

Periodontal conditions in patients with coronary heart disease: a case–control study

Carin Starkhammar Johansson¹,
Arina Richter², Åsa Lundström¹,
Helene Thorstensson³ and
Nils Raval¹

¹Centre for Oral Rehabilitation, Linköping, Sweden; ²Department of Cardiology, University Hospital, Linköping, Sweden; ³Department of Periodontology, Institute for Postgraduate Dental Education, Jönköping, Sweden

Starkhammar Johansson C, Richter A, Lundström Å, Thorstensson H, Raval N. Periodontal conditions in patients with coronary heart disease: a case–control study. J Clin Periodontol 2008; 35: 199–205. doi: 10.1111/j.1600-051X.2007.01185.x.

Abstract

Aim: This study examined periodontal conditions in patients with coronary heart disease (CHD) and subjects with no history of CHD.

Material and Methods: Participants were 161 patients (40–75) with severe angina pectoris (diagnosed as CHD by coronary angiography) who subsequently underwent percutaneous coronary intervention and 162 control subjects with no history of CHD. Periodontal status was recorded. Bone loss was determined on radiographs. Periodontal disease experience was classified into five groups according to Hugoson & Jordan.

Results: Periodontal disease experience groups 4 and 5 were more common in the CHD group (25%) compared with the control group (8%). The mean bone level (the distance from the CEJ to the most coronal level of the alveolar bone) was 3.0 ± 1.0 mm in CHD subjects and 2.6 ± 0.8 mm in controls. CHD patients had significantly lower numbers of natural teeth, higher numbers of periodontal pockets 4–6-mm and higher bleeding on probing (%). In a stepwise regression analysis, the factor *periodontal disease experience groups 4+5* gave an odds ratio of 5.74 (2.07–15.90) for having CHD after controlling for smoking and age.

Conclusion: Severe periodontal disease expressed by several clinical and radiographic parameters was more prevalent among subjects with CHD than among controls. Analysis, the factor *periodontal disease experience groups 4+5* gave an odds ratio of 5.74 (2.07–15.90) for having CHD after controlling for smoking and age.

Key words: alveolar bone level; coronary heart disease; number of teeth; periodontal disease

Accepted for publication 15 November 2007

Atherosclerosis is the leading cause of premature death in the industrial world. Coronary atherosclerosis is the most frequent cause of coronary heart disease (CHD), and plaque disruption with superimposed thrombosis is the main cause of myocardial infarction. In recent

years, a key role for inflammation has been established, suggesting that inflammatory processes underlay all phases of atherosclerosis, from the initial formation of plaques to their progression and rupture, which lead to clinical events such as unstable angina, acute myocardial infarction, and sudden death (Ross 1999, Libby et al. 2002, Vita & Keaney 2002). Growing evidence has also linked common chronic infections to atherosclerosis and CHD and the triggering of acute coronary events (Danesh et al. 1997, Kaski & Cox 1998).

Much interest has been directed to a possible contributory role of chronic oral inflammatory diseases in athero-

genesis and CHD. Mattila et al. (1989) reported a correlation between dental infections and myocardial infarction in males. Even after adjusting for known risk factors, dental infections were significantly correlated to death in CHD (Mattila et al. 1995). Several other authors also proposed an association between periodontitis and CHD (DeStefano et al. 1993, Beck et al. 1996, Joshipura et al. 1996, Wu et al. 2000, López et al. 2002, Geerts et al. 2004, Geismar et al. 2006).

In 1996 Beck et al. presented a hypothesis suggesting that there could be an underlying inflammatory response trait that places an individual at high risk for developing periodontal disease and

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.

This study was supported by The Public Dental Service, Östergötland County, Sweden and The Public Dental Service, Jönköping County, Sweden.

atherosclerosis. The authors also pointed out that on-going periodontal infections harbour Gram-negative anaerobic microorganisms, which increase the burden of endotoxins. Lipopolysaccharides in particular are known to initiate and exacerbate atherogenesis. Periodontal pathogens such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Prevotella intermedia* have been identified in atherosclerotic plaque (Haraszthy et al. 2000, Fiehn et al. 2005).

Periodontitis and CHD share several risk factors, for example, smoking and diabetes (Thorstensson & Hugoson 1993, Grossi et al. 1994, Haffajee & Socransky 2001). It has been claimed that this might be one of the explanations for the association between the two diseases (Hujoel et al. 2002b, Ylostalo & Knuutila 2006). Both arteriosclerosis and periodontitis are complex multifactorial chronic inflammatory diseases. But the studies with different outcomes regarding a potential association between periodontitis and CHD have differed in study design, study population, and definitions of periodontal disease and cardiovascular diseases.

The aim of this study was to describe periodontal conditions in a group of patients with CHD documented by coronary angiography and to compare them with a group of matched controls with no history of CHD.

Material and Methods

Selection of subjects

The present study is prospective and has a matched case-control design. The study population was recruited from the consecutive patients referred to the Department of Cardiology, University Hospital, Linköping, Sweden, for coronary angiography from October 2000 to November 2003 because of known or suspected angina pectoris. During this period, Linköping University Hospital served all of Östergötland and Jönköping Counties concerning invasive cardiac procedures.

All patients aged ≤ 75 years who had significant coronary stenosis and who subsequently underwent percutaneous coronary intervention (PCI) were included consecutively. During the actual period, 187 patients fulfilled the inclusion criteria. Two patients refused to participate in the study when they were asked at the Department of Cardi-

ology. Two patients died in the period between the PCI and the dental examination and four were excluded due to full-mouth dentures. Seventeen patients declined to participate in the study when they were offered a dental examination. After the dental examination, another patient was excluded because of newly diagnosed diabetes mellitus. Thus, data from 161 patients in the test group were analysed.

The control group comprised 162 subjects recruited from the Swedish population register. Eight hundred subjects, 200 in each 10-year interval between 40 and 80 years of age, were randomly chosen. Thus, each test subject could be matched by age, gender, and community to several controls with no symptoms of heart disease. The first control subject on the list was asked to participate in the study, and subsequent controls were contacted consecutively. Only four refused to participate in the study.

To avoid, as much as possible, potential confounding influences on the study parameters, we chose these exclusion criteria: diabetes mellitus, rheumatoid arthritis, malignant diseases, acute infections, and concurrent medication with general glucocorticoids. Smoking was not an exclusion criterion because we wished to include a statistically sufficient number of subjects in a reasonable time period.

The study protocol was approved by the Ethics Committee of Linköping University, Linköping, Sweden, and all participants gave their written, informed consent to participate in the study.

Periodontal examination

The periodontal conditions of the test subjects were clinically examined 3–6 months after PCI. For each test subject, a matched control subject was examined. All subjects were examined clinically in a specialist clinic by two calibrated periodontists. Before the start of the study, the two periodontists examined five periodontal patients who were not involved in the study clinically to determine intra-examiner reproducibility. The results of the clinical evaluations of plaque, probing pocket depth (PPD), and bleeding on probing (BOP) for each periodontist were then compared. In only four sites was a disagreement in $PPD > 1$ mm found. The examination of the cardiac patients was not performed blinded for ethical reasons.

Periodontal conditions were scored according to Lindhe & Nyman (1975). The following clinical parameters were recorded:

Number of remaining teeth – number of teeth, excluding third molars, that remained.

Dental plaque – presence/absence of visible plaque at the gingival margin on four surfaces (buccal, lingual, mesial, and distal) of each tooth corresponding to scores 2 and 3 of the plaque index system of Silness & Loe (1964), calculated in per cent of the total number of available sites (PII%).

PPD – the distance from the gingival margin to the bottom of the probed pocket, determined using a manual periodontal probe (HuFriedy PCP 11, Chicago, IL, USA). Depth was scored to the nearest whole millimetre at four surfaces on each tooth. Pockets were recorded if they were 3 mm or deeper at buccal and lingual sites and 4 mm or deeper at mesial and distal sites according to Lindhe & Nyman (1975).

BOP – presence or absence of bleeding following probing at the bottom of the pocket. The percentage of the total number of sites that bled was recorded.

Radiographic examination

A set of full-mouth intra-oral radiographs, including bitewing projections, was taken for each subject using a standardized parallel technique (Eggen 1969). This included an individualized number of intra-oral radiographs taken with an *Eggenhällare*, according to departmental routine. The quality of the radiographs was very high, and only four proximal sites in the material were unreadable. One radiographic parameter was recorded:

Alveolar bone level – measured in millimetres along the root surface from the cemento-enamel junction (or from the most apical part of an interfering restoration) to the most coronal level at which the width of the periodontal ligament space was considered normal. Two calibrated periodontists made all the measurements blinded. Measurements were then compared, and in cases of disagreement, a consensus was reached.

Classification according to severity of the periodontal disease experience

Based on clinical and radiographic findings, all individuals were classified

according to these criteria, which are modifications of Hugoson & Jordan (1982):

Group 1. Healthy or almost-healthy gingival units, normal alveolar bone height, and ≤12 bleeding units in the molar–premolar regions.

Group 2. Gingivitis, normal alveolar bone height, and >12 bleeding gingival units in the molar–premolar regions.

Group 3. Alveolar bone loss around the majority of the teeth not exceeding 1/3 of normal bone height.

Group 4. Alveolar bone loss around the majority of the teeth ranging between 1/3 and 2/3 of normal bone height.

Group 5. Alveolar bone loss around the majority of the teeth exceeding 2/3 of normal bone height and presence of angular bony defects and/or furcation defects.

Two of the authors scored the participants independently. Classification of each subject was made blinded to whether the subject was a CHD patient or a healthy control. Results were then compared. Only minor disagreements occurred in the scoring of a few subjects, and the examiners then discussed scoring to reach a consensus.

Questionnaire variables

The participants answered a questionnaire covering a wide variety of items related to oral factors, general health, lifestyle, and social environment. These variables were explored:

Educational level – 9-year compulsory school, secondary school, or university.

Professional status – professionally active or retired.

Smoking habits – non-smoker (never smoked), former smoker, or smoker.

Snuff habits – never used snuff, former snuffer, or snuffer.

Oral hygiene measures – use of a toothbrush and interdental cleaning (dental floss, interdental toothpicks, interdental brushes) twice a day, daily, more seldom, or never.

Statistical methods

All calculations and statistical analyses were made using the SPSS® 13.0 software package (SPSS for Windows NT 4.0, SPSS Inc., Chicago, IL, USA). An experienced statistician performed all statistical analyses. To determine the number of participants in the test and control groups that would be needed

to give the study results significance, a power analysis with 80% power at a 5% significance level was performed in pre-study planning.

The Mann–Whitney test was used to compare the clinical variables between the test and control groups. The five periodontal experience disease groups were regrouped into three groups for analysis: groups 1+2 (individuals with no bone loss), group 3 (individuals with moderate periodontal disease experience), and groups 4+5 (individuals with severe periodontal disease experience). Stepwise logistic regression analyses were performed to evaluate differences between the test and control groups based on predictor variable. The level of statistical significance was set at *p* = 0.05.

Results

Table 1 lists the background characteristics of patients and controls. The test group comprised 132 men and 29 women, the control group 133 men and 29 women. Mean age in the test group was 61.0 ± 8.9 years (range 33–76) and in the control group 62.0 ± 8.8 years (range 35–78). At the time of the periodontal examination, one patient and six controls were above age 75. Sixty-nine per cent of the controls had a high educational level compared with 45% in the CHD group. Fifty-eight per cent of this group were professionally active and 42% retired while 50% of the controls still worked and 50% were retired.

Only 9% (*n* = 15) of the patients and 13% (*n* = 21) of the controls were current smokers. Eleven of the test subjects and seven controls smoked <10 cigarettes/day. In total 36% (*n* = 57) of the test subjects and 52% (*n* = 84) of the controls claimed that they had never smoked. The use of snuff was equally prevalent; 8% of each group used snuff. Years since quitting smoking differed between the groups. Forty-five patients with CHD reported that they had ceased smoking in the 10 years preceding our examination and 44 in the last 5 years. Corresponding figures for the control group were 49 and 8.

Dental habits were nearly equal in the test and control groups. Subjects in both groups (CHD: 89%, control: 88%) visited a dentist at least once a year, and around one-third (CHD: 28% and control: 36%) visited a dental hygienist once a year or more often. All subjects (CHD and control: 100%) stated that they brushed their teeth at least once a day. Many (CHD: 41%, control: 42%) performed interdental cleaning daily.

Table 2 presents periodontal conditions. Number of remaining teeth differed significantly between the groups. CHD patients had on average 22.7 ± 5.5 teeth compared with controls who had 24.6 ± 4.1 teeth (*p* = 0.002). PII% differed significantly between the groups (*p* = 0.05). BOP% was also significantly higher in test than in control individuals (*p* = 0.009).

Significantly more periodontal pockets with a 4–6-mm probing depth were found in the CHD than in the control group (*p* = 0.007). Deep pockets with a

Table 1. Number and distribution (%) of coronary heart disease patients (test) and healthy controls according to gender, education, professional status, and tobacco use

Variable	Test <i>n</i> = 161		Control <i>n</i> = 162	
	<i>n</i>	%	<i>n</i>	%
Gender				
Men	132	82	133	82
Women	29	18	29	18
Age (age+X; SD)	61 (8.9)		62 (8.8)	
Education				
9-year compulsory school	89	55	51	31
Higher	72	45	111	69
Tobacco use				
Current smokers	15	9	21	13
Former smokers	89	55	57	35
Non-smokers	57	36	84	52
Snuff users	13	8	13	8
Former snuff users	6	4	7	4
Never used snuff	142	88	142	88

Standard deviations are noted in parentheses.

probing depth of >6 mm occurred only seldom in both groups. Mean bone level calculated on an individual basis differed significantly between the groups ($p = 0.0001$), with a mean bone level of 3.0 ± 1.0 mm in the CHD group and 2.6 ± 0.8 mm in the control group.

Periodontal disease experience was more prevalent among CHD patients than in healthy controls. Figure 1 shows that 25% ($n = 40$) of the cardiac patients had severe periodontitis (groups 4+5), compared with 8% ($n = 13$) of the controls. This difference is highly significant ($p = 0.0001$). Moderately advanced periodontitis (group 3) was equally distributed in both groups. Forty per cent of the controls and 24% of the CHD patients ($p = 0.0001$) had no bone loss (groups 1+2).

Regression analysis

Table 3 presents the results of the stepwise logistic regression analysis. Because age distribution and smoking habits differed in the two groups, these differences were adjusted statistically by comparing the two groups at common values of these two variables. In a first step in the logistic regression analysis, age and smoking habits were entered into the model. In a second step, periodontal experience groups 1–5 were included. In a third step, missing teeth, and in a fourth step, periodontal pockets >3 mm were introduced into the model to explain the remaining differences between the two groups.

After adjusting for age and smoking, the risk of CHD was found to increase with age, periodontal disease experience groups 4+5, more missing teeth, and higher number of deep probing pockets (4 mm or more). The factor periodontal disease experience groups 4+5 had an odds ratio (OR) of 5.74 and a high confidence interval (CI) of 2.07–15.90.

In addition, a logistic regression analysis including only never smokers from test ($n = 56$) and control ($n = 84$) groups was performed. The OR for the factor periodontal disease experience groups 4+5 was 5.5 ($p = 0.064$).

Discussion

This study found periodontal disease to be more prevalent among patients with CHD than among healthy subjects. The test group consisted of a well-defined group of patients with CHD. All test

Table 2. Clinical parameters in the group of coronary heart disease patients (test) and the control group: number of remaining teeth, % surfaces with visible plaque at the gingival margin, bleeding on probing (BOP), number of probing pocket depths (PPD), and bone level

Variable	Test		Control		<i>p</i>
	mean SD	range	mean SD	range	
Number of teeth	22.7 (5.5)	5–28	24.6 (4.1)	8–28	0.002
Plaque (%)	46.0 (26.0)	0–100	39.0 (21.5)	0–90	0.05
BOP (%)	27.0 (19.6)	0–100	21.0 (16.3)	0–68	0.009
4–6 mm PPD	14.0 (12.4)	0–68	10.7 (10.3)	0–49	0.007
>6 mm PPD	0.6 (1.7)	0–16	0.4 (1.0)	0–6	0.328
Bone level (mm)	3.0 (1.0)	1.1–7.6	2.6 (0.8)	1.0–5.9	0.0001

Mann–Whitney test.

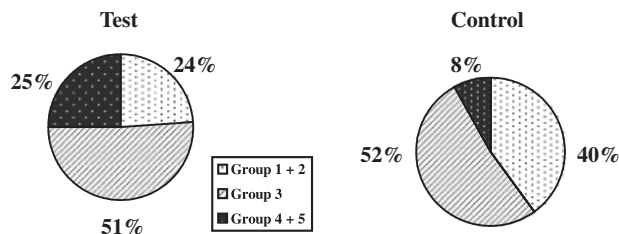


Fig. 1. Distribution of heart disease patients (test) and healthy controls according to periodontal disease experience group (Hugoson & Jordan 1982). Group 1: healthy or almost-healthy gingival units, normal alveolar bone height, and ≤ 12 bleeding units in the molar–pre-molar regions. Group 2: gingivitis, normal alveolar bone height, and >12 bleeding gingival units in the molar–pre-molar regions. Group 3: alveolar bone loss around the majority of the teeth not exceeding 1/3 of normal bone height. Group 4: alveolar bone loss around the majority of the teeth ranging between 1/3 and 2/3 of normal bone height. Group 5: alveolar bone loss around the majority of the teeth exceeding 2/3 of the normal bone height and presence of angular bony defects and/or furcation defects.

Table 3. Results of the stepwise logistic regression analysis with coronary heart disease as the dependent variable

	<i>p</i> -value	Odds ratio (95% CI)	
Smoking			Adjusted for step 1
No	0.209	1.41 (0.83–2.40)	
Yes (current and former)			
Age	0.005		
Periodontal disease experience group			Adjusted for step 2
1+2			
3	0.121	1.64 (0.88–3.08)	
4+5	0.001	5.74 (2.07–15.90)	
Missing teeth	0.004	1.10 (1.03–1.18)	Adjusted for step 3
Periodontal pockets >3 mm	0.025	1.08 (1.01–1.15)	Adjusted for step 4

CI, confidence interval.

subjects suffered from severe angina pectoris. The diagnosis was confirmed with coronary angiography, and all patients had coronary stenosis that required PCI. To reduce the risk of confounding factors, and thereby allow the relation between CHD and periodontitis to be studied, we excluded patients and controls if they had other diseases (such as diabetes and acute

infections) or medications (such as general glucocorticoids) that could influence periodontitis.

Test subjects were compared with subjects with no history of CHD. These controls had never had any symptoms of heart disease and had never visited a doctor because of cardiac problems. It would have been desirable to confirm their coronary health with a coronary

angiography, but this was not possible for ethical and economic reasons.

This prospective study had a case-control design. Thus, the findings depended on the subjects included in the study. In such a study design, findings reflect the subset of CHD defined by the cases, and one condition for validity is that controls are derived from and represent the population from which the cases derive. Generalization to the population level should be made with caution, although the number of participants in our study was based on a power analysis to reduce the risk of random differences between the groups.

Twenty-five per cent of the patients with CHD had severe alveolar bone loss compared with 8% of the controls. This agrees with other studies (Beck et al. 1996, 2001, Persson et al. 2002). In another study of women with CHD, no difference in bone level was found (Buhlin et al. 2005). This might reflect differences between genders or in study design. In our study, we considered the number of women too small to be analysed separately. Absence of alveolar bone loss, as assessed in radiographs, was significantly higher among healthy subjects than among heart patients, 40% and 24%, respectively. This is in line with the results reported by Elter et al. (2004) where subjects with high attachment loss and a high number of lost teeth had a higher risk of CHD compared with subjects with low attachment loss and a low number of lost teeth. We also found a significant correlation between the number of teeth on a subject level and CHD. Other authors, Paunio et al. (1993), Joshipura et al. (1996), and Lagervall et al. (2003) reported similar findings. The mean number of remaining teeth in both groups was high in our study; but the range was wide. This might reflect the fact that dental status in the Swedish population is rather good (Hugoson et al. 2005). We chose to exclude four edentulous subjects because we did not know why the teeth had been extracted. Periodontal health cannot be measured on an edentulous subject. Of course, there is a risk for bias in number of teeth when edentulous subjects are excluded. Our study had only two edentulous subjects, so we thought exclusion of these could have only a minor influence on results.

We found several between-group differences in the clinical variables. Patients with heart disease had higher

numbers of 4–6-mm deep pockets compared with controls. Some investigators have found that gingival inflammation per se might be a risk factor for cardiovascular disease (Bazil et al. 2002, Buhlin et al. 2002). We found a significantly higher level of BOP among the patients with heart disease compared with the controls. Plaque scores differed between the groups. The CHD group had significantly more plaque, but this difference was on a lower statistical level. This might indicate that plaque scores alone are unable to explain the difference between the groups.

We found that patients in periodontal disease experience groups 4 and 5 had a statistically significant risk (OR = 5.74) of CHD, although the CI was wide, 2.07–15.90. This was after adjusting for smoking and indicates that severe periodontitis at least doubles the risk of CHD. This level agrees with studies on cerebrovascular ischaemia (Grau et al. 1997, Wu et al. 2000). The wide CI in our study indicates that, on an individual patient level, differences in periodontal disease influence on the risk of CHD are large. This is in accordance with the results presented by Geerts et al. (2004) who reported an OR of 6.5 (95% CI: 1.8–23) between coronary artery disease and periodontitis.

The proposed association between cardiovascular disease and periodontitis has also been questioned. In a prospective cohort study, Hujoel et al. (2000) found no convincing evidence of a causal association between periodontal disease and cardiovascular disease. Moreover, the risk of CHD in edentulous people was not lower than in people with periodontitis (Hujoel et al. 2001). The hypothesis that periodontitis increases the risk of future coronary events in patients with established CHD was not confirmed in an American epidemiological study (Hujoel et al. 2002a). In middle-aged and elderly men, self-reported periodontal disease was not found to be significantly correlated with cardiovascular disease (Howell et al. 2001). Some authors have questioned whether there is a true association between periodontitis and cardiovascular disease in smokers or whether the association is coincidental (Hujoel et al. 2000). In the present study, few participants were smokers (CHD: 15, control: 21). But a large proportion of the subjects were former smokers. Number of years since quitting smoking differed between the groups.

That information might be inaccurate. How former smoking influences periodontal disease is not clearly understood. To investigate the influence of smoking, we analysed two subgroups of individuals from the test and control populations that had never smoked. In the logistic regression analysis, we found that the impact of belonging to periodontal disease experience groups 4+5 in the non-smoking subgroups was similar to that in the subgroups in the total material but on a non-significant level ($p = 0.064$). This might be another indication of an association between periodontal disease and coronary atherosclerosis. But since the size of the non-smoker groups was random, no power analysis was made. Thus, conclusions should be drawn with caution.

In our study, we found that grouping individuals in five subgroups according to Hugoson & Jordan's (1982) classifications for periodontal severity was useful. This index involves estimations of radiographic alveolar bone level and angular defects. It gives the reader a reasonably good description of periodontal status and severity of periodontal disease. Furthermore, we found that patients with CHD had more past or present periodontal problems, that is, fewer teeth, more 4–6-mm deep pockets, more plaque, and more BOP. Similar results regarding clinical variables were reported by Geerts et al. (2004). D'Aiuto et al. (2004) showed in a pilot intervention study that non-surgical periodontal therapy significantly decreased serum mediators and markers of acute inflammation. This is in accordance with results presented in several recent studies (D'Aiuto et al. 2006, Elter et al. 2006, Blum et al. 2007, Tonetti et al. 2007). Modern medical treatment of cardiovascular disease is being increasingly directed towards modification of the inflammatory component of atherosclerosis. Whether prevention and treatment of periodontitis is particularly efficient in decreasing the risk of CHD remains unanswered. Furthermore, studies elucidating these issues are necessary.

In conclusion, CHD, confirmed by recent coronary angiography, was strongly associated statistically with periodontitis (expressed by several clinical and radiographic parameters). The findings are consistent with a number of prospective studies that appear to imply that periodontitis is an important aetiological factor in CHD.

Acknowledgements

The authors thank Elisabeth Logander, research nurse, Department of Cardiology, University Hospital, Linköping, for her work in recruiting subjects and Dr. Birgit Ljungquist, Jönköping, for all statistical analyses.

References

- Bazil, A., Bissada, N. F., Nair, R. & Siegel, B. P. (2002) Periodontal assessment of patients undergoing angioplasty for treatment of coronary artery disease. *Journal of Periodontology* **73**, 631–636.
- Beck, J. D., Elter, J. R., Heiss, G., Couper, D., Mauriello, S. M. & Offenbacher, S. (2001) Relationship of periodontal disease to carotid artery intima-media wall thickness: the atherosclerosis risk in communities (ARIC) study. *Arteriosclerosis, Thrombosis, and Vascular Biology* **21**, 1816–1822.
- Beck, J. D., Garcia, R., Heiss, G., Volkonas, P. S. & Offenbacher, S. (1996) Periodontal disease and cardiovascular disease. *Journal of Periodontology* **67**, 1123–1137.
- Blum, A., Kryuger, K., Mashiach Eizenberg, M., Tatour, S., Vidger, F., Laster, Z. & Front, E. (2007) Periodontal care may improve endothelial function. *European Journal of Internal Medicine* **18**, 295–298.
- Buhlin, K., Gustafsson, A., Ahvne, S., Jansky, I., Tabrizi, F. & Klinge, B. (2005) Oral health in women with coronary heart disease. *Journal of Periodontology* **76**, 544–550.
- Buhlin, K., Gustavsson, 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.
- D'Aiuto, F., Parker, M., Brett, P. M., Ready, D. & Tonetti, M. S. (2004) Periodontitis and atherogenesis: causal association or simple coincidence? A pilot intervention study. *Journal of Clinical Periodontology* **31**, 402–411.
- D'Aiuto, F., Parkar, M., Nibali, L., Suvan, J., Lessem, J. & Tonetti, M. S. (2006) Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomised controlled clinical trial. *American Heart Journal* **151**, 977–984.
- Danesh, J., Collins, R. & Peto, R. (1997) Chronic infections and coronary heart disease: is there a link? *Lancet* **350**, 430–436.
- 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.
- Eggen, S. (1969) Standardiserad intraoral röntgenteknik. *Sveriges Tandläkarförbunds Tidning* **17**, 867–872.
- Elter, J. R., Champagne, C. M., Offenbacher, S. & Beck, J. D. (2004) Relationship of periodontal disease and tooth loss to prevalence of coronary heart disease. *Journal of Periodontology* **75**, 782–790.
- Elter, J. R., Hinderliter, A. L., Offenbacher, S., Beck, J. D., Caughey, M., Brodala, N. & Madianos, P. N. (2006) The effect of periodontal therapy on vascular endothelial function: a pilot trial. *American Heart Journal* **151**, 47.
- Fiehn, N. E., Larsen, T., Christiansen, N., Holmstrup, P. & Schroeder, T. V. (2005) Identification of periodontal pathogens in atherosclerotic vessels. *Journal of Periodontology* **75**, 731–736.
- Geerts, S. O., Legrand, V., Charpentier, J., Albert, A. & Rompen, E. H. (2004) Further evidence of the association between periodontal conditions and coronary artery disease. *Journal of Periodontology* **75**, 1274–1280.
- Geismar, K., Stoltze, K., Sigurd, B., Gytelberg, F. & Holmstrup, P. (2006) Periodontal disease and coronary heart disease. *Journal of Clinical Periodontology* **77**, 1547–1554.
- Grau, A., Buggle, F., Ziegler, C., Schwarz, W., Mouser, J., Tasman, A. J., Buhler, A., Banish, C., Belcher, H. & Hacker, W. (1997) Association between acute cerebrovascular ischemia and chronic and recurrent infection. *Stroke* **28**, 1724–1729.
- Grossi, S., Zambon, J., Ho, A., Koch, G., Dunford, R., Machtei, E., Norderyd, O. & Genco, R. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of Periodontology* **65**, 260–267.
- Haffajee, A. D. & Socransky, S. S. (2001) Relationship of cigarette smoking to attachment level profiles. *Journal of Clinical Periodontology* **28**, 257–265.
- Haraszthy, V. I., Zambon, J. J., Trevisan, M., Zeid, M. & Genco, R. J. (2000) Identification of periodontal pathogens in atheromatous plaques. *Journal of Periodontology* **71**, 1554–1560.
- Howell, T. H., Ridker, P. M., Ajani, U., Hennekens, C. H. & Christen, W. G. (2001) Periodontal disease and risk of subsequent cardiovascular disease in US male physicians. *Journal of the American College of Cardiology* **38**, 1273–1274.
- Hugoson, A. & Jordan, T. (1982) Frequency distribution of individuals aged 20–70 years according to severity of periodontal disease. *Community Dentistry and Oral Epidemiology* **10**, 187–192.
- Hugoson, A., Koch, G., Göthberg, C., Nydell Helkimo, A., Lundin, S. Å., Norderyd, O., Sjödin, B. & Sondell, K. (2005) Oral health of individuals aged 3–80 years in Jönköping, Sweden during 30 years (1973–2003). I. Review of findings on dental care habits and knowledge of oral health. *Swedish Dental Journal* **4**, 125–138.
- Hujoel, 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., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2001) Examining the link between coronary heart disease and the elimination of chronic dental infections. *Journal of American Dental Association* **132**, 883–889.
- Hujoel, P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2002a) Pre-existing cardiovascular disease and periodontitis: a follow-up study. *Journal of Dental Research* **81**, 186–191.
- Hujoel, P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2002b) Periodontitis-systemic disease association in the presence of smoking – causal or coincidental? *Periodontology* **2000** **30**, 51–60.
- Joshi, K. J., Rimm, E. B., Douglass, C. W., Trichopoulos, D., Asheiro, A. & Willet, W. C. (1996) Poor oral health and coronary heart disease. *Journal of Dental Research* **75**, 1631–1636.
- Kaski, J. C. & Cox, I. (1998) Chronic infection and atherogenesis. *European Heart Journal* **19**, 366–367.
- Lagervall, M., Jansson, L. & Bergstrom, J. (2003) Systemic disorders in patients with periodontal disease. *Journal of Clinical Periodontology* **30**, 293–299.
- Libby, P., Ridker, P. M. & Maseri, A. (2002) Inflammation and atherosclerosis. *Circulation* **105**, 1135–1143.
- Lindhe, J. & Nyman, S. (1975) The effect of plaque control and surgical pocket elimination on the establishment and maintenance of periodontal health. A longitudinal study on periodontal therapy in cases of advanced disease. *Journal of Clinical Periodontology* **2**, 67–79.
- López, R., Oyarzún, M., Naranjo, C., Cumsille, F., Ortiz, M. & Baelum, V. (2002) Coronary heart disease and periodontitis – a case-control study in Chilean adults. *Journal of Clinical Periodontology* **29**, 468–473.
- Mattila, K. J., Neiminen, M. S., Valtonen, V. V., Rasi, V. P., Keaniemi, Y. A., Syrjanen, S. L., Jungel, P. S., Isoumala, M., Hietaniemi, K. & Jokinen, M. (1989) Association between dental health and myocardial infarction. *British Medical Journal* **298**, 779–781.
- Mattila, K. J., Valtonen, V. V., Neiminen, M. S. & Huuttanen, J. K. (1995) Dental infections and the risk of new coronary events: prospective study of patients with documented coronary artery disease. *Clinical Infectious Diseases* **26**, 719–734.
- Paunio, K., Impivaara, O., Tiekso, J. & Maki, J. (1993) Missing teeth and ischemic heart disease in men aged 45–64 years. *European Heart Journal* **14** (Suppl. K), 54–56.
- Persson, R. E., Hollender, L. G., Powell, V. L., MacEntee, M., Wyatt, C. C. L., Kiyak, H. A. & Persson, G. R. (2002) Assessment of periodontal conditions and systemic disease in older subjects. II: focus on cardiovascular disease. *Journal of Clinical Periodontology* **29**, 803–810.
- Ross, R. (1999) Atherosclerosis – an inflammatory disease. *New England Journal of Medicine* **340**, 115–126.
- Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavica* **22**, 112–135.

- Thorstensson, H. & Hugoson, A. (1993) Periodontal disease experience in adult long-duration insulin-dependent diabetics. *Journal of Clinical Periodontology* **20**, 352–358.
- Tonetti, M. S., D’Aiuto, F., Nibali, L., Donald, A., Storry, C., Parkar, M., Suvan, J., Hingorani, A. D., Wallace, P. & Deanfield, J. (2007) Treatment of periodontitis and endothelial function. *New England Journal of Medicine* **1**, 911–920.
- Vita, J. A. & Keaney, J. F. Jr. (2002) Endothelial function: a barometer for cardiovascular risk? *Circulation* **106**, 640–642.
- Wu, T., Trevisan, M., Genco, R., Dorn, J. P., Falkner, K. L. & Sempos, C. T. (2000) Periodontal disease and risk of cerebrovascular disease. *Archives of Internal Medicine* **160**, 2749–2755.
- Ylostalo, P. V. & Knuutila, M. L. (2006) Confounding and effect modification; possible explanation for variation in the results on the association between oral and systemic diseases. *Journal of Clinical Periodontology* **33**, 104–108.

Address:
 Carin Starkhammar Johansson
 Centre for Oral Rehabilitation
 SE-581 85 Linköping
 Sweden
 E-mail: carin.starkhammar@lio.se

Clinical Relevance

Scientific rationale for the study: When linking periodontitis to CHD, it is important to describe the diagnostic criteria for CHD. Participants in the study group suffered from severe angina pectoris, diagnosed as

CHD by coronary angiography, and all underwent PCI.

Principal findings: The statistical analysis confirmed a strong correlation between CHD and periodontitis in clinical and radiographic parameters.

Practical implications: The results of this study strengthen results from several other studies that propose a link between periodontitis and CHD. Periodontal disease might be a risk factor to be considered in CHD patients.