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Analysis of periodontal risk profiles in adults with or without a history of myocardial infarction

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Abstract

Background: An association between periodontitis and cardiovascular diseases has been suggested.

Aims: To study whether a combination of clinical variables in a functional risk diagram enhanced the ability to differentiate between subjects with or without an immediate history of acute myocardial infarction (AMI).

Material and Methods: A functional periodontal pentagon risk diagram (PPRD) with five periodontal risk vectors was created. The surface of individual PPRDs was calculated using data from 88 subjects with recent AMI and 80 matched control subjects with no history of AMI.

Results: Age, gender, number of remaining teeth (mean value: 21.1 versus 21.6 teeth), smoking status, and pocket probing depth (PPD) distribution did not differ by group. Gingival recession was greater in control subjects (mean difference: 5.7, SD: ± 1.9, $p < 0.01$, 95% CI: 1.8–9.6). Bone loss $\geqslant 4.0$ mm was at all levels studied was significantly greater in subjects with AMI and bone loss $\geqslant 50\%$ ($\geqslant 4$ mm) was the best individual predictor of AMI ($\beta = 2.99$, $p < 0.000$, 95% CI: 7.5–53.4). Only PPRD scores were associated with AMI status when factors not included in the PPRD were studied ($\beta = 22.1$, SE: 5.9, $p < 0.0001$, 95% CI: 10.3–33.7). The best association between AMI status and study variables was the combination of $\geqslant 4$ mm of bone loss $\geqslant 50\%$, proportion of bleeding on probing (%BOP), %PPDs $\geqslant 6$ mm, and tooth loss (Nagelkirke $r^2 = 0.46$).

Conclusions: The combination of five periodontal parameters in a PPRD added predictive value, suggesting that comprehensive data should be used in studies of associations between periodontitis and heart diseases. Radiographic evidence of bone loss was the best individual parameter.

Key words: acute myocardial infarction; heart disease; periodontitis; radiograph; risk assessment; smoking

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Analysis of epidemiological data has yielded conflicting interpretations about the associations between chronic periodontitis (CP) and cardiovascular diseases (CVDs) (Mattila et al. 1989, 1993, 1995, 2000, DeStefano et al. 1993, Paunio et al. 1993, Beck et al. 1996, Joshipura et al. 1996, Loesche et al. 1998, Arbes et al. 1999, Emingil et al. 2000, Hujoel et al. 2000, 2001, 2002, Wu et al. 2000, Howell et al. 2001, Jansson et al. 2001, Katz et al. 2001, Buhlin et al. 2002, Muller 2002, Persson et al. 2002). A screening of the National Library of Medicine (PubMed) revealed that within the last 10 years, there are approximately 30 review articles and six original cross-sectional studies. There are also approximately three epidemiological studies providing data used in several publications. These studies were not specifically designed for the purpose of assessing the association between CVD and CP. This has added ambiguity to the literature. Considering the overall health significance of a potential association between having periodontitis and being at risk for CVDs, the research data for conclusions are marginal at best and require additional studies.

Unfortunately, studies on CP and CVD have used different criteria for the identification of both periodontal and cardiovascular status. Thus, various periodontal indices such as the Community Periodontal Index for Treatment Needs (CPITN) (Katz et al. 2001) or the Russell index (De Stefano et al. 1993, Hujoel et al. 2000, 2001, 2002) have been used. Other studies have included loss of attachment at various percentage

of sites (Arbes et al. 1999) or probing depths at various cutoff levels (Emingil et al. 2000, Matilla et al. 2000). Bone height and clinical probing depth values to identify subjects with or without periodontitis have been used in some studies (i.e. Matilla et al. 1993, Beck et al. 1996, Jansson et al. 2001, Persson et al. 2002). Likewise, significant differences in the criteria to define CVDs have been used in the above-cited studies.

Single clinical periodontal outcome measures are poor predictors of progression of periodontitis (Hujoel et al. 1997, Badersten et al. 1990). A low proportion of teeth with bleeding on probing (BOP) identified in recall programs suggest low risk for progression of CP (Lang et al. 1990, Joss et al. 1994). Other studies have indicated that a multi-level analysis is needed to predict the progression of periodontitis (Axtélius et al. 1999).

Cigarette smoking has in several studies been identified as one of many factors associated with an increased risk for periodontitis (i.e. Krall et al. 1999, Bergström et al. 2000, Bergström & Boström 2001). The association between smoking and periodontitis appears to be dose dependent (Haber et al. 1993). Cigarette smoking is also a significant contributory risk factor in heart disease (Schnohr et al. 2002, Tilling et al. 2002). Thus, both heart disease and periodontitis have smoking as a shared contributory risk factor.

Studies have shown that in the absence of compliance in a recall program, the risk for tooth loss increases (Kocher et al. 2000). Although one study has suggested that tooth loss cannot be well predicted by assessment of clinical attachment loss (Hujoel et al. 1997), other studies have identified that clinical data can be useful in predicting future tooth loss, especially in older subjects (Warren et al. 2002).

Methods for the assessment of alveolar bone height using either direct measurement with millimeter-graded rulers or more elaborate projection methods including the employment of digital imaging and computer software programs have been commonly used in periodontal research (Papapanou et al. 1988, Papapanou & Lindhe 1992, Brägger et al. 1994, Fourmousis et al. 1998, Persson et al. 1998).

The purpose of the present study was to assess the sensitivity of individual periodontal parameters or combinations thereof and the ability to discriminate patients with heart disease.

Materials and Methods

The study protocol was approved by the Institutional Review Board at the University of Lund, Sweden. All participating subjects in the study had signed informed consent. A group of 88 consecutive subjects who had been admitted to the Central Hospital, Kristianstad and with a diagnosis of acute myocardial infarction (AMI) defined by a cardiologist were enrolled.

Once the subjects were stabilized and released from the hospital, they received a thorough periodontal examination comprising a full-mouth series of dental radiographs and a comprehensive oral examination with assessments of the presence of plaque, BOP, and pocket probing depths (PPDs) (at four surfaces per tooth (mesi-buccal, mid-buccal, disto-buccal and mid-lingual). The proportional distributions for the presence of plaque, BOP, probing depth <4.0 mm, 4.0–5.0 mm, $\geqslant 6$ mm were calculated and used as subject-based data for the analysis. The number of teeth with visible signs of gingival recession, and the number of remaining teeth were also accounted for. A control group of 80, age-, gender-, and social group-matched control subjects without a history of AMI also consented to participate in the study and received the same comprehensive oral and medical examination as the AMI subjects.

The appropriate number of intra-oral radiographs for each subject was taken using Kodak ekta-speed film (Eastman Kodak company, Rochester, NY, USA). The films were processed using an automatic film processor (Dürr, Periomat plus, Dürr Dental GmbH & Co. KG, Bietigheim Bissingen, Germany). The films were digitized and computer processed using a custom-made image analysis software program (Brägger et al. 1994, Fourmousis et al. 1998). The distance between the cemento-enamel junction (CEJ) and the marginal bone level (BL) was assessed at the mesial and distal of each tooth. In the event the CEJ was not identifiable, a reference point such as the margin of a dental restoration was used. The alveolar BL was defined as the most apical location of the bone.

One examiner (G. R. P.) who was blinded to group belongings, trained and calibrated in reading intra-oral radiographs analyzed all the radiographic images (Persson et al. 2002). The proportional subject-based distribution of mesial and distal sites with a recorded distance $\geqslant 4.0$ mm between CEJ and BL was calculated using $>10\%$ through $>60\%$ of teeth as different cutoff levels to define periodontitis severity. The 4.0 mm distance was chosen as the critical cutoff based on previously published data (Persson et al. 1998).

The periodontal pentagon risk diagram (PPRD)

The principles of how the functional risk diagram was constructed have been presented elsewhere (Lang & Tonetti 2003).

Briefly, a pentagon where the five vectors would include information about the following: (1) the proportion of sites with BOP, (2) the number of sites with a PPD $\geqslant 6.0$ mm, (3) the number of teeth lost in the past deducted from a total of 28 teeth, (4) the proportion of mesial/distal sites with evidence of a distance CEJ to BL $\geqslant$ 4 mm, (5) smoking status with regard to pack/year. Data points for these five parameters were entered in a PC using the Excel software program (Office XP, Microsoft, Redmond, WA, USA). The surface area encompassed by scores for the different vectors was calculated. The scoring model and codes used to identify the position on each vector are presented (Table 1). The surface area that could be outlined between the five different risk scores was calculated and used as the composite PPRD risk score.

Table 1. Coding system used for the pentagon risk diagram

Vector score	Bleeding on probing (%)	Number of sites with probing depth $\geqslant 6.0$ mm	Number of teeth lost	% bone loss $\geqslant 4.0$ mm (%)	Smoking (pack/year)
0	0–4	0–1	0	0–9	0
1	5–9	2–3	1–2	10–19	1–39
2	10–16	4–5	3–4	20–29	40–89
3	17–25	6–7	5–6	30–39	90–189
4	26–35	8–9	7–8	40–49	190–364
5	36+	10+	9+	50+	365+

Statistical methods

The SPSS 10.1 software for PC was used (SPSS, Chicago, IL, USA). Descriptive statistics were used to present the study characteristics. Independent *t*-tests were performed for parametric data with normal distribution. Spearman rank correlation coefficients were studied to identify variables associated with AMI status. Binary logistic Wald regression analysis was performed to identify factors and models associated with AMI. Receiver operator characteristic (ROC) curves were also studied.

Results

The mean age of the study population was 62.7 years (SD: ±9.1). On average, they had 21.3 remaining teeth (SD: ±8.0). Only 13.1% of the subjects were women. The smoking characteristics demonstrated that 33.1% never smoked, 22.7% had quit smoking, and 44.2% were current smokers. Among smokers, the estimated average number of pack/year was 305 (SD: ±180). The distribution of PPRD scores is presented for the 168 subjects (Table 2).

Statistical analysis failed to demonstrate significant differences by age, and gender or AMI status for the number of remaining teeth (mean value: 21.1 versus 21.6 teeth) and smoking status. The proportions of sites with BOP and the presence of plaque differed significantly by AMI status. Thus, the proportion of BOP scores ($p<0.01$) and plaque scores ($p<0.02$) were higher among subjects with AMI. In contrast, the extent of gingival recession was significantly greater among control subjects (mean difference: 5.7, SD: ±1.9, $p<0.01$, 95% CI: 1.8–9.6). Statistical analysis failed to demonstrate differences by AMI status for the distribution of probing pocket depths (PPDs) ⩾6.0, 4–5, or <4 mm. Furthermore, statistical analysis failed to demonstrate differences in radiographic evidence of periapical lesions by AMI status.

Radiographic evidence of alveolar bone loss ⩾4.0 mm defined as the proportions of sites with bone loss from 10% of sites or more were at all levels significantly greater in subjects with AMI ($p<0.001$). A statistically significant difference was also found for the PPRD scores between subjects with or without a history of AMI, with significantly higher scores among subjects with AMI (mean difference: 18.5, SE: ±5.5, $p<0.001$, 95% CI: 7.5–29.3). Examples of a PPRD for a representative subject with AMI (Fig. 1) and for a control subject (Fig. 2) are presented.

The relationship between variables not included in the PPRD (subject age, plaque score, extent of gingival recession, the number of peri-apical lesions) and the PPRD was further studied by linear regression analysis using AMI status as a dependable variable. Only the PPRD scores were associated with AMI status ($\beta = 22.1$, SE: 5.9, $p<0.0001$, 95% CI: 10.3–33.7). When the variables included in the PPRD (%BOP, PPDs ⩾6.0 mm, tooth loss, bone loss, and smoking status) were studied by binary logistic Wald regression analysis and with AMI status as the dependable variable, the best-fit model included the combination of proportional bone loss at the 50% cutoff level, % BOP, and % PPDs ⩾6.0 mm (Nagelkirke $r^2 = 0.46$) (Table 3). Thus, with increasing significance the PPRD gained as a factor to identify AMI status by including information about: (1) Bone loss ⩾50%, (2) %BOP, (3) %PPDs ⩾6.0 mm, and (4) number of teeth lost. Nevertheless, ROC curve analysis demonstrated that radiographic 50% cutoff level remained the best individual predictive parameter followed by the PPRD score (Fig. 3).

Table 2. Subject-based (%) distribution of subject-based data used for the pentagon risk diagram

Vector	Bleeding on probing	Probing depth ⩾6.0 mm	Remaining teeth	Bone loss ⩾4.0 mm	Smoking (pack/year)
0	5.0	55.9	20.6	8.7	34.5
1	0.0	15.7	19.9	16.5	0.7
2	3.9	12.7	9.3	21.4	2.7
3	4.0	7.8	15.2	27.2	18.2
4	5.9	4.1	9.3	33.0	10.1
5	81.2	3.8	25.7	37.9	33.8

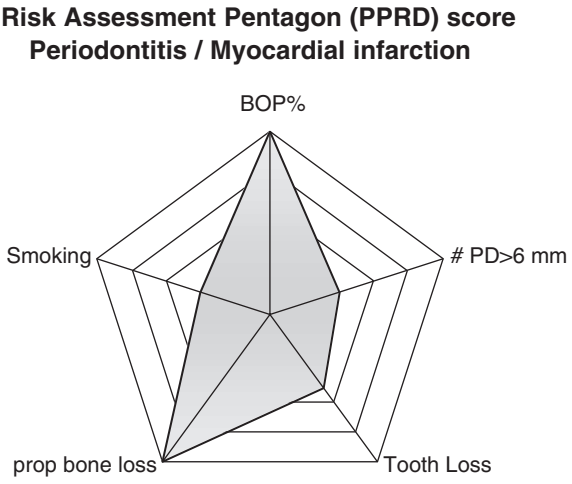


Fig. 1. Periodontal pentagon risk diagram (PPRD) for a representative subject with acute myocardial infarction.

Discussion

Studies that have demonstrated a statistically significant association between periodontal and CVDs have most commonly used PPD data in combination with evidence of alveolar bone loss to define the presence or severity of periodontitis (Matilla et al. 1993, Beck et al. 1996, Jansson et al. 2001, Persson et al. 2002).

Given the fact that most clinical parameters have poor predictability in assessing risk for periodontal disease progression, it is not surprising that the same parameters individually showed poor ability in identifying patients with AMI. However, the present study demonstrated that the combination of information on radiographic evidence of bone loss, proportions of sites with BOP, and PPDs ⩾6 mm showed good ability in identifying patients with a history of recent AMI.

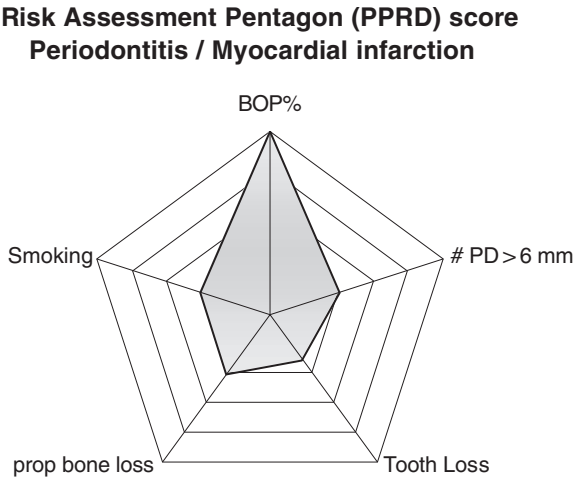


Fig. 2. Periodontal pentagon risk diagram (PPRD) for a representative subject with no history of myocardial infarction. The noticeable difference from the PPRD profile in Fig. 1 is the difference in radiographic score.

Table 3. Binary logistic Wald regression analysis – variables in the equation

Step	Variables included	β	SE	Wald	df	Sig.	Exp(*B*)	Nagelkirke r^2
step 1	bone loss $\geqslant$50%	2.347	0.413	32.217	1	0.000	10.454	0.322
	constant	− 1.431	0.336	18.171	1	0.000	0.239	
step 2	bone loss $\geqslant$50%	2.767	0.464	35.536	1	0.000	15.906	0.371
	tooth loss	0.070	0.027	6.562	1	0.010	1.072	
	constant	− 2.884	0.672	18.447	1	0.000	0.056	
step 3	bone loss $\geqslant$50%	3.057	0.500	37.435	1	0.000	21.273	0.406
	tooth loss	0.079	0.028	7.833	1	0.005	1.082	
	PD $\geqslant$6.0 mm	− 0.096	0.044	4.828	1	0.028	0.908	
	constant	− 2.956	0.689	18.430	1	0.000	0.052	
step 4	bone loss $\geqslant$50%	2.995	0.501	35.781	1	0.000	19.994	0.459
	BOP	.026	0.010	7.239	1	0.007	1.026	
	PD $\geqslant$6.0 mm	− 0.128	0.048	7.017	1	0.008	0.880	
	tooth loss	0.073	0.030	5.891	1	0.015	1.076	
	constant	− 4.076	0.853	22.811	1	0.000	0.017	

Variable(s) entered in (a) step 1: PAROD50; (b) step 2: LOSS; (c) step 3: PD6PLUS; (d) step 4: BOP.
BOP: bleeding on probing; PD: probing depth.

In the present study, it became obvious that the number of remaining teeth did not differ by AMI status while other studies found an association between additional tooth loss and heart disease (Paunio et al. 1993, Joshipura et al. 1996, Loesche et al. 1998). Tooth loss is a very difficult parameter to consider because many reasons exist for extraction of teeth including factors such as: (1) irrational to treat, (2) periodontitis, (3) caries, (4) endodontic complications, (6) accidents, and (7) costs of care. The radiographic data from the present study suggested that many subjects regardless of whether they were in the AMI or control group had one or more teeth that had either received endodontic treatment or had radiographic evidence of severe periapical lesions.

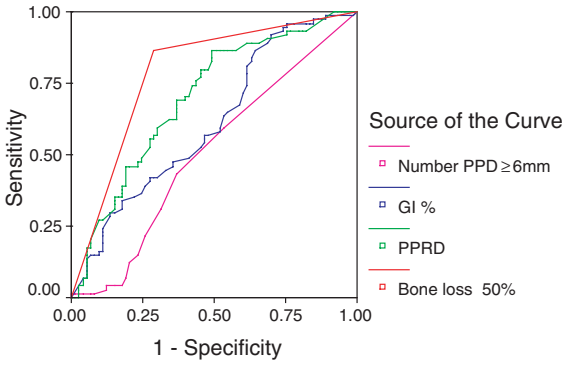
Our findings that subjects with AMI had more alveolar bone loss were consistent with others also using evaluation of radiographs (Beck et al. 1996, Persson et al. 2002). Reports using NHANES I (Hujoel et al. 2000, 2001, 2002) could not consider radiographic evidence of bone loss, which may explain why a significant association between CVD and CP could not be demonstrated. Furthermore, the clinical threshold values used for the analysis of the NHANES I data were most likely not discretionary for periodontitis (Genco et al. 2001). In fact, our results suggest that subject-based clinical information about proportional PPD provided no additional benefit in the differentiation between subjects with AMI or not.

The functional risk diagram used specific cutoff values for maximum scores beyond which the PPRD would not consider the condition. For example, proportional BOP at the 60% level would not have been recorded as worse than 36% BOP. This may be the reason why the PPRD did not demonstrate exclusively better predictive values than bone loss $\geqslant$4 mm at the 50% level. Additional work is needed to define the higher cutoff values for some of the vectors in the functional risk diagram.

Subjects with AMI had a higher proportion of BOP and plaque, but not higher proportions of PPDs $\geqslant$ 6.0 mm. Neither had they lost more teeth. They did have significantly more evidence of alveolar bone loss. This suggests that specific risk factors are involved in subjects with AMI and periodontitis. *Streptococci* present in supra-gingival plaque may be associated with both gingivitis and CVD (Herzberg & Meyer 1996, Meyer et al. 1998). It is also important to recognize that one of the key pathogens associated with CVD, *Chlamydia pneumonia*, carries heat shock protein 60 on its cell surface, which is considered as a factor that may trigger a host hyper-inflammatory response that may lead to arteriosclerosis (Huittinen et al. 2002). *Porphyromonas gingivalis*, generally associated with periodontitis, also carries heat shock protein 60 and that is expressed in serum from subjects with periodontitis (Yamazaki et al. 2002). Therefore, cross-reactivity between these pathogens may exist.

The reason why subjects with AMI had higher proportions of BOP and plaque might also be that they had recently experienced a serious medical condition. Furthermore, they may not have maintained their daily oral hygiene routines. It is also possible that their new medications such as anticoagulant drugs could have resulted in increased gingival bleeding during the periodontal examination process. Thus, the assessments of gingival inflammation and the presence of plaque may not have been representative of their overall long-time bleeding and plaque scores.

Significant oral treatment needs were found among many of the subjects and regardless of AMI status. The findings from the radiographic assessment suggested that a rather extensive and most



Area Under the Curve

Test Result Variable(s)	Area	Std. Error [a]	Asymptotic Sig. [b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Bone loss ≥ 50%	.789	.039	.000	.712	.865
PPRD	.699	.043	.000	.614	.784
GI %	.613	.046	.018	.522	.704
PPD ≥ 6 mm	.505	.048	.909	.411	.600

a. Under the nonparametric assumption
b. Null hypothesis: true area = 0.5

Fig. 3. Receiver operator characteristic curve in relation to acute myocardial infarction for the bone height loss estimation at 50% or more, the periodontal pentagon risk diagram (PPRD) score, proportion of bleeding on probing (%BOP), and the proportion of pocket probing depth ⩾6.0 mm. The inserted table provides information about curve relationships.

likely long-lasting progression of periodontitis was behind the subjects who experienced AMI. What the current data might suggest is that subjects who have clear evidence of alveolar bone loss, a history of smoking, poor oral hygiene, extensive BOP, and with minor or no gingival recession would most likely be at risk for a future AMI. Such subjects should expediently be referred for a cardiology consult.

Subjects at risk may benefit from an active smoking cessation program and effective periodontal preventive measures such as those described by Axelsson & Lindhe (1981), which could provide cost-effective treatment to prevent AMI. However, additional studies are required to further explore the explanatory mechanism of the disease process before intervention programs can be recommended.

In conclusion, the present study demonstrated that subject-based data on the proportion of BOP and plaque were significantly higher in subjects with AMI. However, the primary explanatory variable for the association between AMI and CP was the presence of proportional alveolar bone loss ⩾4 mm.

The combination of five periodontal parameters in a PPRD added predictive value, suggesting that comprehensive data should be used in studies of associations between periodontitis and heart diseases. Radiographic evidence of bone loss was the best individual parameter.

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References

Arbes, S. J. Jr., Slade, G. D. & Beck, J. D. (1999) Association between extent of periodontal attachment loss and self-reported history of heart attack: an analysis of NHANES III data. *Journal of Dental Research* **78**, 1777–1782.

Axelsson, P. & Lindhe, J. (1981) The significance of maintenance care in the treatment of periodontal disease. *Journal of Clinical Periodontology* **8**, 281–294.

Axtélius, B., Söderfeldt, B. & Attström, R. (1999) A multilevel analysis of factors affecting pocket probing depth in patients responding differently to periodontal treatment. *Journal of Clinical Periodontology* **26**, 67–76.

Badersten, A., Nilvéus, R. & Egelberg, J. (1990) Scores of plaque, bleeding, suppuration and probing depth to predict probing attachment loss. 5 years of observation following non-surgical periodontal therapy. *Journal of Clinical Periodontology* **17**, 102–107.

Beck, J., Garcia, R., Heiss, G., Vokonas, P. S. & Offenbacher, S. (1996) Periodontal disease and cardiovascular disease. *Journal of Periodontology* **67**, 1123–1137.

Bergström, J. & Boström, L. (2001) Tobacco smoking and periodontal hemorrhagic responsiveness. *Journal of Clinical Periodontology* **28**, 680–685.

Bergström, J., Eliason, S. & Dock, J. (2000) A 10 year prospective study of tobacco smoking and periodontal health. *Journal of Periodontology* **71**, 1338–1347.

Brägger, U., Schield, U. & Lang, N. P. (1994) Effect of Chlorhexidine (0.12%) rinses on periodontal tissue healing after tooth extraction. (II) Radiographic parameters. *Journal of Clinical Periodontology* **21**, 422–430.

Buhlin, K., Gustafsson, A., Håkansson, J. & Klinge, B. (2002) Oral health and cardiovascular disease in Sweden: Results of a national questionnaire survey. *Journal of Clinical Periodontology* **29**, 254–259.

DeStefano, F., Anda, R. F., Kahn, H. S., Williamson, D. F. & Russell, C. M. (1993) Dental disease and risk of coronary heart disease and mortality. *British Medical Journal* **306**, 688–691.

Emingil, G., Buduneli, E., Aliyev, A., Akilli, A. & Atilla, G. (2000) Association between periodontal disease and acute myocardial infarction. *Journal of Periodontology* **71**, 1882–1886.

Fourmousis, I., Tonetti, M. S., Mombelli, A., Lehmann, B., Lang, N. P. & Brägger, U. (1998) Evaluation of tetracycline fiber therapy with digital image analysis. *Journal of Clinical Periodontology* **25**, 737–745.

Genco, R. J., Trevinen, M., Wu, J. & Beck, J. D. (2001) Periodontal disease and coronary heart disease. *Journal of American Medical Association* **285**, 40–41.

Haber, J., Wattles, J., Crowley, M., Mandell, R., Joshipura, K. & Kent, R. (1993) Evidence for cigarette smoking as a major risk factor for periodontitis. *Journal of Periodontology* **64**, 16–23.

Herzberg, M. C. & Meyer, M. W. (1996) Effect of oral flora on platelets: possible consequences in cardiovascular diseases. *Journal of Periodontology* **67**, 1138–1142.

Howell, T. H., Ridker, P. M., Ajani, U. A., Hennekens, C. H. & Christen, W. G. (2001) Periodontal disease and risk of subsequent cardiovascular disease in U.S. male physicians. *Journal of American College of Cardiology* **37**, 445–450.

Huittinen, T., Leinonen, M., Tenkanen, L., Manttari, M., Virkkunen, H., Pitkanen, T., Wahlstrom, E., Palosuo, T., Manninen, V. & Saikku, P. (2002) Autoimmunity to human heat chock protein 60 *Chlamydia pneumoniae* infection, and inflammation in predicting coronary risk. *Arteriosclerosis Thrombosis and Vascular Biology* 431–437.

Hujoel, P. P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2000) Periodontal disease and coronary heart disease risk. *Journal of American Medical Association* **284**, 1406–1410.

Hujoel, P. P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2001) Examining the link between coronary heart disease and the elimination of chronic dental infections. *Journal of the American Dental Association* **132**, 883–889.

Hujoel, P. P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2002) Pre-existing cardiovascular disease and periodontitis: a follow-up study. *Journal of Dental Research* **81**, 186–191.

Hujoel, P. P., Leroux, B., DeRouen, T. A., Powell, L. V. & Kiyak, H. A. (1997) Evaluating the validity of probing attachment loss as a surrogate for tooth mortality in a clinical trial on the elderly. *Journal of Dental Research* **76**, 858–866.

Jansson, L., Lavstedt, S., Frithiof, L. & Theobald, H. (2001) Relationship between oral health and mortality in cardiovascular diseases. *Journal of Clinical Periodontology* **28**, 762–768.

Joshipura, K. J., Rimm, E. B., Douglass, C. W., Trichopoulos, D., Ascherio, A. & Willett, W. C. (1996) Poor oral health and coronary heart disease. *Journal of Dental Research* **75**, 1631–1636.

Joss, A., Adler, R. & Lang, N. P. (1994) Bleeding on probing. A parameter for monitoring periodontal conditions in clinical practice. *Journal of Clinical Periodontology* **21**, 402–408.

Katz, J., Chaushu, G. & Sharabi, Y. (2001) On the association between hypercholesterolemia, cardiovascular disease and severe periodontal disease. *Journal of Clinical Periodontology* **28**, 865–868.

Kocher, T., Konig, J., Dzierzon, U., Sawaf, H. & Plagmann, H. C. (2000) Disease progression in periodontally treated and untreated patients -a retrospective study. *Journal of Clinical Periodontology* **27**, 866–872.

Krall, E. A., Garvey, A. J. & Garcia, R. I. (1999) Alveolar bone loss and tooth loss in male cigar and pipe smokers. *Journal of the American Dental Association* **130**, 57–64.

Lang, N. P., Adler, R., Joss, A. & Nyman, S. (1990) Absence of bleeding on probing. An indicator of periodontal stability. *Journal of Clinical Periodontology* **17**, 714–721.

Lang, N. P. & Tonetti, M. S. (2003) Periodontal risk assessment (PRA). *Oral Health andPreventive Dentistry* **1**, 7–16.

Loesche, W. J., Schork, A., Terpenning, M. S., Chen, Y. M., Dominguez, B. L. & Grossman, N. (1998) Assessing the relationship between dental disease and coronary heart disease in elderly U.S. veterans. *Journal of American Dental Association* **129**, 301–311.

Mattila, K. J., Asikainen, S., Wolf, J., Jousimies-Somer, H., Valtonen, V. & Nieminen, M. (2000) Age, dental infections, and coronary heart disease. *Journal of Dental Research* **79**, 756–760.

Mattila, K. J., Nieminen, M. S., Valtonen, V. V., Rasi, V. P., Kesaniemi, Y. A., Syrjala, S. L., Jungell, P. S., Isoluoma, M., Hietaniemi, K. & Jokinen, M. J. (1989) Association between dental health and acute myocardial infarction. *British Medical Journal* **298**, 779–781.

Mattila, K. J., Valle, M. S., Nieminen, M. S., Valtonen, V. V. & Hietaniemi, K. L. (1993) Dental infections and coronary atherosclerosis. *Atherosclerosis* **103**, 205–211.

Mattila, K. J., Valtonen, V. V., Nieminen, M. & Huttunen, J. K. (1995) Dental infection and the risk of new coronary events: prospective study of patients with documented coronary artery disease. *Clinical Infectious Diseases* **20**, 588–592.

Meyer, M. W., Gong, K. & Herzberg, M. C. (1998) *Stretpococcus sanguis* induced platelet clotting in rabbits and hemodynamic and cardiopulmonary consequences. *Infection & Immunity* **66**, 5906–5914.

Muller, H. P. (2002) Does chronic periodontitis play a role in the pathogenesis of cardiovascular and cerebrovascular diseases? *Gesundheitswesen* **64**, 89–98.

Papapanou, P. N. & Lindhe, J. (1992) Preservation of probing attachment and alveolar bone levels in 2 random population samples. *Journal of Clinical Periodontology* **19**, 583–588.

Papapanou, P. N., Wennström, J. & Gröndahl, H-G. (1988) Periodontal status in relation to age and tooth type. A cross-sectional radiographic study. *Journal of Clinical Periodontology* **15**, 469–478.

Paunio, K., Impivaara, O., Tiekso, J. & Maki, J. (1993) Missing teeth and ischaemic heart disease in men aged 45–64 years. *European Heart Journal* **14**, 54–56.

Persson, R. E., Hollender, L. G. & Persson, G. R. (1998) Assessment of alveolar bone levels from intraoral radiographs in subjects between ages 15 and 94 years seeking dental care. *Journal of Clinical Periodontology* **25**, 647–654.

Persson, R. E., Hollender, L. G., Powell, L. V., MacEntee, M. I., Wyatt, C. C., Kiyak, H. A. & Persson, G. R. (2002) Assessment of periodontal conditions and systemic disease in older subjects I. Focus on cardiovascular diseases. *Journal of Clinical Periodontology* **29**, 769–802.

Schnohr, P., Jensen, J. S., Scharling, H. & Nordestgaard, B. G. (2002) Coronary heart disease risk factors ranked by importance for the individual and community. A 21 year follow-up of 12 000 men and women from the Copenhagen City Heart Study. *European Heart Journal* **23**, 620–626.

Tilling, K., Sterne, J. A. & Szklo, M. (2002) Estimating the effect of cardiovascular risk factors on all-cause mortality and incidence of coronary heart disease using G-estimation: the atherosclerosis risk in communities study. *American Journal of Epidemiology* **15**, 710–718.

Warren, J. J., Watkins, C. A., Cowen, H. J., Hand, J. S., Levy, S. M. & Kuthy, R. (2002) Tooth loss in the very old: 13–15-year incidence among elderly Iowans. *Community Dentistry and Oral Epidemiology* **30**, 29–37.

Wu, T., Trevisan, M., Genco, R. J., Dorn, J. P., Falkner, K. L. & Sempos, C. T. (2000) Periodontal disease and risk of cerebrovascular disease: the first national health and nutrition examination survey and its follow-up study. *Archives of Internal Medicine* **160**, 2749–2755.

Yamazaki, K., Ohsawa, Y., Tabeta, K., Ito, H., Ueki, K., Oda, T. & Seymour, G. J. (2002) Accumulation of human heat shock protein 60 reactive T-cells in the gingival tissues of periodontal patients. *Infection & Immunity* **70**, 2492–2451.

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