What's New in Dentistry

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Periodontal disease is positively associated with coronary heart disease. Within the past decade, epidemiological studies have suggested an association between periodontal disease and coronary heart disease. It has been suggested that the spread of bacteria and bacterial products from the periodontal lesion to the bloodstream may contribute to arteriosclerosis and coronary heart disease. However, whether an association between periodontal disease and coronary heart disease could be causal is still uncertain. Another explanation for the observed association could be that the two disease entities share common risk factors. Therefore, a study published in the *Journal of Periodontology* (2006;77:1547–1554) tested the hypothesis that a high prevalence of periodontal disease among coronary heart disease patients could be explained, at least in part, by mutual risk factors. This was a case-control study composed of 250 subjects. Of these, 110 individuals had verified coronary heart disease, and 140 individuals served as controls without coronary heart disease. Information on diabetic status, smoking habits, alcohol consumption, physical activity, household income, body weight and height, triglycerides, and serum cholesterol was obtained. In addition, full-mouth probing depths, clinical attachment loss, bleeding on probing, and alveolar bone level on radiographs were recorded. Then, multiple logistic regression models were created to determine associations. The results of this study showed that for participants younger than 60 years, only risk factors such as smoking and diabetic status entered the multivariate analysis. For the alveolar bone loss group, there was a significant association with coronary heart disease for participants older than 60 years, with the odds ratio being 6.6. For participants younger than 60 years, there was no association. Thus, the present study has shown a positive, highly age-dependent association between periodontal disease and coronary heart disease that could be attributed only to diabetes and smoking to some extent.

Laser irradiation of titanium does not negatively affect osteoblast attachment. Osseointegrated implants have demonstrated a high success rate for more than 30 years. However, peri-implantitis may lead to implant failure if no treatment is established. Some recent studies have shown positive results using laser irradiation to control peri-implant infection. Lasers may reduce bacterial accumulation and affect implant surface decontamination. However, do lasers affect the surface of the titanium implant surface after irradiation, which could negatively affect osseointegration and the attachment of osteoblasts to the implant surface? A study published in the *International Journal of Oral and Maxillofacial Implants* (2006;21:232–236) examined the attachment of osteoblasts to titanium surfaces after laser irradiation using scanning electron microscopic analysis. Four different types of titanium disks were used: machined, hydroxyapatite coated, sandblasted, and titanium plasma sprayed. These disks were divided into three groups, and both carbon dioxide and biolase instruments were used. The third group received no laser irradiation and served as the control group. Then, the titanium disks were immersed in a cell culture medium to determine if osteoblasts would attach to the irradiated and control titanium surfaces. The findings of this study demonstrate that osteoblasts could be grown on all of the surfaces. In fact, pseudopodia and a spread of cells that demonstrated maturation were observed on the laser-irradiated titanium disks. In conclusion, the data show that laser irradiation of titanium surfaces may promote osteoblast attachment and further bone formation.

Diabetes enhances periodontal disease in specific ways. The progression of periodontal disease may be affected by systemic conditions, such as diabetes. Type 2 diabetes significantly increases the risk for periodontal disease with either attachment loss or bone loss as a criterion. However, it is not known whether diabetes primarily affects periodontitis by enhancing bone loss or by limiting osseous repair. A study published in the *Journal of Dental Research* (2006;85:510–514) used a diabetic animal model, in which bone resorption and formation could be separately examined, to test the hypothesis that type 2 diabetes would aggravate periodontal disease by both increasing bone loss and limiting reparative bone formation. Ligatures were placed around a molar tooth in each animal, and the ensuing inflammation, loss of attachment, bone loss, and bone formation were measured. One quadrant in each animal had no ligature

attached and served as the control. The amount of bone resorption, as well as the reparative capacity, was evaluated 2 and 4 days after the ligatures had been placed as well as 4 and 9 days after the ligatures had been removed. The results showed that diabetes increased the intensity and duration of the inflammatory infiltrate. Four days after removal of the ligatures, the type 2 diabetic group had significantly higher osteoclast numbers and activity. The amount of new bone formation following resorption was 2.4- to 2.9-fold higher in the healthy versus the diabetic animals. Diabetes also decreased the number of bone-lining cells, osteoblasts, and periodontal ligament fibroblasts. In conclusion, diabetes caused a more persistent inflammatory response, greater loss of attachment, more alveolar bone resorption, and impaired new bone formation.

Cochrane Systematic Review critically assesses bone grafting prior to or during dental implant placement. Different indications, numerous alternative techniques, and various biologically active agents and biomaterials are currently used to augment bone prior to or during dental implant placement. However, no evidence-based information currently exists regarding the utility of these various. Therefore, a Cochrane Collaboration Review was performed regarding this topic and was published in the *International Journal of Oral and Maxillofacial Implants* (2006;21:696–710). The purposes of this systematic review were to test whether and when bone augmentation procedures are necessary and which is the most effective augmentation technique for specific clinical indications. An exhaustive search was conducted for all randomized controlled clinical trials comparing different techniques and materials for augmenting bone for implant treatment. A total of 13 randomized clinical trials of 30 potentially eligible trials reporting the outcome of 332 patients were suitable for inclusion. Six trials evaluated techniques for vertical and/or horizontal bone augmentation. Four trials evaluated techniques of bone grafting for implants placed in extraction sockets, and three trials evaluated techniques to treat fenestrated implants. This systematic review concludes that major bone-grafting procedures of extremely resorbed mandibles may not be justified. Bone-guided regeneration procedures and distraction osteogenesis can be used to augment bone vertically, but it is unclear which is most efficient. More bone was regenerated around the fenestrated implants with nonresorbable barriers than without barriers. Bone morphogenetic proteins may enhance bone formation around implants grafted with Bio-Oss, but there was no reliable evidence supporting the efficacy of other active agents, such as platelet-rich plasma, in conjunction with implant treatment.

Chlorhexidine varnish has a positive caries-inhibiting effect in children. Chlorhexidine has been studied for a few decades as a potent antimicrobial agent used for the chemical control of plaque and caries prevention. A meta-analysis on the use of chlorhexidine-containing gels, mouthwashes, and toothpastes showed a 46% caries-reducing effect of chlorhexidine treatment. In recent years, a strategy for caries prevention has been to prolong the chlorhexidine-releasing effect by placing the chemical in a varnish form that can be applied to the teeth of children during caries-prone years. A study published in the *Journal of Dental Research* (2006;85:469–472) investigated the caries-inhibiting effect of a chlorhexidine varnish in the pits and fissures of first permanent molars. The sample for this study consisted of 460 six- to seven-year-old children. These subjects were enrolled in a 2-year randomized controlled trial. A split-mouth design was employed. One group of permanent first molars received the chlorhexidine varnish in the pits and fissures at baseline, 6, 12, and 18 months and another group at baseline, 3, 12, and 15 months. Control molars did not receive the varnish application. Adherence to the treatment protocol was good, and the dropout rate was low at 17%. Blinded examiners performed the dental examinations, and the caries-inhibiting effects of the two chlorhexidine varnish application schemes were compared. There were small differences between the two application schemes. However, there was a statistically significant short-term reduction in caries between treated and control groups. The prevented fraction of caries was 25% after 2 years and 9% 1 year after termination of the trial. This suggests a positive short-term benefit from the use of the chlorhexidine varnish in the pits and fissures of permanent first molars.