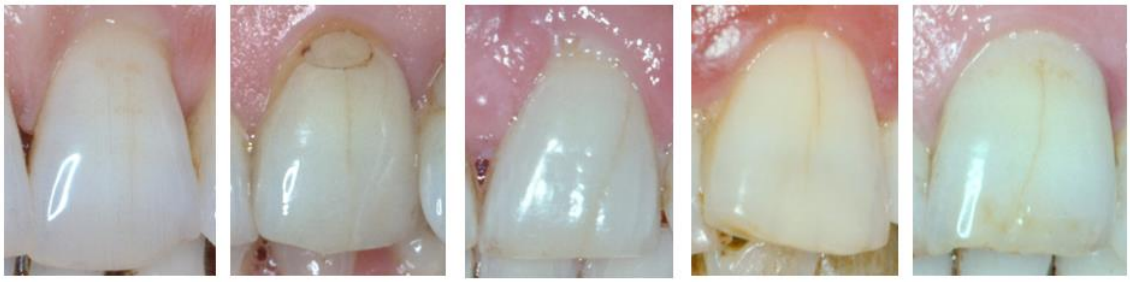

Figure 3.8 Development of hairline cracks on the facial aspect of central incisors.

Chapter 6

The long-term outcome (LTO) index

1. Introduction

The LTO index is the result of a comprehensive retrospective long-term study (Martinez-Canut, Llobell & Romero 2017) designed to characterise the baseline

status of patients in relation to their long-term outcome. This enabled baseline characterisation of patients that experienced higher tooth loss rates in the long-term., which in turn enabled the development of the LTO index (Long-term outcome index). In parallel, two different profiles of patients (Type 1 and Type 2) that lost more teeth, associated with bruxism, were identified. That is to say, two different ways of losing the teeth associated with bruxism.

This study's sample consisted of 174 moderate and severe chronic periodontitis patients following periodontal maintenance for a mean 20 years.

The association between the long-term outcome of these patients, smoking, bruxism and routine clinical and radiographic parameters that are also associated with these habits, was analysed. These parameters were gingival inflammation based on the Gingival Index (GI), gingival recession, presence of radio-opaque subgingival calculus, tooth wear (incisal and occlusal attrition and abfraction), vertical and circumferential bone defects, furcation defects and increased tooth mobility.

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5. Development of the LTO index

The LTO index was developed on the basis of the analysed variables, enabling baseline characterisation of patients experiencing worse long-term outcomes. This index consisted of a simple addition of one score for each variable involved. Thus, the final value ranged from the presence of 0 to 5 of the following variables: 1. Fewer radio-opaque subgingival calculus deposits; 2. Mean Gingival Index below 1.7; 3. Vertical and circumferential bone defects and/or grade II and III furcation defects; 4. Mean vestibular gingival recession > 1.5 mm, and 5. Abfraction.

Chapter 7 focuses on the assessment and interpretation of the parameters defining this index. The information provided represents a practical approach to interpreting

routine clinical and radiological periodontal parameters within the scope of prognosis.

Table 6-4 shows the distribution of patients according to the index. For tooth loss 0, 71 patients out of 74 (96%) presented an index of 0 to 2. For tooth loss >2, 45 out of 51 (97.7%) presented an index of 3 to 5.

The mean tooth loss corresponding to an index of 4 and 5 were respectively 2.7 and 4.6. For tooth loss >2 teeth, index values of 3, 4 and 5 matched the number of teeth lost ± 1 in 43 out of 55 patients (78.1%). The higher the value of the Index, especially with bruxism and smoking, the higher the resulting tooth loss rate and the accuracy of the Index (Spearman correlation 0.680, $p = 0.0001$).

Table 6-4. Distribution of patients with the corresponding value of the LTO index and the mean index value within each sub-sample of TL according to smoking and bruxism

	n. patients	**n. teeth lost**	n. patients	**n. patients with the corresponding LTO value (0 to 5) of the index**						**Mean index value / pt.**
				0	**1**	**2**	**3**	**4**	**5**	
TL 0										
Total	74									1.51
S- B-	28	0		3	17	8				1.17
S- B+	37	0			12	22	2	1		1.78
S+ B-	6	0			5	1				1.16
S+ B+	3	0				3				2
TL 1-2										
Total	45									2.48
S- B-	10	1 – 2		1	4	3	2			1.68
S- B+	23	1 – 2			1	12	6	4		2.56
S+ B-	5	1 – 2				1	3	1		3
S+ B+	7	1 – 2				1	4	2		3
TL > 2										
Total	55									
S- B-	3									2
		3	3			3				
S- B+	10									3.6
		3	3				2	1		
		4	4				3		1	
		5	2					1	1	
		6	1					1		
S+ B-	5									3
		3	2				1	1		
		4	1				1			
		> 4	2			1	1			
S+ B+	37									4
		3	11				7	3	1	
		4	11				1	4	6	
		5	6				3	2	1	
		> 5	9					5	4	

TL, tooth loss due to periodontal disease; S, heavy smoking; B, bruxism; n. pts, number of patients; n. teeth lost; number of teeth lost

Table 6.4 Distribution of patients with the corresponding value of the LTO index and the mean index value within each sub-sample of TL according to smoking and bruxism.

According to the results of one of our studies (Martinez-Canut et al. 2017 a), the most reliable predictors of an unfavourable outcome were baseline vertical bone defects and/or furcation defects associated with increased attrition and/or abfraction, especially in smoking patients. Therefore, the identification of emerging abfraction, vertical defects, and furcation defects at early stages of disease might help to make a more precise diagnosis and institute the most appropriate prophylactic and therapeutic measurements for a patient at risk of losing more teeth.

Searching for the earliest signs in these patients, emerging abfraction and gingival recession should be considered. This very early abfraction does not consist of tooth wear in the strictest sense, but of dark intrinsic decolouration close to the cementoenamel junction. These lesions appear simultaneously with initial gingival recession. If this developing recession occurs in the presence of wide gingival margins and perhaps a thickened vestibular osseous process, bruxism might be present. If smoking is involved, the patient could reasonably be identified as having an initial higher risk of tooth loss.

Figure 6-1 presents the baseline status and the long-term situation of two young patients. The top images correspond to a female non-smoking patient with mild to moderate bruxism. This lady did not lose teeth and developed grade 1 and 2 abfraction. The bottom images correspond to a male patient with heavy bruxism and heavy smoking who lost several teeth as well as breaking an implant. The only clinical signs were emerging abfraction and localised gingival recession.

However, emerging abfraction is characterised by very subtle changes that could go unnoticed. Chapter 7 addresses this issue in more detail.

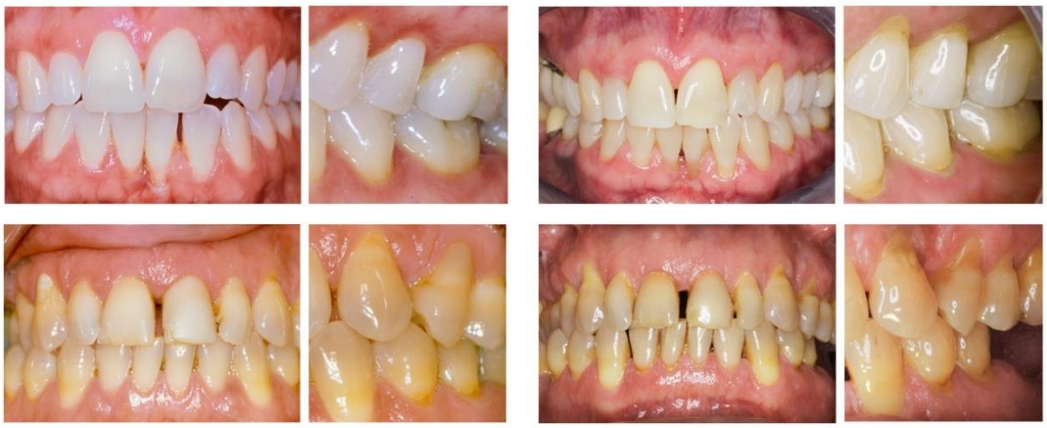

Figure 6.1 Baseline status and the long-term situation of two young patients.

6. Characterisation of patients that lose more teeth associated with bruxism: Type I and Type II patients

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Chapter 7

Clinical and radiological parameters defining the LTO index

This chapter addresses the assessment and interpretation of those parameters that define the LTO index. Since this is a pioneering index handling routine clinical and radiological parameters from the perspective of prognosis, the authors understand that this tool needs to be comprehensively validated and therefore represents the starting point for further research.

The assessment and interpretation of the LTO index is a challenging and fascinating task that can fortunately be retrospectively assessed with well-documented long-term follow-up cases. Despite the statistically significant association of the five parameters of the LTO index with the long-term outcome in our research, most of these factors are not definitive predictors in themselves and thus need to be interpreted simultaneously.

The LTO index is based on the presence or absence of these five parameters:

- 1. Radiographically visible subgingival calculus
- 2. Gingival inflammation: a mean Gingival index above or below 1.7
- 3. Vertical or circumferential bone defects and/or grade II and III furcation lesions
- 4. Generalised vestibular gingival recession, above or below 1.5 mm
- 5. Abfraction

An LTO 5 patient, with the highest predisposition to experiencing tooth loss, would neither present baseline subgingival calculus nor significant gingival inflammation, but will present bone defects and/or furcation lesions, generalised gingival recession and abfraction.

The most relevant parameter is the third, which is the one on which we shall mainly focus our attention.

Strikingly, it has been generally assumed that two parameters of this index increase the risk of periodontal disease progression and the resulting tooth loss (subgingival calculus and gingival inflammation) while these parameters actually associate with a better outcome. Both parameters have been found to be associated with the presence and progression of periodontal disease during the natural course of the disease. However, our research revealed that these factors were clearly associated with a better outcome from the perspective of prognosis in patients following periodontal maintenance.

We will now focus on the details to assess and interpret these five parameters.

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3. Vertical or circumferential bone defects and/or furcation lesions

These lesions are the most relevant parameter of the LTO index. Based on our records, patients concentrating higher tooth loss rates presented, in comparison with patients that do not lose teeth, a fivefold increase in vertical and circumferential bone defects and a fivefold increase in grade II and III furcation defects.

Periodontal defects are associated with the main patient-related prognostic factors of severe periodontitis, smoking and bruxism. Our research deepened knowledge of this issue by analysing comparable sub-samples according to smoking and bruxism. In the absence of both factors, 31.7% of patients lost a mean 0.54 teeth, attributed to severe periodontitis. In the presence of smoking, 62.5% lost a mean 1.6 teeth. In the presence of bruxism, 47% lost a mean 1.1 teeth, and in the presence of smoking and bruxism, 93.6% lost a mean 4 teeth. The statistical analysis revealed smoking and bruxism, either isolated or combined, to be statistically associated with tooth loss.

A close examination of our clinical and radiological records enabled differentiation of the variety of vertical, circumferential and furcation defects in relation to smoking, bruxism and the long-term outcome to bring us closer to the prognostic meaning of these lesions. Utilising this approach, a classification of periodontal defects was developed and will be introduced later on.

The radiographs shown in figure 7.12 would suggest a generalised horizontal bone loss pattern. However, different osseous defects can be distinguished: grade III furcation defect in the first lower right molar, grade II furcation defect in the second lower left molar and an emerging circumferential defect in the upper first right premolar. Upper first molars seem to depict an emerging furcation defect that would be compatible with a developing vertical defect, as will be addressed next.

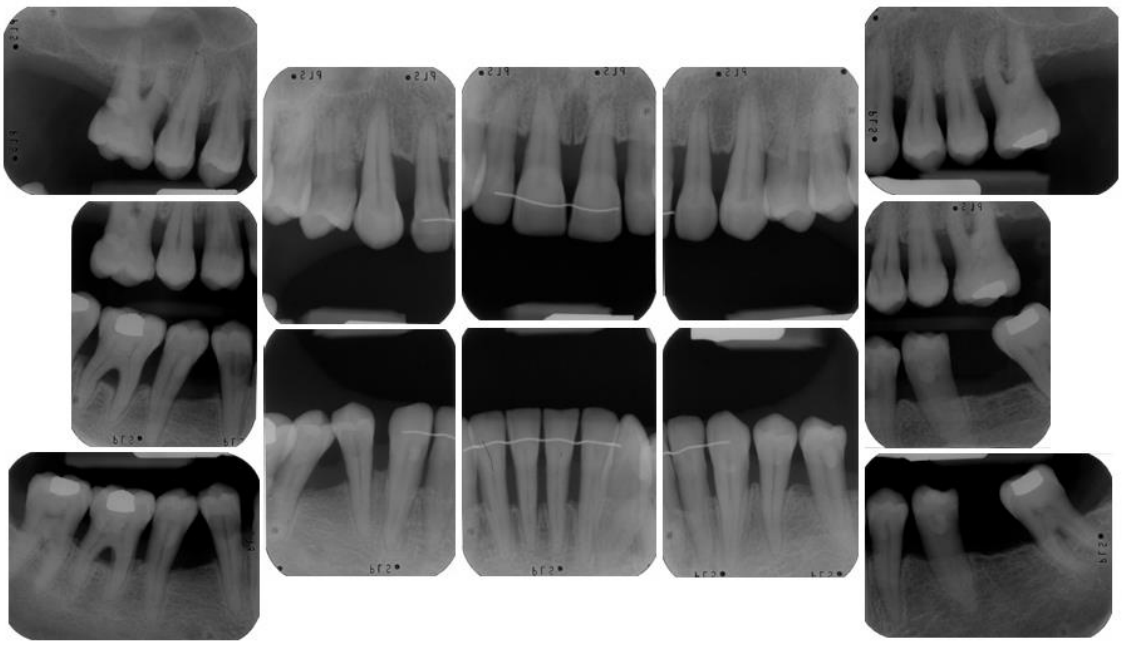

Figure 7.12 Different osseous defects can be distinguished radiographically.

On the one hand, there are periodontal defects resulting from attachment loss due to periodontal disease *per se* and most probably characterised by a horizontal bone loss pattern. On the other hand, attachment loss can be altered by the impact of the patient-related factors of smoking and bruxism. For instance, certain vertical and circumferential osseous defects can develop on already lost bone, combining a previous horizontal bone loss pattern with a vertical bone loss pattern. These situations might experience quite different outcomes in comparison with an isolated horizontal or vertical bone loss pattern.

The same would apply to furcation involvement. These lesions can develop consistently with a horizontal bone loss pattern but can also develop in the absence of interproximal bone loss or associated with a vertical defect. On the contrary, an intact furcation area can be found close to a vertical defect.

In the absence of smoking and bruxism, patients usually present no vertical defects or one defect, and rarely more than two. The same would apply to grade II and III

furcation lesions. The isolated effect of bruxism was associated with a threefold increase in the mean vertical defects per patient while the isolated effect of smoking was associated with a threefold increase in the mean grade II and III furcation defects per patient (Martinez-Canut, Llobell & Romero 2017).

The simultaneous impact of smoking and bruxism seems to define a very particular scenario characterised by a sixfold increase in the mean vertical and grade II and III furcation defects per patient in comparison with the absence of smoking and bruxism. Most of these patients lost teeth and 79% lost more than two teeth.

Interestingly, these differences extend beyond the mere distribution of these lesions, so that

these different periodontal defects present quite a different prognostic meaning. Some defects are clear predictors of a worse outcome (with an index value of 1) while other defects do not have a significant prognostic meaning and can even predict a better outcome (with an index value of 0).

The above information seems to be a fascinating area that questions some traditional concepts and broadens our view of periodontal defects, as is detailed next.

3.1. Vertical defects *versus* furcation lesions.

The conventional distinction between furcation and vertical or circumferential defects has rather weak scientific support other than the location of the defect. Vertical defects are associated with bruxism while furcation defects are associated with smoking. However, in certain situations, it does not seem possible to differentiate a vertical defect on molars of a furcation lesion and this seems to be associated with the simultaneous impact of bruxism and smoking. This is addressed next.

3.2. Vertical defects.

The available information on vertical defects has certainly been scarce. The intra-bony component of a vertical defect has been associated with a reduced probability of tooth loss (Muzzi et al. 2006) whereas certain risk assessment tools consider vertical defects to be a risk factor (Page et al. 2002, Lindskog et al. 2010). Data supporting the latter has been limited to an animal study (Lindhe & Svamberg 1974) and a study in a non-treated population in which the vertical defects were associated with further bone loss (Papapanou & Wennström, 1991). Research efforts have focused on the treatment of these lesions without a complete understanding of its development and prognostic meaning.

The attempts to categorise these defects have been limited to differentiating vertical from circumferential osseous defects and assessing the deepness and number of walls of the defect.

3.3. Furcation lesions.

The attempts to categorise furcation lesions have consisted of a horizontal furcation involvement classification: grade I, II and III (Hamp et a. 1975), and two vertical furcation involvement classifications (Tarnow & Fletcher 1984, Tonetti et al.2017). These latter classifications might be useful from a prognostic perspective. However, there may be additional characteristics worthy of further research.

The distribution of grade II and III furcation involvement according to smoking and bruxism clearly differs from vertical defects. In the absence of smoking and bruxism as well as in the presence of isolated bruxism, the percentage of patients presenting grade II and III furcation lesions is around 25%, with a mean 0.5 for grade II and a mean 0.5 for grade III per patient. In the presence of smoking, this percentage increases to 68% with a twofold increase in the mean grade II and III furcation lesions per patient. The simultaneous impact of smoking and bruxism increases the percentage to 76% with a sixfold increase in the mean grade II and III furcation involvement.

Grade II and III furcation defects can present quite different morphological characteristics with different prognostic meanings. Besides the aforementioned horizontal and vertical classifications, the relation between the interproximal bone level and the level of the remaining bone inside the furcation area seems relevant. According to this, several types of furcation lesions can be differentiated, with quite different prognostic meanings: predicting a long-term outcome, predicting a better one and lacking significant prognostic meaning. These different prognostic meanings need to be interpreted in consideration of the involvement of smoking, bruxism and the extent of attachment loss.

Before introducing a systematic classification of periodontal defects, four different scenarios are described in Figure 7-13 based on the way smoking and bruxism associate with these defects. This information should be kept in mind for better comprehension of this classification.

SMOKING − & BRUXISM − (41 p.) One or no VD/p., only 5% more than two VD. Regeneration predictable. Few FL. Level of bone loss coincides with the level of interproximal bone loss and there may be no FL despite the existence of interproximal loss. May stabilise. Only 7% lost more than two teeth.	**SMOKING − & BRUXISM + (70 p.)** Twice as many VD and 15% more than two VD. Regeneration depends on control of bruxism. FL do not increase and there may be no FD despite there being an interproximal VD. May stabilise. Only 14% lost more than two teeth. Highly intense bruxism may be associated to VDs that spread towards the root apex.
SMOKING + & BRUXISM − (16 p.) Twice as many VD and 12% more than two VD. Regeneration not predictable. Three times more FD and in some cases no loss of interproximal bone 31% lost more than two teeth.	**SMOKING + & BRUXISM + (47 p.)** Six times more VD and 51% more than two VD. Tooth loss predictable if the defect is circumferential. Five times more FD, in many cases with no loss of interproximal bone. The association of a VD with a FD is common in these cases. Loss of the affected tooth highly predictable. 78% lost more than two teeth.

Figure 7.13 The participation of heavy smoking and bruxism are associated with quite different scenarios

A. Vertical and circumferential bone defects

A. 1. Vertical bone defect on relatively intact periodontium

A. 2. Interproximal vertical bone defect on relatively intact furcation areas

A. 3. Circumferential bone defect on reduced periodontium

A. 4. Deep vertical or circumferential defect involving the apex of the root

B. Furcation defects

B. 1. Furcation lesion on reduced periodontium

B. 2. Furcation lesion on relatively intact periodontium

B.3. Furcation lesion with a vertical or circumferential osseous defect

A. 1. Vertical bone defect on relatively intact periodontium

Certain vertical bone defects develop on relatively intact periodontium, in mild to moderate periodontitis. Due to the absence of significant loss of support, the defect usually presents several walls. This situation more consistently occurs in the absence of smoking. Bruxism and/or occlusal discrepancies might be present. In the absence of heavy bruxism and heavy smoking, this type of lesion usually occurs in isolation. Whenever heavy bruxism is involved, more than one vertical defect may develop. This type of defect presents the best prognosis in terms of periodontal regeneration. Figure 7.14 depicts some examples. This type of defect is usually deep and presents several walls. Guided periodontal regeneration would be highly predictable in the short- and long-term.

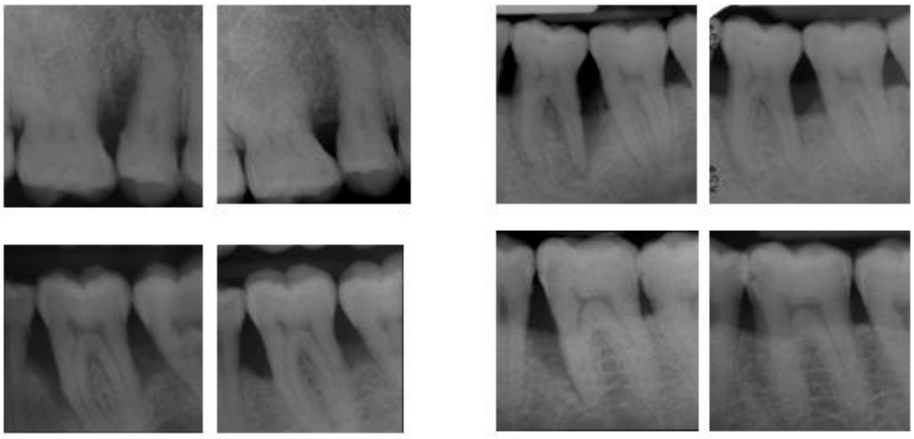

Figure 7.14 Certain vertical osseous defects develop isolated, with relatively intact periodontium in the surrounding areas. Periodontal regeneration is quite predictable in these cases.

In the presence of bruxism, there is a twofold increase in the prevalence of these defects. Almost 50% of these patients develop more than one defect and 20% develop more than two.

The radiographic follow-up shown in Figures 7.15 and 7.16 depict the resolution of several vertical and circumferential defects in a heavy bruxist (clenching) and non-smoking 43-year-old female patient. The defects on the lower second right premolar, upper left central incisor and lower second left molar were successfully treated with guided periodontal regeneration. The defects on the upper right second molar and upper left first premolar were judged to be non-manageable due to the extent of the defect and only conventional periodontal treatment was performed, with a surprising outcome. In our experience, if heavy smoking had been involved in this situation, the outcome would not have been so successful. This would suggest that factors other than the type of defect, such as certain patient-related factors, might be relevant.

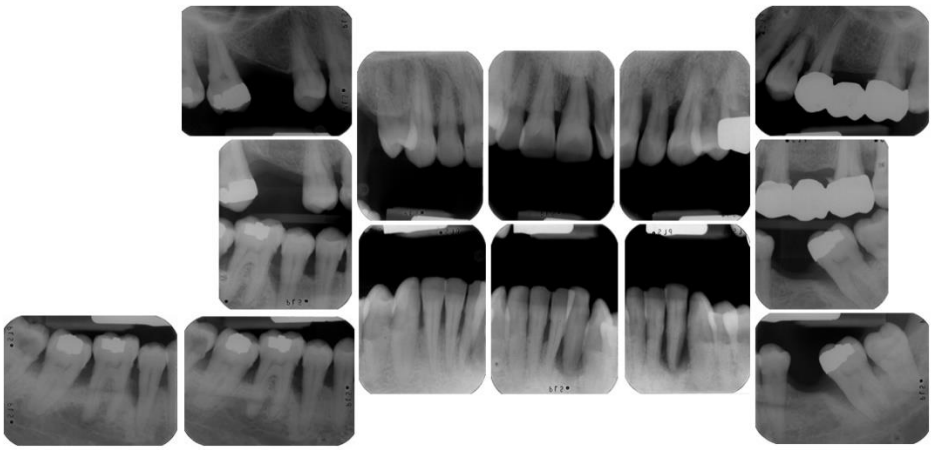

Figure 7.15 Vertical and circumferential osseous defects in a bruxist, non-smoking female patient.

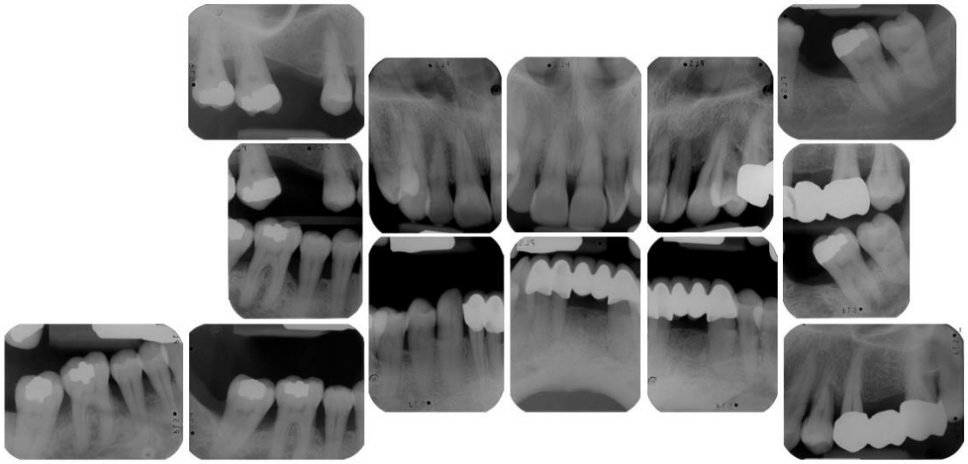

Figure 7.16 Resolution of these defects in the former patient.

These situations might not be that exceptional. The following follow-up (Figure 7.17, 7.18, 7.19 and 7-20) shows the resolution of vertical defects in a heavy bruxist (clenching) and non-smoking 36-year-old female patient.

4.2. The relevance of the periodontal biotype in plaque-associated gingival inflammation

It was suggested that plaque-associated inflammation may result in gingival recession in thin biotypes while it may result in deep pockets in thick biotypes (Olsson & Lindhe 1991). This seems obvious from logical reasoning and during the natural course of periodontal disease. However, in terms of prognosis, in patients following periodontal maintenance and performing relatively good plaque control, it still has to be elucidated whether a thin biotype is a predisposing factor for gingival recession. According to our data, we did not find a thin biotype or an existing gingival recession to be a predisposing factor in non-bruxist patients, since the periodontal condition on these teeth remained stable (Figure 3.16). The opposite was observed in bruxist patients, regardless of the biotype and despite complying with periodontal maintenance and relatively good plaque control.

Figure 7.57 shows the long-term follow-up of three patients undergoing periodontal maintenance for more than two decades. Plaque control measures were quite efficient. Several lower incisors that were facially displaced and/or had thin gingival margins and a certain extent of gingival recession remained stable. Paradoxically, gingival recession did not develop in these suspected predisposed areas but in posterior teeth, associated with the development of mild abfraction. These were non-smoking and non-bruxist patients. According to our findings, abfraction also developed in non-bruxist patients although with much less prevalence and extent of tooth wear.

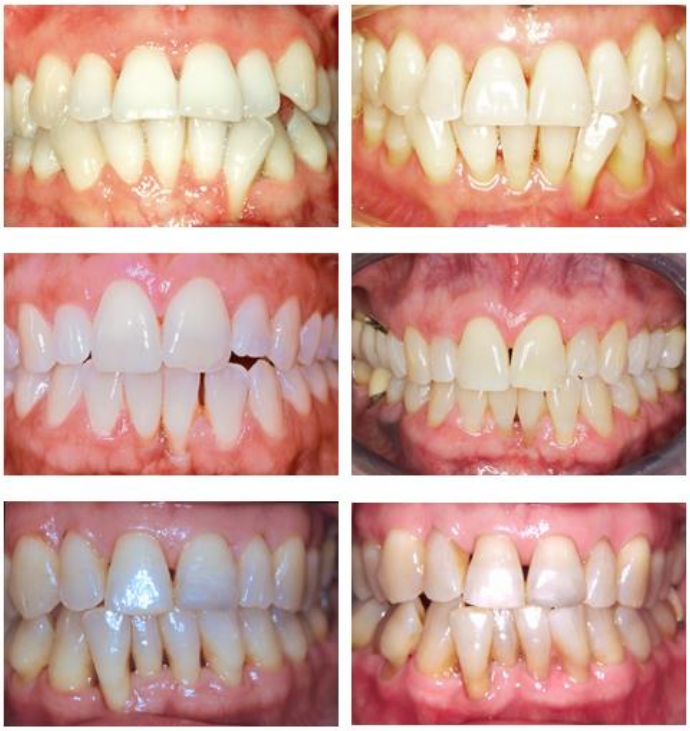

Figure 7.57 Long-term stability of the gingival margins in non-smoking and non-bruxist patients. This was so despite the marginal periodontium was theoretically predisposed to gingival recession.

4.3. Bruxism and gingival recession

The earliest description of gingival recession associated with occlusal trauma, shaping the Stillman's cleft (Stillman 1921) is still something of a myth; a clinical observation lacking clinical studies supporting this association. The few studies on the subject found no relationship between occlusal discrepancies and gingival recession (Harrel & Nunn 2004). This study, however, exclusively analysed certain occlusal features without any clear differentiation between patients with and without bruxism.

The most definitive data supporting the association of bruxism with gingival recession is the fact that abfraction consistently develops in association with localised gingival recession. The only exception would be abfraction emerging on

enamel and therefore located coronally to the cementoenamel junction. Nonetheless, most of this abfraction usually spreads towards the root surface with the accompanying gingival recession. Insofar as the tooth wear index category (Smith & Knight 1984) increases and the apical base of the abfraction extends beyond the cementoenamel junction, the extent of gingival recession consistently increases.

As shown in Figure 7-58, the thick marginal periodontal tissues of this 45-year-old patient with heavy clenching would not theoretically justify the development of gingival recession on the upper left premolars. However, these recessions developed and progressed together with the accompanying abfraction.

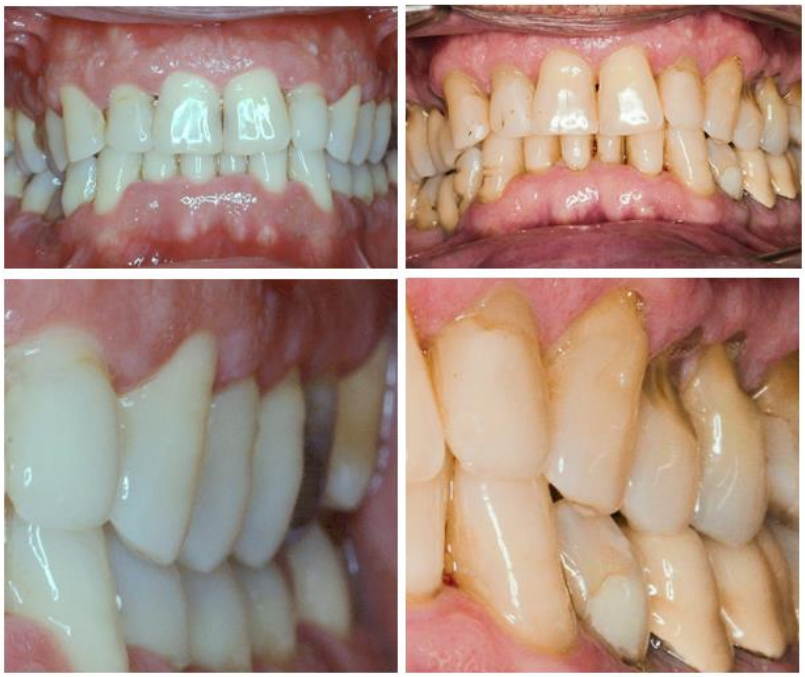

Figure 7.58 Long-term follow-up of a thick marginal periodontium in a heavy bruxist (clenching) patient. Gingival recession developed associated with the development of abfractions.

Figure 7.59 depicts gingival recession associated with abfraction on areas of thick marginal periodontal tissue. These lesions might represent a completely different scenario to that of gingival recession developing in thin marginal tissue. We might question whether the centre left image is actually an abfraction lesion or simply a gingival recession. This issue will be addressed later on.

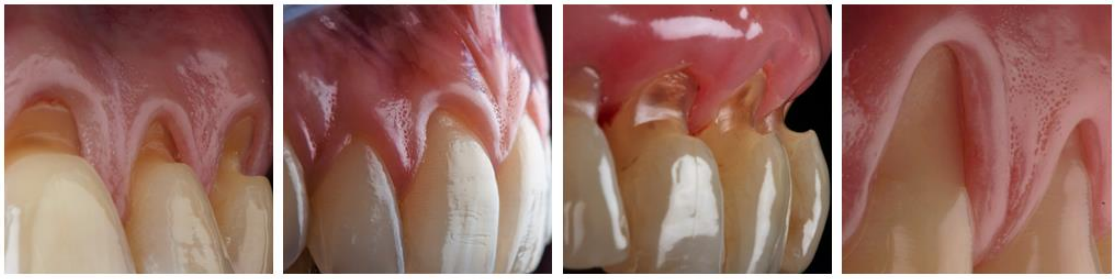

Figure 7.59 Gingival recession associated with abfractions.

4.4. Factors influencing the behaviour of the marginal periodontium

In non-smoking and non-bruxist patients, the extent of gingival recession associated with active periodontal treatment differed depending on the degree of gingival inflammation and probing pocket depth. Assuming these variations, the resulting initial gingival recession was comparable to the extent of gingival recession taking place afterwards, during a mean 25-year follow-up.

In smoking patients, gingival recession associated with active periodontal treatment was much less pronounced in comparison with non-smoking patients.

The periodontal biotype as described in healthy patients is hard to assess in many periodontal patients due to the existing loss of attachment and gingival inflammation. However, assuming this limitation, it is possible to clearly differentiate

extreme situations corresponding to thin and thick periodontal biotypes. Considering this parameter, together with gingival inflammation, the extent of attachment loss and the involvement of smoking and bruxism, it was possible to draw some interesting conclusions on the behaviour of the marginal periodontal tissues after active periodontal treatment and in the long-term.

In the absence of bruxism, the development of gingival recession seemed to occur regardless of the periodontal biotype. The development of abfraction was quite uncommon and the tooth wear index in the cervical area of the teeth was limited to category 1 most of the times. The marginal periodontium remained stable in the long-term, with minimal and even absent gingival recession. Baseline localised gingival recessions in these patients remained stable or progressed to quite a limited extent. The uncommon situations in which no additional recession occurred during the follow-up period corresponded exclusively to non-bruxist patients.
Figure 7.59 shows the follow-up of several thinner biotypes and Figures 7.60 and 7.61 depict the follow-up of thicker biotypes in the absence of bruxism. Several shallow grade I abfractions developed, in all likelihood due to occlusal overload. Many areas with baseline gingival recession or thin gingival margins remained stable.

Due to the high prevalence of bruxism in periodontal patients and the association of gingival recession with bruxism, a high proportion of routinely treated gingival recessions would be associated with bruxism. On the other hand, the prognosis of mucogingival treatment in these situations would be good based on its efficacy and predictability. The hypothetical recurrence of the treated gingival recession might take place in the long-term, as these lesions usually develop.

Besides the commonly utilised Miller (1985) classification of gingival recession, the Pini-Prato et al. (2010) classification on dental surface defects in areas of gingival recession seems, somehow, to suggest different scenarios of gingival recession and represents, to our understanding, a reasonable approach to gingival recession associated with bruxism.

Two categories of this classification are characterized by the loss of the cemento-enamel junction and the dental structure in the recession area. The authors identify these non-carious cervical lesions with traumatic abrasion and erosion. Interestingly, the clinical images presented by the authors would be comparable to the ones presented in this book to illustrate several abfractions on wide gingival margins.

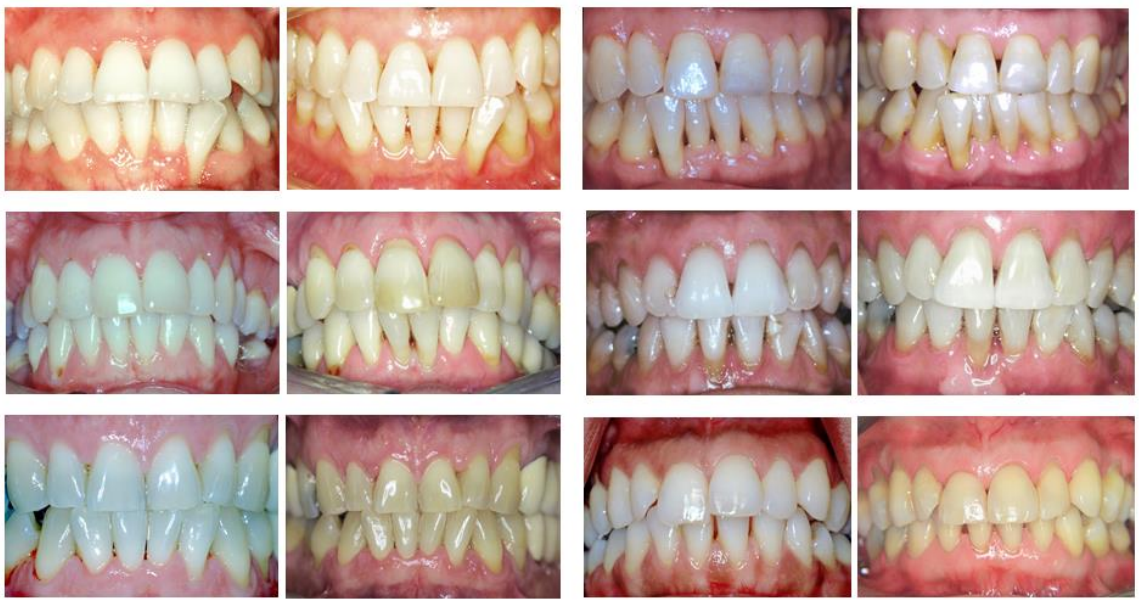

Figure 7.60. Follow-ups of thinner marginal periodontium in the absence of bruxism

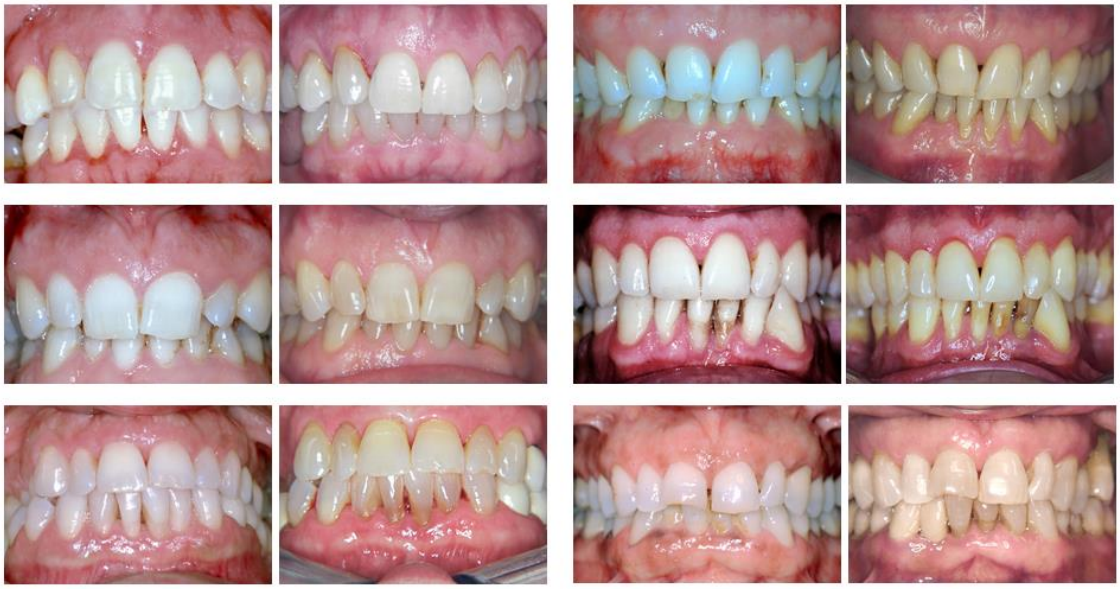

Figure 7.61. Follow-ups of thicker marginal periodontium in the absence of bruxism

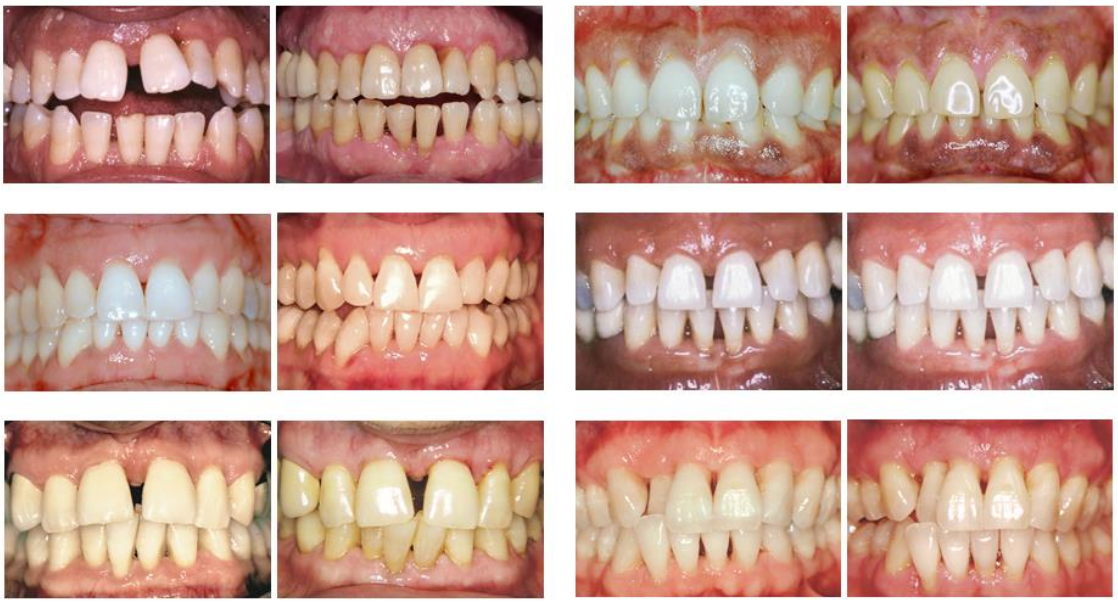

Figure 7.62. Follow-ups of thicker marginal periodontium in the absence of bruxism

In the presence of bruxism, regardless of smoking, the extent of gingival recession occurring during the follow-up did not depend on the Gingival index, but on bruxism. This was the main factor associated with gingival recession, either localised gingival recession associated with abfraction or generalised gingival recession.

In this case, the periodontal biotype seemed to be more relevant. Abfraction developed more frequently in the thick biotype, associated with localised gingival recession surrounding the abfraction. The more severe bruxism was and the more competent the supporting alveolar bone, the higher the extent of cervical tooth wear. Tooth mobility was usually absent. Figures 7.63 and 7.64 show the follow-up of mild to moderate periodontitis in the anterior sextants. Bone loss was ≤ 50%. The periodontal biotype of these patients was rather thick. Recession occurred associated with abfraction.

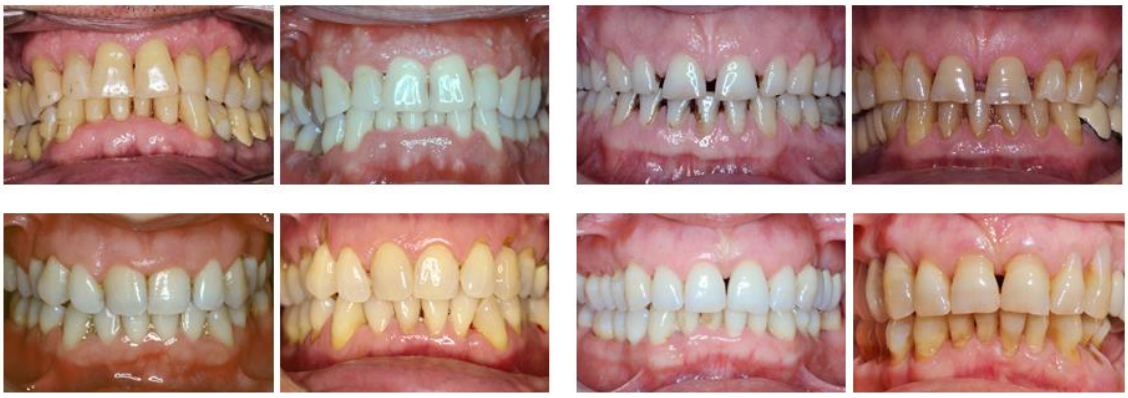

Figure 7.63. Follow-ups of rather thick marginal periodontium in bruxist patients. Gingival recession occurred associated with the development of abfractions.

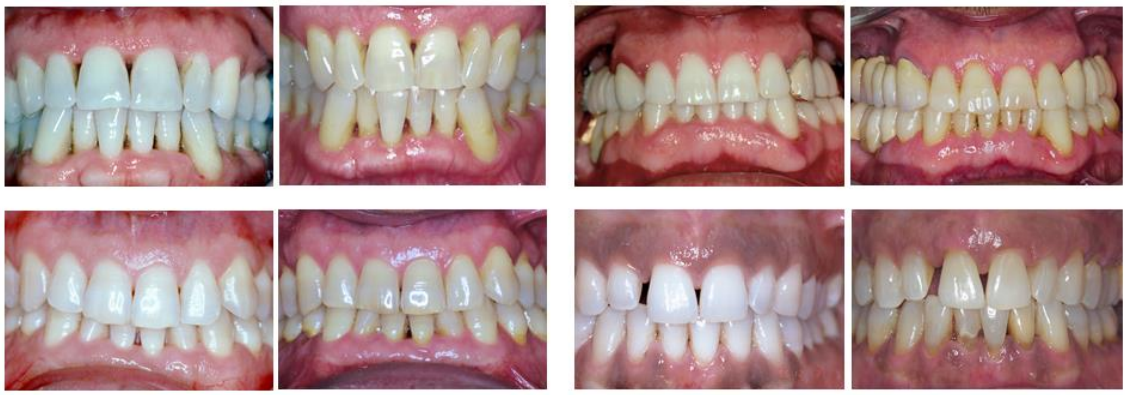

Figure 7.64 Follow-ups of rather thick marginal periodontium in bruxist patients. Gingival recession occurred associated with the development of abfractions.

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5. Abfraction

Abfraction has been associated with bruxism (Xhonga 1977, McCoy 1982), wear facets (Schiller et al. 1985, Bader et al. 1996, Mayhew et al. 1998, Telles et al. 2000), and occlusal disturbances (Miller et al. 2003). The study by Miller et al. (2003) found that 10% of patients with abfraction presented bruxism (eccentric bruxism with increased occlusal attrition), while the remaining 90% presented occlusal disturbances (wear facets, lack of canine guidance and group function).

Furthermore, a similar percentage of patients with eccentric bruxism (8.5%) was found in our research. However, the 90% of occlusal disturbances reported by Miller et al. (2003) might to some extent correspond to the 92.4% of our patients with clenching, occlusal disturbances and abfraction.

The lack of defined criteria to identify centric bruxism would explain the different results and might indicate the possibility of under-diagnosing a relevant and prevalent factor involved in tooth loss. Only 10% of bruxists might present the conventional pattern of increased attrition.

The development of abfraction has been attributed to occlusal forces on the cervical area of the teeth and fall within the multi factorial etiology of non-carious cervical lesions (Grippo et al. 2012). The stress component within this scheme would be the result of biomechanical loading forces exerted on the teeth, causing flexure and ultimate fatigue of susceptible teeth.

However, these lesions have remained a theoretical process supported by engineering analysis using finite element models (Sarode & Sarode 2013). Only one study reporting a 14-year follow-up of a patient with bruxism, abfraction, and occlusal wear was found in the literature (Pintado et al. 2000) when we performed

our research. We were able to observe the presence and progression of these lesions in 174 patients in the long-term.

According to our records, the presence of abfraction was not an exclusive feature of bruxism, since patients without bruxism also developed it. However, these lesions were almost four times more prevalent in bruxist patients, with a two-fold increase in cervical tooth wear in comparison with non-bruxist patients. In parallel, patients concentrating higher tooth loss rates presented, in comparison with patients that lose no teeth, an almost twofold increase in the presence of abfraction, which is thus interpreted as a predictor of a worse long-term outcome, with an LTO index value of 1 for this parameter. The extent of tooth wear may be an indicator of the severity of bruxism, as might also be vertical and circumferential bone defects.

Broadening the perspective on abfraction lesions

The term abfraction is derived from the Latin words "ab" or "away" and "fractio" or "breaking" or (Grippo 1991); that is, "to break away" from the occlusal contact point (Sarode & Sarode 2013). Although the pathognomonic abfraction lesion is the one located on the cervical area of the tooth, abfraction can be located all around the crown and the cervical area.

Up to fifteen types of abfraction have been described (Grippo 1991). The resulting lesion would depend, as well as the multi factorial etiology of non-carious cervical lesions (Grippo et al. 2012), on a wide range of factors. These include, among others, the location, duration, frequency and intensity of the occlusal contact; the anatomical characteristics of the tooth, as well as the composition and structure of enamel and dentin. Furthermore, and to our understanding, they also include the

composition and structure of alveolar bone, which in turn might be somehow associated with the nature of subgingival calculus deposits, as we will address later.

Figure 7.75 depicts several types of abfraction: striations on the palatal aspect of the upper incisors and 'hairline' cracks on the facial aspect of the upper central incisors. The two top right images show some striations and occasional discolorations close to the cementoenamel junction. The two bottom left images depict severe incisal and occlusal attrition (occlusal wear category 3) combined with cusp-tip abfraction or invagination. The centre right image shows a well-defined semilunar abfraction on the root dentin and the bottom left image shows a saucer-shaped abfraction on enamel. To our understanding, the variety of abfraction lesions is such that these are difficult to classify. Furthermore, the shape and location of the abfraction substantially varies insofar as the lesion progresses in the long-term.

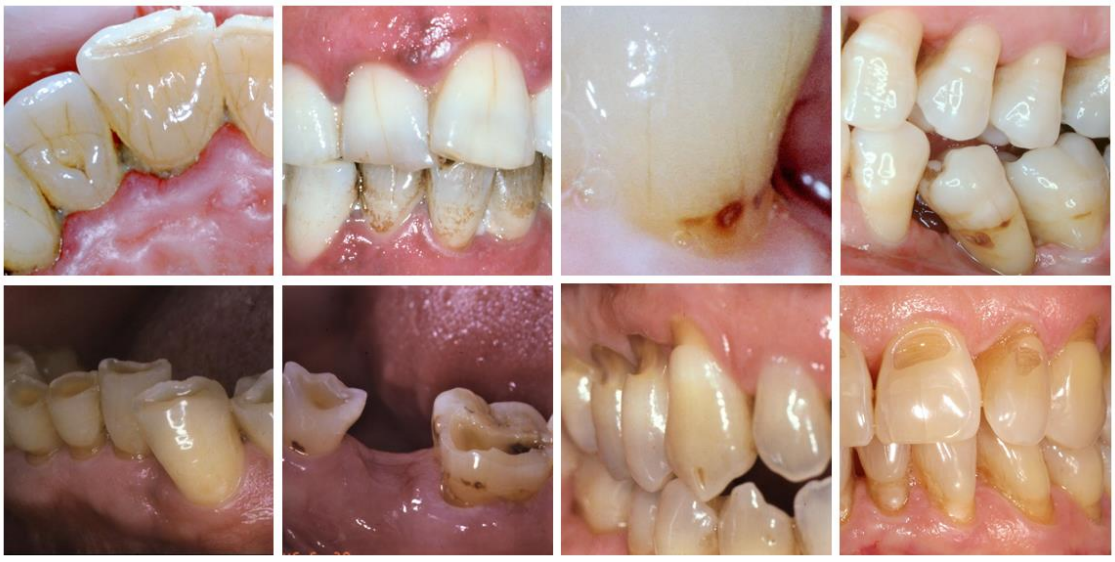

Figure 7.75 Several types of abfraction.

Before looking in greater depth at abfraction lesions, it is worth revisiting the method utilised to assess and evaluate tooth wear. Clinicians usually refer to tooth

wear in qualitative and overall terms, for instance, severe tooth wear. However, a detailed description of occlusal and cervical tooth wear requires the use of tooth wear indices. We implemented the one by Smith and Knight (1984), which is

designed to measure tooth wear *per se*, irrespectively of the aetiology and thus considering the nature of multifactorial tooth wear. Intra- and inter-examiner reproducibility utilizing this index was demonstrated to be within the acceptable range. However, no single index has been universally accepted and there is no index capable of identifying and measuring all tooth wear patterns, as is shown next.

To simplify the use of the Smith and Knight index, only the cervical and occlusal surfaces were considered. Since abfraction lesions also develop on the occlusal and incisal surfaces, occlusal and incisal attrition will be included to make a clearer distinction between both types of tooth wear. Figure 7.76 is quite illustrative in this respect. There are two category 2 abfraction lesions on enamel, the most apical extending towards the root. However, the cusp of the canine does not present an abfraction lesion (cusp-tip invagination), but category 2 occlusal attrition. However, in other instances, occlusal attrition develops together with occlusal abfraction, mainly shaping cusp-tip invagination.

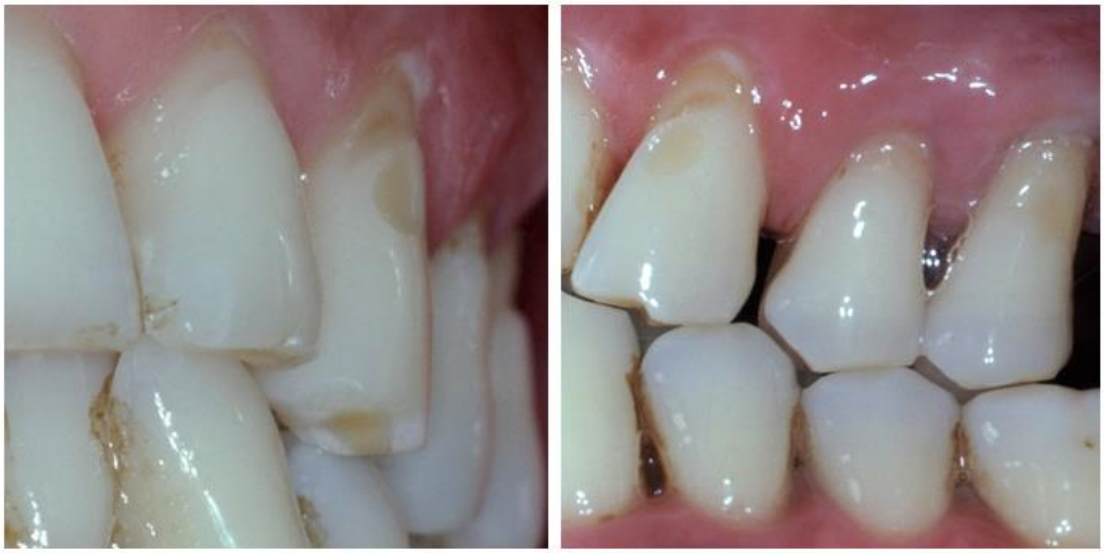

Figure 7.76 Occlusal attrition and abfractions.

The tooth wear index by Smith and Klein differentiates five categories for occlusal attrition and another five for cervical abfraction lesions. However, these categories do not allow for assessment and measurement of certain tooth wear types, mainly abfraction located in areas other than the cervical one, for instance, cusp-tip invaginations, striations, hairline cracks and cusp-tip invagination. To our understanding and from a prognostic perspective, there are two additional signs that should be included: dark discoloration and localised gingival recession.

For occlusal attrition, the categories are as follows: 0, no loss of enamel surface characteristics; 1, loss of enamel surface characteristics; 2, loss of enamel only exposing dentin on less than one third of the surface; 3, loss of enamel exposing more than one third of the surface, and 4, complete enamel loss with secondary dentin exposure and/or pulp exposure. However, in this last category, the enamel does not usually undergo complete loss, since the so-called enamel halo usually remains. While these categories focus on tooth wear, from a prognostic perspective, a localised wear facet (category 1, without dentin exposure) would reveal the

presence of occlusal overload associated with periodontal breakdown, as will be addressed in the next chapter. In parallel, striations, hairline cracks and dark discolorations, which are not included in these categories, prevail in the presence of smoking and bruxism. Many patients with these signs experienced unfavourable long-term outcomes in our records.

These aforesaid occlusal tooth wear categories differ in terms of incisal tooth wear, so category 2 is loss of enamel only exposing dentin, category 3 is loss of enamel and substantial loss of dentin, and category 3 is pulp exposure or secondary dentin. For cervical abfraction lesions, the categories are as follows: 0, no loss of contour; 1, minimal loss of contour; 2, lesion less than 1 mm deep; 3, lesion between 1 and 2 mm deep, and 4, defect deeper than 2 mm with secondary dentin exposure and/or pulp exposure.

We find several arguments to revisit these categories. First, the presence of secondary dentin does not take place exclusively in category 4, since what we termed dark discoloration is intrinsic and can be present in any category of the index, even in the absence of enamel or dentin loss. These discolorations would in all likelihood reveal the presence of reactive dentin. Furthermore, the presence of these punctual discolorations reveals the presence of an emerging abfraction and they are reliable predictors of further tooth wear, besides indicating the presence of bruxism.

Figure 7.77 (two left images) shows category 1 on enamel, close to the cervical area. The two right images show category 1 abfraction in the upper premolars (centre right image) and upper canine and first premolar.

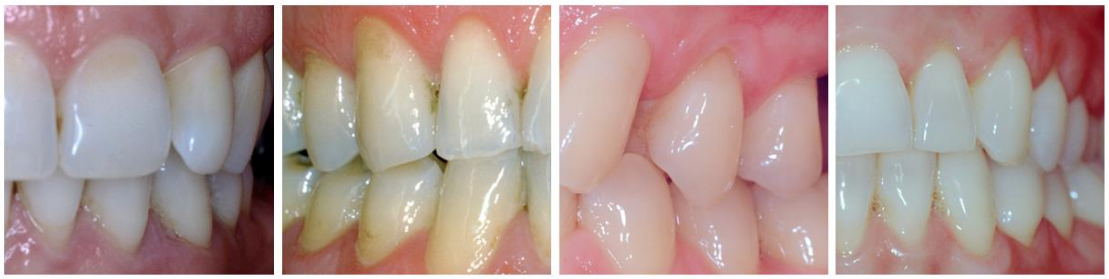

Figure 7.77 Category 1 abfraction.

The identification of emerging cervical abfraction in periodontal patients seems to be a relevant issue due to its prognostic meaning. However, emerging abfraction is characterised by very subtle changes that could go unnoticed. Category 1 consists of minimal loss of contour and this seems difficult to objectively assess, which is why, to our understanding, the identification of localised gingival recession and occasional dark discoloration might be useful complementary signs to be assessed. Localised gingival recession, especially if it develops on thick gingival margins, seems to be a more objective parameter than minimal loss of tooth contour. This approach seems controversial, since until recently, there was no supporting data on the association of gingival recession, abfraction and bruxism.

Figure 7.78 depicts the baseline status and the final outcome after more than 20 years. The only identifiable baseline sign was localised gingival recession in the areas where abfraction developed.

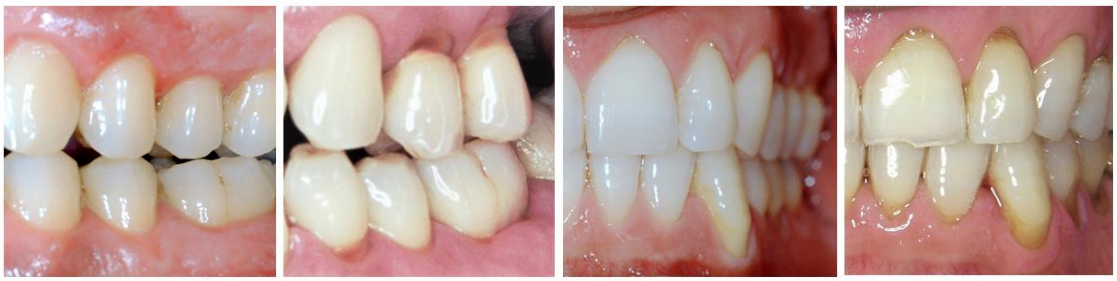

Figure 7.78 Abfractions emerging as localised gingival recession.

Figure 7.79 is quite illustrative. This is a female type 2 patient who will experience an unfavourable long-term outcome after 26 years. The only baseline predictors were gingival recession and dark discoloration on the cervical areas of the lower premolar and molar. It seems logical for gingival recession to be present in the upper anterior teeth with thinner marginal periodontium. However, the marginal periodontium of the lower canine and posterior teeth is thicker, so this periodontium is presumably not predisposed to gingival recession. Thus, the presence of this localised recession in a thick marginal periodontium seems to be a more objective and meaningful parameter than minimal loss of contour. Besides, this patient will not experience increased tooth wear since her teeth present higher mobility.

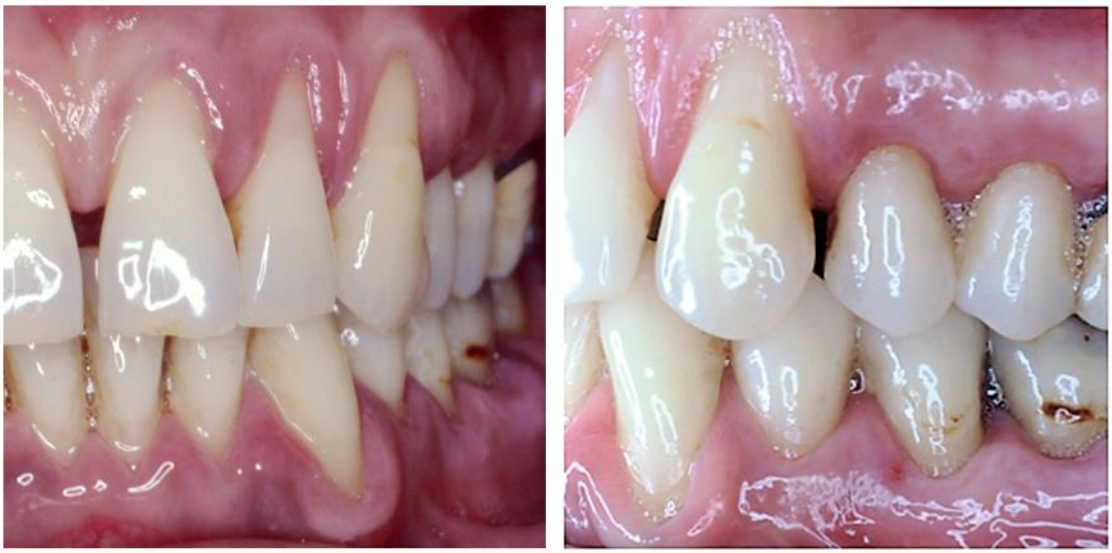

Figure 7.79 Abfractions emerging as localised gingival recession and dark discoloration.

Figure 7.80 shows the development of abfraction in teeth that despite presenting minimal loss of contour on the cervical area, present baseline gingival recession. In the right hand case, baseline gingival recession involves the interproximal area, so it could not be localised gingival recession. In this case, gingival recession would result from generalised attachment loss together with a certain extent of occlusal

overload. On the contrary, the left-hand case could be interpreted as a more genuine localised gingival recession. As a matter of fact, the gingival recession of the upper second premolar shapes the characteristics of the classic Stillman's cleft.

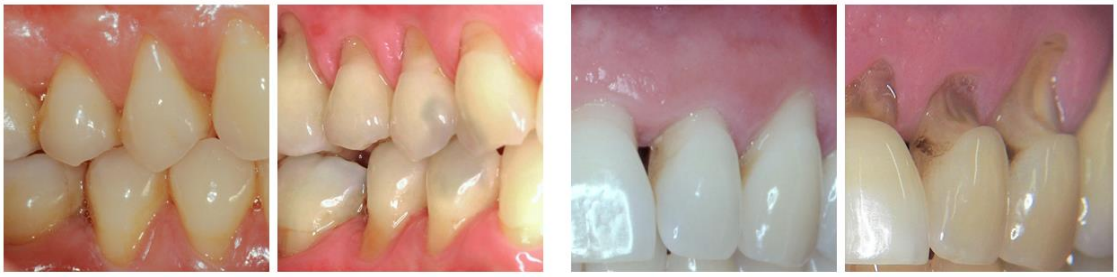

Figure 7.80 Abfractions emerging as localised gingival recession.

We will now focus on what we interpret as occasional dark discolorations. A lot of abfraction starts out as well-defined changes in colour on the enamel or root surface. Furthermore, these changes occur before any loss of tooth surface contour occurs. This colour change consists of an intrinsic discoloration that, to our understanding, resembles reactive dentin. Certainly, reactive dentin seems to develop in deep abfraction, jeopardizing the pulpal tissues, but we did not find a reasonable explanation for the early discoloration of emerging abfraction. Altered collagen fibre metabolism might be involved, especially in heavy smoking patients. Some of these early lesions disappeared as the abfraction progressed, while others remained. The presence of 'hairline' enamel cracks was found to be a common feature associated with this type of emerging abfraction.

Fig 7.81 depicts some examples. The one on the left is hard to identify in the upper first premolar. The one on the right, close to the cementoenamel junction, is the dark discoloration while the more coronal lesion was caries. The dark discoloration remained as such during the entire follow-up. Several follow-ups of these lesions will be presented later on.

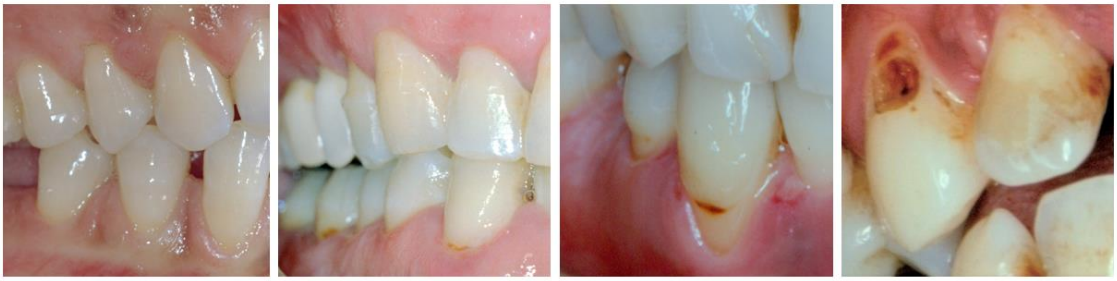

Figure 7.81 Several emerging abfractions.

Figure 7.82 shows category 1 and 2 abfraction lesions in the dentin. Category 1 in the left-hand image; category 2 in the centre left image in the upper canine; category 2 in the upper premolars of in the centre right image. The right hand image depicts category 2 on enamel. This was the patient with the heaviest bruxism (eccentric).

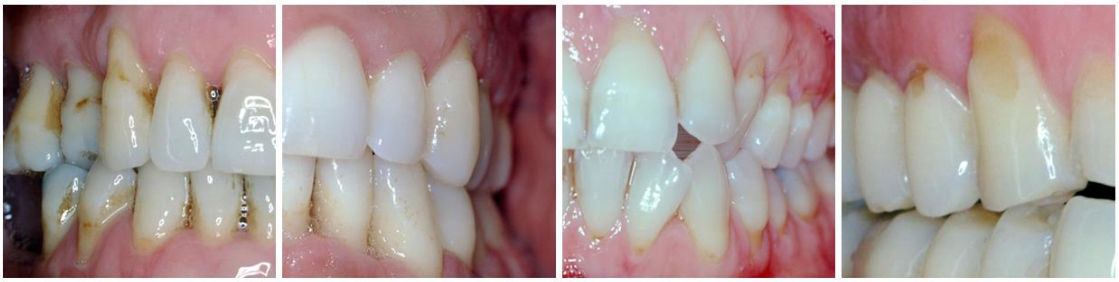

Figure 7.82 Grade 1 and 2 abfraction category

Figure 7.83 depicts category 3 abfraction in several teeth. Only the patient with dark discoloration experienced an unfavourable outcome, losing many teeth. Figure 7.84 shows several category 3 lesions. The abfraction in the two left images are semilunar while the ones in the right-hand images, in the upper premolars and lower premolar, are wedge-shaped with sharp external and internal angles.

Figure 7.83 Category 3 abfractions.

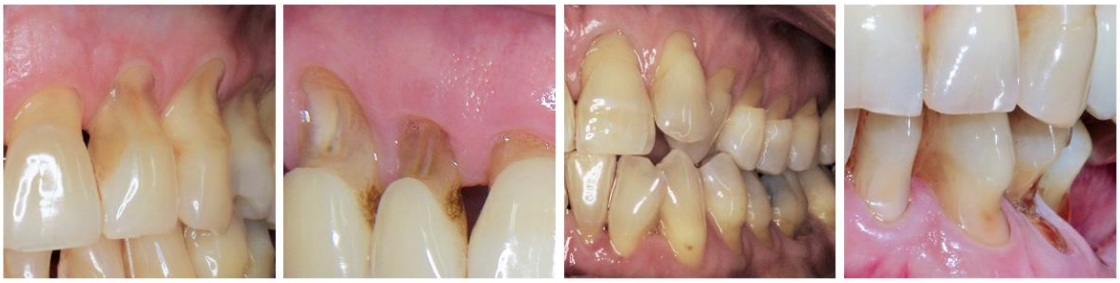

Figure 7.84 Category 3 abfractions.

Next we will look at incisal and occlusal attrition and its differential diagnosis with incisal and occlusal abfraction lesions. Figure 7.85 shows the long-term follow-up of two wear facets that should be interpreted as category 1 occlusal attrition: minimal loss of contour. In the left case, with moderate bruxism, the sharp angles of the vestibular wear facets have rounded with time. The mesiolingual cusp presents an enamel fracture with no defined contours. The right-hand images, also with moderate bruxism, depict a similar progression of the wear facet of the second premolar. The long-term outcome of these patients was favourable.

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6. Classification and prognostic meaning of periodontal osseous defects.

After decades of analysing tooth loss due to periodontitis, we were missing a key detail: teeth are lost in association with certain types of periodontal defects, both furcation and intraosseous defects. In the same way, periodontal regeneration occurs in association with certain types of periodontal defects, in certain patients. After analysing over an average of 25 years how hundreds of defects evolved, in relation to loss of support per se, bruxism, smoking, and tooth loss, we were able to develop the classification of periodontal defects for molars and single-rooted teeth, which we incorporated into Perioproject 2.0.

In section 3 of this chapter (Vertical or circumferential bone defects and/or furcation lesions) a systematic classification of periodontal defects was introduced, according to four different scenarios based on the way smoking and bruxism associate with these defects.

6.4.1 Basic criterium de define periodontal osseous defects in molars

This classification has the peculiarity of simultaneously interpreting the loss of support and the remaining bone support. As it is shown in Figure 6.2, when stablishing the relationship between the level/levels on interproximal bone (blue lines) and the location of the fornix of the furcation (yellow dot), in defects 1 and 2, the fornix of the furcation locates coronal to the interproximal bone levels. The contrary occurs in defect 3, so the fornix of the furcation is located apically to the interproximal bone levels. It would suggest an “early” furcation lesion, in the absence of significant interproximal bone loss.

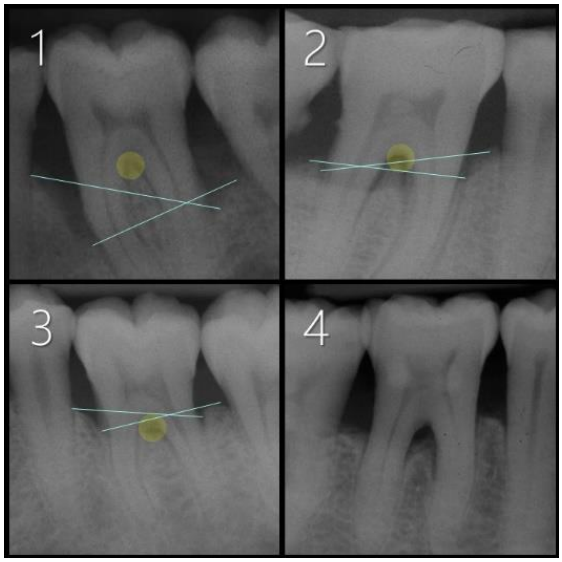

Figure 6.2

6.4.2. PODs classification index for molars

Figure 6.3 depicts images representing the classification for molas

- Defect 0 (horizontal)

Horizontal bone loss without intraosseous component and intact furcation

- Defect 1

Any vertical and or horizontal interproximal defect, with the base of the defect apical to the fornix of the furcation, and intact furcation. These defects are mainly vertical or craters. The progression of this defect towards the furcation would result in the complex defect 2.

- Defect 2

Any interproximal defect with FL II. The fornix of the furcation tends to be located coronal to the most apical interproximal bone levels. Since it was difficult to identify the lesion in upper molars, clinical identification of FL II is required, besides

the indicative criterion of defects 2 in lower molars. The interproximal defect could be intrabony (vertical or crater) or horizontal.

- Defect 3

Any FL II or III with intact interproximal bone, or minimal BL (<20%). The coronal base of the FL III, or the location of radiodensity changes in FL II, tend to be located apical to the interproximal bone levels.

The vertical extent of FL II or III can be located from 1 to 4 mm apical to the fornix of the furcation. In upper molars, widening of the periodontal space in the furcation area or around the roots, shaping circumferential defects, are reliable signs, together with clinical detection of FL II or III. These defects usually evolved to the following defect, with increased interproximal BL.

- Defect 4

A defect 3 with interproximal BL 20%-40% in one or two interproximal surfaces, with FL III in lower molas, and FL II or III in upper molars. Many defects in upper molars were difficult to classify, not even with flap access, since these were complex, with a variable extent of FL involvement, depending on the root anatomy. So, due to limitations to differentiate defects 2 and 4 in upper molars, the type of defects in lower molars would be an indicative criterion.

Two patterns of progression can be distinguished: interproximal horizontal BL and interproximal vertical or circumferential BL, extending towards the furcation and around the roots.

- Defect 5

This is the advanced defect with FL III and interproximal bone loss >40%. It progressed with either a horizontal or a vertical or circumferential defect pattern.

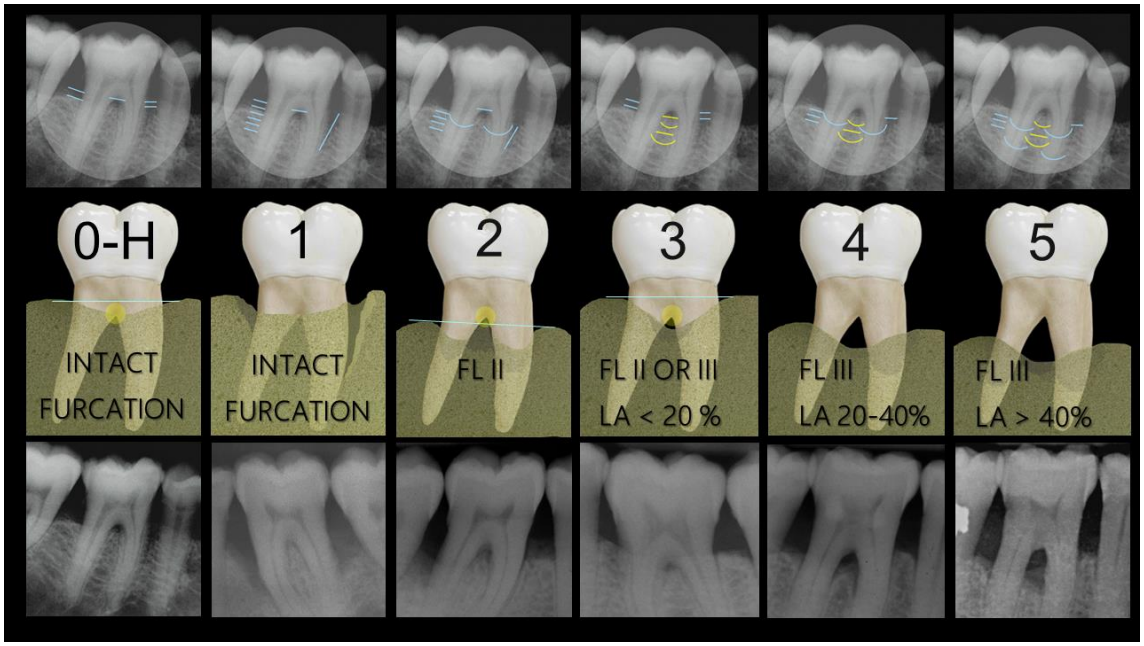

Figure 6.3 PODs classification for molars

6.4.3. PODs classification index for non-molar teeth

Figure 6.9 depicts images representing the classification for non-molar teeth.

Defect H (horizontal): horizontal bone loss without intraosseous component. Bone loss would range from 10% to 70%. Bone loss exceeding 70% would associate with intraosseous component.

Defect 1: Any vertical interproximal defect, without bone loss in the other side.

Defect 2: Any vertical interproximal defect, with bone loss in the coronal third of the root in the other side

Defect 3: Any vertical interproximal defect, with bone loss reaching half the root length in the other side

Defect 4: Interproximal bone loss reaching half the root length, with a circumferential intraosseous component in the middle third of the root

Defect 5: Interproximal bone loss reaching half the root length, with a circumferential component reaching the apical third of the root

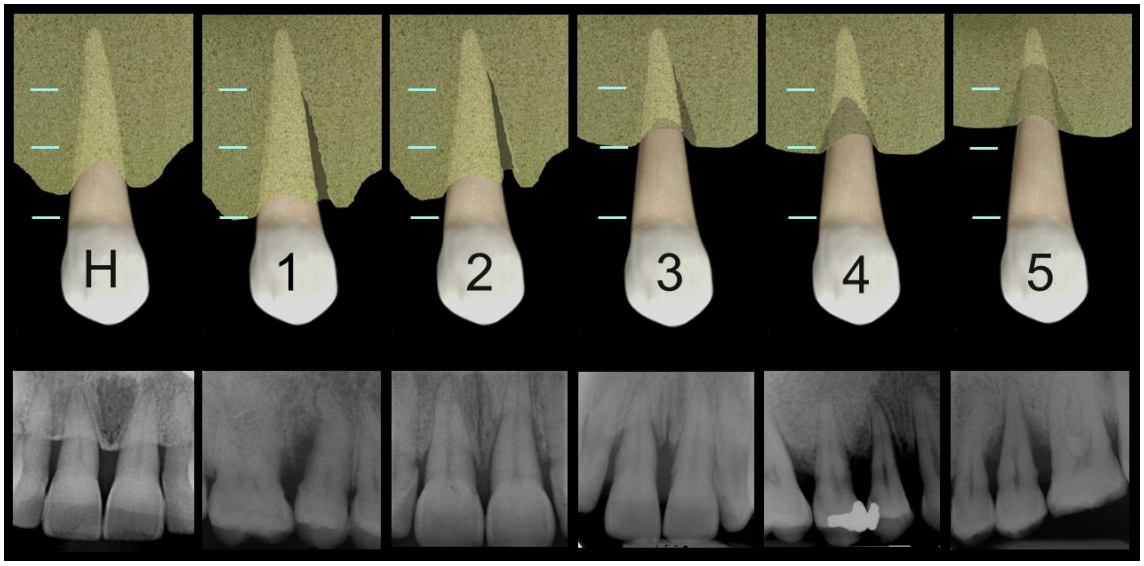

Figure 6.9 PODs classification for non-molar teeth

Defects 1 and 2, in the absence of smoking, would respond favorably to periodontal regeneration. Defect 3 would respond in a more unpredictable way, favorably or unfavorably. Defects 4 and 5 would be more unstable and associate with tooth loss.

Defect H with high bone loss levels, in the absence of smoking and bruxism, could maintain stable, and would justify surprising long-term maintenance of teeth with minimal bone support.

The following are examples of the evolution of these defects.

D 0, or horizontal bone loss, appears to have required little attention compared to the other periodontal defects. However, this D H is worth exploring further. Its prognosis would not depend so much on the bone loss level; in other words, a 40% bone loss would not necessarily present a better prognosis than a 70% bone loss, and a 70% bone loss would not necessarily have a bad prognosis.

Therefore, ranking D H according to the percentage of bone loss, in the absence of risk factors other than the loss of support itself, would not be useful for the prognosis (Figure 6.10)
In addition, a D 0 can occur at one point in time and may progress to D 2, 3, 4 or 5, depending on the prognostic factors involved (Figure 10 and 11).

Chapter 9

Implementing the LTOP system in daily practice

This Chapter consists of a practical seminar on periodontal prognosis.
Implementing the LTOP system for each of the periodontal patients that will be

introduced can be an enriching exercise. It might increase clinical experience and skills and provide more a solid basis to assign periodontal prognosis.
Twenty cases of periodontitis will be presented, enabling the reader to assign overall, individual and comprehensive periodontal prognosis based on data on patient-related factors, the clinical images, the parameters assessed in the periodontal chart and the radiological images. The final long-term outcome is presented at the end of each case, detailing the actual survival time of each tooth. These images have been reduced, so that the reader can easily avoid seeing them before assigning his/her own periodontal prognosis.

CASE 1

This is a 36-year-old female patient whose chief complaint was spontaneous gingival bleeding and bleeding upon mastication. The patient had also recently noticed a diastema developing between the central incisors. The last prophylaxis was performed many years ago.

Her parents lost their teeth early on in life. This patient made it clear that her main concern was whether she was going to lose her teeth, especially those that had lost almost half the periodontal support and the ones experiencing the sudden diastema.

This patient had never smoked and was not aware of any sign or symptom compatible with bruxism. As far as she could remember, her last dental prophylaxis had been performed a couple of years before.

The extent of gingival inflammation is evident in the clinical image (Figure 9-1-1), with generalised category 3 Gingival Index, besides the increased probing pocket depth. The reader can use the clinical and radiological data (Figures 9-1-2, 9-1-3, and 9-1-4) to assign overall and individual tooth prognosis assuming that this patient will strictly comply with periodontal maintenance.

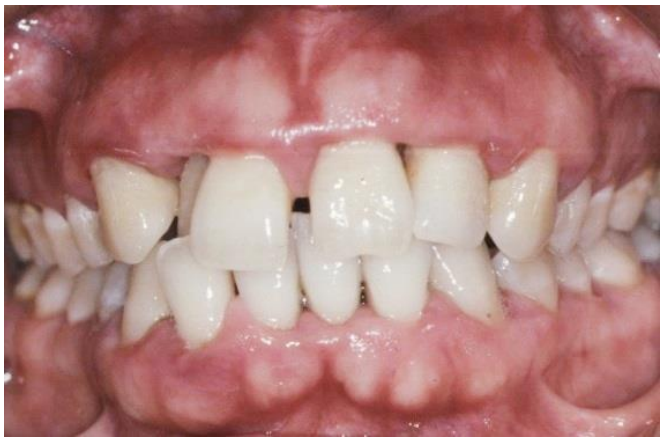

Figure 9.1.1

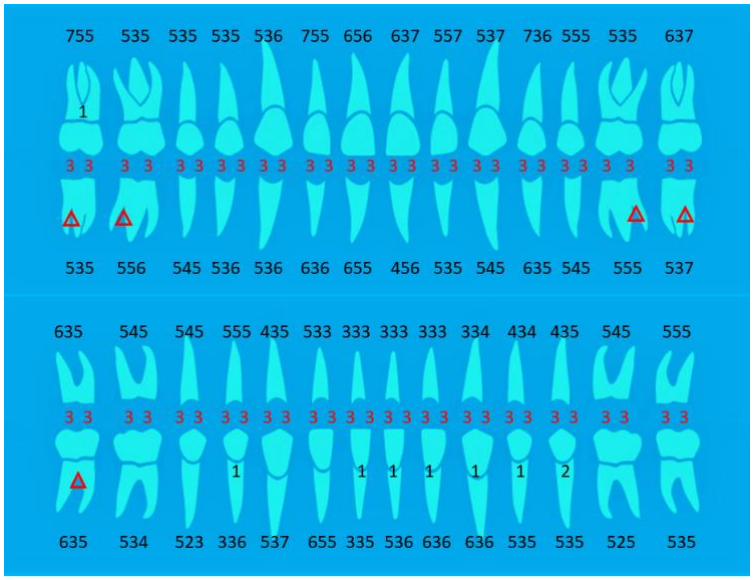

Figure 9.1.2

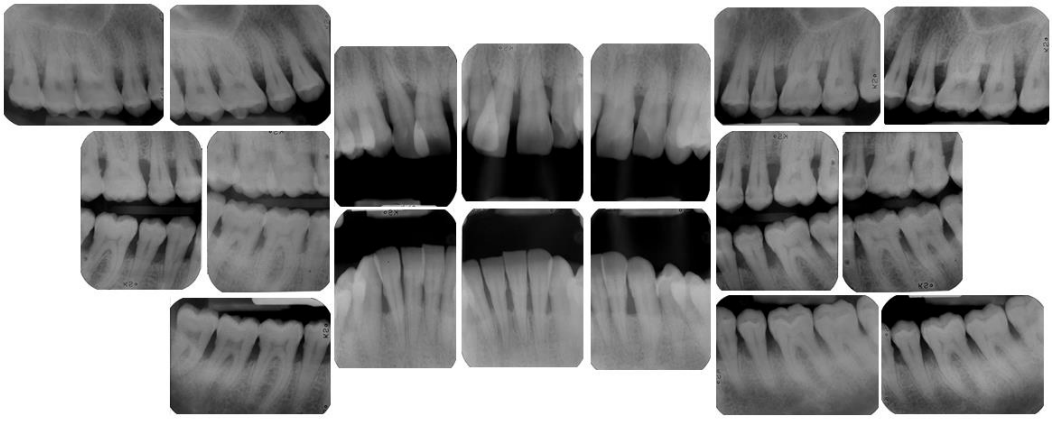

Figure 9.1.3

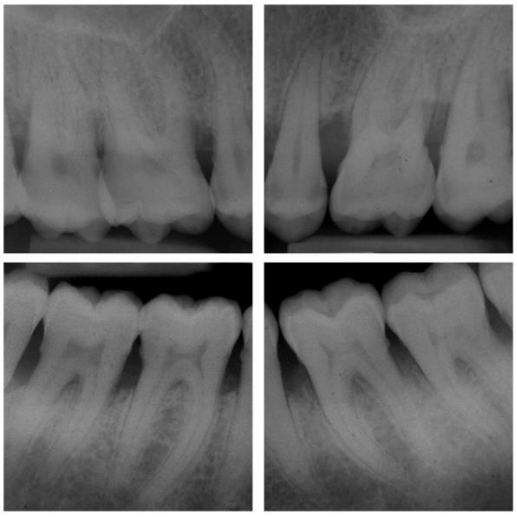

Figure 9.1.4

Overall, individual and comprehensive periodontal prognosis

Overall prognosis

Overall prognosis

This patient presented a low risk of tooth loss according to an LTO index category of 0. This category corresponded to the presence of heavy subgingival calculus (0), increased gingival inflammation as shown by a mean Gingival Index above 1.7 (0), absence of furcation lesions and/or vertical defects (0), absence of generalised gingival recession (0) and absence of abfraction or localised gingival recession in areas of thick gingival margins (0).

Certainly, several molars present grade II furcation lesions, but these lesions have better prognosis, since they correspond to the following type of furcation lesion: - B. 1. Furcation lesion on reduced periodontium (Chapter 7, section 3.) or defect D 2. A high percentage of molars with this type of lesion, in these patients, can be maintained in the long-term.

In addition to the assigned category LTO 0, this patient does not present smoking and bruxism. So what we have here is complementary information to assign overall prognosis: in the absence of smoking and bruxism patients present the characteristics shown in Figure 9-1-5, as already addressed and according to our data.

Since smoking and bruxism are not involved, the only two additional relevant predictors would be severe periodontitis and non-replaced missing teeth. In other words, overall prognosis would depend on the severity of the disease and the capability of restoring missing teeth.

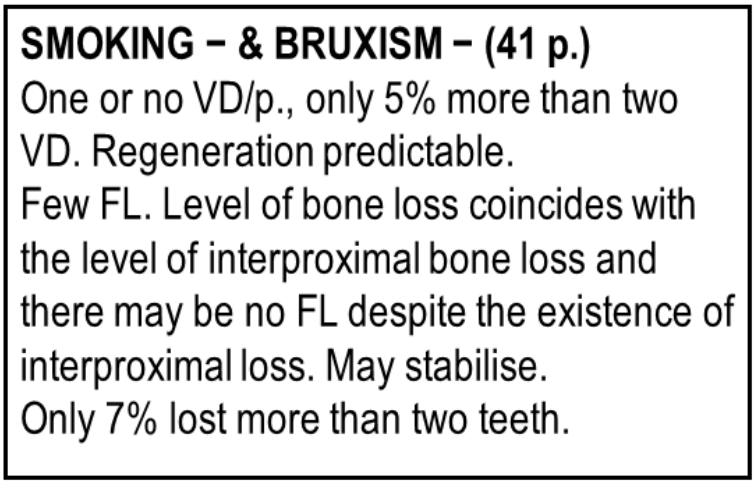

Figure 9.1.5

Individual tooth prognosis

Perioproject assigned survival times of 12 to 22 years to several teeth. In this particular case, these intervals correspond to the intermediate category of tooth-related factors in the absence of the patient-related factors of smoking and bruxism. These teeth remained stable during the follow-up period (Figure 9-1-6).

Comprehensive prognosis

In our database, no single tooth assigned a survival time of 12-22 years was lost in LTO 0 patients despite severe attachment loss in patients complying with periodontal maintenance. For LTO 0 to 3 patients, between 0% to 5% of these teeth were lost. If this particular patient presented teeth assigned intermediate survival times (9 to 20 years and 6 to 20 years) or shorter survival times (5 to 18 years and 4 to 13 years), between 3% and 32% and between 21% and 59% of these teeth would be lost, respectively. The teeth lost usually presented attachment loss above 70% or were upper molars and lower second molars with short and conical roots.

Thus, almost 40% to 60% of teeth with the highest periodontal involvement could be maintained in the long-term. Nonetheless, in the absence of bruxism and smoking, the percentage of teeth assigned shorter survival times is quite low.

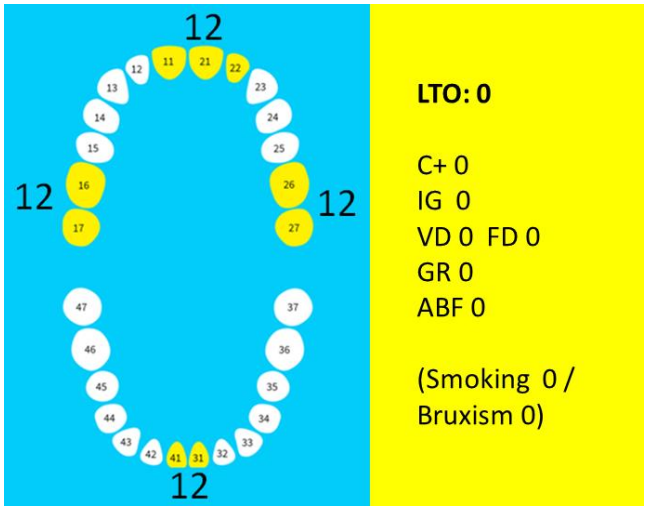

Figure 9.1.6

Actual outcome

This patient did not lose any teeth due to periodontitis despite the extent of attachment loss in several teeth. The length of the follow-up was 24 years. The second upper left molar was lost due to endodontic complications.

The diastema between the central incisors closed during active periodontal therapy without the use of any orthodontic appliance. The spontaneous repositioning of these incisors took place after removing the granulomatous tissue following scaling and root planning, together with occlusal adjustment. The spontaneous resolution of this diastema in the presence of increased gingival inflammation is a common outcome that can go unnoticed.

Figure 9-1-7 shows the clinical images of the response to active periodontal therapy (left image) and after 24 years (right image). The extent of gingival

recession after active treatment was comparable to gingival recession taking place during the entire follow-up.

Figures 9-1-8 and 9-1-9 show the radiographs after three years and at the end of the follow-up respectively.

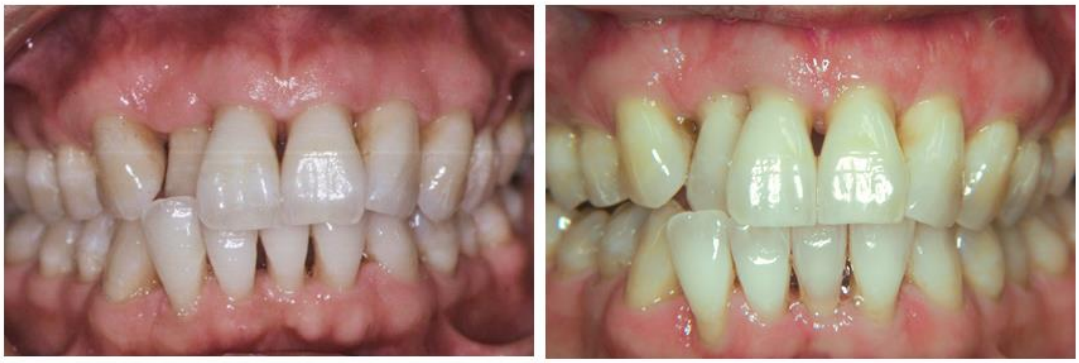

Figure 9.1.7

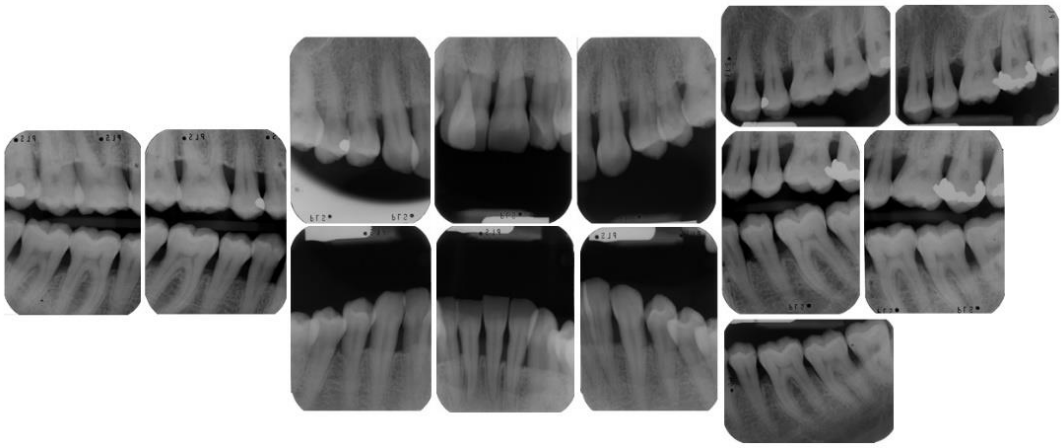

Figure 9.1.8

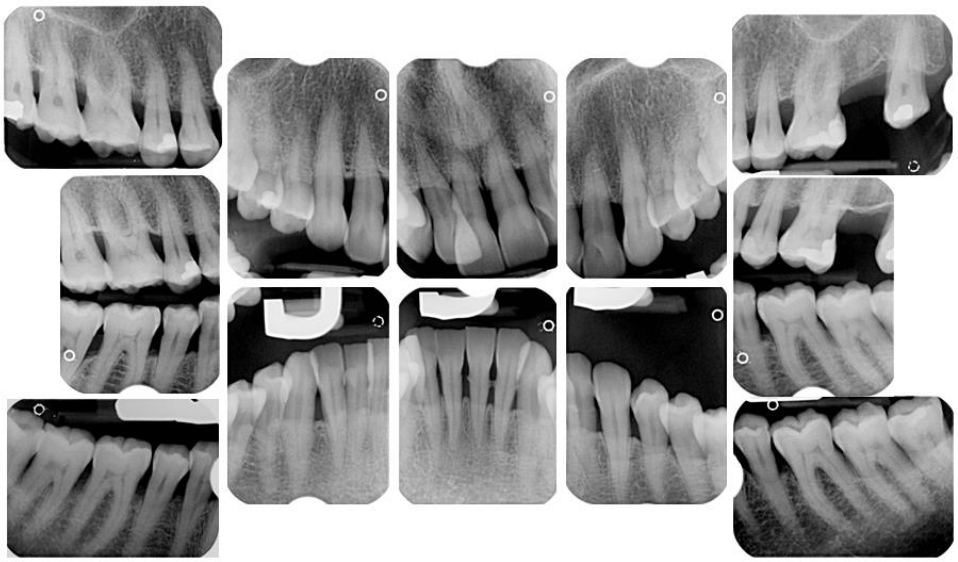

Figure 9.1.9