Purpose: The purpose of this study was to analyze periimplantitis lesions in man as they present in biopsies obtained from implant sites exhibiting clinical signs of inflammation and progressive bone loss.

Materials and Methods: Soft tissue biopsies were obtained from 12 implant sites in six patients. The implants had been in function between 4 and 21 years and were, with one exception from the maxilla. The clinical and radiographic examination performed prior to biopsy revealed that all sites exhibited advanced bone loss, severe inflammation including suppuration, swelling and/or fistula, and seven of the 12 implants were mobile at the time of biopsy. A soft tissue biopsy was obtained from either the mesial or distal aspect of each site and sections were prepared for histometric and morphometric analysis.

Findings and Conclusion: All of the biopsies from the implant failure and periimplantitis treatment groups (including mobile and non-mobile implants at removal) had similar histopathological characteristics. A keratinized oral epithelium outlined the biopsies and was continuous with a pocket epithelium. The marginal aspect of the pocket epithelium was wide and exhibited rete ridges that projected into the infiltrated connective tissue. In most sections the inflammatory cell infiltrate reached a position that was apical of the pocket epithelium. The results of the morphometric assessments (MA) of the composition of the inflammatory cell infiltrate showed that in apical, ulcerated areas of the pocket epithelium, deposits of microbial plaque could be observed. The MA also showed that about 60% of the lesions were occupied by inflammatory cells (38.8% plasma cells, 10% collagen, and 9.3% vascular structure, 5.7% fibroblasts, 5.2% macrophages, 6.6% lymphocytes, 4.2% PMN’s, and 20.3% residual tissue).

The size of the connective tissue at mobile implant sites was on the average 3.39 squared mm while the corresponding non-mobile implants was 3.88 squared mm. The author concludes that this indicates that the presence of inflammatory lesions in the periimplant soft tissues may not have been influenced by implant mobility.
Purpose: Local adverse effects involving the use of Emdogain® have not been reported in the literature to date. The purpose of this case report is to describe two examples of external inflammatory resorption following surgical root surface debridement and the use of Emdogain®.

Materials and Methods: The two cases described were both seen at the Department of Periodontology of the Eastman Dental Hospital. Both non-smoking patients presented with generalized chronic periodontitis with no other modifying risk factors and clear medical histories. After both cases received initial full mouth non-surgical therapy and oral hygiene instructions they presented 6 weeks later in need of further intervention. At this point Case 1 had a 3 wall defect on the distal of 31 and case 2 had a 2 wall defect on the distal of 22. Both defects were exposed, cleaned of all granulation tissue, the root surfaces debridged, conditioned with Pref-Gel® (EDTA), Emdogain® placed per the manufacturers instructions, flaps were replaced, sutured, and post-op instructions given. Radiographs taken before treatment and at 6 months to assess healing.

Findings: Case 1- post-op appointments uneventful until 6 months. Pain, BOP, 10mm pocket, radiographic radiolucency, tooth diagnosed with external inflammatory root resorption. Tooth 31 extracted and histopathologic section confirmed diagnosis. Case 2- post-op appointments uneventful until 2 years. Pain, no BOP, radiographic radiolucency coronal to the level of the re-generated bone, tooth diagnosed with external inflammatory root resorption. Tooth 22 extracted and histopathologic section confirmed diagnosis.

Conclusion: While these two cases report external inflammatory root resorption following the treatment of intra-bony defects with Emdogain® and Pref-Gel® (EDTA), the true prevalence of this problem and the contribution of the materials used in the procedure to the inflammatory resorption seen are unknown. The exact nature of the cellular effects of Emdogain® are still relatively unclear and further work is necessary to establish this.

**Purpose:** To study the incidence, magnitude, duration and nature of bacteremia in individuals with periodontitis, gingivitis and clinically healthy periodontium after chewing, tooth brushing and scaling.

**Materials and Methods** 60 systemically healthy individual took part in this study. The participants were categorized in to three groups: 1-Periodontitis, having at least 10 sites with probing pocket depth PPD >5mm. 2-Gingivitis, having GI >1.5 (Loe and Silness 1963) and radiographically alveolar bone 2mm from CEJ. 3-Healthy periodontium, GI>.5. Exclusion criteria were antibiotic treatment within the last 6 month, patients with prosthetic heart valve and etc. Full mouth periodontal registration was performed which includes PPD, GI, PI, BOP and CAL. FMX were also taken before the start of the study. All participant were exposed to the three experimental procedure which were: 1- chewing gum for 10 min, 2- supervised tooth brushing for 2 min, 3- full mouth scaling. 2ml of blood was discarded before drawing the blood for bacteremia analysis. 9ml of Blood samples at baseline and at .5, 10 and 30 min after each procedure were examined for bacteremia. Detection of microorganism in the Blood sample was done by lysis filtration method. A 5 % level of significance was used in the statistical analyses.

**Findings:** None of the 60 individual was detectably bacteremic before the three experimental procedures. Following chewing 4 of periodontitis patient (20%) were bacteremic, where as none of the periodontally healthy individual or gingivitis patients showed bacteremia. Following tooth brushing only one periodontitis individual (5%) was bacteremic but none from the other group. After 30 min bacteremia was found in another individual. Bacteremia after scaling occurred in 2 of the healthy participants, 4 of the gingivitis individual and 15 of the periodontitis patient. The magnitude of bacteremia decrease within 30 min. After 30 min bacteremia was detectable in four individual, 1 after chewing, 1 after tooth brushing and 2 occurred after scaling. A total of 163 isolates were collected from 29 bacteremic episodes in 23 of the 60 individual. The isolates were from 2 healthy individuals, 4 gingivitis patients and 17 periodontitis patient after chewing, tooth brushing and scaling. Among the streptococcus specie S. mitis and S. oralis were predominant and among G-ve there were P. intermedia and F. nucleatum. After scaling the incidence and magnitude of bacteremia were significantly higher in the periodontitis group than in the gingivitis group and the healthy control group.

**Conclusions:** Individual with periodontitis has an increased risk of developing bacteremia during everyday events such as chewing and tooth brushing.

**Purpose:** The aim of this study was to calculate stresses and Tsai-Wu strength ratio in the cervical area of the mandibular molar during grinding, clenching, and mastication, as well as theoretical investigation of the mechanism of cervical lesion formation in teeth.

**Materials and Methods:** The study of stresses in mandibular first molars during grinding, clenching, and mastication was conducted with finite element analysis (FEA). Computational simulation of mastication of a bolus with high elastic modulus, including grinding and clenching, was performed. Pairs of contact elements were used between the bolus and occlusal surfaces of the teeth. The analysis was nonlinear. During these simulations, the pressure exerted on the occlusal surface and the state of stresses in the mandibular molar were calculated. To evaluate the strength of anisotropic tooth tissues, the Tsai-Wu failure criterion was applied. This criterion considers the difference in strength of materials due to tensile, compressive, and shear stresses.

**Results:** The highest pressure was exerted on the lingual cusp of the mandibular molar in the closing phase of mastication, preceding the maximal intercuspal position. The highest values of normal stress were seen along the Y axis. The compressive stress occurred on the lingual surface, and tensile stress occurred on the buccal surface of the mandibular molar. The concentration of the highest tensile stresses, 24.4 MPa, in the direction perpendicular to the prisms, appeared in the enamel near the CEJ at the buccal surface of the tooth. In this area, the theoretical Tsai-Wu ratio reached 3.2. According to this criterion, elements in which the value exceeded 1 were damaged and, thus, were removed from the model. A small cervical lesion formed near the CEJ. In subsequent simulations of the mastication cycle, the tensile stresses in the enamel along the DEJ exceeded the strength of this tissue. Enamel elements in which the Tsai-Wu ratio was higher than 1 were eliminated. Thus, an enamel overhanging fragment was created, separated from the dentin by a fissure. Application of minimal horizontal force caused a fracture of this fragile, unsupported enamel fragment.

**Conclusion:** 1. In a simulation model, the mastication of a bolus of high elastic modulus, grinding, and clenching evoked considerable overload of the lingual cusp of the mandibular molar. In the buccal cervical area, tensile stress appeared that theoretically exceeded the strength of the enamel. The Tsai-Wu strength ratio in this area exceeded 1.
2. According to the 2D FEA, overloading of teeth may result in damage of the enamel at the CEJ and initiate a cervical lesion.