

'periodontal charting, profitable or not' study evaluation



**ASSIGN
BUSTER**

Is ' Periodontal charting profitable or not' study consistent with CDC prevalence study?

The information provided in ' Periodontal charting, profitable and not' essay quotes the results from US CDC (Centers for Disease Control) study which reported that 47. 2% of people over the age of 30 have some form of periodontal disease (Eke et. al. 2012). The essay goes on to assert that high prevalence number indicates the value of Perio charting as it can help identify, track and treat this disease which may otherwise go unnoticed. I agree with the remarks made by essay that skipping perio charting would lead to missed opportunity for profits.

However, I do not completely agree with the rule of thumb proposed for hygiene department to perform periodontal services in about 35% of cases. I would have liked if they had instead said that hygiene department should conduct periodontal charting for nearly 100% of cases above aged 30 because of high prevalence. Doing so will equip them to monitor the progression of this disease and to take preventive treatment at the earliest. Treating periodontal disease is very important especially because it not only helps prolong the longevity of the dentition but also reduce the risk of cardiovascular disease, stroke and diabetes.

My thoughts on why Periodontal charting is not completed on a routine basis.

Periodontal charting is time consuming and tedious as proper charting requires measurements on six surfaces per tooth. This makes the appointment lengthier and in the interest of patient satisfaction may often be skipped.

<https://assignbuster.com/periodontal-charting-profitable-or-not-study-evaluation/>

The process gets more time consuming when it is single handed recording (absence of assistant) as it requires leaving the instrument after the reading, typing the numbers on the system and then repeating the same for next tooth.

Also, periodontal charting being a diagnostic examination requires ' poking gums' as per patient and therefore requires huge patient co-operation.

To save time, often clinicians do sub-optimal charting based on less than six measurements per tooth. This saves some time, however may not give the most accurate reading.

If I observe my hygienist copying over previous charting in the records.

I would have a separate discussion with my hygienist, where I would like to hear their point of view. Once I have listened to their point of view, I would like to explain some of the benefits of proper and optimal periodontal charting. I would offer my thoughts by saying that in the long run a proper periodontal charting is beneficial to the health of patient as it can improve longevity of the teeth and reduce patient's risk to cardiovascular disease, stroke and diabetes. It can therefore help in establishing trust of the patient and ensure continuous visits.

Educating patients about the benefits of such tedious examination can also lead to greater co-operation.

I would also try to facilitate speedier charting by incorporating one or more of following, as suggested in (Hu, 2018)

Voice activated charting systems

Working with a partner

Use instruments that make probing faster.

Current theories on progression

Experimental gingivitis and Burst Hypothesis study

Experimental gingivitis study was conducted on a small population involving just 12 subjects, which were all part of same university set up. The objective of study were two fold – i) to produce gingivitis in subjects with healthy gingivae by withdrawing all efforts towards oral cleaning and ii) to study the sequence of changes in the microbial flora.

The study reported that the time required in subjects to develop clinical gingivitis varied between 10 and 21 days, with the variation most likely due to individual host defense capability. Reinstitution of oral hygiene resulted in healthy gingival conditions.

Given that the study focused on a very small group, with an aim to just induce gingivitis and study floral change, no attempt was made to do any statistical analysis or suggest any model on the progression of the disease from gingivitis to periodontitis.

However some remarks on disease progression were made such as – as plaque ages, it causes alteration in local environment favoring the growth of

bacterial flora, and that the abolition of oral hygiene procedures results in rapid increase of oral debris.

The Burst hypothesis study is a review study which focused on prevailing concepts on periodontal disease progression at the time of publication. The study refutes the 'continuous destructive' disease progression model and instead shows that most of current evidence (at the time of publication) points to disease progression in 'bursts', thereby presenting alternate theories in periodontal disease progression (Socransky et. al. 1984)

The continuous destructive model prevailed for many decades and was largely the result of epidemiological studies in human population, where all results indicated that severity of disease increased by age (Scherp 1964, Russell 1967, Axelsson & Lindhe 1978, 1981, Loe et. al 1978), thereby adding credibility to the continuous model.

However contradicting results of the nature highlighted below were obtained in other studies leading to questioning the credibility of continuous model:

- Results of the longitudinal studies on a group of patients showed significant increase (2-5 mm) in probeable attachment level in 5. 7% of monitored sites (Goodson et al. 1982) within one year, and such rates of change were not consistent with the continuous destructive model.
- Approximately 50% of sites showing no loss in first three years showed attachment loss in next monitoring period, and in contrast of the sites showing some attachment loss in initial period, about 66% of them showed no attachment loss in subsequent follow up period (Lindhe et.

al. 1983). Data from animal studies also showed that disease did not progress equally (Lindhe et. al. 1975).

- Rapid destructive disease was brought under control by then unknown mechanism (Kennedy and Polson, 1973).

Another model – the *asynchronous multiple burst model* was suggested indicating disease may occur in peaks during certain periods in individual's life. The asynchronous multiple burst model differs from random burst model because it indicates that multiple sites may show breakdown within reasonably short period of time followed by prolonged periods of remission (Socransky et. al. 1984). Although this study offered alternate models, differing from continuous model, these models required further testing and experimentation.

Drawbacks of earlier models

Earlier studies have attempted to resolve the dynamic conditions of all sites via single equations. In a way they were attempting to come up with a one size fits all model, when in reality the periodontal disease progression appeared more complex . On one hand was the ' linear, continuous' model which overestimated changes, and on other hand was the ' burst' model which slightly underestimated the changes (Ralls et. al. 1986; Allen et. al. 1995). To add more complexity to this, there were observations which reported that certain sites improved while others were deteriorating, and in a cyclical manner.

Given that the periodontal data is structured on multiple levels such as the patient-level, tooth-level, site-level and repeated-level measurements,

summary statistical approaches (i. e., mean or sum scores) or the maximum value of the site-level evaluation have been applied for patient-level evaluations. However, such aggregation of site-level information causes a loss of information (Cho et. al. 2015). Traditional statistical models overlooked multilevel structures and such overgeneralization lead to type I errors (Sterne et. al. 1990) and potential misinterpretations. Therefore, they did not obtain conclusive results and do not provide useful information for clinicians.

Current theories on progression

To overcome the drawback of earlier models, multilevel modeling approach was attempted in an effort to unify the aspects of both ‘burst’ and ‘continuous’ models, as both models seemed valid at different levels and different points of time (Gilthorpe et. al. 2003).

In a recent prospective cohort study, multi-level modelling (site level, tooth level and patient level) was used to analyze 18, 834 sites distributed on 3, 139 teeth in 124 patients, and data were collected 5 times over a 24-month follow-up period (Nomura et. al. 2017). One of the reported observation from this study was that both linear and burst type progression occurred at the same time within the same patients with more than half of the teeth presenting burst-type progression sites accompanied by linear-type progression sites. Maxillary premolars and anterior teeth tended to show burst-type progression. The parameters identified in this study may guide practitioners in determining the type and extent of treatment needed and that appropriate clustering is must for proper clinical assessment of risk.

The results from this study were compelling. Of the 18, 834 sites investigated in this study, 1. 1% showed improvement, 197. 0% were stable, and 1. 9% sites progressed during the 24-month follow-up period. At the patient level, 32. 3% patients had only stable sites, 16. 9% had stable and improved sites, 24. 2% had stable and progressed sites, and 26. 7% had stable, improved and progressed sites. At the tooth level, most of the teeth (89. 2%) had only stable sites, and only 0. 4% of the teeth had both improved and progressed sites. Progressed and improved teeth were observed in only 6. 4% and 3. 4% of the patients, respectively. These results clearly show that in significant percentage of patients the progression and improvement proceeded simultaneously.

As to the determinants of progression, the tooth surfaces and the CAL at baseline were statistically significant in determining changes to CAL value. The level of progression was highly dependent on the type of tooth. Carriers of *A. actinomycetemcomitans* were at a greater risk of CAL progression. Multiple studies employing mixed effects modeling have shown that of the multiple levels, the contribution of patient-level factors was small compared with that of tooth- or site-level factors. The salivary levels of *P. gingivalis* and *A. actinomycetemcomitans* were statistically significant determinants of CAL progression, and their significance was more than that of tooth and site level factors (Nomura et. al. 2017).

In conclusion I can say that both 'burst' and 'continuous' theories of progression hold their validity but they manifest at different levels (site, tooth, patient) and can occur at the same time in a patient but in different teeth, or at same tooth in a patient but at different times. Also, as per recent <https://assignbuster.com/periodontal-charting-profitable-or-not-study-evaluation/>

studies it has been found that progression of periodontal diseases happen in an acute manner and these episodes are recurrent.

The age of some of the articles actually do not impact their relevance, as the observations noted in each article would still stand if experiments were carried out today under similar conditions. The older articles were an attempt to model the disease progression, and current theories uphold the results from previous studies but goes on to show that multiple models are in play at same time and at different regions across the patient's teeth.

[Management of periodontal disease]

As per the current studies, host response plays a major role in management of periodontal disease depending on the resolution phase of acute inflammation, which involves the lipo-oxygenase and cyclo-oxygenase pathways in restoring homeostasis.

Resolution of inflammation is an active process rather than a passive decay of pro-inflammatory signals. Activation of pro resolving molecules neutralizes and eliminates inflammatory leukocytes and thereby prevent pathology (Van Dyke, 2000; Serhan CN, 2005). Although resolvins and protectins stimulate anti-inflammatory and proresolving pathways similar to the lipoxins, their mechanisms of action differ, with binding occurring to distinct sites on inflammatory cells (Serhan et. al. 2008) Such proresolving functions result in a shift in inflammatory response to a shorter resolution interval, which may help to prevent progression (Hasturk et. al. 2007)

[Current thinking on the prevalence of periodontal disease]

<https://assignbuster.com/periodontal-charting-profitable-or-not-study-evaluation/>

As per a large study conducted by U. S. Centers for Disease Control and Prevention (CDC), 47. 2% of people over the age of 30 have some form of periodontal disease. Periodontal disease afflicts over 70% of those over 65 and is a major contributor to tooth loss. This condition is more common in men (56. 4%) than women (38. 4%), those living below the federal poverty level (65. 4%), those with less than a high school education (66. 9%), and current smokers (64. 2%) (Eke et. al., 2012).

The world Health Organization (WHO) has maintained global oral health data bank using community periodontal index (CPI) (World Health Organization, 2005).

The proportion of adolescents with calculus deposits ranged from 35% to 70% in developing countries while it ranged from 4% to 34% in developed nations. Compared with developed countries, developing nations have higher prevalence of calculus and bleeding on probing among adolescents. The risk of periodontal disease increases with the advancing age that is why the high prevalence of periodontal disease is seen among elderly population.

Periodontal disease is the main cause of tooth extraction in adults aged ≥ 40 years. (Reich and Hiller, 1993)

Several factors increase the risk of periodontal diseases such as smoking, diabetes, poor oral hygiene, stress, heredity, crooked teeth, hormonal changes in female, defective fillings etc. These risk factors, modifiable and non-modifiable, contribute toward the clinical significance of periodontal diseases. Smoking is one of the most important risk factors for periodontitis, and the reduction in periodontal disease prevalence is related to the drop in

smoking rates (Bergstrom J, 2014). The smokers are 3 times more likely to have a severe form of periodontal disease than non-smokers (Johnson and Hill, 2004)

On its part, periodontal disease is likely to cause 19% increase in the risk of cardiovascular disease, and this increase in relative risk reaches to 44% among individuals aged 65 years and over. Type 2 diabetic individuals with severe form of periodontal disease have 3. 2 times greater mortality risk compared with individuals with no or mild periodontitis (Nazir, 2017). Therefore, the reduction in the incidence and prevalence of periodontal disease can reduce its associated systemic diseases and can also minimize their financial impact on the health-care systems. (Nazir, 2017)

It is therefore in the best interest of community and health practitioners that they all get familiar with perio-systemic link and risk factors, and on part of dental practitioners to ensure proper due diligence is exercised for specialized periodontal care.

- Allen KM, Hausmann E. Model fit and measurement outcome in attachment measurements: a simulation study. J Periodontal Res. 1995; 30, 15-22. PMID: 7722843
- Axelsson, P, & Lindhe, J, (1978) Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. Journal of Clittical Periodontology 5, 133-151,
- Axelsson, P, & Lindhe, J, (1981) The significance of maintenance care in the treatment of periodontal disease. Journat of Clinical Periodotitology 8, 281- 294,

- Bergstrom J. (2014) Smoking rate and periodontal disease prevalence: 40-year trends in Sweden, 1970-2010. *J Clin Periodontol.* 41: 952–7.
- Cho Y, Kim HY. Analysis of periodontal data using mixed effects models. *J. Periodontal Implant Sci.* 2015; 45, 2–7. PMID: 25722920
<https://doi.org/10.5051/jpis.2015.45.1.2>
- Eke PI, Dye B, Wei L, Thornton-Evans G, Genco R. Prevalence of periodontitis in adults in the united states: 2009 and 2010. *J Dent Res.* 2012; 91(10): 914-920. doi: <https://doi.org/10.1177/0022034512457373>
- Gilthorpe MS, Zamzuri AT, Griffiths GS, Maddick IH, Eaton KA, Johnson NW. Unification of the “burst” and “linear” theories of periodontal disease progression: a multilevel manifestation of the same phenomenon. *J Dent Res.* 2003; 82, 200–205. PMID: 12598549
<https://doi.org/10.1177/154405910308200310>
- Goodson, J, M,, Tanner, A, C, R,, Haffajee, A, D,, Sornberger, G. C, & Socransky, S, S, (1982) Patterns of progression and regression of advanced destructive periodontal disease. *Journal of Clinical Periodontology* 9, 472-481.
- Hasturk H, Kantarci A, Goguet-Surmenian E, et al. Resolvin E1 regulates inflammation at the cellular and tissue level and restores tissue homeostasis in vivo. *J Immunol.* 2007; 179: 7021–7029.
- Hu-Friedy (2018). PERIODONTAL CHARTING: PROFITABLE OR NOT. Retrieved from <https://www.hu-friedy.com/blog/perio-charting>
- Johnson GK, Hill M. (2004) Cigarette smoking and the periodontal patient. *J Periodontol.* 75: 196–209.

- Kennedy, J, E! & Poison, A. M, (1973) Experimental marginal periodontitis in squirrel monkeys. *Journal of Periodontology* 44, 140-144.
- Loe, H,, Anerud, A,, Boysen, H, & Smith, M, (1978) The natural history of periodontai disease in man. The rate of periodontai destruction before 40 years of age, *Journal of Periodontoigy* 49, 607-620,
- Lindhe, J,. Hamp, S,-E, & Loe, H, (1975) Plaque induced periodontal disease in beagle dogs. A 4-year clinical, roentgenographical and histometrical study, *Journai of Periodontai Research* 10, 243-255
- Lindhe J, Haffajee AD, Socransky SS (1983) Progression of periodontal disease in adult subjects in the absence of periodontal therapy. *J Clin Periodontol.*; 10, 433-442
- Nomura, Yoshiaki & Morozumi, Toshiya & Nakagawa, Taneaki & Sugaya, Tsutomu & Kawanami, Masamitsu & Suzuki, Fumihiko & Takahashi, Keiso & Abe, Yuzo & Sato, Soh & Makino-Oi, Asako & Saito, Atsushi & Takano, Satomi & Minabe, Masato & Nakayama, Yohei & Ogata, Yorimasa & Kobayashi, Hiroaki & Izumi, Yuichi & Sugano, Naoyuki & Ito, Koichi & Yoshie, Hiromasa. (2017). Site-level progression of periodontal disease during a follow-up period. *PLOS ONE*. 12. e0188670. 10. 1371/journal. pone. 0188670.
- Russell, A, L, (1967) Epidemiology of periodontai disease, *Internationai Dental Journal* 17, 282-296,
- Ralls SA, Cohen ME. Problems in identifying “ bursts” of periodontal attachment loss. *J Periodontol*. 1986; 57, 746-752. PMID: 3467060 <https://doi.org/10.1902/jop.1986.57.12.746> 50.

- Reich E, Hiller KA. (1993) Reasons for tooth extraction in the western states of Germany. Community Dent Oral Epidemiol. Dec; 21(6): 379-83.
- Scherp, H, (1964) Current concepts in periodontal research: epidemiological contributions. Journal of the American Dental Association 68, 667-675,
- Serhan CN. (2005) Novel omega-3-derived local mediators in anti-inflammation and resolution. Pharmacol Ther. 105: 7-21.
- Serhan CN, Chiang N. (2008) Endogenous pro-resolving and anti-inflammatory lipid mediators: A new pharmacologic genus. Br J Pharmacol. 153(Suppl 1): S200-S215
- Socransky SS, Haffajee AD, Goodson JM, Lindhe J. (1984) New concepts of destructive periodontal disease. J Clin Periodontol. 11: 21-32. doi: 10.1111/j.1600-051X.1984.tb01305.x.
- Sterne JA, Curtis MA, Gillett IR, Griffiths GS, Maiden MF, Wilton JM, Johnson NW. (1990) Statistical models for data from periodontal research. J Clin Periodontol. 17, 129-137. PMID: 2180989
- Van Dyke TE. (2007) Control of inflammation and periodontitis. Periodontol 2000. 45: 158-166.
- World Health Organization. WHO Global Oral Health Data. 2005. Available from: http://www.who.int/oral_health/databases/niigata/en.