University of Muenster – Dental School
Department of Periodontology
Provisional Director: Prof. Dr. Dag Harmsen

Multifactorial risk assessment of tooth loss in subjects receiving periodontal therapy

Submitted in Fulfilment of the
Requirements for the Degree
doctor medicinae dentium
(Dr. med. dent.)

The Faculty of Medicine,
University of Muenster

by
Faggion, Clóvis Mariano Junior
Sao Miguel do Oeste / Brasilien
2005

Gedruckt mit Genehmigung der Medizinischen Fakultät der Westfälischen Wilhelms-Universität Münster

University of Muenster – Dental School
Department of Periodontology
Provisional Director: Prof. Dr. Dag Harmsen

Multifactorial risk assessment of tooth loss in subjects receiving periodontal therapy

Submitted in Fulfilment of the
Requirements for the Degree
doctor medicinae dentium
(Dr. med. dent.)

The Faculty of Medicine,
University of Muenster

by
Faggion, Clóvis Mariano Junior
Sao Miguel do Oeste / Brasilien
2005

Dekan: Univ.- Prof. Dr. med. Heribert Jürgens

1. Berichterstatter: Univ.-Prof. Dr. med. dent. Thomas F. Flemmig

2. Berichterstatter: Prof. Dr. med. dent. Edgar Schäfer

Tag der mündlichen Prüfung: 28.09.06

University of Muenster – Dental School

Department of Periodontology

Provisional Director: Prof. Dr. Dag Harmsen

Referent: Univ.-Prof. Dr. med. dent. Thomas F. Flemmig

Koreferent: Prof. Dr. med. dent. Edgar Schäfer

Zusammenfassung

Multifactorial risk assessment of tooth loss in subjects receiving periodontal therapy

Clóvis Mariano Faggion Junior

Ziel der Studie: Ziel dieser retrospektiven Studie über einen Zeitraum von 8 bis 15 Jahren war die Bestimmung von Zahnverlust und der diesem zugrunde liegenden Risikofaktoren bei erwachsenen Patienten, die sich in parodontologischer Behandlung befanden. Hiermit sollte ein Modell zur Vorhersage von Zahnverlust erstellt werden.
Material und Methoden: Von insgesamt 780 kaukasischen Patienten, die in den Jahren 1989/90 an einer Universitätsklinik eine systematische Parodontitistherapie erhielten, wurden von 198 Patienten (Durchschnittsalter 47.58 SD +/- 10.42) die Befunde ausgewertet. Klinische und röntgenologische Daten wurden zu Beginn, nach Abschluss der Initialtherapie sowie nach weiteren 8 – 15 Jahren, im Laufe der unterstützenden Parodontitistherapie erhoben. Die Compliance der Patienten wurde anhand der Frequenz von Terminen zur unterstützenden Parodontitistherapie bestimmt. Mit Hilfe der multifaktoriellen Regressionsanalyse wurde auf mehreren Ebenen (Patient, Zahn, Messstelle) der Einfluss verschiedener Parameter auf den Zahnverlust bestimmt (Alveolarknochenverlauf, Taschensondierungstiefe (TST), Zahnsensibilität, Zahnmobilität, Vorhandensein von Restaurationen, einwurzelige versus mehrwurzelige Zähne, Plaqueindex (PI), Blutung auf Sondierung (BAS), Frequenz der unterstützenden Parodontitistherapie sowie allgemein anamnestischer Befund).
Ergebnisse: 166 Zähne (3,64 % der gesamten Zähne) gingen während der Initialtherapie verloren und 249 Zähne (5,46 % der gesamten Zähne) im Laufe der unterstützenden Parodontitistherapie. Die Regressionsanalyse der Daten von ausschließlich den Zähnen, die im Laufe der unterstützenden Parodontitistherapie extrahiert wurden, zeigte einen deutlichen Zusammenhang von Diabetes, Zahnmobilität, mehrwurzeligen versus einwurzeligen Zähnen, Zahnsensibilität und Zunahme des Alveolarknochenverlustes mit Zahnverlusten ($p < 0,05$). Teilweise ließen sich die erhaltenen Ergebnisse durch das Modell der Regressionsanalyse erklären ($R^2 = 0,15$).
Schlussfolgerung: Bei diesem Modell der Regressionsanalyse wurden sowohl zahn- als auch patientenbezogene Parameter als Prädiktoren für Zahnverlust identifiziert. Des Weiteren können die Ergebnisse dieser Studie möglicherweise zu einer Verbesserung der klinischen Entscheidungsfindung und der gesamten zahnmedizinischen Behandlung führen, wenn sie in einem Modell zur Vorhersage von Zahnverlust angewandt werden.

Tag der mündlichen Prüfung: 28.09.06

Abstract

Aims of the study: This 8 through 15-year retrospective study was performed to assess tooth loss and its underlying risk factors in an adult population receiving periodontal therapy aiming the construction of a predictor model for tooth loss.

Materials and Methods: 198 patients (mean age 47.58 SD +- 10.42) from a group of 780 caucasian people who received systematic periodontal treatment in 1989/1990 in a university setting had their charts recorded. Clinical and radiographic data were obtained at baseline, after accomplishment of the initial periodontal therapy and subsequently 8 through 15 years of supportive periodontal therapy. Compliance of the patients was assessed through the recall frequency during the supportive periodontal therapy. Multilevel logistic regression
using three different levels of analysis (patient, tooth and site level) was used to test the influence of various parameters on tooth loss (alveolar bone level (ABL), pocket probing depth (PPD), tooth sensitivity, tooth mobility, presence of restorations, single versus multi rooted teeth, plaque index (PI), bleeding on probing (BOP), frequency of supportive periodontal therapy and systemic health).

Results: 166 teeth (3.64% of all teeth) were lost during initial therapy and 249 teeth (5.46% of all teeth) during the supportive periodontal therapy, respectively. Logistic regression analysis, with only data from teeth extracted during supportive periodontal therapy period, revealed a strong association of diabetes, tooth mobility, multi-rooted vs. single-rooted teeth, tooth sensitivity and increment in alveolar bone loss with tooth loss ($P<0.05$). The model of regression explained partially the results obtained ($R^2= 0.15$).

Conclusions: In this model of regression, both tooth- and patient-related parameters were identified as predictors for tooth loss. In addition, the study findings, when applied in a predictor model for tooth loss, may have the potential to improve clinical decision making and subsequent overall dental treatment.

1. Introduction

1 . 1 Etiopathogenesis and Treatment of Periodontitis

Periodontal diseases affect the tooth supporting structures and cover a group of inflammatory entities with bacteria as the main etiologic factor. Gingivitis and periodontitis, the most common manifestation of the disease, share the same clinical signs of inflammation. However, loss of clinical attachment is observed just in the latter. It is well established that the reduction or even eradication of periodontal pathogenic bacteria usually brings the gingival tissues to a healthy state (Loe et al. 1965, Theilade et al. 1966, Listgarten, 1988). Nevertheless, in some patients merely antiinfective therapy may not be sufficient to effectively treat and control periodontitis due to the complexity and multifactorial features within the process of periodontal destruction. Some important concepts related to the onset and development of periodontits are described below.

1 . 1 . 1 Dental biofilm definition

The number of bacteria colonising some soft tissue surfaces as mucosa and skin exceeds the number of cells forming human body (Tlaskalová-Hogenová et al. 2004). To maintain a healthy state, a harmonious interaction between bacteria and the host is beneficial, yet even crucial. Within this biocenosis, the physiologic desquamation of epithelium and the innate and acquired immune responses are usually able to regulate the body homeostasis.

However, under specific environmental conditions, bacteria adhere to moist surfaces within an extracellular polymer, forming a structured community of microorganisms known as biofilm (Hoyle & Costerton 1991). Bacteria within this complex environment are a major etiologic factor for infectious diseases especially when localized in implants or catheters placed in various parts of the body, such as urinary tract (Choong & Whitfield 2000), heart (Holman et al. 2004) and joints (Neut et al. 2004).

Under the specific condition within the oral cavity, microorganisms are able to adhere to teeth or other hard surfaces, such as endosseal implants or prosthetic restorations. Its unique characteristics to resist to the innate and immune responses and antimicrobial agents (Bowden & Hamilton, 1998) makes this mass of bacteria a potential source of periodontal infections (Sockransky & Haffajee 2002). The majority of microorganisms within a biofilm may be essentially nonpathogenic. However, they may cause inflammation if they become more in numbers due to various factors such as grow rate, nutritional status, temperature, pH or early exposures to low doses of antibiotic or if the host becomes compromised (Costerton et al. 2003).

When attached to tooth surface, biofilm resides in the supra and subgingival areas and may have distinct composition (Sockansky & Haffajee 2002). *Actynomices species* are predominant in supragingival plaque in both, healthy and diseased patients (Ximenez-Fyvie et al. 2000). Similar to supragingival plaque, *Actynomices species* represent the majority of subgingival microrganisms. However, a higher number, proportion and prevalence of species with potential to promote periodontal destruction such as *Porphyromonas gingivalis, Tannerella forsythensis, Treponema denticola, Prevotella intermedia, Prevotella nigrescens* are also present in the subgingival milieu (Socransky & Haffajee 2002).

1 . 1 . 2 Dental biofilm formation

A considered “healthy gingiva” shows pink color and firm consistency with no evident signs of clinical inflammation. However, histologically, an inflammatory infiltrate is generally present (Lindhe et al. 1997). The process of dental biofilm formation starts with the adhesion of gram-positive microorganisms to the enamel surface (Yao et al. 2003). This first account of bacteria is important for the biofilm formation because it provides surfaces for attachment, or modifies the environment, for subsequently occurring colonizers (Li et al. 2004). These microorganisms grow and spread onto tooth and hard tissues and are known as “primary colonizers” (Nishihara & Koseki 2004). If this layer of bacteria is not mechanically disrupted from the tooth surface, other previously planctonic bacteria may enter the biofilm community. At this stage, the various microorganisms benefit from each other, with dead or lysed bacteria serving as nutrients for the remaining organisms (Theilade & Theilade 1970). As the layers of bacteria increase, the inner community of the plaque changes from aerobes to facultative anaerobes due reduced oxygen supply and a stable situation with an obligate anaerobic environment in the inner layers becomes established. (Bradshaw et al. 1998).

1 . 1 . 3 Pathogenesis of Periodontitis

The mechanism of periodontal pocket formation is yet not fully understood. A probable explanation for this mechanism is that the diffusion of microbial products from supragingival biofilm to the surface of gingival epithelium increases the flow of gingival crevice fluid from periodontal tissues (Nishihara & Koseki 2004). These events

create a propitious subgingival environment allowing specific periodontal bacteria to grow and produce substances such as proteases and other toxins.

Pathogenic bacterial products destroy the extracellular matrix and attack the collagen structure of host cell membranes or may activate humoral immune system, which also may deteriorate the adjacent tissues and develop periodontal pockets ((Darveau et al. 1997, Lindhe et al. 1997).

However, not all patients subgingivally harbouring periodontal pathogens will develop periodontitis. Moreover, onset and progression of periodontal destruction depend on the presence of various factors occurring simultaneously (Socransky & Haffajee 1992). A fundamental prerequisite is virulence of the periodontal pathogens. Interestingly, various strains of same species may not have similar potential for periodontal destruction. An example of difference of bacterial virulence is the variation of the *P. gingivalis* fimbria genotypes in healthy and diseased periodontal sites. Type II fim A organisms seem to be found in periodontitis patients more frequently than other strains (Amano et al. 2004, Missailidis et al. 2004). Hence, the pathogenicity of periodontal bacteria from same species might be regulated by important genetic components.

Another prerequisite for the development of periodontitis is the impairment of the host immune system. For example, functional polymorphisms may be linked to the severity of periodontal destruction. In a study with smoker patients, subjects being positive for IL-1α and IL-1ß polymorphisms had 4 times higher risk for attachment loss when compared to genotype-negative smokers (Meisel et al. 2002). In other study, a same comparison was made regarding to tooth loss. Patients positive for IL-1 α and IL-1ß had 2.7 times increased risk of tooth loss from periodontal diseases compared to Genotype-

negative individual (McGuire & Nunn, 1999). Therefore, subjects with certain genotypes may be more susceptible to periodontitis than others.

Another factor that may affect the host resistance is the influence of some systemic diseases on the development of periodontitis such as e.g. diabetes mellitus. It is well established that poorly controlled diabetes increases the risk for periodontal destruction (Wilson 1989, Tervonen & Oliver 1993, Oliver & Tervonen 1994, Soskolne 1998, Reeds 2000, Mattson & Cerutis 2001). In addition, the severity of the attachment loss seems to be directly related to the level of diabetes control. The poorer the diabetes control, the more severe is the loss of attachment (Guzman et al. 2003). A possible mechanism that may explain the influence of the abnormal regulation of glucose metabolism on periodontal tissues is the impairment host resistance due to the dysfunction of neutrophils and excessive tissue destruction by bacteria (McMullen et al. 1981). Moreover, an exaggerated cellular inflammatory response in diabetic patients also may increase the severity of periodontitis with an enhanced production of tumor necrosis factor- α, interleukin 1-ß and prostaglandin E_2 (Salvi et al. 1997).

Regarding the patients smoking habit, there is an irrefutable body of evidence confirming it as an important risk factor for the development of periodontitis (Bergstrom 1989, 2003, Haber et al. 1993, Grossi et al. 1994, 1995, Axelsson et al. 1998, Kinane & Chestnutt 2000, Haffajee & Socransky 2001). Tobacco and smoking use induce modifications in both innate and immune mechanisms of host response (Barbour et al. 1997). As a local effect of nicotine, the first line of neutrophil defense might have its functions impaired by smoking exposure such as phagocitosis and protease inhibitor production (Persson et al. 2001). In addition, these cells of the monocyte/macrophage lineage might be also less activated in smokers, resulting in a

subproduction of determined cytokines that stimulate B-cell proliferation. Thus, the rise of the amount of Ig serum levels in periodontal patients is impaired, causing a reduction of function of the humoral response (Graswinckel et al. 2004).

Taken together, there is a broad body of evidence to affirm that numerous factors have a significant influence on the development of periodontitis. Thereby, the treatment and control of periodontitis may become complex. Thus, a reasonable strategy for the correct treatment and control of disease should include antimicrobial therapy to reduce the numbers of the pathogenic bacteria as well as the assessment of the various factors that contribute for the modification of the normal immunologic host response.

1 . 1 . 4 Treatment of Periodontitis

Due to the solid evidence, regarding bacteria as the main etiology of periodontitis, disruption and reduction of the load of the biofilm adhered to the tooth surface are the current standard treatment (Loesche et al. 2002, Petersilka et al. 2002, Axelsson et al. 2004, Adriaens & Adriaens 2004, Umeda et al. 2004). The elimination of bacterial biofilm and subgingival calculus comprises initially nonsurgical debridement by root instrumentation with manual or power-driven instruments. The aim of the therapy is to reduce as much as possible the sub- gingival biofilm and calculus. It has been suggested that a reduction of 99% of the bacterial content may be achieved immediately after scaling and root planning (Petersilka et al. 2002). Despite the significant reduction of periodontal pathogens after the nonsurgical therapy, eradication off all bacteria is virtually impossible due to the limitation of current modalities of therapy and the ability of bacteria to adapt to the hard tissue anatomy (Petersilka et al. 2002). Clinically,

scaling and root planning reduce pocket depth and signs of periodontal inflammation such as bleeding on probing. Furthermore, gain of clinical attachment may be achieved. After completing the initially perfomed nonsurgical phase, periodontal flap procedures may be indicated in remaining deep pockets, aiming to achieve a further reduction of pocket probing depth or to reestablish physiologic periodontal conditions (Kaldahl et al. 1996).

Supportive periodontal therapy consisting of frequently performed professional supra- and subgingival redebridement is required after the initial therapy in order to control the possible reeinfection and to prevent from further attachment loss. (Lindhe & Nyman 1984, Axelsson & Lindhe1981, Axelsson et al. 2004).

Also, in cases of severe chronic or aggressive periodontal disease, systemic or local use of antibiotics may improve the outcome and lasting stability of the therapy when used in combination with the mechanical debridement procedures (Flemmig et al. 1998, Herrera et al. 2002, Haffajee et al. 2003, Page 2004).

1 . 2 Periodontal Diseases and Tooth Loss

Periodontitis has the potential to damage the tooth supporting tissues and cause tooth loss. Various epidemiological studies have shown that periodontal disease and caries are the most important reasons for tooth loss (Manji et al. 1988, Ong 1996). For example, in a survey study performed in Canada (Murray & Kay 1997), 165 dental practioners provided information on 6134 patients receiving dental treatment. 1710 teeth were extracted during a reference week of treatment. 64.8% of these teeth had periodontal disease and caries as the main reasons for extraction. Another study (Agerholm & Sidi

1988) had even a greater percentage of extracted teeth having caries and periodontal disease as the main reason for their extractions (74%). Regarding the relationship between tooth loss and age, older people may be more susceptible to tooth loss than younger and some authors also suggest that periodontal disease account for the majority of tooth loss in subjects older than 40 (Cahen 1985, Locker 1996, Sayegh et al. 2004). In a 4-year prospective study (Sayegh et al. 2004), 3069 lost teeth were assessed from 2200 selected patients from a low-level socio-economic population seeking dental care. Stepwise logistic regression demonstrated less likelihood to occur tooth extraction at 40 years or less. Furthermore, regarding the type of extracted teeth, the regression analysis has shown in this study a higher probability of tooth loss due to periodontitis in incisors followed with lower premolars. These results are in accordance to other studies that indicate periodontitis as the main reason for extraction of incisors (Manji et al. 1988, Locker et al. 1996).

Thereby, there is strong evidence implicating periodontitis as an important etiologic factor for tooth loss. Therefore, the correct treatment and control of the disease should be considered an important step in the overall dental treatment in order to avoid or reduce tooth extraction.

1 . 3 Socio-economic and functional Impact of Tooth Loss

It is unequivocal, that total and partial edentulism can have a negative impact on life. Some authors suggest that a minimum of 20 teeth may be necessary to keep or restore a normal chewing function and improve the oral status satisfaction of the patients (Kayser

1981, Elias & Sheiham 1999). Another author suggests that at least one occluding pair of molars is necessary to achieve this objective (Sarita et al. 2003).

Therefore, dentistry has a determinant role towards both, social as well as somatic aspects. The importance of functional factors as biting and chewing and the esthetic and communication components has been researched (Osterberg et al. 1982, Kayser 1990). These two authors concluded that the social functions might be even more relevant in the individual's subjective need than a functional dentition. Steele et al. 2004 analysed two national samples aiming to assess how age and tooth loss have influence on the impact of oral health on the regular life. A scale has been used, Oral Health Impact Profile (OHIP), to measure the negative influence of oral conditions on daily life. When the effect of the dependent variable age was eliminated from the statistical regression analysis, loss of teeth was associated with high levels of OHIP. Thus, tooth loss induced a reduction of quality of life in both population samples.

Tooth loss may also have a substantial impact on public economy policy. Complex prosthetic treatments are sometimes deemed necessary to reestablish esthetic and normal oral function of total or partial edentulous patients. Even people with most teeth in function may need some kind of prosthetic treatment due to esthetic reasons. Prosthetic dentistry involves various segments of society such as dental surgeons, dental product companies, dental technicians, dental insurance companies and demands a huge amount of resources. In Germany, 3 billion Euros are paid by the Statutory Health Insurances for prosthetic treatments. Since the insurance only covers roughly 50 % of the total cost, the overall costs amount to approximately 6 billion Euros per year (KZBV Jahrbuch 2003). Comparatively, this amount corresponds in average seven times more expenses in restorative than in periodontal and preventive procedures (Fig. 1).

It is reasonable to think that a tool, which could directly aid dental practioners in determining the prognosis of teeth, would improve the clinical decision making process. In addition, the correct oral care planning would avoid wasting resources and over and under treatment due to the optimization of the dental treatment.

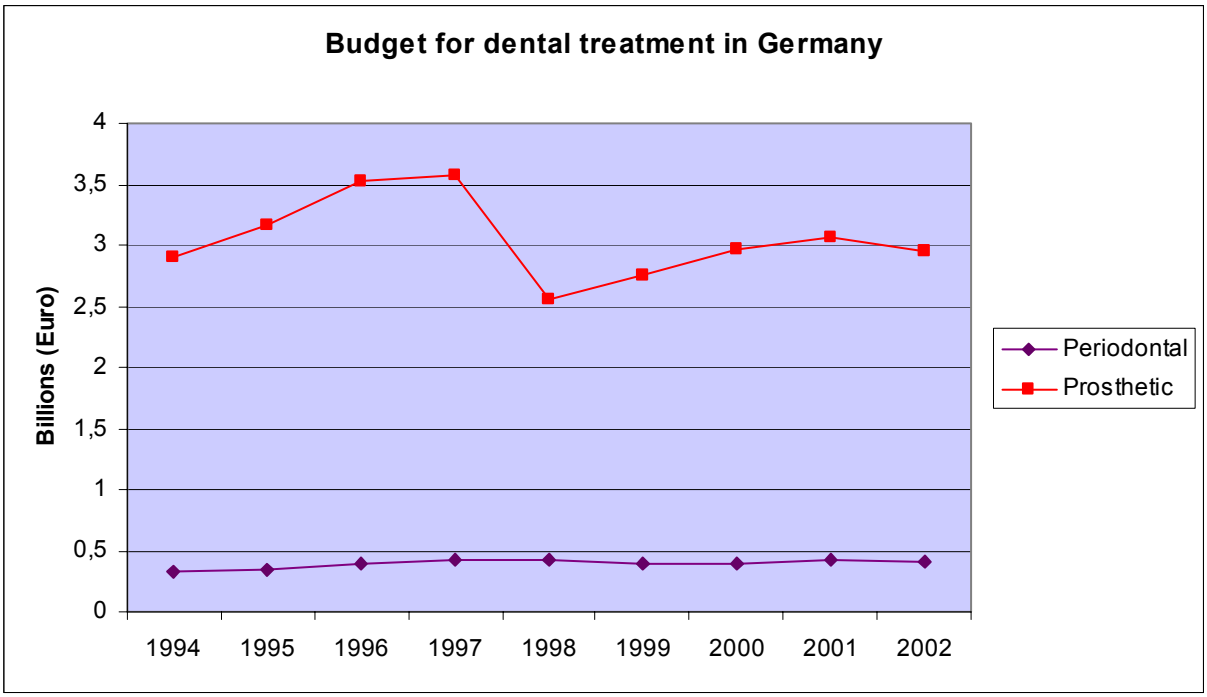

Fig. 1 Presentation of the expenses paid by the Statutory Health Insurances of services related to periodontal and prosthetic treatments in Germany from 1994 to 2002 (KZBV Jahrbuch 2003).

1 . 4 Risk Assessment in Periodontal Treatment

1 . 4 . 1 Definition of Keywords in Periodontal Risk Assessment

In periodontology, risk assessment is a relatively new tool. Due to the prior belief that all gingivitis would inevitably progress to periodontitis and that nonspecific bacteria were the main etiology of the disease, periodontal treatment was focussed, until 80´s, on the elimination of the bacteria as the causal factor. Following this paradigm, the prevalence of periodontitis was extremely high and little error in prediction resulted when everyone was categorized as having the disease (Beck 1994). However, the paradigm shifted with the specific plaque hypothesis and multifactorial components as etiology of disease. Not all gingivitis patients develop periodontitis and thus, the prevalence and incidence are not as high as it was estimated. Hence, certain people or populations are at higher risk than others to develop periodontitis and consequent attachment loss (Loe et al. 1986, Beck 1994). Some concepts about the terminology of risk for diseases will be described to establish the basis for further discussion.
Risk is the probability that an event might occur in the future. In epidemiology, risk factors are described as objective findings that indicate a strong probability of developing unwanted outcomes in people who do not yet have a given disease (Fletcher et al. 1988). In order to assess how strong this probability is, a cause-effect relationship between the factor and disease is generally determined through various studies in humans. A summary about the terminology of possible factors related to periodontitis is demonstrated in Table 1.

Term	Meaning	Strenght of cause-effect	Example
Sufficient cause	It refers to any condition, characteristic, or presence of which, the disease will always occur	*****	Genetic anomaly
Necessary cause	It is the main etiology, but its presence does not necessarily result in presence of disease	****	Mycobacterium tuberculosis
Risk factor	It may be part of the causal chain of periodontitis or exposes the patient to the cause of disease. The factor is found to be consistently associated to the periodontitis process in longitudinal studies.	***	Pathogenic bacteria in the periodontal pocket
Risk indicator	It may be a risk factor but it has been only demonstrated by means of cross-sectional studies.	**	HIV infection
Risk predictor	It is not a part of the causal chain but may be predictive for the increase of periodontitis.	*	History of periodontitis

Table 1. Terminology of risk in epidemiology. The highest term (first column) has the strongest association with the disease. In the third column, five asterisks mean the strongest cause-effect relationship, whereas one asterisk means the weakest cause-effect relationship (adapted from Page & Beck, 1997 and Nunn ME 2003).

Sufficient and necessary causes terms suggest the strongest type of causal relationship because it has been already established their role in the causal chain of disease. The other three terms are determined when assessed in different study designs (Beck 1994, Page & Beck 1997, Nunn 2003). *Risk factors* are assessed by randomized controlled clinical trials and prospective cohort studies (Beck 1994, Nunn 2003) which give the strongest evidence (after the sufficient and necessary causes) of a potential causal relationship between risk and disease (Nunn 2003). *Risk indicators* were only demonstrated in cross-sectional studies and therefore, they need to be investigated in stronger designs of studies (cohort prospective and/or randomized clinical trials) in order to be accepted as risk factors. A *risk predictor* has been associated with disease in

cross-sectional or longitudinal studies, but without a biological relationship with the disease (Beck 1994, Pihlstrom 2001).

1 . 4 . 2 Assessing the Risk for Tooth Loss

Despite all recent forthcomings in periodontal risk assessment, assigning a definitive prognosis to a periodontally affected tooth remains an uncertain task. In daily dental practice, a variety of clinical and laboratory-based criteria is used to create a prognosis on possibly deteriorating attachment level or tooth loss (McGuire 1991, 1996). Furthermore, even dentists specialized in periodontal treatment have been proven to have widely heterogeneous opinions regarding the criteria suitable for risk assessment. Using traditional approaches for treatment planning, the severity of periodontal disease is measured almost exclusively by estimating radiographic bone loss or by the prevalence of deep periodontal pockets (Persson et al. 2003). However, interestingly there is a large body of evidence that severely periodontally diseased teeth may not necessarily be lost in the near future. Classical studies on the long-term effect of periodontal therapy assigned a questionable prognosis to a tooth which had furcation involvement, non-eradicable deep pockets, extensive alveolar bone loss, or marked tooth mobility. Using these criteria in a representative study, out of more than 2000 teeth considered to have a questionable prognosis approximately only one third was lost during the study period of more than 20 years (McGuire & Nunn 1996). Data like these demonstrate the clinical difficulty to assign an exact prognosis to a periodontally involved tooth or dentition.

Another important aspect in assessing risk for tooth loss is its multifactorial feature. Clinical decision making should not be based only in periodontal parameters. In fact, other local factors such as caries, restorations and endodontic lesions may also have influence on the long-term tooth survival. In the literature, there are various studies that analyze the reasons for tooth loss in different population, indicating the influence of these parameters on tooth loss (Niessen & Weyant 1989, Eckerbom et al. 1992, Reich & Hiller 1993, Vignarajah S. 1993, Hiidenkari et al. 1996, Murray et al 1996, Stabholz et al. 1997, Haddad et al. 1999, McCaul et al. 2001, Sayegh et al. 2004, Da´ameh D 2005, Richards et al. 2005, Oginni FO 2005).

As explained in topic 1.2, periodontal diseases and caries are the most common reasons for tooth loss and therefore the weight of these entities on tooth prognosis should be seriously considered. Furthermore, similarly to the effect on development of periodontitis (see topic 1.1.3), systemic factors such as diabetes and tobacco consume seem to have also some negative impact on tooth survival.

As a consequence of this current multifactorial concept of risk, there is an increasing body of literature dealing with the achievement of an accurate analysis of these local and systemic factors related to tooth loss or to allow an adequate disease management (see Tables 2 and 3).

Among these studies, a calculated model of risk prediction in periodontology was proposed recently (Page et al. 2002, 2003). It analyses patient-, tooth- as well as site-based data in untreated patients to determine the risk of developing periodontal disease. However, this model just gives results for the patient- rather than site-based risk for the development of periodontal disease and lacks from the disadvantage that the data were derived from non-treated patients only.

Therefore, it appears necessary to establish a model based on the data of patients receiving periodontal treatment. In addition, this model should be constructed analyzing all possible parameters, local and systemic, related to tooth loss. Such a multifactorial forecast model may serve as an important tool for the overall improvement of dental care in subjects suffering from periodontitis.

Tooth-based significant predictor parameters for tooth loss assessed at baseline

Study	Design	Number of Patients	Periodontal Treatment	Duration-years	Bone loss	CAL	PPD	Tooth Decay	Tooth Mobility
Jansson et al. 2002	Cohort	513	No	20	Yes				
König et al. 2002	Cohort-retrospective	142	Yes	10					Yes
Gilbert GH et al. 2002	Cohort	687	No	4		Yes		Yes	Yes
Baelum et al. 1997	Cohort	587	No	10				Yes	
Mcguire & Nunn 1996	Cohort-retrospective	101	Yes	5	Yes		Yes		Yes

Table 2. Studies showing various significant tooth-based parameters related to tooth loss and assessed at baseline by regression analysis (CAL: clinical attachment loss, PPD: pocket probing depth).

Patient-based significant predictor parameters for tooth loss assessed at baseline

Study	Design	Number of Patients	Periodontal Treatment	Duration-years	Smoking	Diabetes	Bone loss	CAL	PPD	Tooth Decay	BOP	PI
Fardal et al. 2004	Cohort	100	Yes	10	Yes							
Jansson et al. 2002	Cohort	513	No	20								Yes
Shimazaki et al. 2003	Cohort	418	No	6								Yes
König et al. 2002	Cohort-retrospective	142	Yes	10	Yes							
Matthews et al. 2001	Cohort-retrospective	335	Yes	10				Yes				
Machtei et al. 1999	Cohort	415	No	5			Yes	Yes				
Baelum et al. 1997	Cohort	587	No	10				Yes		Yes		
Slade et al. 1997	Cohort	693	No	2	Yes				Yes	Yes		
Mcguire & Nunn 1996	Cohort-retrospective	100	Yes	5	Yes							Yes
Locker J et al. 1996	Cohort	491	No	3				Yes		Yes		
Holm, G 1994	Cohort	273	No	10	Yes							Yes
Burt BA et al. 1990	Cohort	195	No	28				Yes				

Table 3. Studies showing various significant patient-based parameters related to tooth loss and assessed at baseline by regression analysis (CAL: clinical attachment loss, PPD: pocket probing depth, BOP: bleeding on probing, PI: plaque index, / means no assessment in the study or no significant parameter).

1 . 5 Problem Definition

The aim of this cohort retrospective study was to assess the relevant risk factors for tooth loss, measured at baseline, in a population of patients having periodontal treatment. A second objective was to develop a forecast model for tooth loss, based on the analysed significant risk factors.

2 . Materials and Methods

2 . 1 Patient Sample and Data Assessment

This retrospective cohort study was based on the records of 198 patients who were treated and maintained in a period of 8 through 15 years at the Department of Periodontology at the University of Münster Clinic in Germany. This is a specialty clinic for periodontal diseases and receives referrals from general dental practioners in the community and other counties, other departments of the dental school and physicians.

From a total of 1257 subjects treated at the Department of Periodontology, University of Münster, Germany in 1989 and/or 1990 the charts of 780 patients were retrievable and assessed. Only patients with charts presenting the information determined by the inclusion criteria were entered into the study. Details of the inclusion/exclusion parameters are described below on table 4.

Inclusion	**Exclusion**
Initial periodontal therapy completed in 1989 and/or 1990 Diagnosis of: - Aggressive Periodontitis - Chronic Periodontitis and/or - NUP	Periodontal therapy not completed Incomplete or no radiographs at baseline Incomplete or no clinical data at baseline

Table 4. Criteria used for the inclusion of subjects in the study. NUP means necrotizing ulcerative periodontitis

386 out of 780 subjects were excluded from the study due to the reasons described in table 4 and the remaining 394 subjects were included into the study. 181 of these subjects continued to receiving periodontal supportive therapy at the Department of Periodontology over a period

of 8 to 15 years, while 213 patients who had been receiving SPT at the Department for a minimum of five years following initial therapy were no longer treated there. Those 213 patients were contacted by mail and asked to give permission for obtaining their dental information from their current dentists. 60 patients responded and gave the permission to contact their private dentists. After that, their dental practioners were contacted by mail in order to have the periodontal charts assessed. Out of those, 17 dental professionals provided the requested information of 17 patients to be analysed. Therefore, a final number of 198 (181 + 17) patients were statistically analysed in this study (see fig. 2).

The study protocol was approved by the Research Ethic Committee of the Medical Chamber and The University of Münster.

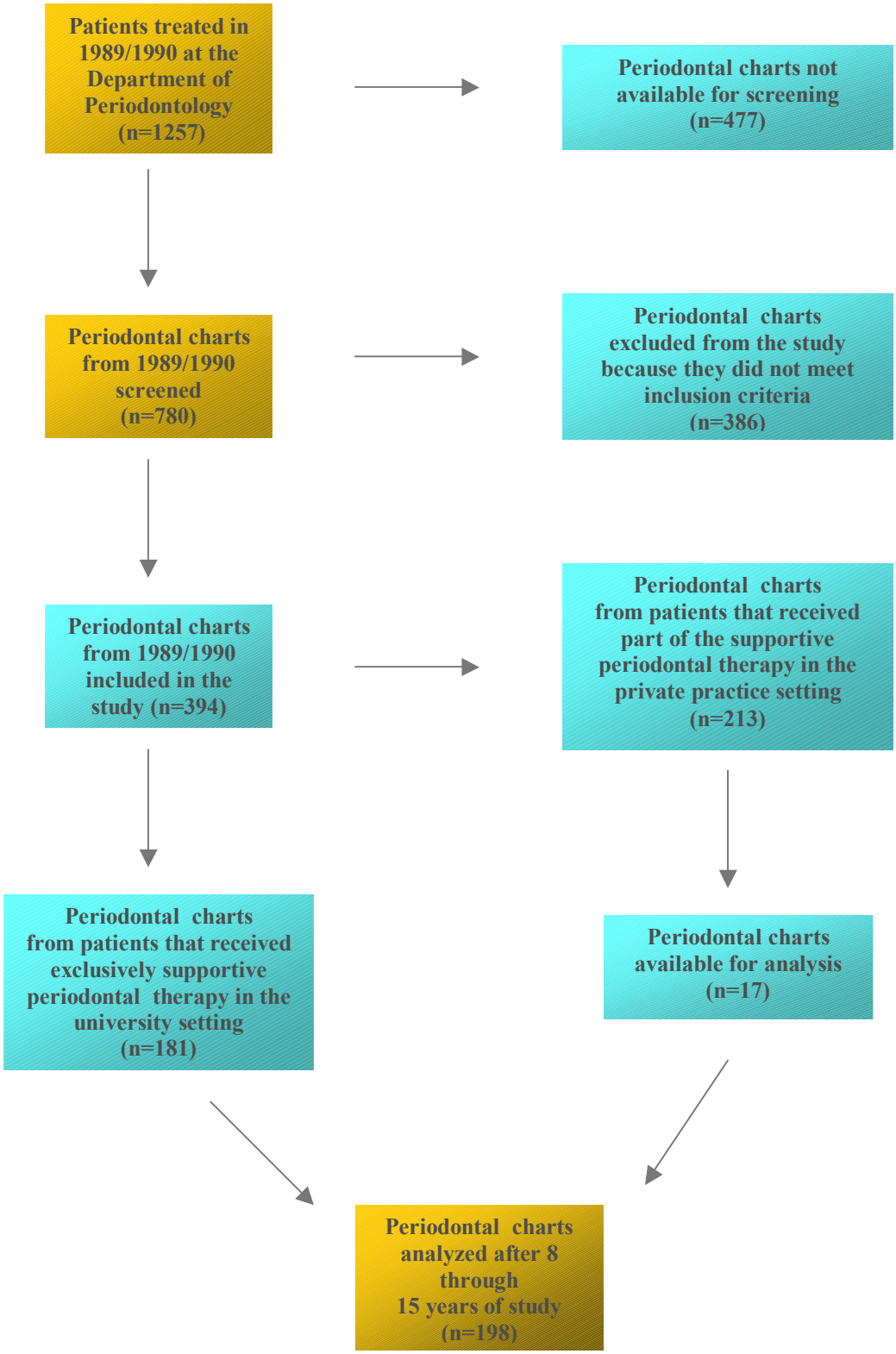

Figure 2 demonstrates the process for the inclusion and assessment of patients in the study.

The parameters and indices assessed at baseline were part of the normal procedures for the initial periodontal examination applied in the Department of Periodontology in the years 1989 and 1990 (see table 5). All available data were entered into excel spreadsheets and posteriorly analyzed for statistical purposes.

Assessed parameters	All patients (n=198)	
	Baseline	End of study
Patient history	X	
History of tooth loss	X	X
Pocket probing depth	X	X
Plaque index	X	
Bleeding on probing	X	
Tooth mobility	X	X
Tooth sensitivity	X	X
Radiographic assessment	X	

Table 5. Assessed parameters at baseline and after 8 through 15 years of study.

2 . 1 . 1 Patient History

Data assessment was restricted to recording findings such as cardiovascular diseases, diabetes, allergy and head and neck radiation. Since at baseline no information was assessed in the patient history on tobacco consumption, there could be no data generated on the smoking status of the assessed patient population.

2 . 1 . 2 History of Tooth Loss

Tooth loss was assessed during the initial and supportive periodontal therapy (see topic 2.3). Parameters such as patient history, alveolar bone loss, pocket probing depth, tooth mobility, frequency of supportive periodontal therapy, plaque index and bleeding on probing, related to tooth loss, were recorded.

2 . 1 . 3 Pocket Probing Depth

A manual periodontal probe was used to assess pocket probing depth at baseline, after the initial therapy and after 8 through 15 years. The distance from the gingival margin to the bottom of the pocket was recorded. Just the most affected side of the tooth (mesial or distal) was used in the descriptive and regression analysis.

2 . 1 . 4 Plaque Index and Bleeding on Probing

The oral hygiene and the gingival inflammation levels were assessed at baseline and after initial therapy by the approximal plaque index (API) (Lange et al. 1977) and the sulcus-bleeding index (SBI) (Mühlemann und Son 1971), respectively. Plaque index and bleeding on probing were also assessed in the end of the study using the plaque control record (O´Leary et al. 1972).

2 . 1 . 5 Tooth Mobility

The mobility was assessed at baseline, after initial therapy and in the end of the study and it was classified by degrees I to III (Lindhe and Nyman 1977).

2 . 1 . 6 Radiographic Assessment (alveolar bone level)

All 198 patients received full mouth radiographic evaluation using the Paralleling Technique (Langland & Sippy 1966) at baseline. Regarding the methodology for the alveolar bone level examination, the values at baseline were assessed in the most affected side of the tooth (mesial or distal). The distances between cement-enamel-junction and the most apical level of the alveolar crest were measured. When a vertical bone defect was present in the side to be measured, the distance between cement-enamel-junction and the most coronal level of the bone defect was also recorded. Finally, the distance between cement-enamel-junction and root apex was obtained. A remark on the alveolar bone level measurement is worth to be done. It is well known that physiologically there is a distance between alveolar bone level and cement-enamel-junction and therefore, for this reason, in the measurement we do not have 100% bone level.

All measurement were performed with a millimetered slide rule by one researcher (CF). The percentage of bone level in the most affected side was obtained using a proportion formula with the distances described above.

2 . 1 . 7 Caries and Restorations

Besides the periodontal diagnostics, information on tooth decay, fillings, crowns, and inlays was recorded for further statistical analysis. Within the subsequently performed regression analysis model (see Chapter 2.4) a tooth showing decay or any kind of restoration material was assessed as “restored”. Teeth without any signs of restorative treatment irrespective of the periodontal status were considered as “nonrestored”, respectively. Radiographically, there was difficulty to differ crowns from “overlays” and MOD restorations from “inlays”. Therefore, the term “restored” was used to designate all types of restorative procedures.

2 . 1 . 8 Missing Values

All information that could not be recorded for statistical analysis was considered as a missing value and therefore not included in the study. Data such as systemic diseases, plaque index, bleeding on probing, tooth condition, tooth sensitivity and tooth mobility that could not be retrieved from the periodontal charts were excluded from the statistical analysis. Furthermore, data regarding alveolar bone level, alveolar bone loss progression and pocket probing depth that were not readable or recorded on the chart were also considered missing values and consequently excluded from the study.

2 . 2 Performed Therapy

The periodontal treatment procedures were performed by overall 23 dental surgeons who participated in the postgraduation course at the Department of Periodontology in the years of 1989 and 1990. 30 patients have received initial periodontal therapy from the head of the department. The subjects received a hygiene phase prior to the initial therapy. This phase consisted of oral hygiene instructions, supragingival scaling and polishing procedures. After the improvement of the compliance, patients received open flap procedures and subgingival debridement. The performed periodontal therapy was intended to allow a clear visualization of the subgingival area. There was no intention to actively eliminate periodontal pockets or to perform any means of bone recontouring. After the initial therapy, patients were maintained under supportive periodontal therapy with sessions consisted of the reinforcement of oral hygiene instructions, tooth cleaning and supra-gingival scaling. The recall intervals and the necessity of further active therapy in form of subgingival debridement and/or periodontal surgery were defined, as the operator deemed necessary.

2 . 3 Tooth loss assessment

Type of tooth extracted and date of extraction were normally recorded in three periods (before the initial therapy, immediately after initial therapy and after 8 through 15 years of study) by the dental surgeons who participated in the postgraduation course at the Department of Periodontology. One researcher (CF) retrieved these information comparing directly the radiographic films from the initial therapy and after 8 through 15 years (when available) and the written data from the three periods already described. On the chart, a tooth was considered lost or extracted when it was marked missing by one of the postgraduate dental surgeons. Reasons for tooth extraction were not usually recorded.

2 . 4 Data Analysis

Clinical and radiographic data were obtained during the patients' first appointment at the department. Having completed the initial therapy, patients were reviewed in order to analyse the preliminary results of the treatment. In this appointment, clinical measurements were repeated and the patients were placed on an individualized periodontal maintenance programme. After 8 through 15 years (between years of 1998 and 2004), clinical data from 198 patients were reassessed (see fig. 3).

Fig 3. Time line of the study. Data assessment (198 patients) was performed at baseline, after the initial therapy and in the end of the study. SPT means supportive periodontal therapy

2 . 5 Statistical Analysis

The assessed data were analysed using descriptive statistics. Furthermore, regression analysis was performed using the SAS software package (Release 8.02) and S-PLUS 6.1. In order to analyse the influence of different parameters measured at baseline on further tooth loss a Generalized Linear Model was established and fitted via Generalized Estimating Equations. Statistical significant independent variables were identified by means of a backward selection procedure. A Wald Test was used to analyse the statistical significance of each parameter within the model. A second regression analysis was performed only with the selected significant variables set at $P<0.05$ aiming the construction of a more robust model.

The predicted values were estimated by odds ratios (OR) which are given together with 95% confidence intervals (CI). Graphical methods were applied and a Person´s chi square Test was performed to ensure that the final model adequately fitted the data.

All statistical analysis were performed at the Department for Statistics and Biomathematics at the University of Münster.

3 . Descriptive Results

3 . 1 Baseline

The epidemiologic results assessed at baseline were presented in two groups. The first one included 4559 teeth that had radiographic and clinical examination at baseline and in the end of the study and formed the whole sample. The second one presented only teeth that remained after initial therapy. The assessment of various patient-, tooth- and site-based parameters and conditions was performed. The results are described below.

3 . 1 . 1 Whole Sample (198 patients and 4559 teeth)

3 . 1 . 1 . 1 Systemic Conditions

In the whole sample (n=198 patients), 27 patients declared to have cardiovascular diseases at baseline. Six patients declared to be suffering from diabetes (fig. 4). The data of six patients with cardiovascular diseases and three patients with diabetes were considered as missing values and therefore not included in the study.

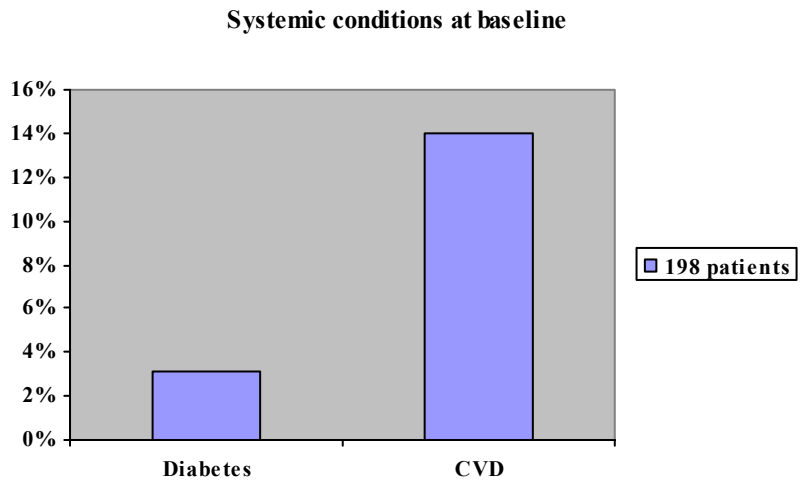

Fig 4. Prevalence of systemic diseases in the whole sample (n=198) (in percentage). CVD means cardiovascular diseases. Diabetes is related to types II and I.

3 . 1 . 1 . 2 SPT – Frequency Distribution

Patients were kept under supportive periodontal therapy for different periods.

Figure 5 demonstrates the treated patients who were followed for 8 to 15 years.

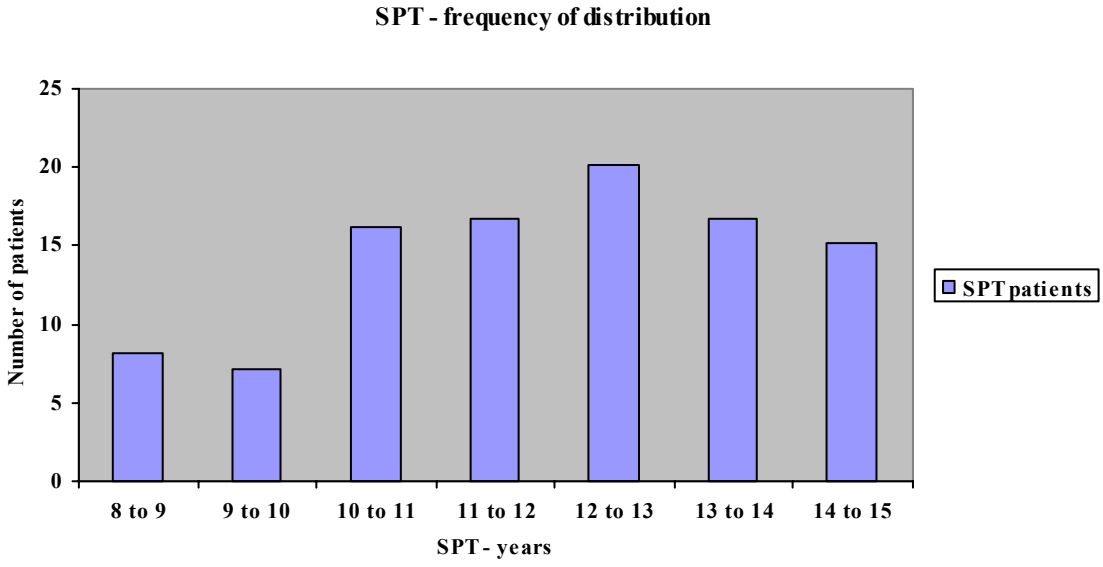

Figure 5. Frequency distribution of the patients who participated in the study (n=198) divided into various groups of supportive periodontal therapy (SPT) follow up (mean 11.80 years, Standard Deviation 2.27).

Overall, a number of 4559 teeth were assessed at baseline. Out of those, 2210 teeth were in upper jaw (48.5 % of the sample) and 2349 in lower jaw (51.5% of the sample). Single-rooted were more present than multi-rooted teeth (3173 and 1386), 69.6% and 30.4%, respectively (see fig. 6a and 6b).

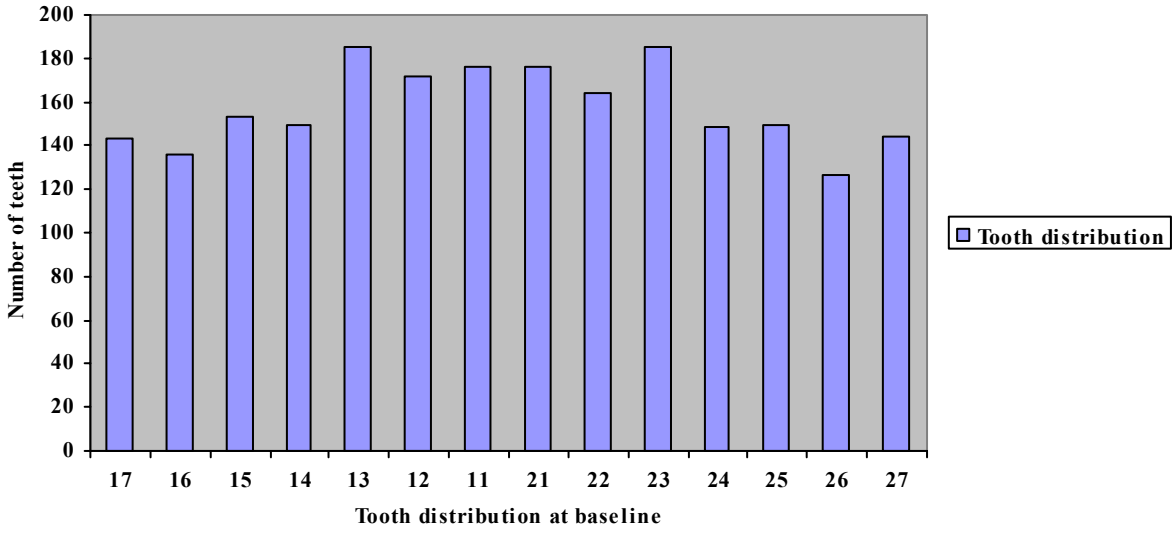

Fig. 6a. Distribution of overall assessed teeth at baseline in the upper jaw

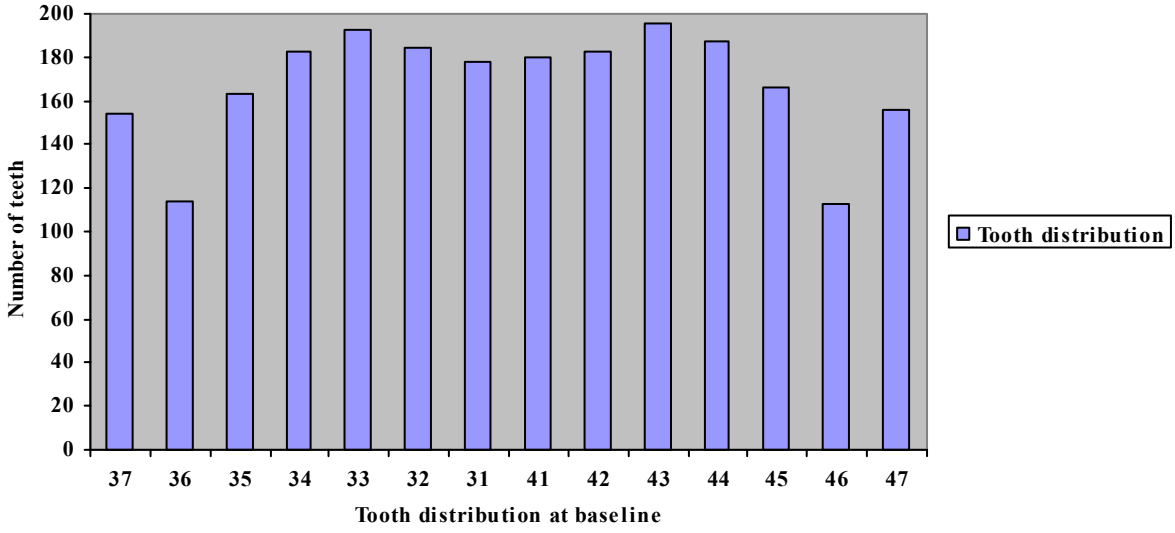

Fig. 6b. Distribution of overall assessed teeth h at baseline in the lower jaw

3 . 1 . 1 . 3 Bone Level

Regarding the initial overall periodontal bone level, 198 patients were assessed in the whole sample. The most radiographically affected side of the tooth (mesial or distal) was used in order to have the alveolar bone level descriptively measured. The same reference was valid for the percentage of bone level and the regression analysis assessment.

Overall, in the whole sample, 3291 teeth could be recorded for statistical analysis. Teeth were divided into different strata of bone level. The percentage values of bone level at baseline was related to the distance between cement-enamel-junction (CEJ) and the most apical level of the interproximal alveolar bone crest level (ABL).

It should be noted, that the term "bone loss" was explicitly not used due to the difficulty to determine a threshold for bone loss. In other words, due to the variable physiologic distance between CEJ and alveolar crest, it was not possible to determine with accuracy the limit between physiologic bone level and pathologic bone loss. Figure 7 shows the number of assessed teeth divided into different levels of alveolar bone.

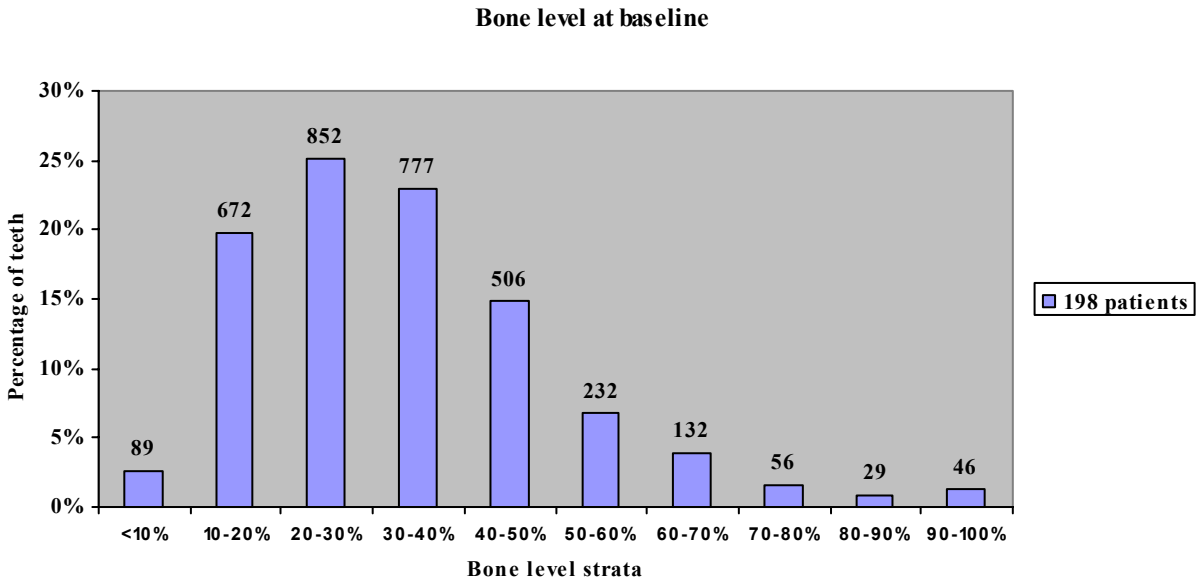

Fig 7. Overall number of teeth in patients (n=198) divided into various strata of bone at baseline (in percentage). Numbers above the columns indicate absolute numbers of teeth in each strata of bone at baseline. In this sample, the data of 1168 teeth (25.6%) could not be retrieved and therefore were considered as missing values.

3 . 1 . 1 . 4 Pocket probing depth

Periodontal pocket measurement was also performed in whole sample (n=198). Overall, 4507 teeth were available for statistical analysis. Fig 8 shows the values of the sample divided into various strata of pocket probing depth.

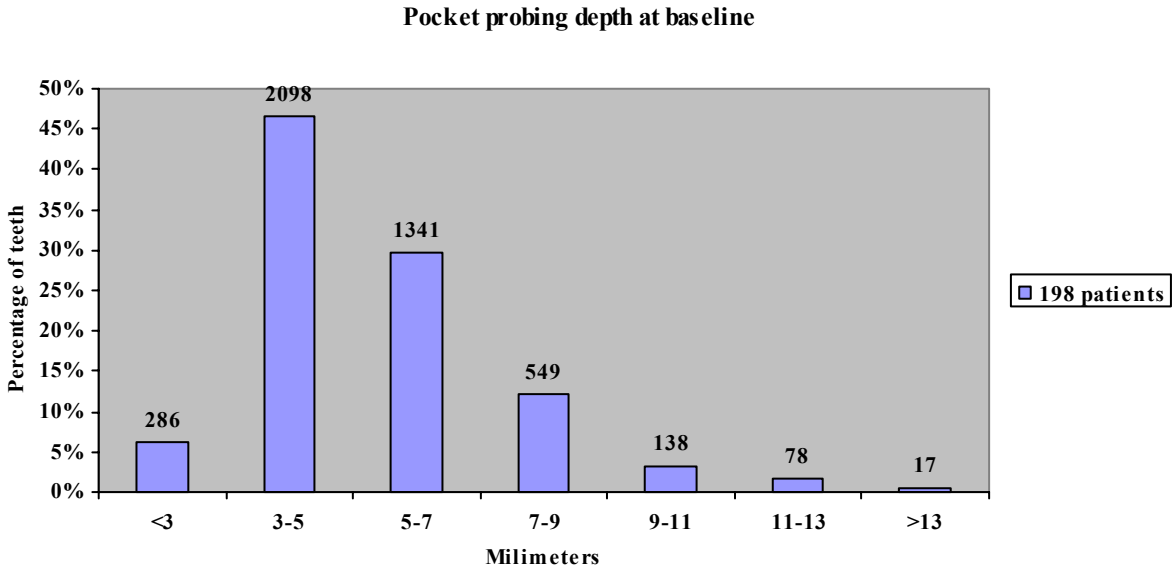

Fig 8. Pocket probing depth (PPD) measurements of the whole sample (n=198) assessed at baseline. The values above the columns mean the overall number of teeth distributed into various strata of pocket depth at baseline. In this sample, the data of 52 teeth (1.1%) could not be retrieved and therefore were considered as missing values.

3 . 1 . 1 . 5 Tooth Mobility

Tooth mobility was also assessed in the whole sample (n=198). 3245 teeth were available for assessment in this group (see fig. 9).

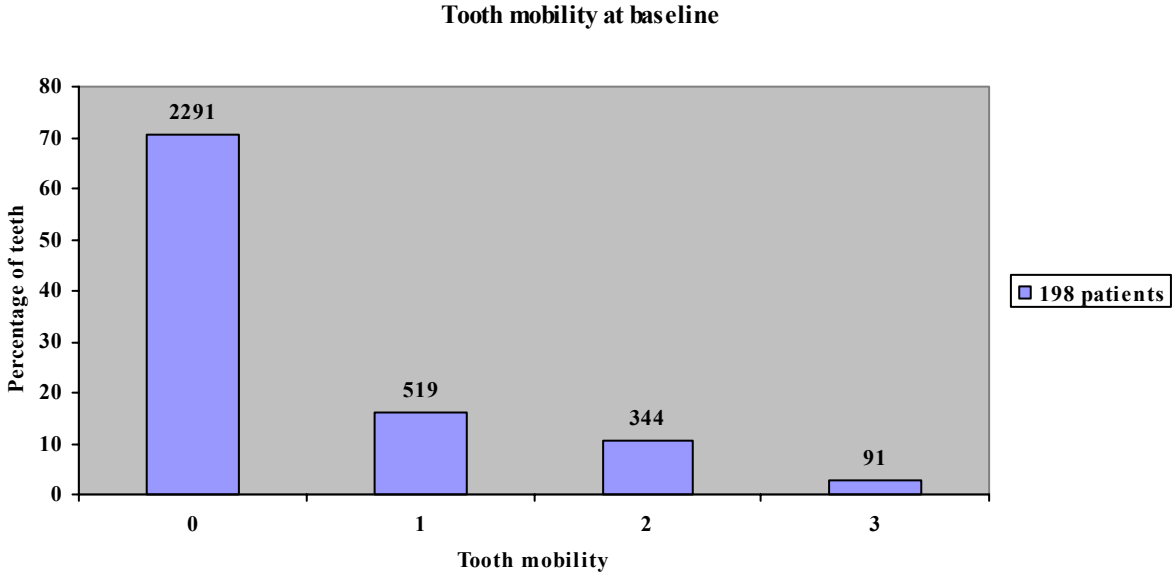

Fig. 9. Tooth mobility distribution in the whole sample (n=198) assessed at baseline. Values above the columns indicate the absolute numbers of teeth into each strata of tooth mobility at baseline. In this sample, the data of 1314 teeth (28.8%) could not be retrieved and therefore were considered as missing values.

3 . 1 . 2 Sample with Teeth that Remained After Initial Therapy

3 . 1 . 2 . 1 Systemic Conditions

In this sample, 24 patients declared to have cardiovascular diseases at baseline. Five patients declared to be suffering from diabetes (fig. 10). The data of five patients with cardiovascular diseases and two patients with diabetes were considered as missing values and therefore not included in the study.

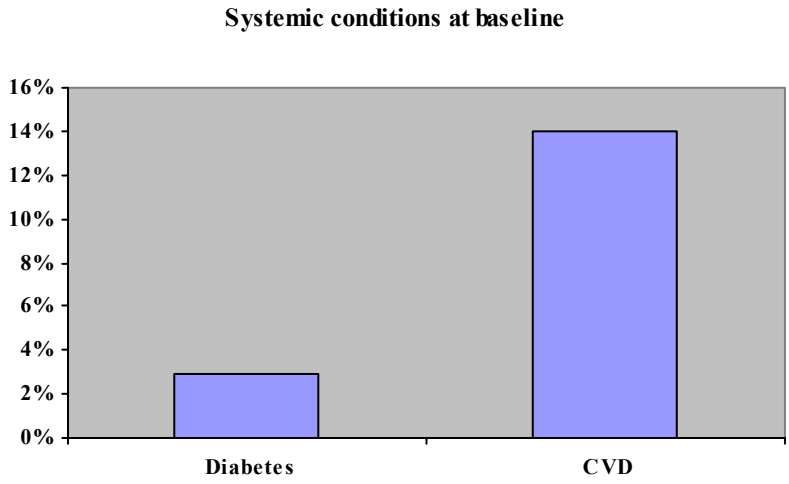

Fig 10. Prevalence of systemic diseases in the sample with teeth that remained after initial therapy (in percentage). CVD means cardiovascular diseases. Diabetes is related to types II and I.

3 . 1 . 2 . 2 SPT – Frequency Distribution

Figure 5 demonstrates the frequency distribution of the patients who had teeth extracted only after initial therapy

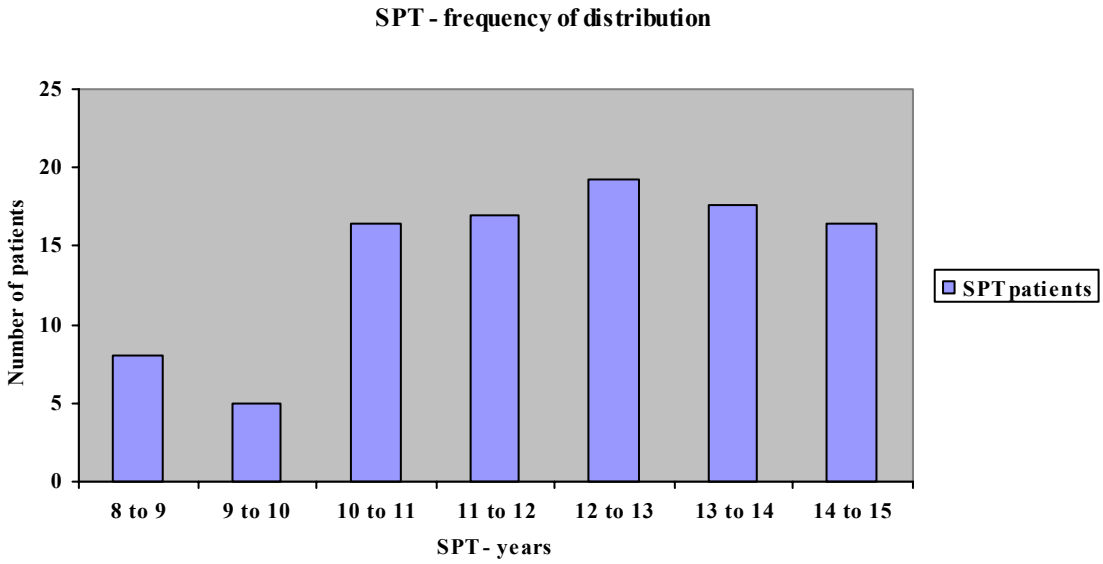

Figure 11. Frequency distribution of the patients who had teeth extracted only after initial therapy (n=176) divided into various groups of supportive periodontal therapy follow up (SPT) (mean 11.90 years, Standard Deviation 2.24).

The information on 3745 teeth that remained after initial therapy distributed in 176 patients could be retrieved. Out of those, 1806 teeth were in upper jaw (48.2 % of the sample) and 1939 in lower jaw (51.8% of the sample). Single-rooted were more present than multi-rooted teeth (2642 and 1103), 70.5% and 29.5%, respectively (see fig. 12a and 12b).

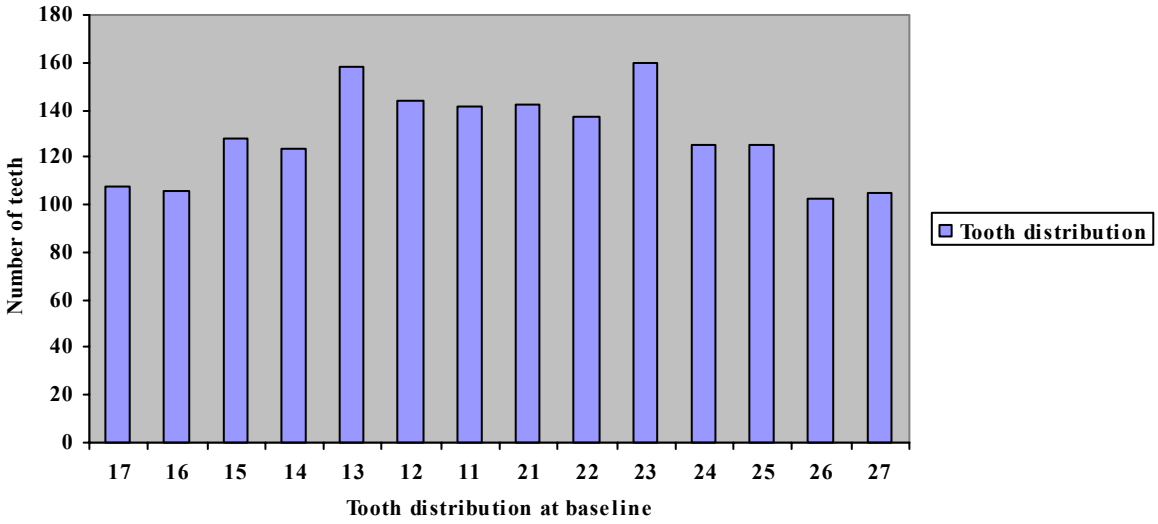

Fig. 12a. Distribution of the teeth that remained after the initial therapy (upper jaw).

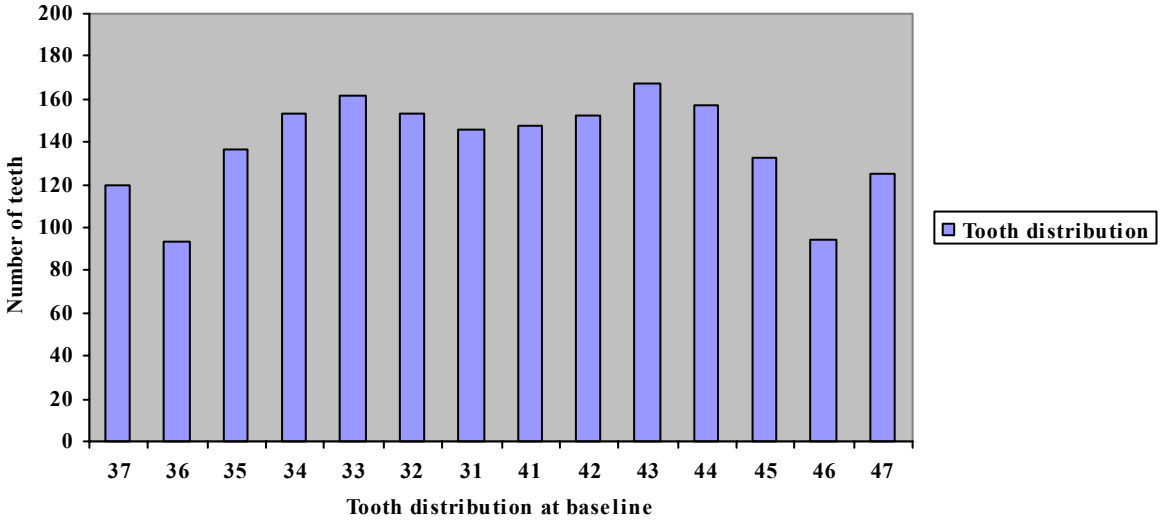

Fig. 12b. Distribution of the teeth that remained after the initial therapy (lower jaw).

3 . 1 . 2 . 3 Bone Level

In this sample, 3291 teeth could be recorded for statistical analysis. Teeth were divided into different strata of bone level. The percentage values of bone level at baseline was related to the distance between cement-enamel-junction (CEJ) and the most apical level of the interproximal alveolar bone crest level (ABL).

It should be noted, that the term “bone loss” was explicitly not used due to the difficulty to determine a threshold for bone loss. In other words, due to the variable physiologic distance between CEJ and alveolar crest, it was not possible to determine with accuracy the limit between physiologic bone level and pathologic bone loss. Figure 13 shows the number of assessed teeth divided into different levels of alveolar bone.

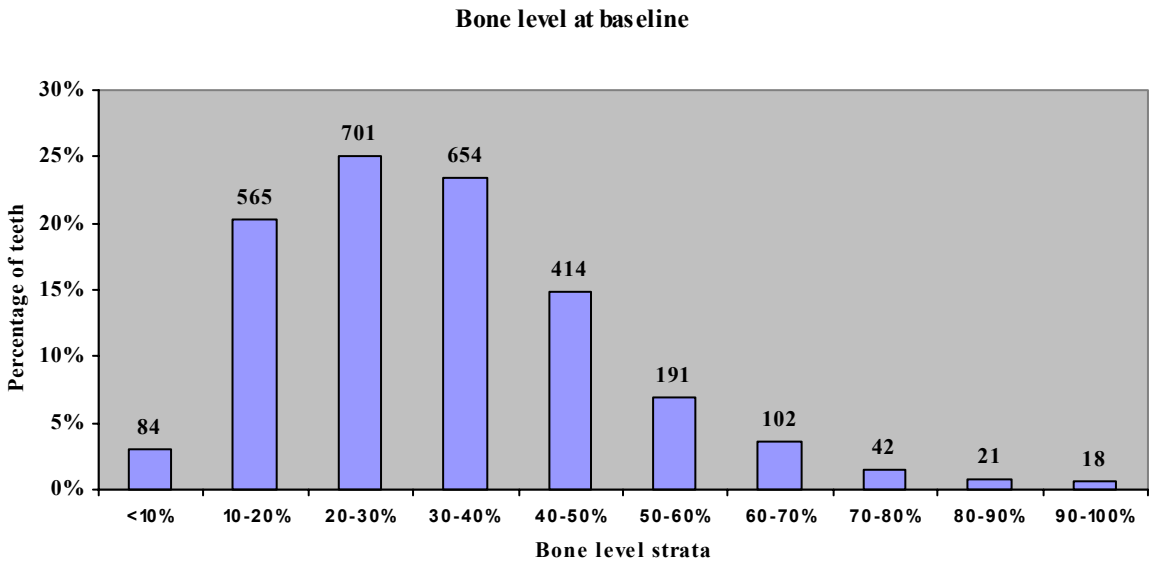

Fig 13. Overall number of teeth that remained after initial therapy divided into various strata of bone (in percentage). Numbers above the columns indicate absolute numbers of teeth in each strata of bone. In this sample, the data of 953 teeth (25.4%) could not be retrieved and therefore were considered as missing values.

3 . 1 . 2 . 4 Pocket Probing Depth

Periodontal pocket was also assessed in teeth that remained after initial therapy. Overall, 3717 teeth were available for statistical analysis. Fig 14 shows the values divided into various strata of pocket probing depth.

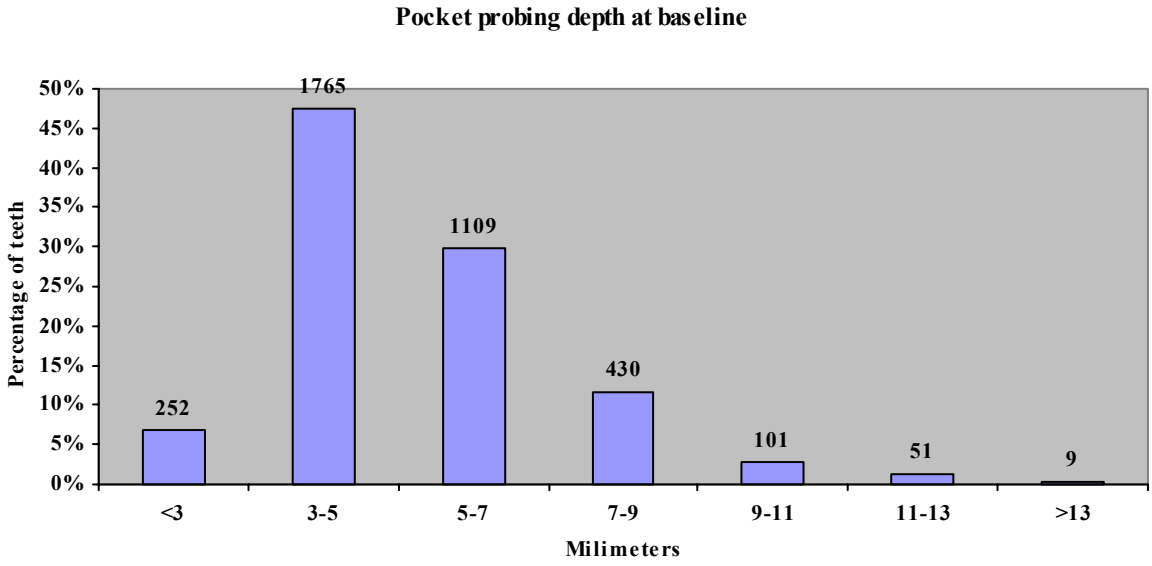

Fig 14. Pocket probing depth (PPD) measurements of the teeth that remained after initial therapy. The values above the columns mean the overall number of teeth distributed into various strata of pocket depth. In this sample, the data of 28 teeth (0.7%) could not be retrieved and therefore were considered as missing values.

3 . 1 . 2 . 5 Tooth Mobility

Tooth mobility was also assessed in teeth that remained after initial therapy. 2660 teeth were available for assessment in this sample (see fig. 15).

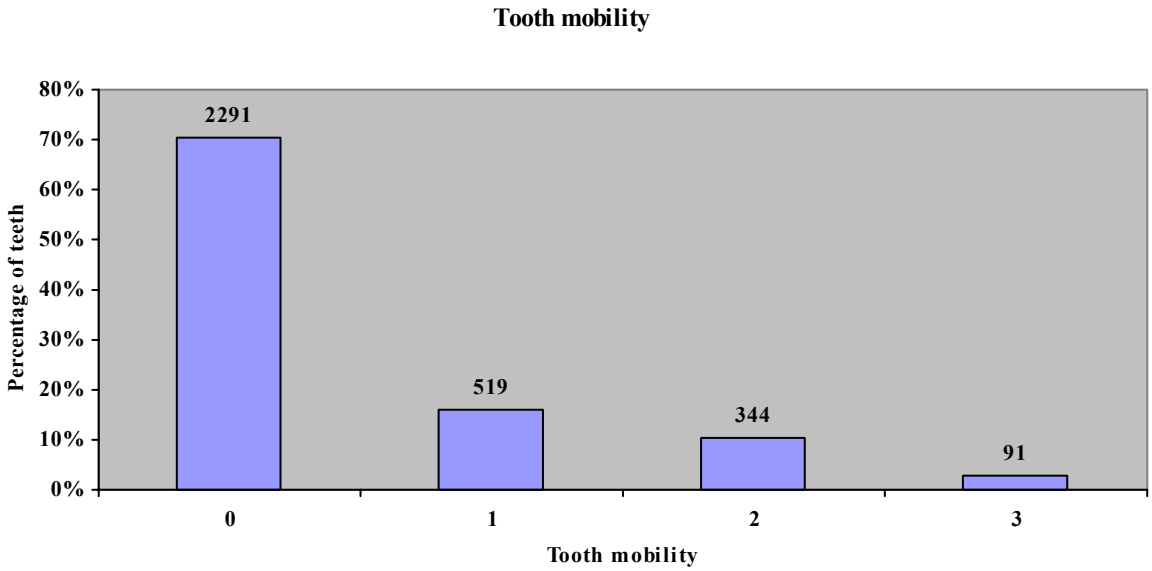

Fig 15. Mobility of the teeth that remained after initial therapy. The values above the columns mean the overall number of teeth distributed into different levels of mobility. In this sample, the data of 1085 teeth (29%) could not be retrieved and therefore were considered as missing values.

3 . 2 Outcome of Therapy

The results of the influence of various parameters assessed at baseline on tooth loss occurring during initial therapy and after 8 through 15 years of supportive periodontal therapy are depicted below in distinct sections. In addition, the relationship between tooth loss and patient history and the information on types of surgical procedures performed during the 8 through 15 years of study are also demonstrated.

3 . 2 . 1 Initial Therapy (Tooth Loss)

166 teeth (35.54% of all extracted teeth) were lost during initial therapy. Due to the difficulty to define the precise period of extraction, 52 extracted teeth were considered as missing values and therefore not included in the analysis.

Table 6 shows the tooth loss in the 198 patients group. A graphic presentation of the anterior and posterior lost teeth is depicted in Figures 16 and 17.

Number of Teeth Baseline	Tooth Loss Overall	Tooth Loss Initial Therapy	Tooth Loss SPT	Teeth with undefined Data of Extraction
4559	467	166	249	52
100%	10.24%	3.64%	5.46%	

Table 6. Percentage of tooth loss during the various periods of therapy. SPT means supportive periodontal Therapy.

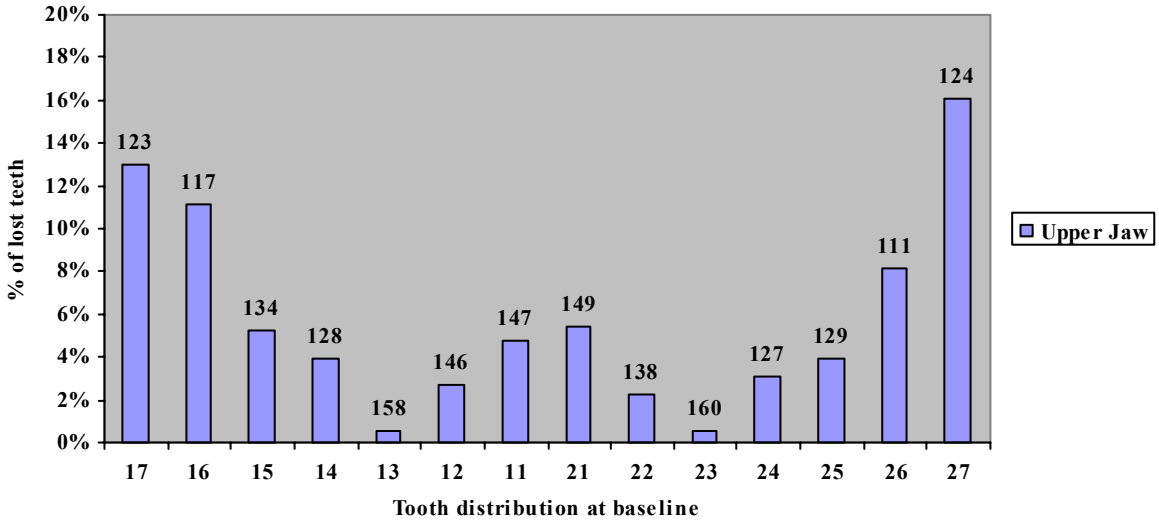

Fig. 16. Graphic representation of tooth loss (percentage) in the upper jaw during initial therapy. The values above the columns mean the overall number of teeth distributed at baseline

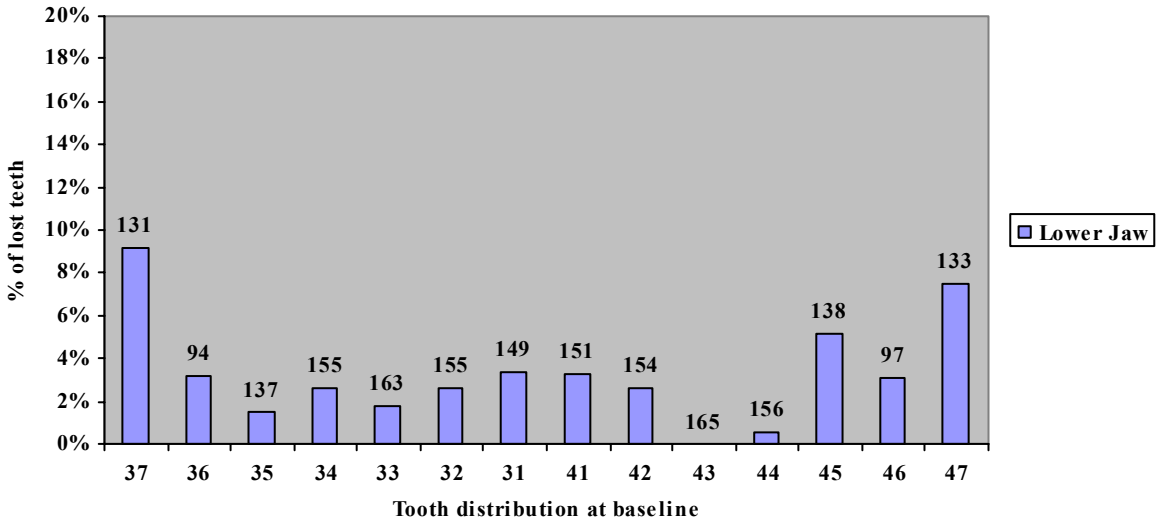

Fig 17. Graphic representation of tooth loss (percentage) in the lower jaw during initial therapy. The values above the columns mean the overall number of teeth distributed at baseline

3 . 2 . 1 . 1 Tooth loss and Bone Level

The influence of initial alveolar bone level on subsequently occurring tooth loss during initial therapy was assessed. To allow a precise data presentation, extracted teeth were grouped into

various strata of bone level at baseline and the percentage of lost teeth within each group was determined (figure 18).

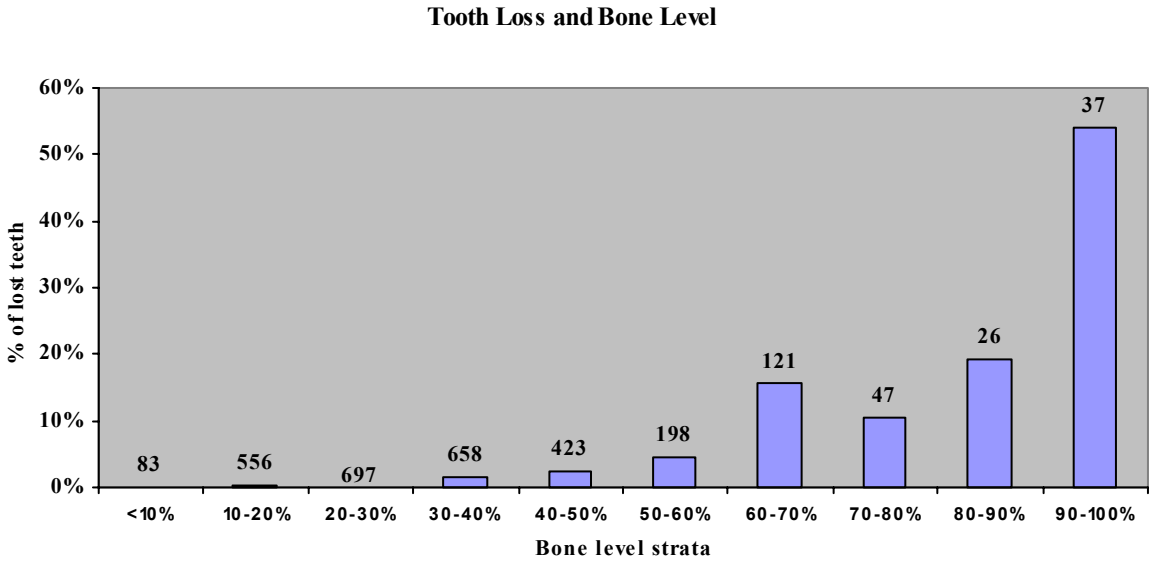

Fig 18. Relationship between alveolar bone level at baseline and tooth loss during initial therapy. Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above the columns) divided into various level of alveolar bone at baseline.

3 . 2 . 1 . 2 Tooth Loss and Pocket Probing Depth

To assess the influence of pocket probing depth on tooth loss, teeth were divided at baseline into various strata exhibiting different levels of periodontal pockets. The percentage of lost teeth after 8 through 15 years of study was assessed. The results from the sample of 198 patients are depicted on fig. 19.

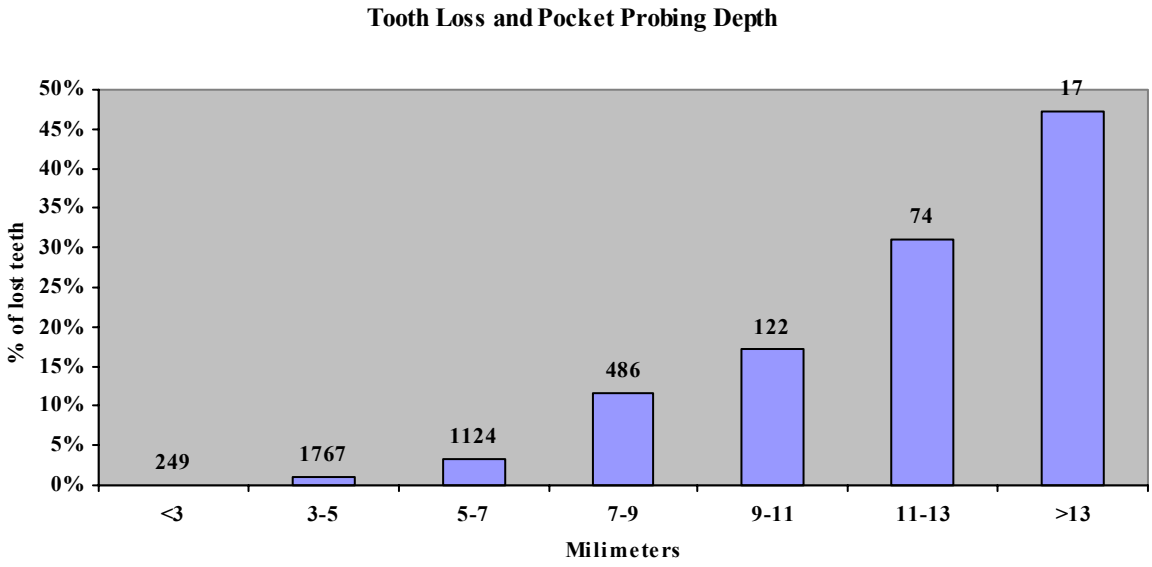

Fig 19. Relationship between pocket probing depth at baseline and tooth loss during initial therapy. Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above the columns) divided into various level of pocket probing depth at baseline.

3 . 2 . 1 . 3 Tooth Loss and Tooth Mobility

The relationship between tooth mobility at baseline and tooth loss during initial therapy was assessed. Figure 20 depicts the distribution of the number of teeth at baseline divided into different levels of mobility and their percentage of tooth loss within each level.

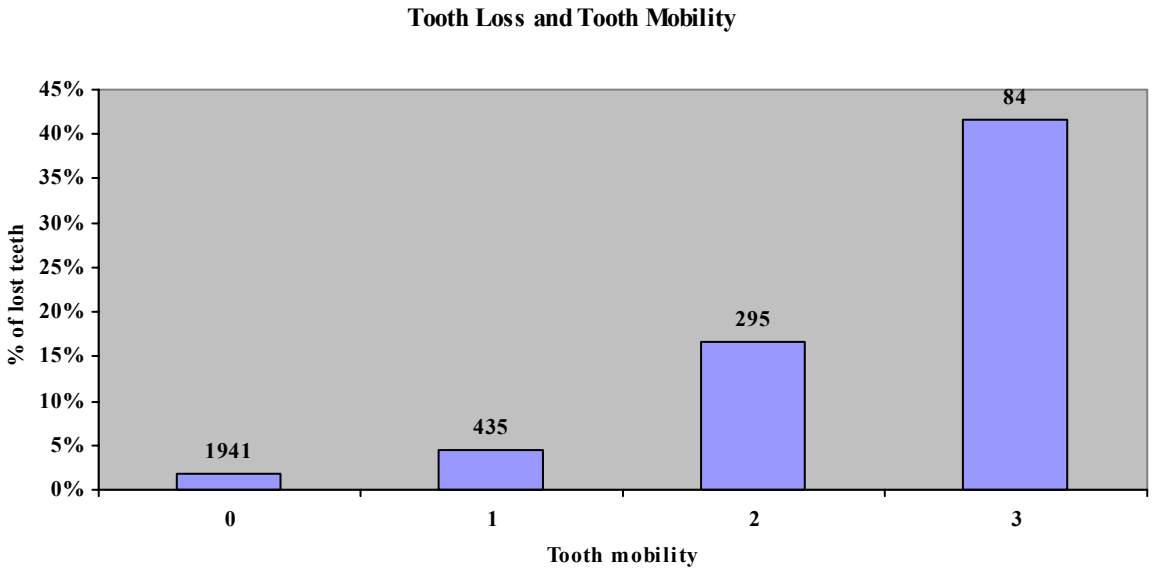

Fig. 20. Relationship between tooth mobility at baseline and tooth loss during initial therapy.
Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above the columns) divided into various level of tooth mobility at baseline.

3 . 2 . 2 Supportive Periodontal Therapy (Tooth Loss)

249 teeth (5.46% of all teeth) were lost during the subsequently 8 through 15 years of study. Due to the difficulty to define the precise period of extraction, 52 extracted teeth were considered as missing values and therefore not included in the analysis (see fig. 21a and 21b).

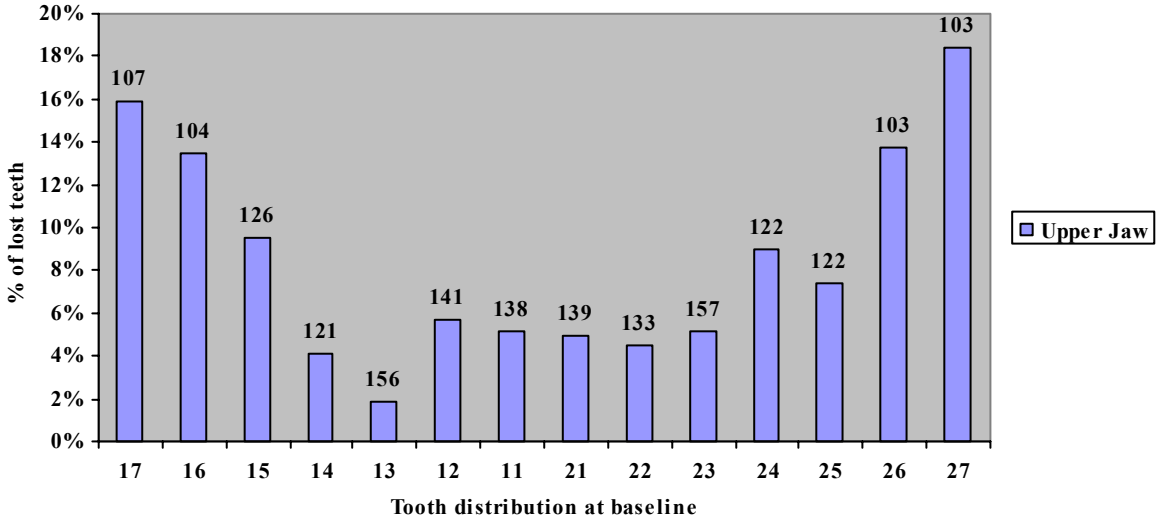

Fig. 21a. Graphic representation of tooth loss (percentage) in upper jaw during supportive periodontal therapy.

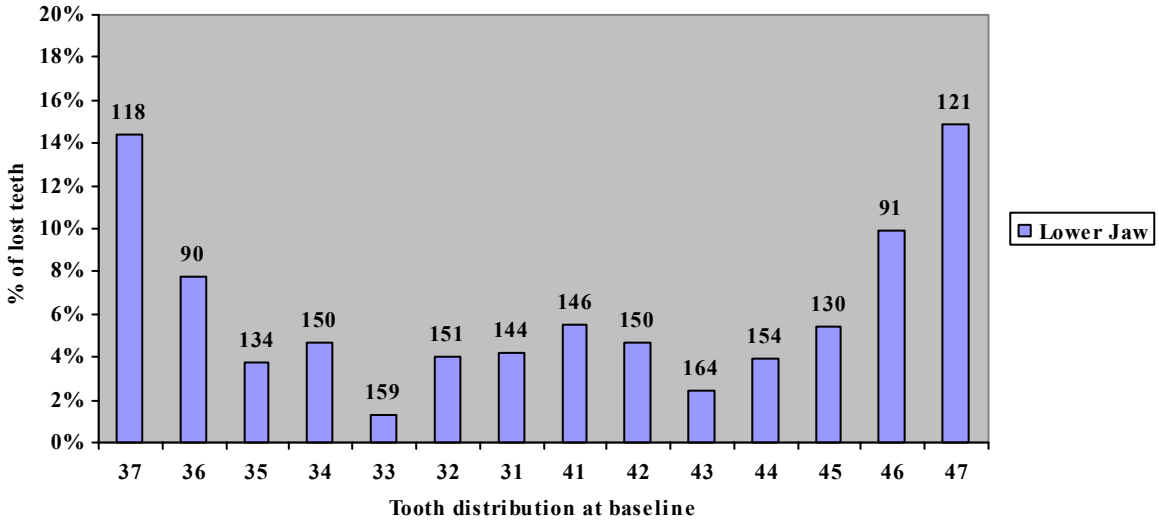

Fig. 21b. Graphic representation of tooth loss (percentage) in lower jaw during supportive periodontal therapy.

3 . 2 . 2 . 1 Tooth loss and Bone Level

The influence of initial alveolar bone level on subsequently occurring tooth loss during supportive periodontal therapy was assessed. To allow a precise data presentation, extracted teeth were grouped into various strata of bone level at baseline and the percentage of lost teeth within each group was determined (figure 22).

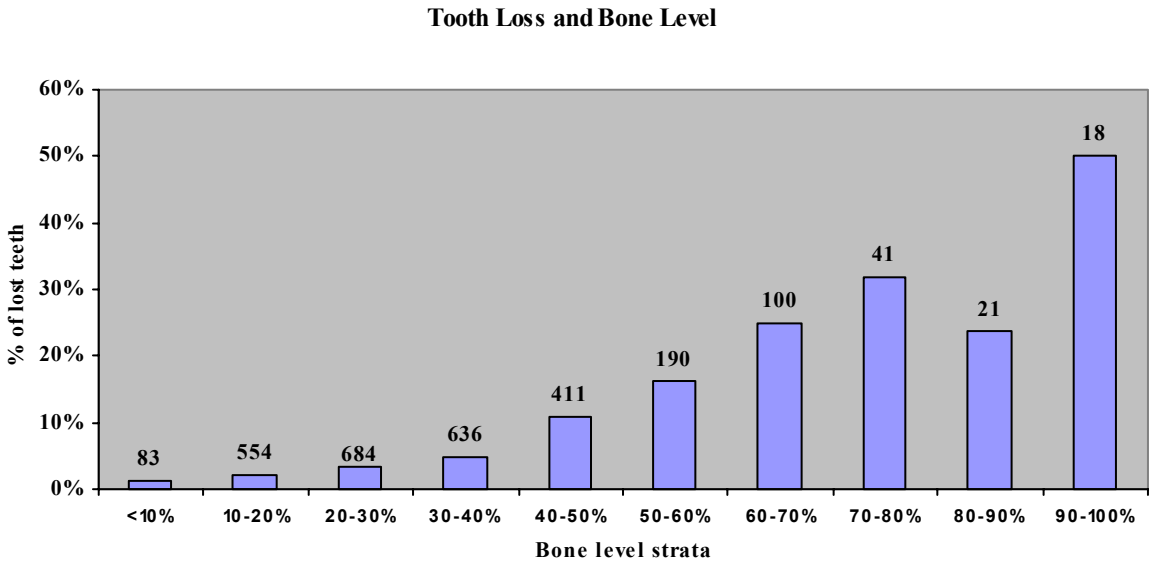

Fig 22. Relationship between alveolar bone level at baseline and tooth loss during supportive periodontal therapy. Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above he columns) divided into various levels of alveolar bone at baseline.

3 . 2 . 2 . 2 Tooth Loss and Pocket Probing Depth

To assess the influence of pocket probing depth on tooth loss during the supportive periodontal therapy period, teeth were divided at baseline into various strata exhibiting different levels of periodontal pockets. The percentage of lost teeth after 8 through 15 years of study are depicted on fig. 23.

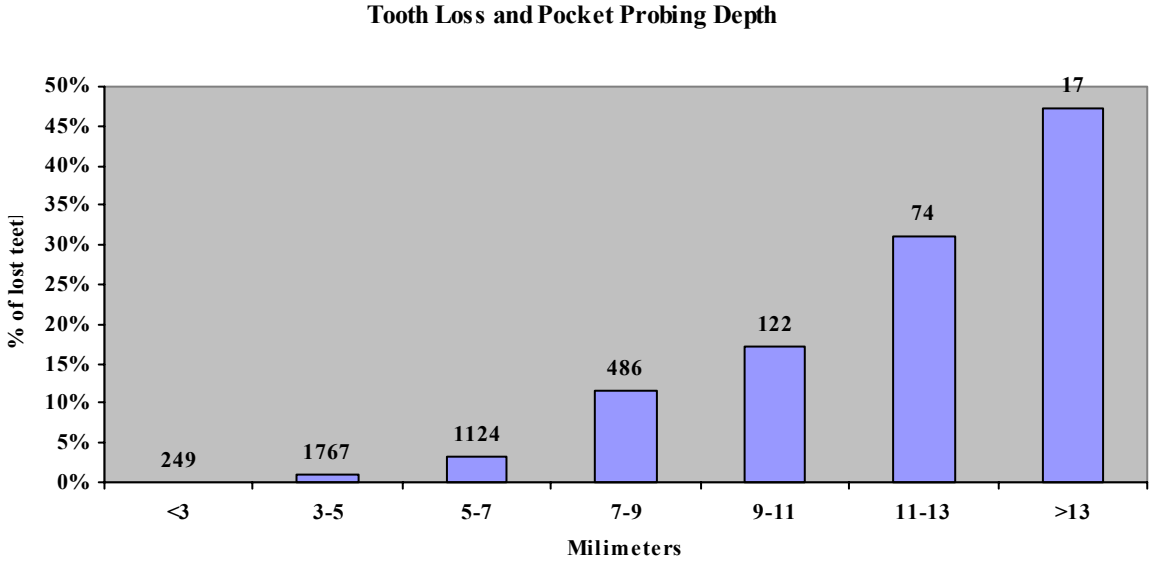

Fig 23. Relationship between pocket probing depth at baseline and tooth loss during supportive periodontal therapy. Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above the columns) divided into various levels of pocket probing depth at baseline.

3 . 2 . 2 . 3 Tooth Loss and Tooth Mobility

The relationship between tooth mobility at baseline and tooth loss during supportive periodontal therapy was assessed. Figure 24 depicts the distribution of the number of teeth at baseline divided into different levels of mobility and their percentage of tooth loss within each level.

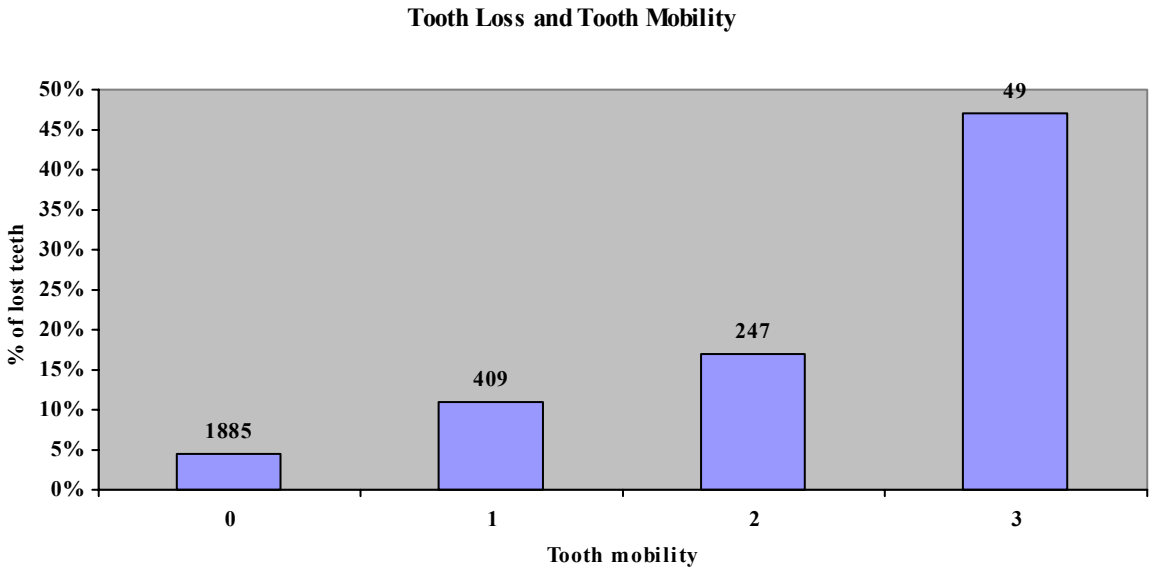

Fig. 24. Relationship between tooth mobility at baseline and tooth loss during supportive periodontall therapy. Y-axis represents the percentage of tooth loss from each group of patients (number of subjects above the columns) divided into various levels of tooth mobility at baseline.

3 . 2 . 2 . 4 Tooth Loss and Supportive Periodontal Therapy (SPT)

The relationship between SPT and tooth loss was assessed. Patients were divided into groups according to the Hirschfeld classification (Hirschfeld & Wasserman 1978):

Well-maintained group (WM), lost 0 to 3 teeth

Downhill group (DH), lost 4 to 9 teeth

Extreme Downhill group (EDH), lost 10 to 23 teeth

151 patients, were included into the well-maintained group, 24 into the downhill and 2 into the extreme downhill group (see table 7). 20 patients had their charts data not assessed due the difficulty to determine the exact period of tooth extraction.

groups	Hirschfeld/Wasserman study	This study	SPT during 8-15 years of study (average)
WM	83.2%	84.83%	16 times
DH	12.6%	13.48%	17 times
EDH	4.2%	1.12%	19 times

Table 7. Relationship between patients from this study and Hirschfeld/Wasserman´s study regarding the inclusion of teeth into well-maintained, downhill and extreme downhill groups.

3 . 2 . 3 Tooth Loss and Patient History

128 patients out of the whole sample had lost one or more teeth during the time of study. 20 patients out of them had a history of heart disease at baseline. Regarding to diabetes status, 3 patients from the group that has lost any teeth (n=128), confirmed this diagnosis at baseline. Due to the incomplete patient history, it was not possible to verify substantially the relation between tooth loss and history of the patient.

3 . 2 . 4 Performed Surgical Procedures

Overall, 935 periodontal surgeries were performed during 8 through 15 years of study in 136 patients. The average number of surgeries in these patients was 3.11 procedures per patient. The information on 27 patients could not be retrieved and therefore it was considered as missing values.

4 . Regression Analysis Results

Two regression analysis were performed separately. The first one was performed with teeth that were extracted during initial therapy. The second regression analysis was carried out with teeth extracted during the supportive periodontal therapy phase. The results are depicted below.

4. 1. Teeth Extracted During Initial Therapy

4. 1. 1. Tooth- and Site-Level Based Analysis

Tooth mobility, alveolar bone level, infectious diseases, caries/restoration status and pulpal status (negative tooth sensitivity) were factors significantly related to tooth loss (CI=95%, $P<0.05$). However, number of roots was not a significant parameter in this model of regression. A reduced percentage level of alveolar bone at baseline was a strong predictor for further tooth loss (OR 1.0558 for 1% bone level reduction, $P<0.0001$, CI=1.0395 – 1.0725; OR 1.7219 for 10% bone level reduction, $P<0.0001$, CI=1.4725 – 2.0136; OR 2.9650 for 20% bone level reduction, $P<0.0001$, CI=2.1683 – 4.0545). This means e.g. that an increase of 1% of bone level reduction will lead to a rise in the likelihood of tooth loss of almost 6%, while e.g. an increase of 10% of bone level reduction may rise the possibility of tooth loss to 72% compared to a tooth with normal distances between CEJ and ABL (90% of bone level: 100% bone level minus the physiologic distance).

Following the same pattern, increase of tooth mobility was a relevant predictor for further tooth loss when compared to absence of tooth mobility (mobility II vs 0: OR 3.2170, P=0.0292, CI=1.1254 – 9.1956; mobility III vs 0: OR 7.0700, P=0.0003, CI=2.4325 – 20.5490).

Tooth sensitivity was also a relevant parameter to predict further tooth loss. After the dichotomization of the values (positive and negative tooth sensitivity), teeth with endodontic treatment or having an evident apical lesion (assessed through the analysis of the radiographic examination) had more probability to be lost than vital teeth (OR 3.8210, P=0.0010, CI=1.7169 – 8.5038).

When the tooth condition was analysed, present restorations or decays were also considered risk factors for further tooth loss (OR 4.5497, P=0.0061, CI=1.5393 – 13.4473). Table 8 summarizes the significant risk factors for tooth loss and their respective OR measurements assessed at baseline.

4. 1. 2. Patient Level

Infectious disease (hepatitis) was a strong predictor for future tooth loss (OR 29.0826, P<0.0001, CI=15.0542 – 56.1835). Cardiovascular diseases, allergy, head and neck irradiation, time interval between the performance of periodontal maintenance therapy, bleeding on probing, pocket probing depth and plaque index were not associated in this model of regression with further tooth loss (see table 8). This model of regression explained partially the results (R^2=0.35).

Risk factors	Estimate (OR)	Confidence Interval	P value
Bone loss (increment 1%)	1.0558	1.035/1.0725	<0.0001
Bone loss (increment 10%)	1.7219	1.4725/2.0136	<0.0001
Bone loss (increment 20%)	2.9650	2.1683/4.0545	<0.0001
Tooth mobility II vs 0	3.2170	1.1254/9.1956	0.0292
Tooth mobility III vs 0	7.0700	2.4325/20.5490	0.0001
Infectious disease	29.0826	15.0542/56.1835	<0.0001
Caries and/or restored tooth	4.5497	1.5393/13.4473	0.0061
Tooth sensitivity negative	3.8210	1.7169/8.5038	0.010

Table 8. Summary of the estimate Odds Ratio with their respective confidence intervals and significant P values obtained from the data assessed at baseline

4. 2. Teeth Extracted During SPT Phase

4. 2. 1. Tooth- and Site-Level Based Analysis

Tooth mobility, alveolar bone level, number of roots, and pulpal status (negative tooth sensitivity) were factors significantly related to tooth loss (CI=95%, $P<0.05$). However, restoration and caries status were not significant parameters in this model of regression. A reduced percentage level of alveolar bone at baseline was a strong predictor for further tooth loss (OR 1.0452 for 1% bone level reduction, $P<0.0001$, CI=1.0279 – 1.0628; OR 1.5559 for 10% bone level reduction, $P<0.0001$, CI=1.3165 – 1.8387; OR 2.4207 for 20% bone level reduction, $P<0.0001$, CI=1.7333 – 3.3808). This means e.g. that an increase of 1% of bone level reduction will lead to a rise in the likelihood of tooth loss of 4%, while e.g. an increase of 10% of bone level reduction may rise the possibility of tooth loss to 63% compared to a tooth with normal distances between CEJ and ABL.

Following the same pattern, increase of tooth mobility was a significant predictor for further tooth loss when compared to absence of tooth mobility (mobility III vs. 0: OR 5.9329, P=0.0005, CI=2.1877 – 16.0897).

Regarding the anatomy of the teeth (single or multi-rooted), molars and first upper pre-molars were considerable more susceptible to further loss than second upper pre-molars, lower pre-molars, canines and incisors (OR 1.8043, P=0.0247, CI=0.0751 – 1.1053).

Tooth sensitivity was also related to further tooth loss. After the dichotomization of the values (positive and negative tooth sensitivity), teeth with endodontic treatment or having an evident apical lesion had more probability to be lost than vital teeth (OR 2.5415, P=0.0004, CI=1.5137 – 4.2672). Table 9 summarizes the significant risk factors for tooth loss and their respective OR measurements assessed at baseline.

4. 2. 2. Patient Level

Diabetes was a strong predictor for future tooth loss (OR 3.5896, P<0.01, CI=1.3030–9.8892). Cardiovascular diseases, allergy, head and neck irradiation, time interval between the performance of periodontal maintenance therapy, infectious diseases, probing pocket depth, bleeding on probing and plaque index were not associated in this model of regression with further tooth loss (see table 9). This model of regression also explained partially the results (R^2=0.15)

Risk factors	Estimate (OR)	Confidence Interval	P value
Bone loss (increment 1%)	1.0452	1.0279/1.0628	<0.0001
Bone loss (increment 10%)	1.5559	1..3165/1.8387	<0.0001
Bone loss (increment 20%)	2.4207	1.7333/3.3808	<0.0001
Tooth mobility III vs 0	5.9329	2.1877/16.0897	0.0005
Multirooted tooth	1.8043	0.0751/1.1053	0.0247
Diabetes	3.5896	1.3030/9.8892	0.0134
Tooth sensitivity negative	2.5415	1.5137/4.2672	0.0004

Table 9. Summary of the estimate Odds Ratio with their respective confidence intervals and significant P values obtained from the data assessed at baseline

5 . Predicting Tooth Loss (Practical Tool)

The objective of the first regression analysis (teeth extracted during initial therapy) was to obtain the decision criteria for extracting teeth. The results demonstrated how the various variables related to tooth loss had influence on the clinicians´decision making process. A table was built with the significant parameters (table 10) in order to demonstrate the tendency for tooth extraction when some variables were associated. The red colour indicates a higher probability in deciding for extraction and blue in deciding for tooth maintenance.

The second regression analysis was performed with teeth extracted during the supportive periodontal therapy phase aiming to identify significant parameters related to tooth loss. These variables formed the basis for the development of a risk assessment tool for tooth loss during the maintenance phase. The predictor model was developed using a formula generated from this logistic regression analysis. The logarithmic chance (Odds) of tooth loss was modelled in dependence of a linear combination of certain measured variables that form the basis for the computation of the Odds Ratios. A table was built using the predictor model described above in order to access the risk of tooth loss when various variables are analyzed

together (table 11). The objective was to create a practical tool for the dental practioner to determine tooth prognosis during supportive periodontal therapy. As in the first regression analysis, a scale of colour was used in order to quantify the risk of tooth loss with light blue showing highest and red lowest survival rate. The risk for tooth loss column shows the probability of a tooth to be lost in the next 8 through 15 years (table 11).

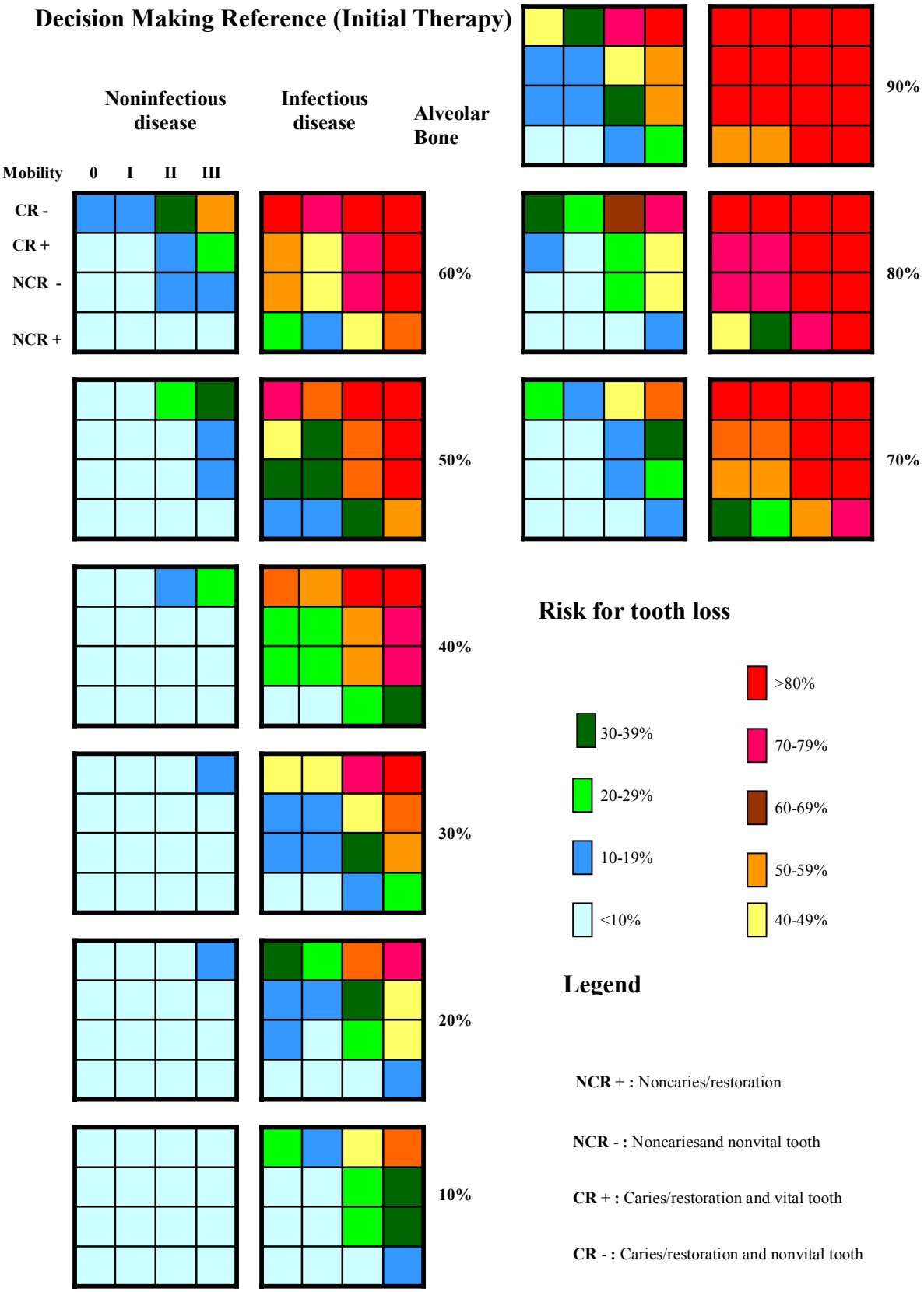

Table 10 indicates the decision criteria for tooth extraction during initial therapy. The red colour indicates a higher probability in deciding for extraction and blue in deciding for tooth maintenance.

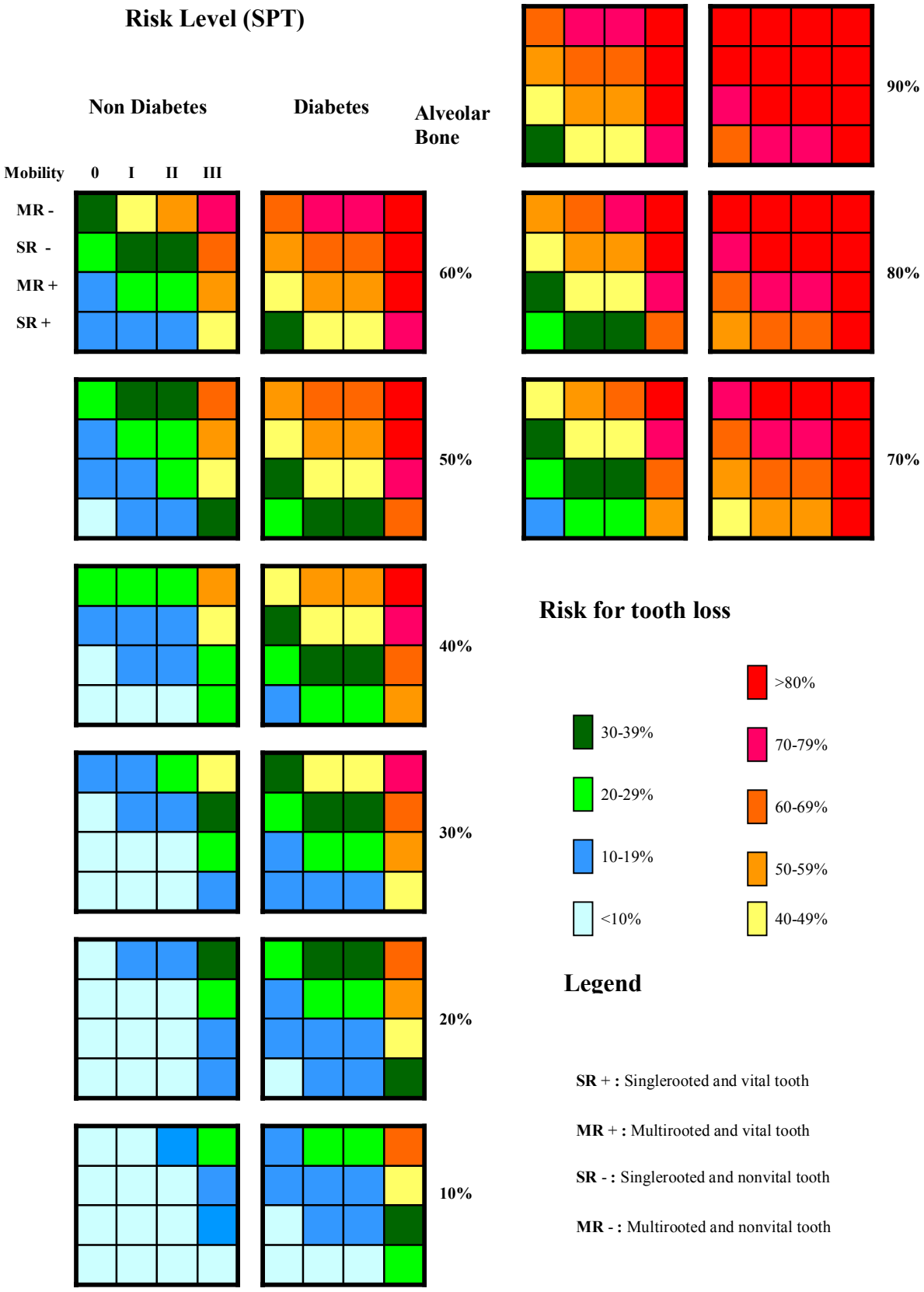

Table 11 indicates the risk for tooth loss when various variables are interacting together.

How to use this table:
- Identify the table relating to the tooth within the alveolar bone level strata at baseline
- Choose the cell that corresponds to the tooth anatomy, tooth sensitivity, tooth mobility and tooth status (caries or no caries)
- The risk of this tooth to be lost in 8 through 15 years will be depicted in the risk for tooth column (in the middle of the page) (adapted from "the Assessment and Management of Cardiovascular Risk", Evidence-Based Best Practice Guideline, 2003)

6 . Discussion

Various studies have identified different predictors for tooth loss in patients receiving or not periodontal treatment (Mcguire, 1996,1996, Baelum et al. 1997, Machtei et al. 1999, Matthews et al. 2001, König J et al. 2002, Jansson et al. 2002, Richards et al. 2005). However, these studies only have assessed the predictors separately, without a meaningful analysis of periodontal disease and other factors leading to tooth loss. Therefore, these information may be not helpful in clinical decision making due to the multifactorial components of the periodontal disease and tooth loss. This is the first study that identifies and organizes various variables related to tooth loss in periodontal treated patients, within a tool that may inform the probability of long-term tooth survival.

The effectiveness of the periodontal treatment achieving stable periodontal conditions has been repeatedly demonstrated. However, clinical measurements as pocket probing depth, absence of bleeding or suppuration, tooth mobility and presence of furcation may be not sufficient to predict long-term survival of an individual tooth (Renvert & Persson 2002). A periodontally involved tooth with deep pockets, increased mobility and bone loss is believed to be more prone to extraction. Otherwise, it can also not be assured that a stable tooth will be in function for a long time. As already explained, tooth loss has a multifactorial nature and therefore its assessment should be focussed toward this concept.

The difficulty to assign tooth prognosis was brought up in the dental literature in a series of studies that questioned the way dental practioners performed tooth extraction (Mcguire 1991, Mcguire & Nunn 1996, 1996, 1999). The authors argue that the prognosis assignment has usually an empirical and rather not scientific background. In addition, they suggest that the decision of tooth extraction varies depending on knowledge and experience of the practitioner.

In these series of studies, some clinical parameters related to tooth loss were investigated. Similar to the current results, the authors have found that initial tooth mobility and alveolar bone loss were related to tooth loss. However, some results are in disagreement with data assessed here. In McGuire´s study, diabetic and nondiabetic patients had similar tooth survival rate after the completion of the study. In the present study, diabetes was strongly associated with future tooth loss. Diabetic patients had almost 4 times more chance to have their teeth lost after 8 through 15 years than nondiabetics. Although the current sample of diabetes-affected patients is rather small, the results obtained in this investigation are in principle in agreement with various studies that have associated non-controlled diabetes with the worsening of periodontal condition and further tooth loss (Wilson 1989, Tervonen & Oliver 1993, Oliver & Tervonen 1994, Soskolne. 1998, Reeds 2000, Mattson & Cerutis 2001). A possible explanation for the discrepancy of results between McGuire's and the others studies is that in the former study all patients were categorized as having controlled diabetes. Some authors suggest similar periodontal conditions and prevalence of tooth loss in both well-controlled and nondiabetic patients (Tervonen & Oliver 1994, Sbordone et al. 1998). In other words, well-controlled diabetic patients might have the same response for periodontal treatment as disease-free patients and this might generate similar long-term results. However, in the present study, information about the metabolic control of diabetes mellitus could not be retrieved from the patient history and therefore a hypothetical high prevalence of poorly controlled patients might explain the higher prevalence of tooth loss in diabetic patients.

In the current study, it was also found that initial and increasing alveolar bone loss was related to further tooth loss. The results are in agreement with studies that have demonstrated that alveolar bone loss and clinical attachment loss may be in some way predictors for treatment outcomes and further tooth loss (Gilbert et al. 2002, Renvert et al. 2004). The analogy between levels of alveolar bone and attachment for clinical assessment of disease has been

demonstrated in the literature and one can be a surrogate from other (Papapanou & Wennström 1989, Machtei et al. 1997). However, the assessment of the level of attachment in the daily dental practice is not usual. Otherwise, the assessment of variation of alveolar bone level by a dental practioner may be performed by the measurement of normal intra-oral radiographs and can be therefore a more realistic situation. We preferred to use the term "bone level" instead of "bone loss" at baseline because of the difficulty to infer a standard minimum threshold for pathologic alveolar bone condition (Persson et al. 1998).

Type of tooth was another local factor related to future tooth loss. The most frequently extracted teeth in this study were multi-rooted. Also in the regression analysis multi-rooted had 80% more chance to be lost after 8 through 15 years than single-rooted teeth. This result is in agreement with various studies that have shown more difficulty to treat and control multi- than single-rooted periodontally affected teeth (Loos et al. 1989, Kaldahl et al. 1990, Wang et al. 1994).

One major limitation of this study is the lack of smoking status data. In 1989/1990 this question was not part of the medical questionnaire of the Department of Periodontology and therefore the data assessment was not possible. Tobacco use has been thoroughly demonstrated in the dental literature as an important predictor for the worsening of periodontal conditions, i.e. clinical attachment loss (Albandar et al. 2000, Calsina et al. 2002, Bergstrom J, 2004) and alveolar bone loss (Bolin et al. 1993,Bergstrom J, 2004), respectively. There are also evidences that tobacco consume may increase the risk for tooth loss. This subject will be discussed in-depth through the synopsis of some studies because of the importance of smoking in the pathogenesis of periodontitis and its potential effect on treatment outcome.

The effect of three forms of tobacco use (cigar, pipe and cigarette) on alveolar bone loss and tooth loss was assessed in a cohort prospective study covering up to 23 years and involving 690 males (Krall et al. 1999). Relative risk (RR) was used as a measurement for risk of future

tooth loss. The values for cigarette smokers, pipe smokers and cigar smokers were significantly more elevated than nonsmokers (RR=1.6, CI=95% 1.5-1.7 ; RR=1.6, CI=95% 1.4-1.9 and RR=1.3, CI=95% 1.2-1.5, respectively). In another longitudinal study involving 273 subjects, stratified on age and followed for 10 years (Holm G. 1994), the increase of risk for tooth loss was associated to the age and the number of cigarettes consumed daily. Patients up to 50 years old, smoking more than 15 cigarettes a day, were found to have the highest relative risk of losing teeth (RR=4.55, CI=95% 1.9-11.1). The significant discrepancy between RR values in Krall´s (Krall et al. 1999) and Holm´s (Holm G. 1994) studies (1.6 and 4.55, respectively) might be associated to the threshold for cigarette smoking exposure used in Holm´s study (Holm G. 1994) and the differences in the study methodology. The analysis of the influence of smoking cessation and its temporal relationship to tooth loss was described in another longitudinal study with 977 males who were followed-up over a period of 3 to 26 years (mean SD=18 +-7 years). Both sample of men who went on to quit smoking cigarettes and men who smoked throughout the study showed to be homogeneous at baseline regarding clinical oral measures and social behaviour. The results showed more probability of tooth loss in subjects that continuously smoked during the study (RR=2.4, CI=95% 2.2-2.7) than in nonsmokers. After the adjustment for age and baseline number of teeth, these estimates dropped considerably after the continuous cigarette smokers have quitted (RR=1.5, CI=95% 1.4-1.7). These evidences confirm that smokers are more prone to lose teeth than nonsmokers. Regarding the prevalence of tobacco consume, the smoking behaviour seems to be widespread in the contemporary society. A national survey on the use of psychoactive substances has been conducted in Germany every two to five years since 1980. The questionnaire survey of year 2000 included a population of 8139 participants (randomized sample with 45.5% response rate) between 18-59 years. In this investigation, a smoker was defined as a person who has smoked in the last 30 days some form of tobacco (cigarette, cigar, pipe, chewing and snorting tobacco). A person was considered non-smoker when he or

she has not consumed overall more than 100 cigarettes (respectively cigars and pipes). Former smoker was a person who has used more than 100 cigarettes (respectively cigars and pipes) but was abstinent at the time of survey.

In this sample, 39% of males and 31% of females respectively, responded as current smokers. It corresponds to an estimated population of 16.7 million smokers in the age group 18-59 years.

The smoking habit appears to be stable in Germany. The same national survey demonstrates that the smoking rate variation among males, between years 1990 and 2000, increased in the youngest ages (18-24 years) in the first half of the decade. After 1995, all age groups have showed a decrease in the number of smokers. In the female group, there was a decrease in the first half of decade for subjects between 18-39 years old. From 1995-2000 there was an increase of the number of smokers in the groups 18-24 and 40-59 years old. The intermediate group (25-39 years old) showed a substantially reduction of the number of smokers.

Regarding the trend of smoking, it can be concluded that, overall, males are smoking less since the half of last decade. Females show an ambiguous pattern, with two age-strata groups (18-24 and 40-59 years) having an increase of smoking rates and the remained group showing a slightly decrease (25-39 years).

The data from the present study is homogeneous regarding gender (52% females and 48% males) and age (mean age 47.58, SD +- 10,42) and, as explained before, current and former smokers were not identified in this sample due to the lack of this kind of information at baseline.

Interestingly, another variable at patient level (infectious hepatitis) was strongly related to tooth loss in the regression analysis with teeth extracted during initial therapy. It may be suggested that infectious hepatitis had a strong influence on the dental surgeon's extraction decision and, therefore, it might indicate that a more radical approach was thought to be needed in these patients.

In the present study, teeth with deep periodontal pockets were more extracted that teeth with shallow pockets and these findings confirm the validity of deep periodontal pockets as predictors for tooth loss (Copeland et al. 2004). However, the influence of pocket probing depth assessed at baseline on tooth loss was not confirmed in the present model of regression. A possible explanation for that is the difficulty to compare repeatedly probing pocket measurement due to the difference between the numbers of assessed sites. In 1989/1990 the pocket probing assessment was performed in only 2 reference points (vestibular - mesial and distal). Nowadays, the clinical pocket probing assessment changed considerably and 6 reference points are used. Hence, a reliable comparison between this parameter and tooth loss is difficult.

In this study, the overall tooth loss after 8 through 15 years was 10.24%. This result is in accordance with the literature (Tonetti et al. 2000, Checchi et al. 2002, König et al. 2002) and confirms that most periodontal patients with good compliance have a good chance to keep their teeth in function for long periods of time. In a study by Tonetti et al. (2000) 8.8% of all initially treated teeth were lost, however the average time span of the study was only 5.6 years. König et al. (2002) extracted 7.9% of all teeth assessed at baseline. A possible explanation for this difference between results is the number of teeth that were removed during the initial therapy phase. König et al. (2002) and Tonetti et al. (2000) have extracted during initial therapy 63% and 54% of all lost teeth, respectively. In the sample assessed here, 40% of all extracted teeth (3.64% of all teeth) were removed during this phase and a substantial higher number of teeth were extracted during the supportive periodontal therapy (249 teeth or 5.46% of all teeth). It is meaningful to explain that, in the present study, 52 from the 467 extracted teeth were considered as missing values due to the impossibility to identify the exact period of extraction and were not included in this analysis. Therefore, in our study, more teeth with questionable prognosis were initially kept, resulting in more teeth extracted along the study.

We also compared different groups of patients (Hirschfeld & Wasserman 1978, see chapter 3.2.2.4) who lost teeth with the number of periodontal maintenance appointments, but we have not found any statistically difference between groups. This result is in agreement with the data obtained by Rosen et al. (1999) who concluded that despite different time spans between SPT, similar effect on changes of probing depths or probing attachment levels were to be achieved. In other words, there was not statistically significant or clinical relevant difference in further disease progression between groups that received heterogeneous SPT frequencies. Hence, the logistic regression in our study failed to show any relationship between frequency of SPT and tooth loss. Other information, compared to the Hirschfeld and Wasserman´s study, was the percentage of patients included into the three different groups proposed by the authors. In the present study, only 1.12% of the patients were included into the extreme downhill group (patients who lost 10-23 teeth) and the data confirms that most periodontal patients may keep their teeth for long periods with appropriate supportive therapy. The strategy of running two distinct regression analysis is due to the characteristics of risk assessment in distinct periods of treatment. The risk assessment in teeth extracted during the initial therapy phase is entirely dependent on the dentist's treatment decision and therefore the effect of periodontal treatment on tooth loss is difficult to be assessed. The second regression analysis is clearly more related to the outcome of periodontal therapy, in spite of some relationship between dentist's decision and risk assessment may be present. Therefore, the forecast model for tooth loss only used the variables related to teeth extracted during supportive periodontal therapy and this may give us a more reliable model. However, the model might have some limitations of use when some variables are categorized into distinct levels. This is the case of the alveolar bone level parameter. The heterogeneous tooth distribution at baseline into various strata of bone, with few teeth included into <10% and more than >60% bone level categories might suggest that the predictor model does not work very well in these levels. The same is valid for the tooth mobility III, which has few teeth

included into this level of mobility at baseline. Therefore, the few data in these categories might imply in a less reliable model.

The R^2 value from the regression analysis performed with teeth extracted during SPT is in agreement with the results from other two studies, with the models explaining partially the results. The first one analysed the influence of some variables on tooth loss (König et al. 2002). In this retrospective study the R^2 value was 0.12 whereas a slightly higher value was obtained in the present study (R^2=0.15). Another study (Paulander et al 2004) assessed some risk factors for periodontal bone loss in 50-year-old individuals. The findings in this study were similar to our results with the R^2 values varying from 0.12 to 0.15.

The present study also demonstrated a difference between R^2 values from the regression analysis with teeth extracted during both initial and supportive periodontal therapy ($R^2(1)$=0.35 and $R^2(2)$=0.15, respectively). A possible explanation for this difference is the length of the supportive periodontal therapy. Probably the lower value for the $R^2(2)$ is due to other variables than the ones included in the model, that may have had influence on tooth loss in the period of supportive periodontal therapy. The short period for tooth loss assessment in the regression analysis with teeth extracted during initial therapy probably reduced the influence of other variables than the ones included in the model. In other words, the short time span between the baseline and tooth extraction during initial therapy possibly generated a more precise model.

A risk assessment model using clinical parameters was already described in the dental literature (Page et al. 2002, 2003). However, there are important considerations that are worth to be mentioned. In the Page et al. (2002, 2003) study a risk scale was used to assess the risk of future periodontal deterioration at patient level. In other words, patients were grouped into various levels of risk (scale of 1 to 5) that indicate the probability of these subjects to develop periodontitis in the future. However, the assessment of tooth loss as the true endpoint for the dental treatment instead of clinical outcomes surrogates should be considered (Hujoel et al

1995). A model that organizes patients into levels of risk may generate inaccurate information for clinical decision making (for example, a complex restorative treatment that demands a critical decision about the maintenance or not of a strategic tooth). Furthermore, in the Page´s study the vast majority of patients (92% of subjects over the 15-year period of study) have had no periodontal treatment.

The predictor model for tooth loss, which has the ability to assess the prognosis of a periodontal treated tooth through the analysis of various parameters affecting its long-term survival, is featured to be applied at tooth level. This is an advantage in clinical decision making because a prognosis on the true endpoint of the therapy can be assessed.

7 . Conclusions

The findings presented in this study provide meaningful data for the construction of a risk assessment model for tooth loss in patients receiving periodontal therapy over an observation period from 8 through 15 years. The risk assessment chart constructed with this model, which explains 15% of the observed variability in tooth loss, may be useful in determining the individual prognosis of a tooth under periodontal therapy and therefore in guiding treatment decisions.

8. Literature

Adriaens PA, Adriaens LM. Effects of nonsurgical periodontal therapy on hard and soft tissues. Periodontol 2000 2004; **36**: 121-45.

Agerholm DM, Sidi AD. Reasons given for extraction of permanent teeth by general dental practioners in England and Wales. Br Dent J 1988. 164: 345-348.

Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. J Periodontol. 2000 Dec;71(12):1874-81.

Amano A, Nakagawa I, Okahashi N, Hamada N. Variations of Porphyromonas gingivalis fimbriae in relation to microbial pathogenesis. J Periodontal Res 2004; **39**(2): 136-42.

Axelsson P, Nystrom B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. J Clin Periodontol 2004; **31**(9): 749-57.

Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. J Clin Periodontol 1998; **25**(4): 297-305.

Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. J Clin Periodontol. 1981; **8**(4): 281-94.

Baelum V, Luan WM, Chen X, Fejerskov O. Predictors of tooth loss over 10 years in adult and elderly Chinese. Community Dent Oral Epidemiol 1997; 25(3): 204-10.

Barbour SE, Nakashima K, Zhang JB, Tangada S, Hahn CL, Schenkein HA, Tew JG. Tobacco and smoking: environmental factors that modify the host response (immune system) and have an impact on periodontal health. Crit Rev Oral Biol Med. 1997; **8**(4): 437-60.

Bergstrom J. Influence of tobacco smoking on periodontal bone height. Long-term observations and a hypothesis. J Clin Periodontol. 2004 Apr;31(4):260-6.

Bergstrom J. Tobacco smoking and chronic destructive periodontal disease. Odontology. 2004 Sep;92(1):1-8. Review

Bergstrom J. Tobacco smoking and risk for periodontal disease. J Clin Periodontol. 2003; **30**(2):107-13.

Bergstrom J. Cigarette smoking as risk factor in chronic periodontal disease Community Dent Oral Epidemiol 1989; **17**(5): 245-7.

Bolin A, Eklund G, Frithiof L, Lavstedt S. The effect of changed smoking habits on marginal alveolar bone loss. A longitudinal study. Swed Dent J. 1993;17(5):211-6.

Bowden GH, Hamilton IR. Survival of oral bacteria. Crit Rev Oral Biol Med 1998; **9**(1): 54-85.

Bradshaw DJ, Marsh PD, Watson GK, Allison C. Role of Fusobacterium nucleatum and coaggregation in anaerobe survival in planktonic and biofilm oral microbial communities during aeration. Infect Immun 1998; **66**(10): 4729-32.

Burt BA, Ismail AI, Morrison EC, Beltran ED. Risk factors for tooth loss over a 28-year period. J Dent Res 1990; **69**(5): 1126-30.

Cahen PM, Frank RM, Turlot JC. A survey of the Reasons for Dental Extractions in France. J Dent Res 1985; **64**(8): 1087-1093.

Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. J Clin Periodontol. 2002 Aug;29(8):771-6.

Checchi L, Montevecchi M, Gatto MR, Trombelli L. Retrospective study of tooth loss in 92 treated periodontal patients. J Clin Periodontol 2002 ; **29**(7): 651-6.

Choong S, Whitfield H. Biofilms and their role in infections in urology. BJU Int. 2000; **86**(8): 935-41.

Copeland LB, Krall EA, Brown LJ, Garcia RI, Streckfus CF. Predictors of tooth loss in two US adult populations. J Public Health Dent 2004; 64(1): 31-37.

Costerton W, Veeh R, Shirtliff M, Pasmore M, Post C, Ehrlich G. The application of biofilm science to the study and control of chronic bacterial infections. J Clin Invest 2003; **112**(10): 1466-77.

Da'ameh D. Reasons for permanent tooth extraction in the North of Afghanistan. J Dent. 2005 May 19

Darveau RP, Tanner A, Page RC. The microbial challenge in periodontitis. Periodontol 2000. 1997; 14:12-32.

Eckerbom M, **Magnusson T**, **Martinsson T**. Reasons for and incidence of tooth mortality in a Swedish population. Endod Dent Traumatol. 1992 Dec;8(6):230-4.

Elias AC, Sheiham A. The relationship between satisfaction with mouth and number, position and condition of teeth: studies in Brazilian adults. J Oral Rehabil 1999; **26**(1):53-71.

Flemmig TF, Milian E, Karch H, Klaiber B. Differential clinical treatment outcome after systemic metronidazole and amoxicillin in patients harboring Actinobacillus actinomycetemcomitans and/or Porphyromonas gingivalis. J Clin Periodontol 1998; **25**(5):380-7.

Gilbert GH, Shelton BJ, Chavers LS, Bradford EH Jr. Predicting tooth loss during a population-based study: role of attachment level in the presence of other dental conditions. J Periodontol 2002 ; **73**(12): 1427-36.

Graswinckel JE, van der Velden U, van Winkelhoff AJ, Hoek FJ, Loos BG. Plasma antibody levels in periodontitis patients and controls. J Clin Periodontol 2004; **31**(7): 562-8.

Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R, Zambon JJ, Hausmann E. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. J Periodontol 1995; **66**(1):23-9.

Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, Norderyd OM, Genco RJ. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. J Periodontol 1994; **65**(3):260-7.

Guzman S, Karima M, Wang HY, Van Dyke TE. Association between interleukin-1 genotype and periodontal disease in a diabetic population. J Periodontol 2003; **74**(8): 1183-90.

Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993; **64**(1): 16-23.

Haddad I, **Haddadin K**, **Jebrin S**, **Ma'ani M**, **Yassin O**. Reasons for extraction of permanent teeth in Jordan. Int Dent J. 1999 Dec;49(6):343-6.

Haffajee AD, Socransky SS, Gunsolley JC. Systemic anti-infective periodontal therapy. A systematic review. Ann Periodontol 2003; **8**(1): 115-81.

Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles.

J Clin Periodontol 2001; **28**(4): 283-95.

Herrera D, Sanz M, Jepsen S, Needleman I, Roldan S. A systematic review on the effect of systemic antimicrobials as an adjunct to scaling and root planing in periodontitis patients. J Clin Periodontol 2002 ;**29** Suppl 3: 136-59; discussion 160-2.

Hiidenkari T, **Parvinen T**, **Helenius H**. Missing teeth and lost teeth of adults aged 30 years and over in south-western Finland. Community Dent Health. 1996 Dec;13(4):215-22.

Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. J Periodontol 1978; **49**(5): 225-37.

Holm G. Smoking as an additional risk for tooth loss. J Periodontol 1994; **65**(11): 996-1001.

Holman WL, Rayburn BK, McGiffin DC, Foley BA, Benza RL, Bourge RC, Pinderski LJ, Kirklin JK. Infection in ventricular assist devices: prevention and treatment. Ann Thorac Surg 2003; **75**(6 Suppl): S48-57.

Hoyle BD, Costerton JW. Bacterial resistance to antibiotics: the role of biofilms. Prog Drug Res 1991; **37**: 91-105.

Jansson L, Lavstedt S, Zimmerman M. Prediction of marginal bone loss and tooth loss--a prospective study over 20 years. J Clin Periodontol 2002; **29**(8): 672-8.

Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP. Responses of four tooth and site groupings to periodontal therapy. J Periodontol 1990; **61**: 173-179.

Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. J Periodontol. 1996; **67**(7): 675-81.

Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of

periodontal therapy: I. Response to 4 therapeutic modalities. J Periodontol 1996; **67**(2): 93-102.

Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: II. Incidence of sites breaking down. J Periodontol 1996; **67**(2):103-8.

Kassenzahnärztliche Bundesvereinigung. Jahrbuch 2003.

Kayser AF. How much reduction of the dental arch is functionally acceptable for the ageing patient? Int Dent J. 1990 ; **40**(3): 183-8.

Kayser AF. Shortened dental arches and oral function. J Oral Rehabil 1981; **8**(5): 457-62.

Kinane DF, Chestnutt IG. Smoking and periodontal disease. Crit Rev Oral Biol Med 2000; **11**(3): 356-65.

Konig J, Plagmann HC, Ruhling A, Kocher T. Tooth loss and pocket probing depths in compliant periodontally treated patients: a retrospective analysis. J Clin Periodontol. 2002; **29**(12): 1092-100.

Lange DE, Plagmann HC, Eenboom A, Promesberger A. [Clinical methods for the objective evaluation of oral hygiene] Dtsch Zahnarztl Z 1977; **32**(1): 44-7.

Langland OE, Sippy FH. A study of radiographic longitudinal distortion of anterior teeth using the paralleling technique. Oral Surg Oral Med Oral Pathol 1966; **22**(6): 737-49.

Li J, Helmerhorst EJ, Leone CW, Troxler RF, Yaskell T, Haffajee AD, Socransky SS, Oppenheim FG. Identification of early microbial colonizers in human dental biofilm. J Appl Microbiol 2004; **97**(6): 1311-8.

Lindhe J. Clinical periodontology and implant dentistry. 3rd edition. Munksgaard

International Publishers 1997.

Lindhe J, Nyman S. Long-term maintenance of patients treated for advanced periodontal disease. J Clin Periodontol 1984; **11**(8): 504-14.

Listgarten MA. The role of dental plaque in gingivitis and periodontitis. J Clin Periodontol 1988; **15**(8): 485-7.

Locker D, Ford J, Leake JL. Incidence of and risk factors for tooth loss in a population of older Canadians. J Dent Res 1996; **75**(2): 783-9.

Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 16 years of age. J Clin Periodontol 1986; 13(5): 431-445.

Loesche WJ, Giordano JR, Soehren S, Kaciroti N. The nonsurgical treatment of patients with periodontal disease: results after five years. J Am Dent Assoc 2002; **133**(3): 311-20.

Loos B, Nylund K, Claffey N, Egelberg J. Clinical effect of root debridement in molar and non-molar teeth. J Clin Periodontol 1989; **16**: 498-504.

Machtei EE, Hausmann E, Dunford R, Grossi S, Ho A, Davis G, Chandler J, Zambon J, Genco RJ. Longitudinal study of predictive factors for periodontal disease and tooth loss. J Clin Periodontol 1999; **26**(6): 374-80.

Machtei EE, Hausmann E, Grossi S, Dunford R, Genco RJ. The relationship between radiographic and clinical changes in the periodontium. J Periodontal Res 1997; **32**(8): 661-666.

Manji F, Baelum V, Fejerskov O. Tooth mortality in an adult rural population in Kenya. J

Dent Res 1988; **67**(2): 496-500.

Matthews DC, Smith CG, Hanscom SL. Tooth loss in periodontal patients. J Can Dent Assoc 2001; **67**(4): 207-10.

Mattson JS, Cerutis DR. Diabetes mellitus: a review of the literature and dental implications. Compend Contin Educ Dent 2001; **22**(9): 757-60, 762, 764 passim; quiz 773.

McCaul LK, **Jenkins WM**, **Kay EJ**. The reasons for extraction of permanent teeth in Scotland: a 15-year follow-up study. Br Dent J. 2001 Jun 23;190(12):658-62.

McGuire MK, Nunn ME. Prognosis versus actual outcome. IV. The effectiveness of clinical parameters and IL-1 genotype in accurately predicting prognoses and tooth survival. J Periodontol 1999; **70**(1): 49-56.

McGuire MK, Nunn ME. Prognosis versus actual outcome. III. The effectiveness of clinical parameters in accurately predicting tooth survival. J Periodontol 1996; **67**(7): 666-74.

McGuire MK, Nunn ME. Prognosis versus actual outcome. II. The effectiveness of clinical parameters in developing an accurate prognosis. J Periodontol 1996; **67**(7): 658-65.

McGuire MK. Prognosis versus actual outcome: a long-term survey of 100 treated periodontal patients under maintenance care. J Periodontol 1991; **62**(1): 51-8.

McMullen JA, Van Dyke TE, Horoszewicz HU, Genco RJ. Neutrophil chemotaxis in individuals with advanced periodontal disease and a genetic predisposition to diabetes mellitus. J Periodontol 1981; **52**(4): 167-73.

Meisel P, Siegemund A, Dombrowa S, Sawaf H, Fanghaenel J, Kocher T. Smoking and polymorphisms of the interleukin-1 gene cluster (IL-1alpha, IL-1beta, and IL-1RN) in

patients with periodontal disease. J Periodontol 2002; **73**(1): 27-32.

Missailidis CG, Umeda JE, Ota-Tsuzuki C, Anzai D, Mayer MP. Distribution of fimA genotypes of Porphyromonas gingivalis in subjects with various periodontal conditions. Oral Microbiol Immunol 2004; **19**(4): 224-9.

Muhlemann HR, Son S. Gingival sulcus bleeding--a leading symptom in initial gingivitis. Helv Odontol Acta 1971; **15**(2):107-13.

Murray H, **Locker D**, **Kay EJ**. Patterns of and reasons for tooth extractions in general dental practice in Ontario, Canada. Community Dent Oral Epidemiol. 1996 Jun;24(3):196-200.

Murray H, Clarke M, Locker D, Kay EJ. Reasons for tooth extractions in dental practices in Ontario, Canada according to tooth type. Int Dent J 1997; **47**: 3-8.

National Center for Health Statistics. Current smoking among adults aged 18 years and over: United States, 1997-2003.

Neut D, van Horn JR, van Kooten TG, van der Mei HC, Busscher HJ. Detection of biomaterial-associated infections in orthopaedic joint implants. Clin Orthop 2003; (413):261-8.

New Zealand Guidelines Group (NZGG). The assessment and management of cardiovascular risk. Evidence-Based Best Practice Guideline, December 2003.

Niessen & Weyant. Causes of tooth loss in a veteran population. J Public Health Dent. 1989 Winter;49(1):19-23.

Nishihara T, Koseki T. Microbial etiology of periodontitis. Periodontol 2000 2004 ;**36**: 14-26.

Nunn ME. Understanding the etiology of periodontitis: an overview of periodontal risk

factors. Periodontol 2000. 2003; **32**: 11-23.

Oginni FO. Tooth loss in a sub-urban Nigerian population: causes and pattern of mortality revisited. Int Dent J. 2005 Feb;55(1):17-23.

O'Leary TJ, Drake RB, Naylor JE. The plaque control record. J Periodontol 1972; **43**(1):38.

Ong G. Periodontal reasons for tooth loss in an Asian population. J Clin Periodontol 1996; 23: 307-309.

Osterberg T, Steen B. Relationship between dental state and dietary intake in 70-year-old males and females in Goteborg, Sweden: a population study. J Oral Rehabil 1982; **9**(6): 509-21.

Page RC. The microbiological case for adjunctive therapy for periodontitis. J Int Acad Periodontol 2004; **6**(4 Suppl): 143-9.

Page RC, Martin J, Krall EA, Mancl L, Garcia R. Longitudinal validation of a risk calculator for periodontal disease. J Clin Periodontol 2003; **30**(9): 819-27.

Page RC, Krall EA, Martin J, Mancl L, Garcia RI. Validity and accuracy of a risk calculator in predicting periodontal disease. J Am Dent Assoc 2002; **133**(5): 569-76.

Page RC, Beck JD. Risk assessment for periodontal diseases. Int Dent J 1997; **47**(2): 61-87.

Persson RE, Hollender LG, Persson GR. Assessment of alveolar bone levels from intraoral radiographs in subjects between ages 15 and 94 years seeking dental care. J Clin Periodontol 1998; **25**(8): 647-654.

Persson GR, Attstrom R, Lang NP, Page RC. Perceived risk of deteriorating periodontal

conditions. J Clin Periodontol 2003; **30**(11): 982-9.

Persson L, Bergstrom J, Ito H, Gustafsson A. Tobacco smoking and neutrophil activity in patients with periodontal disease. J Periodontol. 2001; **72**(1): 90-5.

Petersilka GJ, Ehmke B, Flemmig TF. Antimicrobial effects of mechanical debridement. Periodontol 2000. 2002; **28**: 56-71.

Reich and Hiller. Reasons for tooth extraction in the western states of Germany. Community Dent Oral Epidemiol. 1993 Dec;21(6):379-83.

Renvert S, Persson GR. A systematic review on the use of residual probing depth, bleeding on probing and furcation status following initial periodontal therapy to predict further attachment and tooth loss. J Clin Periodontol 2002; Suppl **3**: 82-89.

Renvert S, Persson GR. Patient-based assessments of clinical periodontal conditions in relation to alveolar bone loss. J Clin Periodontol 2004; 31(3):208-13.

Richards W, **Ameen J**, **Coll AM**, **Higgs G**. Reasons for tooth extraction in four general dental practices in South Wales. Br Dent J. 2005 Mar 12;198(5):275-8.

Rosen B, Olavi G, Badersten A, Ronstrom A, Soderholm G, Egelberg J. Effect of different frequencies of preventive maintenance treatment on periodontal conditions. 5- Year observations in general dentistry patients. J Clin Periodontol 1999; **26**(4): 225-233.

Salvi GE, Yalda B, Collins JG, Jones BH, Smith FW, Arnold RR, Offenbacher S. Inflammatory mediator response as a potential risk marker for periodontal diseases in insulin-dependent diabetes mellitus patients. J Periodontol 1997; **68**(2): 127-35.

Sarita PT, Witter DJ, Kreulen CM, Van't Hof MA, Creugers NH. Chewing ability of

subjects with shortened dental arches. Community Dent Oral Epidemiol 2003; **31**(5):328-34.

Sayegh A, **Hilow H**, **Bedi R**. Pattern of tooth loss in recipients of free dental treatment at the University Hospital of Amman, Jordan. J Oral Rehabil. 2004 Feb;31(2):124-30.

Sbordone L, Ramaglia L, Barone A, Ciaglia RN, Iacono VJ. Periodontal status and subgingival microbiota of insulindependent juvenile diabetics: a 3-year longitudinal study. J Periodontol 1998: **69**: 120–128.

Shimazaki Y, Soh I, Koga T, Miyazaki H, Takehara T. Risk factors for tooth loss in the institutionalised elderly; a six-year cohort study. Community Dent Health 2003; **20**(2): 123-7.

Slade GD, Gansky SA, Spencer AJ. Two-year incidence of tooth loss among South Australians aged 60+ years. Community Dent Oral Epidemiol 1997; **25**(6): 429-37.

Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. Periodontol 2000 2002; **28**: 12-55.

Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. J Periodontol 1992; **63**(4 Suppl): 322-31.

Soskolne WA. Epidemiological and clinical aspects of periodontal diseases in diabetics. Ann Periodontol 1998; **3**(1): 3-12.

Stabholz A, **Babayof I**, **Mersel A**, **Mann J**. The reasons for tooth loss in geriatric patients attending two surgical clinics in Jerusalem, Israel. Gerodontology. 1997;14(2):83-8.

Steele JG, Sanders AE, Slade GD, Allen PF, Lahti S, Nuttall N, Spencer AJ. How do age and tooth loss affect oral health impacts and quality of life? A study comparing two national samples. Community Dent Oral Epidemiol 2004 ; **32**(2): 107-14.

Tervonen T, Oliver RC. Long-term control of diabetes mellitus and periodontitis. J Clin Periodontol 1993; **20**(6): 431-5.

Theilade E, Wright WH, Jensen SB, Loe H. Experimental gingivitis in man. II. A longitudinal clinical and bacteriological investigation. J Periodontal Res 1966; **1**: 1-13.

Tlaskalova-Hogenova H, Stepankova R, Hudcovic T, Tuckova L, Cukrowska B, Lodinova-Zadnikova R, Kozakova H, Rossmann P, Bartova J, Sokol D, Funda DP, Borovska D, Rehakova Z, Sinkora J, Hofman J, Drastich P, Kokesova A. Commensal bacteria (normal microflora), mucosal immunity and chronic inflammatory and autoimmune diseases. Immunol Lett 2004; **93**(2-3): 97-108.

Tonetti MS, Steffen P, Muller-Campanile V, Suvan J, Lang NP. Initial extractions and tooth loss during supportive care in a periodontal population seeking comprehensive care. J Clin Periodontol 2000; **27**(11): 824-31.

Umeda M, Takeuchi Y, Noguchi K, Huang Y, Koshy G, Ishikawa I. Effects of nonsurgical periodontal therapy on the microbiota. Periodontol 2000 2004; **36**: 98-120.

Vignarajah S. Various reasons for permanent tooth extractions in a Caribbean population—Antigua. Int Dent J. 1993 Jun;43(3):207-12.

Wang HL, Burgett FG, Shjr Y, Ramfjord S. The influence of molar furcation involvement and mobility on future clinical periodontal attachment loss. J Periodontol 1994; **65**: 25-29.

Wilson TG Jr. Periodontal diseases and diabetes. Diabetes Educ 1989; **15**(4):342-5.