Are changes in probing depth a reliable predictor of change in clinical attachment loss?

REVIEWER
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PURPOSE/QUESTION
The authors sought to examine the utility of using changes in probing depth (PD) for predicting changes in clinical attachment loss (CAL) in clinical trials

SOURCE OF FUNDING
Not provided

TYPE OF STUDY/DESIGN
Secondary analysis of data from clinical trials

LEVEL OF EVIDENCE
Level 2: limited-quality patient-oriented evidence

STRENGTH OF RECOMMENDATION GRADE
Not applicable

SUMMARY
Subjects
Data from 363 adults with multiple teeth and with greater or equal to 5 mm probing depths (PD) that bled on probing were pooled from across five clinical trials of nonsurgical periodontal therapy. Baseline and 12-month follow-up data were available for 314 subjects, providing data for 27,718 tooth sites. The study subjects had a mean age of 47.8 years and 53.2% were male; 33.9% were current smokers.

Key Exposure/Study Factor
The primary study factors were changes in PD and CAL over 12 months of follow-up.

Main Outcome Measure
The main outcome was the performance correlations between measured changes in PD and CAL in the same direction from baseline to the final 12-month follow-up. Changes in PD and CAL were assessed at a threshold of ≥3 mm and ≥2 mm, respectively. The utility of measured changes in PD in predicting measured change in CAL after 12 months was assessed by comparing sensitivity, specificity, and positive and negative predictive values.

Main Results
Overall, the results showed low agreement between changes in PD and changes in CAL. In sites with PD ≥3 mm at baseline, only 18% measured a concurrent change of ≥2 mm CAL and ≥2 mm PD (sensitivity of 18%). Sensitivity was higher for sites with >3 mm PD at baseline. About 70% of sites with a measured PD change of 2 mm also had similar measured CAL change in the same direction. Sites without changes in CAL were unlikely to show a measured change in PD. Overall, these results demonstrate that in nonsurgical clinical trials, changes in PD may not be a reliable and sensitive measure for changes in periodontal status over a 12-month period, especially for sites with ≥3 mm PD at baseline.

COMMENTS
The manuscript cautions us on the common practice of using PD to ascertain changes in periodontitis in clinical trials. Notably, direct correlations between PD and CAL can be undermined by a number of clinical factors, including gingival recession or enlargement. In addition, progressive CAL can occur without concurrent deepening of PD. CAL, which is the typical clinical manifestation of periodontitis, should be the primary outcome measure for detecting changes in periodontitis in clinical trials. This is supported by recommendations from the Task Force on Design and Analysis in Oral Health Research, which recommends measures of CAL or alveolar bone support as the standard primary outcome measure in nonsurgical intervention trials.
The result of this study confirms poor concordance between changes in PD and concurrent changes in CAL in nonsurgical intervention trials. A major strength of this study was the large number of subjects, and analyses of pooled teeth and site level data. Overall, the analysis demonstrated that measured PD changes over 12 months were only moderately correlated with measured changes in CAL. At the person level, associations between mean changes in PD and CAL varied inconsistently by site location and baseline PD. Similarly, after treatment, measured associations between PD and CAL varied substantially by tooth type, tooth site, and initial disease severity. However, some relationship was determined between changes in PD and CAL at sites with the deepest PD at baseline, which are sites that are most likely to benefit from the intervention. Sites with no change in PD reliably predicted the absence of a similar change in CAL (high specificity).

It is noteworthy that this study was based on secondary analysis of data pooled from a mix of five nonsurgical periodontal interventions clinical trials (first reported in 1995 and 1998, and one unpublished study) based on different study designs and interventions that range from the use of scaling and root planing to inclusion of locally delivered antimicrobial and anti-inflammatory treatments. It is conceivable that these various treatment modalities will have different impacts on gingival inflammation and gingival enlargement, which can have an impact on measures from the gingival margin (GM) to the cementoenamel junction (CEJ). Similarly, the longitudinal relationships between PD and CAL may progress differently in placebo controls relative to the intervention within (e.g., split quadrant design) and between subjects. Thus, pooling data from intervention and control sites might skew the true effects of the intervention and their subsequent effects on the relationships between PD and CAL. Also, the number of sites and teeth examined per subject per intervention varied considerably and thus may not contribute equally to the analyses. Finally, the analysis was based on changes in PD or CAL defined by a threshold of ≥3 mm or greater and ≥2 mm based on statistical reproducibility criteria and not on clinical criteria.

Overall, this study confirms that in nonsurgical clinical trials, changes in PD may not be a reliable and sensitive measure for changes in periodontal status over a 12-month period. Future studies may benefit from regression modeling of continuous measures of PD and CAL and controlling for important potential confounders of the relationship including clustering of sites within persons.

Disclaimer: The findings and conclusions in this report is that of the author and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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