Recurrence of destructive periodontal disease after treatment. A long term study

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SUMMARY

This study demonstrates the difficulties which arise in statistical analysis derived from data of observations in post-treatment healing and recurrence of periodontal disease. Ten individuals received an initial phase of treatment followed by surgical pocket elimination utilizing replaced flap surgery. Clinical measurements of recession and pocketing were taken pre-operatively and 6 monthly thereafter for a period of four years after treatment. The results have shown that surgical treatment is least benefit in cases of mild pocketing, and of most benefit in cases of severe pocketing. A tendency for reduction in recession levels after two years was observed. Pocketing and attachment levels showed a tendency toward relapse as early as 6 months postoperatively. However, this simple observation masks a complex pattern of tissue remodeling in which some sites remain stable, others improve, while others relapse with time. Improvement may occur concurrently in different sites. Continued improvement in the clinical parameters of pocketing and attachment levels was observable in some sites for up to 2 years after surgery, after which the tendency for recurrent disease became more noticeable. Analyses of changes in attachment levels may be misleading, because they may mask the divergent or convergent trends of recession and pocketing, which may be occurring simultaneously. As bursts of healing as well as bursts of destruction could be observed concurrently over the period of 4 years after treatment, it is suggested that a state of «dynamic equilibrium» is established, in which continued tissue remodeling over a protracted period of time, may give rise to the clinical misconception that little or no changes are occurring in the majority of sites with time.

KEY WORDS:

Statistical analysis, periodontal disease, post-treatment.

RÉSUMÉ

Cette étude démontre les difficultés qui surviennent dans l'analyse statistique issue des données et observations obtenues après traitement, au cours de la guérison et pendant les récidives de la paradontite. Dix patients ont subi une phase initiale de traitement suivie d'une élimination chirurgicale des poches paradontales utilisant le «flap surgery» (rétablissement de la partie recouvrante de la gencive). On a relevé des mesures précises des récessions et des poches paradontales avant l'intervention chirurgicale ainsi que tous les six mois pendant quatre ans. Les résultats ont démontré que dans les cas de paradontite peu sévères, les avantages sont considérables. On a aussi observé une tendance générale à la réduction des niveaux de récession pendant deux ans. On a noté une tendance vers la rechute dès six mois après l'opération. Cette observation d'ordre général déguise cependant un mode complexe d'évolution où certaines zones se stabilisent, d'autres s'améliorent et d'autres se détériorent avec le temps. Le processus d'amélioration peut exister simultanément dans différentes zones. On a pu observer l'amélioration continue des paramètres cliniques des poches et des attachements jusqu'a deux ans après l'intervention chirurgicale, après quoi le syndrome

resurgit. Les tendances divergentes ou convergentes simultanées de la récession ainsi que de la formation des poches sont difficiles à observer seulement par des analyses de changement des niveaux d'attachement. Puisqu'il a été possible d'observer des poussées d'amélioraton et de régression pendant quatre ans après le traitement, on peut conclure qu'un état «d'équilibre dynamique» s'établit, dans lequel un remodelage continu du tissu gingival pendant une période prolongée pourrait conduire à la conception erronée que très peu ou même aucun changement ne se produit avec le temps dans la majorité des zones observées.

MOTS CLEFS:
Analyse statistique, périodontite, après-traitement.

INTRODUCTION

Traditionally, the progression of periodontal diseases have been viewed as a linear progression towards tooth loss in later adult life. In most of the epidemiological studies, the statistical analysis has been based on mean values for different population groups. In an investigation where there are many observations (sites) with little or no change and a few sites with large changes, the use of parametric statistics, such as the means and standard deviations of the changes may not adequately represent the data. The mean value may conceal the few large changes, and is dominated by the small changes. In a similar manner, the absolute mean values, pooled across many sites may conceal information available in the relative or paired changes observed at individual sites. Haffajee *et al.* (1983) have suggested that mean values should be supplemented by analysis of frequency distributions of the sites undergoing major changes. Utilising these statistical techniques, new models of the progression of periodontal disease have been proposed (Goodson *et al.* (1982), Haffajee *et al.*, (1983)).

These models include:

a) The linear progression model which results from continuous breakdown of affected sites with time.

b) The random burst model in which susceptible sites demonstrate sudden short bursts of activity followed by periods of quiescence. The frequency and extent of these bursts of activity vary between individuals, and in different sites within the same individual.

c) The asynchronous multiple burst model in which a number of sites may become highly active during periods in an individual's life at which they are susceptible to the pathological process. The frequency and extent of the destructive bursts of activity varies between individuals. These is some evidence demonstrating bursts of destructive activity occurring at different time intervals (Socransky *et al.* 1984, Socransky & Haffajee 1985).

Goodson *et al.* (1982) have shown that less than 10% of sites in an individual may be subject to destructive periodontal disease in a one year period, while other workers (Socransky & Haffajee, 1985) have described the existence of a small group of individuals who are highly susceptible to periodontal destruction. The existence of different patterns and rates of destructive periodontal disease have been described in untreated cases of periodontitis.

Little is known of the pattern of disease activity after treatment of periodontitis. In long-term studies by Hirschfield & Wasseman (1978) and McFall (1982) recurrent destructive periodontal disease has been shown to occur in a small group of individuals. The groups which showed the greatest amount of tooth loss as a result of recurrent disease (termed the extreme downhill group) comprised 4.2% of the population, while those with more moderate tooth loss (termed the downhill group) accounted for 12.6% of the population. These two groups accounted for the vast majority of the tooth loss reported in these studies. This would tend to imply that the patterns of destruction described in untreated cases of periodontal disease may well be observable in recurrent periodontal disease after treatment.

The aim of this study was to observe and assess what patterns of recurrent disease occurred over a four year period, after periodontal treatment.

MATERIAL AND METHODS

Ten individuals were selected for periodontal surgery after completing an initial phase of treatment which included oral hygiene instruction, supragingival and subgingival scaling and root planing. Criteria for selection to the study included the presence of multiple pockets after completion of the initial phase of

treatment, the absence of relevant medical history, and a consistent ability to maintain high levels of oral cleanliness as monitored by the O'Leary Plaque Control Record (1972) over an extended period of time. Replaced flap surgery was performed, taking care to preserve as much of the gingival tissue especially in the papillary regions to enable the best possible wound coverage in the interdental regions. No systemic antimicrobial agents were prescribed prior to or during the active phase of treatment. Chlorhexidine gluconate mouthwash was prescibed for the first week after surgery, after which no further topical or systemic antimicrobial agents were recommended. The degree of gingival recession and pocket depths were assessed pre-operatively and every six months post-operatively for a period of four years using a Hu-Friedy periodontal probe with a probe tip diameter of 0.62 mms. Pocket depth measurements were taken from the gingival margins to the base of the pockets, and the degree of recession was measured from the cemento-enamel junctions to the gingival margins. The attachment levels were then derived from the summation of the pocket dephts and recession levels at each site. All measurements were taken by the same investigator whose reproducibility had been assessed to be 86% in a pilot investigation. The attachment level was derived by adding the degree of recession to the pocket depth in each case. Measurements were taken at pre-determined sites around the teeth, without the use of stents which were found to prevent adequate pocket depth measurements in deeper pockets and in the less accessible areas in a preliminary pilot study. Assessment sites included the mesial and distal line angles, midfacial, and furcations on both the buccal and lingual surfaces of the teeth.

During the period of study the individuals were all seen at three monthly intervals for scaling and remotivation in oral hygiene procedures. At each visit plaque and gingivitis levels were assessed. The data for pocket depth measurements, recession, and levels of attachment were then analysed. Initially mean values and corresponding standard deviations were derived from each of the groups of pockets and subdivided into mild (0-3 mm), moderate (4 & 5 mm), and severe (greater than 6 mm). A number of methods for statistical analysis of the data were considered. These included tolerance tests, regression analysis, cumulative sum and multiple analysis of variance techniques. As the data could not be assumed to follow a linear progression with time, tolerance tests and regression analyses were not considered appropriate. Cumulative sum and multiple analysis of variance techniques would be appropriate for this type of data, but were not applied as the small size of the sample group in this study did not permit the extraction of meaningful inferences from these robust statistical tests.

The data were therefore only subjected to an analysis of frequency of sites which remained stable or unstable following treatment either in terms of continued long-term healing or in terms of recurrence of disease. Thus the data is presented subdivided into the categories of sites which were better, stable, or worse at each time period during the study.

RESULTS

Plaque and gingivitis levels were assessed at each visit, and these remained consistently low throughout the study.

The results of the statistical analysis are presented in Tables I-IV.

Table I shows the mean values in millimetres and standard deviations for the mild, moderate and severe pockets, for the assessed variables of recession, pocketing and attachment levels. From this table it can be seen that just over 1 mm of recession occurred immediately after treatment, which remained stable until two years after treatment, after which there was a tendency for increased recession.

This was more marked in those pockets which where initially severe than in those which were initially mild or moderate. Reduction in pocket depths, and gain in attachment as a result of treatment was minimal in the mild group, with approximately 50% reduction in the moderate group and about 60% in the severe group. Gain in attachment was also better in the moderate and deep defects than in the mild group.

Thus more reduction in these variables was achieved in the deeper defects. After three years post-operatively the mild group showed a trend for increased pocket depths and loss of attachment, which became evident in all three groups by four years post-operatively. It should be noted that the standard deviation about the means tend to increase with time in all three groups, indicating that higher levels of variation occur with time.

Thus some sites may show greater changes post-operatively than others.

Table II illustrates the results of treatment in respect of post-operative gingival recession. Up to 1 year

TABLE I
Mean values of the clinical variables in millimeters and standard deviations for all patients during the time period under investigation subdivided into those defects which were initially mild, moderate, or severe.

Variable	Weeks	Mild	Moderate	Severe
Recession	Baseline	0.26 (0.64)	0.33 (0.81)	0.026 (0.72)
	6 months	1.01 (1.64)	1.06 (1.14)	1.53 (1.42)
	1 year	0.97 (1.18)	1.02 (1.21)	1.53 (1.42)
	2 years	1.07 (1.16)	1.09 (1.11)	1.61 (1.52)
	3 years	1.29 (1.61)	1.20 (1.25)	1.83 (1.52)
	4 years	1.47 (1.28)	1.44 (1.26)	2.17 (1.66)
Pocketing	Baseline	1.73 (0.44)	3.72 (0.86)	7.33 (1.22)
	6 months	1.39 (0.80)	1.96 (0.98)	2.96 (1.52)
	1 year	1.51 (0.85)	2.16 (0.97)	3.44 (1.60)
	2 years	1.38 (0.79)	2.06 (0.91)	3.32 (1.72)
	3 years	1.47 (0.95)	1.90 (1.14)	2.96 (1.51)
	4 years	1.55 (1.00)	2.09 (1.12)	3.25 (1.76)
Attachment	Baseline	1.99 (0.76)	4.05 (1.19)	7.59 (1.33)
	6 months	2.40 (1.25)	3.02 (1.36)	4.48 (2.03)
	1 year	2.49 (1.39)	3.18 (1.46)	4.96 (2.11)
	2 years	2.45 (1.47)	3.15 (1.55)	4.93 (2.26)
	3 years	2.76 (1.85)	3.10 (1.46)	4.78 (2.07)
	4 years	3.02 (1.74)	3.53 (1.70)	5.42 (2.43)

post-operatively, it can be seen that a small minority of sites in the mild and moderate group had less recession (better) after treatment than at baseline, and approximately 40% of the mild group and 50% of the moderate group showed no change relative to baseline. However, by two years post-operatively, over half of the mild and moderate sites and nearly two thirds of the deep sites showed increased recession. In addition, 15.9% of mild, 14.9% of moderate and 9.8% of deep demonstrated continued recession with time. Increasing numbers of sites with continuing recession were evident with time, particularly after 3 years post-operatively and this tended to be worse in the deep defects than in the shallower defects.

Table III illustrates the results of treatment in terms of pocket depths over the period of study. It can be seen that the vast majority of pockets in the moderate and severe groups improved as a result of surgery, while a sizable proportion of those in the mild groups also benefitted. Half of the pockets in the mild group, however, showed no change (stable) initially although fewer pockets remained in this group over the long term. 43% of the mild sites and 86% of the moderate sites showed continuing improvement (better) six months post-operatively. A slight reduction in the pocketing of these improved sites occurred at 1 year post-operatively, after which these sites maintained their improvements. By contrast all but 1 (0.8%) of the severe sites showed improvement with treatment which was maintained throughout the period of study. About 50% of the mild group and 13% of the moderate group remained stable in the first six months but there was a tendency for change to take place with time as few sites remained in this group. Thus, a significant percentage of sites particularly in the mild group had a tendency to break down with time due to recurrent pocketing. In addition, the severe pockets benefited the most from surgical treatment in terms of long term stability, and the pockets in the mild group derived least benefit from surgery.

Table IV shows the results of treatment as assessed by the overall loss or gain of attachment. It can be seen from this table that greatest gain in attachment was achieved in the moderate (63%) and severe groups (86%), which improved as a result of surgery and remained better throughout the period of study. By contrast only a few defects in the mild group (19%) showed an overall gain in attachment. 41% of the sites in this group remained unaltered after treatment and 40% showed loss of attachment as a result of treatment which tended to increase with time. Thus, the group which demonstrated the least benefit was clearly those sites with initially mild defects of which only 19% demonstrated a gain of attachment as a result of surgery with a substantial percentage actually showing a net loss of attachment.

TABLE II
Results of treatment - To show % changes in recession after surgery relative to baseline.

	Time period	Mild	Moderate	Severe
Better	6 months	3.9%	3.7%	0.8%
	1 year	4.6%	2.9%	1.5%
	2 years	4.6%	4.1%	0.8%
	3 years	3.9%	2.1%	0
	4 years	0.7%	1.7%	0.8%
Stable	6 months	40.1%	43.2%	26.3%
	1 year	48.0%	49.0%	27.8%
	2 years	40.8%	39.8%	33.1%
	3 years	36.2%	38.6%	23.3%
	4 years	27.6%	30.3%	16.5%
Worse	6 months	55.9%	53.1%	72.9%
	1 year	47.4%	48.1%	70.7%
	2 years	54.6%	56.0%	66.2%
	3 years	58.6%	59.3%	76.7%
	4 years	71.7%	68.0%	82.7%
Total sites		152	241	133

TABLE III
Results of treatment - To show the % of pocket depths changing after surgery relative to baseline.

	Time period	Mild	Moderate	Severe
Better	6 months	43.4%	86.3%	99.2%
	1 year	35.2%	81.3%	99.2%
	2 years	46.7%	83.4%	96.7%
	3 years	45.4%	86.7%	97.7%
	4 years	40.8%	83.4%	97.7%
Stable	6 months	50.0%	12.9%	0.8%
	1 year	48.0%	15.4%	0.8%
	2 years	48.0%	13.3%	0.8%
	3 years	34.9%	11.6%	2.3%
	4 years	39.9%	11.7%	1.5%
Worse	6 months	6.6%	0.8%	0%
	1 year	13.9%	3.3%	0%
	2 years	10.5%	3.3%	2.3%
	3 years	19.7%	1.7%	0%
	4 years	15.1%	3.7%	0.8%
Total sites		152	241	133

TABLE IV
Results of treatment - To show the % changes in attachment levels after surgery relative to baseline.

	Time period	Mild	Moderate	Severe
Better	6 months	19.1%	62.7%	86.5%
	1 year	23.7%	63.9%	86.5%
	2 years	23.7%	63.9%	84.9%
	3 years	18.4%	63.1%	84.9%
	4 years	12.9%	57.7%	78.9%
Stable	6 months	41.4%	20.3%	10.5%
	1 year	30.9%	17.8%	7.5%
	2 years	30.9%	20.3%	12.8%
	3 years	31.6%	22.4%	9.0%
	4 years	29.6%	20.7%	13.5%
Worse	6 months	39.5%	13.2%	3.0%
	1 year	45.4%	18.3%	6.0%
	2 years	45.4%	15.8%	7.5%
	3 years	50.0%	14.5%	6.8%
	4 years	57.9%	21.6%	7.5%
Total sites		152	241	133

Although some evidence of loss of attachment was evident in a small percentage of sites with time, the vast majority of the sites in the moderate and severe groups derived a net benefit from surgical treatment.

DISCUSSION OF RESULTS

Hirschfield and Wasserman (1978) have shown that tooth loss occurs after periodontal therapy in a small susceptible group of individuals. As tooth loss represents the extreme endpoint of periodontal disease, this is not representative of recurrence of periodontal destruction after treatment, which in many cases does not take into account sites of recurrent diseases in which tooth loss does not occur. Similarly other research (Socransky *et al.* 1984, Socransky & Haffajee 1985) indicates that active periodontal disease may only occur in a small number of susceptible sites. Haffajee *et al.* (1983) have suggested that mean values alone are not sufficient in the statistical analysis of periodontal disease progression. In this study, the mean values and standard deviations of the data were analysed, and it was found that healing occurred for up to 12 months after treatment, with a tendency for long term recurrence to occur with time in all three clinical parameters evaluated. There was a tendency for overall attachment loss to occur with time which may be attributable predominantly to continued recession. Attachment levels remained stable for two years, after which increasing recession was observed concomitant with loss of attachment.

Pocketing was reduced in proportion to the initial depths, with the greatest reduction occurring in the deepest pockets. This observation is in agreement with Ramfjord *et al.* (1987) who observed only minimal benefit from surgical intervention in the mild and moderate groups. The mild and moderate pockets tended to show recurrent breakdown with time, while the deeper pockets tended to show long term healing. Nevertheless, some evidence of recurrence of disease was seen in all three groups. It would therefore seem that the periodontal tissues do not remain stable after treatment. Jenkins *et al.* (1988) observed that periodontal breakdown occurs within a year of treatment in the absence of maintenance therapy. In this study, 64% of sites remained stable, while 6% improved, and 24% deteriorated. No correlation could be established between either clinical or bacteriological variables, and sites which broke down. In the study described here, patients were seen at three monthly intervals for scaling and remotivation in oral hygiene. As a result, plaque and gingivitis levels remained low throughout the period of investigation. Therefore, breakdown occurred in this study in spite of good oral hygiene and regular maintenance therapy. Recurrence of the disease process with time is not unexpected, but reduction in pocket depths after a burst of destructive activity is of interest

because it is assumed that rapid healing with closure of the pockets after surgery occurs. This may however not be the case, as some sites were undergoing healing at the same time as other sites were subjected to recurrent disease at each assessment during the study. This was particularly noticable in the mild and moderate sites which seemed to have less long term stability than the severe group of sites. This is in agreement with the random burst model proposed by Goodson *et al.* (1982) except that these active and quiescent sites seem to be present not only in the pathogenesis of periodontal disease, but also during the post-operative healing phases. In addition, to the «bursts of destruction» which have been described by these workers, «bursts of healing» may also be occurring as part of the long-term maintenance of the treated periodontal patient.

Recurrent loss of attachment has been shown to occur, particularly after three years post-operatively. However, pocketing and recession were continuously changing throughout this period, with some sites showing improvement and others showing worsening of both of these variables. Thus, the healing of the gingival tissues may be occurring as a result of the interdependence of recession and pocketing, with some improvement in one variable at the same time as worsening of the other to give apparently constant attachment levels. Thus the interplay between recession and pocketing indicates long-term remodelling of the tissues as part of a continuing healing or disease process in different sites, which has been demonstrated throughout the 4 year period of this study.

It is therefore important to appreciate that, in establishing new periodontal disease models and analysing the data in the light of these models it is not sufficient to merely observe changes in mean values supplemented by an analysis of frequency distributions as proposed by Haffajee *et al.* (1983). In addition to these parameters, it necessary to consider the interplay between clinical assessment parameters of soft tissue pocketing, gingival recession, and attachment level changes in order to fully evaluate the progression of the disease process.

Several models of the progression of periodontal disease have been proposed, which include linear progression, random and assynchronous bursts of disease activity. This study has highlighted the fact that concurrent bursts of healing as well as bursts of destruction may occur with time. These bursts of healing may be observed independently in all three of the clinical variables evaluated in this study. As one variably may show worsening at the same time that another shows improvement, it is proposed that in the evaluation of these different clinical variables, a tendency towards the establishment of a «dynamic equilibrium» exists in which remodelling of the tissues occurs continuously, showing both divergence and convergence between the different clinical variables during any period of study. This may present as either worsening, or improvement, or stability in any one of the variables evaluated, but does not give an indication of the overall remodelling process which is taking place within the tissues over a protracted period of time. Thus those studies which have reported that only a small number of sites are actively undergoing change in a given period of time, may only be indicating an imbalance in the dynamic equilibrium which is discernable clinically as change only at the extremes of the distribution of remodelling activity. Thus, studies like the ones by Goodson *et al.* (1982) and Socransky *et al.* (1984) which have indicated that less than 10% of sites may be actively changing at any one period of time do not agree with the observations of this study which indicates that as many as 40% to 60% of sites may be active at any one time. Due to the different degrees of activity between different clinical variables, a cancellation effect results in only a relatively small number of sites demonstrating clinical change.

Deriving a complete statistical model of the processes involved would be a highly complex task. However, two methods exist for simplification of the task. It is possible to estimate mean values and standard deviations for each variable, and these values may be compared using conventional parametric statistics. However, the error estimates include measurement errors, and clinical variation together with a number of potential sources of interaction, since teeth are grouped together for individual patients, so that intra- and inter-patient variation will exist. No separation of the errors into these different sources appears to be reliable, given the relatively small number of patients, the rather large variability, and the likelihood the existence of co-factors not as such recorded.

The summary of the results being a collection of mean values and associated standard deviations are presented in Table I. The reader may observe the differences as a function of time and group but should be warned that these do not permit a robust statistical analysis of trend. Given the quality of the data, it is not judged sensible to attempt to extract more information than the data will support. Thus only

statements of the form: «no change is observable», are considered reasonable to test statistically. It is not considered appropriate to infer a linear model, for example relating clinical variables as a function of time, since clinical models of progression of periodontal disease do not support such a statistical model. The second approach is to view the data from a non-parametric approach, by defining groups and estimating ranks.

Using information from the parametric statistics (mean and standard deviation) three groups (mild, moderate, and severe) and three categories (better, stable and worse) were defined. Tables II and IV are thus contingency tables, presenting the numbers of cases in each group and category as a function of time. Each of these tables indicates changes for a given time only and no attempt has been made to determine whether the sites in which change was observed were consistently changing or not. Extraction of further information about trends from these data is considered ill advised, and the raw data as such is presented to the reader to interpret.

Never the less, some tentative conclusions can be inferred from the data.

CONCLUSIONS

1) Post-operative healing continues over a period of at least 12 months after treatment, and in some sites much longer.

2) After a period of stability, there is a tendency for recurrent disease to be clinically observable particularly in the sites which had mild or moderate pocketing prior to treatment.

3) A large number of sites demonstrate recurrent «bursts of destructive activity» but in addition, a large number of sites demonstrate «bursts of healing».

4) Recurrent bursts of destruction and healing occurring in different clinical variables concurrently result in a «dynamic equilibrium» which may be misinterpreted as being indicative of stability of the tissue with no change taking place.

5) Studies indicating that only a small number of sites are active at any one time may be misleading as they do not take into account the possibility of a large number of sites remaining in a dynamic equilibrium state even though they are active.

6) Statistical modelling of the activity of periodontal disease is highly complex, and should include statistical models based not only on the use of mean values and frequency distributions, but also the interactions between the different clinical assessment variables of recession, pocketing, and change in levels of attachment with time, in order to establish the true nature of the activity which is occurring in the tissues.

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