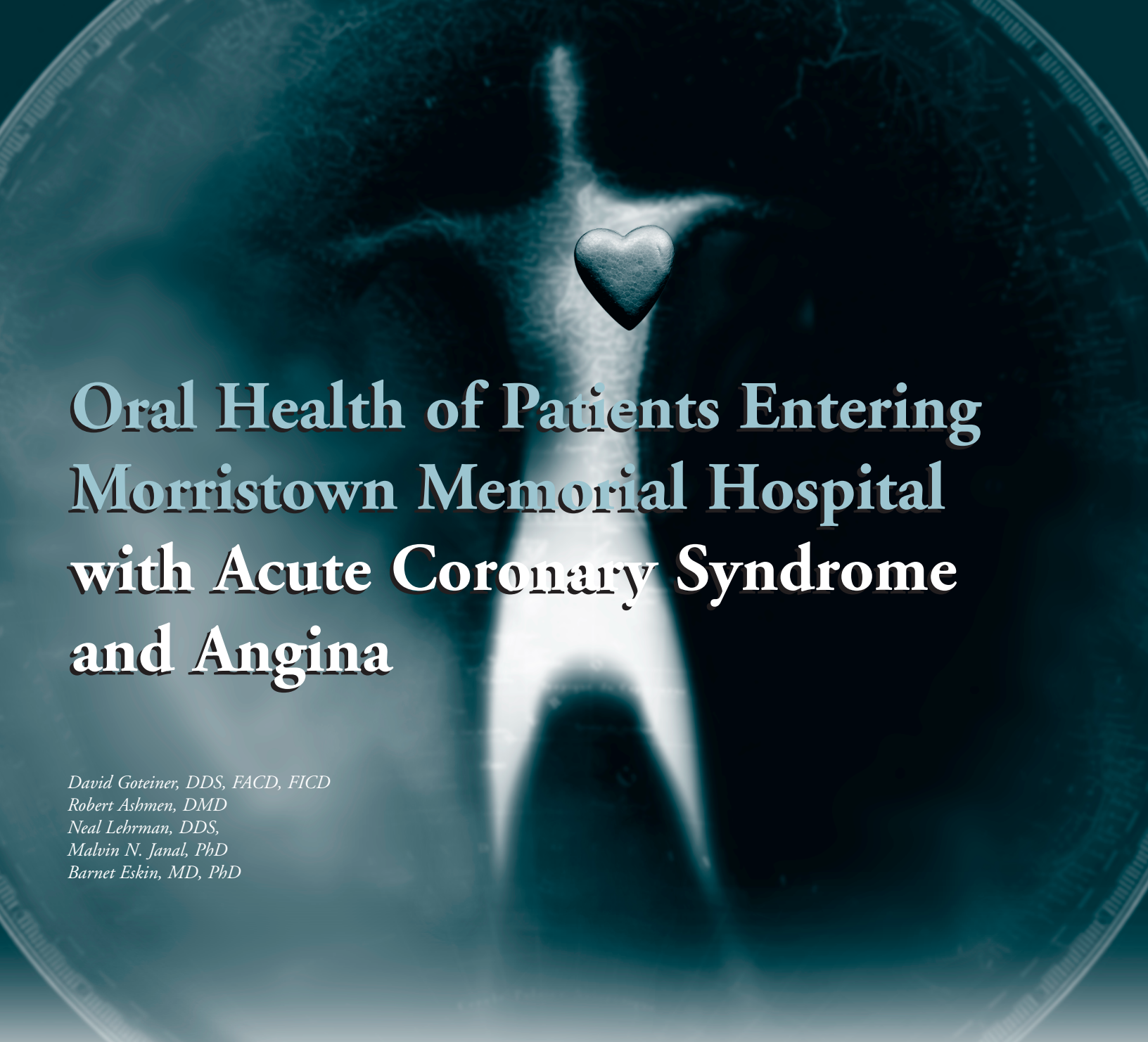

Oral Health of Patients Entering Morristown Memorial Hospital with Acute Coronary Syndrome and Angina

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The development of coronary artery disease (CAD) is attributed to several risk factors, including high serum cholesterol, low serum high density lipoprotein (HDL) levels, smoking, increased levels of C-reactive protein (CRP), hypertension and diabetes.[1,2] These risk factors, however, do not explain all cases of coronary artery disease. Additional risk factors such as chronic periodontitis have been suggested but, to date, no firm linkage has been established.[1, 2, 3, 4]

Some studies have suggested that two potential risk factors are bacterial and viral infections, which may precipitate myocardial pathology in a certain susceptible population.[5, 6] Subclinical infections may also play a role in cardiomyopathy.[7, 8, 9] Loesche suggested that an equilibrium is maintained in the contained periodontal infection when the immune response of the healthy individual keeps the lesion subclinical. If the host immune system is compromised and plaque is permitted to mature, a critical mass may be reached, and the resulting pathology may no longer be localized to the mouth.[1, 10]

Gingivitis and periodontitis are infectious inflammatory diseases of the gingiva and underlying supporting structures of the teeth.[11] *Porphyromonas gingivalis*, *Prevotella intermedia, Tannerella forsythensis* and *Actinobacillus actinomycetemcomitans* are often implicated in the pathogenesis of periodontal disease and unsuccessful therapy.[12] These pathogens can be found in the epithelium and connective tissue and within the cells of the periodontal lesion.[13, 14] Recently these microorganisms have been found incorporated in coronary endothelium, as well as atherosclerotic plaques.[15-23] Kozarov *et al.,* were able to demonstrate that *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* were both viable and invasive.[24] Kuramitsu *et al.,* demonstrated the ability of *Porphyromonas gingivalis* to induce foam cell formation, an initial step in plaque formation, in the presence

of LDL in macrophage cell culture.[25] Pussinen *et al.,* showed that the extent of affected tissue in periodontitis is directly associated with the ability of isolated LDL to activate macrophages *in vitro.*[26]

Geerts *et al.,* recently demonstrated endotoxemia can be induced by gentle mastication and that the levels are higher in patients with severe periodontal disease.[26] Beck *et al.,* concluded that microbial and LPS (endotoxin) exposures may be associated with acute aspects of CAD such as thrombus formation.[27] In support of this, Herzberg and Meyer's *in vitro* study demonstrated that dental bacteremias could induce platelet aggregation.[28] Valtonen has proposed that chronic dental infections, among others, may act synergistically along with classic risk factors, such as a genetic propensity, for a more robust inflammatory response, in the development of various atherosclerotic diseases.[29] In experiments with mice lacking the IL-1 receptor agonist gene, the results suggest that the expression of IL-1 is likely to have a significant role in signaling artery wall damage by affecting lipoprotein metabolism and foam-lesion development.

Purpose

To determine whether ACS/angina patients have more oral disease at the time of hospital admission than a) a group whose diagnosis was chest pain and b) a group who entered the hospital for elective procedures.

Methods and Materials

This study, before it began, was reviewed and approved by the Institutional Review Boards at both the University of Medicine and Dentistry of New Jersey and Morristown Memorial Hospital in Morristown, New Jersey.

The experimental group consisted of one hundred and thirty-seven (137) dentate patients who entered through the emergency room and were admitted and discharged from the cardiac care unit at Morristown Memorial Hospital in Morristown, NJ, with a diagnosis of acute myocardial infarction (AMI), unstable angina or angina (ICD-9CM codes 410-413.9). Since both AMI and unstable angina are part of a spectrum essentially differing only in the sequellae of cell death, they are often referred to as acute coronary syndrome or ACS. After obtaining patient consent, we examined the patient's medical and dental records and contacted the admitting physician. In an attempt to exclude cofounding factors, excluded from the population were patients with insulin-dependent diabetes mellitus (IDDM), multiple diagnoses (multiple DRGs), chronic antibiotic therapy, pregnancy, obesity, or antibiotic regimes or infection at the time of admission. The control groups consisted of:

a) 37 patients who visited the emergency room and were released with a diagnosis of chest pain (Control Group 1).

b) 101 patients signing in at the admissions desk for elective procedures at Morristown Memorial Hospital (Control Group 2). This group was used to collect demographic data for the community.

Background data were obtained from patient records at the hospital and the patient's dentist of record. A supplemental questionnaire was sent to the patient. The study population was asked about their oral habits and if their dentist told them they had periodontitis, "gum disease" or "pyorrhea." The radiographs were assessed by three periodontists. The results were interpreted and assigned a score as follows: no disease (0), mild or up to 20% loss of support (1), moderate or up to 50% (2), severe periodontitis or greater than 50% loss of support (3).[30]

Statistical Analysis:

Data included both discrete measures, summarized as proportions, and continuous measures, summarized as mean, M, and standard deviation, SD. Differences between groups on discrete measures were evaluated by means of the chi-square test. Differences between groups on continuous measures were evaluated by means of t-tests (if only 2 groups), or ANOVA (if more than 2 groups), with post-hoc tests between groups that controlled for experiment-wise error rate. Statistical significance was taken with an alpha of 0.05.

Results

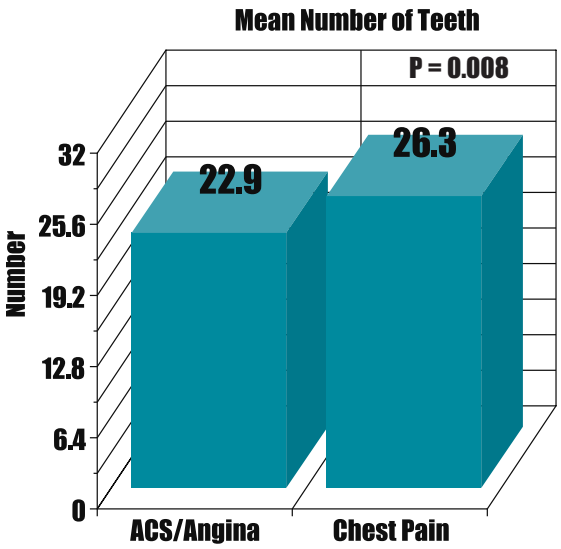
Populations: A total of 137 patients with either ACS or angina were entered into the study as the experimental group. There were 37 patients discharged from the ER with a diagnosis of chest pain who constituted control group 1. The second control group, to assess maintenance frequency, consisted of 101 patients who entered the hospital for elective procedures.

Age: The experimental group had a mean age of 61.4 years while control groups 1 and 2 had mean ages of 57.7 and 61.7 years, respectively (Table A). Ages were not statistically significantly different. When the experimental groups were further divided into groups that had AMI, unstable angina or angina, no statistically significant differences in age were seen.

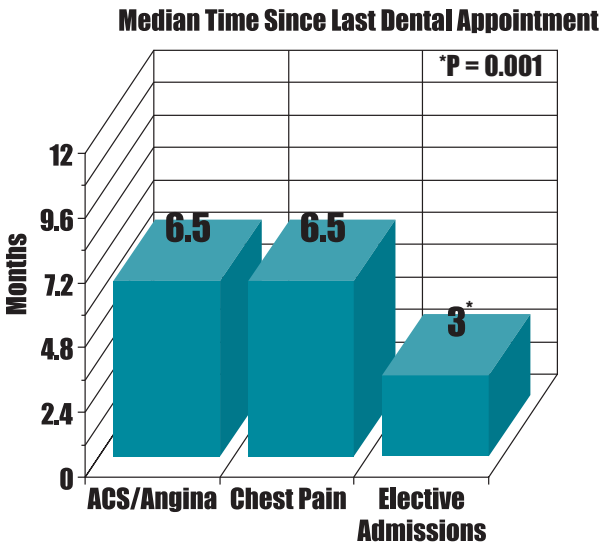
Table A
DEMOGRAPHICS

	Number	Age ±SD	% Female (Number)	NIDDM (Percent)	Smoker Current (%)	Smoker Ever (%)
Experimental Group ACS or Angina	137	61.4 ±12.3	23.3% (32)	22 (16.0%)	25 (18.3%)	50 (36.0%)
Control Group 1 Chest Pain without ACS or Angina	37	57.7 ±11.3	51.4% (19)	7 (18.9%)	8 (24.2%)	10 (30.3%)
Control Group 2 Elective Admissions	101	61.7 ±13.5	45.5% (46)	Unknown	Unknown	Unknown

Graph 1



Graph 2



Gender: Of the 295 total patients in the study, 65% were male and males predominated in all the groups (Table A). There were no statistical differences between genders for all variables studied. The distribution of periodontal pathology was equivalent between the sexes.

Smoking: All the groups had a similar history of smoking, either past or present. There were no significant statistical differences between groups (Table A).

Number of Teeth: The experimental group averaged 22.9 (±7.6) teeth, which was significantly less than the mean of control group 1 (those discharged from the ER without a cardiac diagnosis) of 26.3 (±6.0) teeth (p=0.008). The number of teeth were inversely related to the radiographic bone loss where r=0.424 (p<0.001) (Graph 1).

Dental Demographics: When questioned about the elapsed time since their last visit for either prophylaxis or scaling, there was a statistically significant difference between those who entered the hospital on an emergency basis (experimental group and control group 1) and those electively admitted (control group 2). Many experimental patients could not remember seeing a dentist within the past 10 years. We arbitrarily chose 10 years in determining the median. Those who entered the hospital via the emergency room averaged 6.5 months, while those entering the hospital electively (control group 2) averaged 3.0 months (p=0.001) (Graph 2).

Dentist's Questionnaire/Clinical Findings: Those patients without a dentist, who chose to be examined, did so at the Dental Clinic in Morristown Memorial Hospital. Gingivitis (treated or untreated) was reported in over 75% of both groups and periodontitis (treated or untreated) in over 60% of the experimental group and control group 1 (Table B). Using the z-test for independent proportions, there was a significant difference (p<0.01) of untreated pathology (dental caries, periodontal disease and periapical pathology) in the experimental versus control group 1, of 50.4% and 24.2% respectively (Graph 3). The experimental group had more radiographic bone loss (2.00±0.74) than control group 1 (1.64±0.7), but this was not statistically significant. However radiographic interpretation indicated that the prevalence of severe periodontal pathology was significantly more common in the ACS/angina group than in control

Graph 3

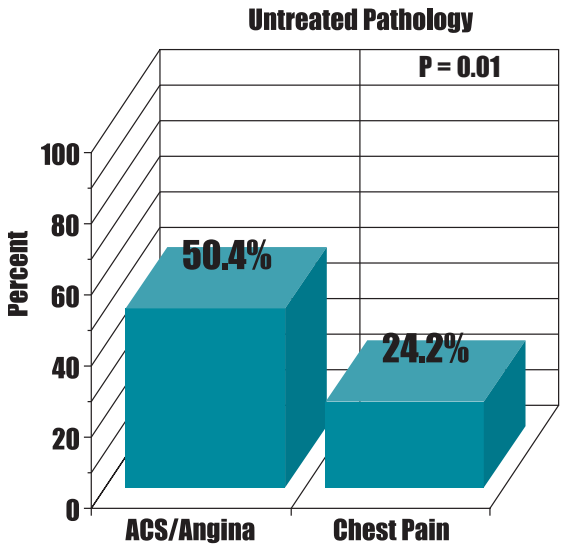
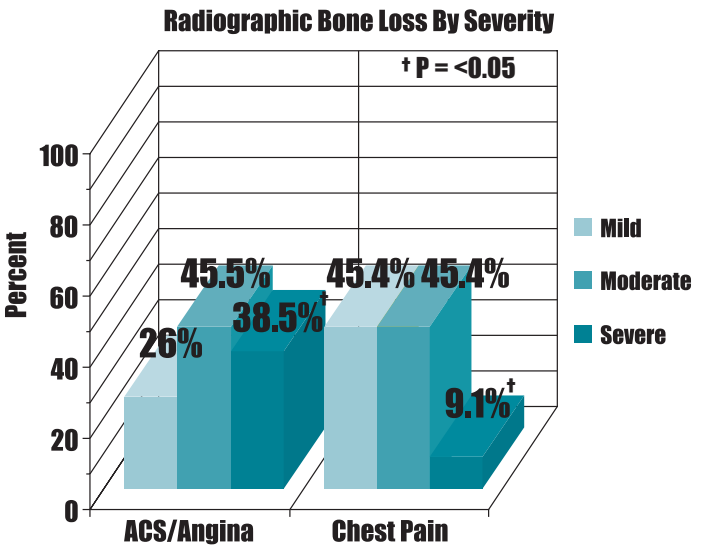


Table B

CLINICAL FINDINGS AS REPORTED BY THEIR DENTISTS

	Experimental Group ACS and Angina	Control Group 1 Chest Pain
	Percent (Number of subjects) [Total in the group]	
Gingivitis Yes	76.3% (100) [131]	75.8% (25) [33]
Periodontitis Yes	67.9% (89)	63.6% (21)

Graph 4



group 1 (38.5 vs. 9.1%, chi square, df =2, p<0.05) (Graph 4). It is interesting to note that the treating dentists underestimated the severity of the patients' periodontal status in all the groups entering the ER.

Discussion

Renvert *et al.,* concluded that of the five parameters studied, radiographic evidence of bone loss was the best individual parameter in associating periodontal disease with acute myocardial infarction.[31] Our study looked at acute myocardial infarction, unstable angina and angina. We chose to use radiographic evidence as our measure of periodontal disease. Machtei *et al.,* concluded that alveolar bone loss assessed from radiographs highly correlates with other quantitative clinical measures of periodontitis such as pocket depth and attachment loss.[32]

Our study only measured the dentate population. Since the edentulous population appears to be as susceptible to bacteremias as the dentate population, due to ill-fitting prostheses, perhaps they should be included in future studies. Slade and co-workers reported that CRP levels did not improve with loss of all teeth. They concluded it may be due to mucosal pathology induced by ill-fitting prostheses.[33]

Although radiographic evidence of the progression of alveolar bone loss was not significant, a trend was evident with severe periodontitis being significantly related. More importantly, the number of remaining teeth appeared related to whether or not a patient entered the hospital on an elective or emergency basis for treatment. Presence of gingivitis, periodontitis and oral pathology coincidental to CAD, is again confirmed by this study.

Perhaps endotoxemia caused by periodontitis and modified by a robust inflammatory response are the proximate cause of some acute coronary event. It may also be possible that dental prophylaxis will delay such an event by reducing the bacterial load in the periodontium.[34] Finally, being edentulous may not prevent bacteremias due to the presence of oral microbes. More research to establish the role of periodontitis in acute coronary episodes is indicated.

Conclusions

Poor dental care may be associated with an acute coronary event. Patients with ACS or angina are more likely to have poorer oral care, less teeth and increased radiographic bone loss.

Acknowledgement

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