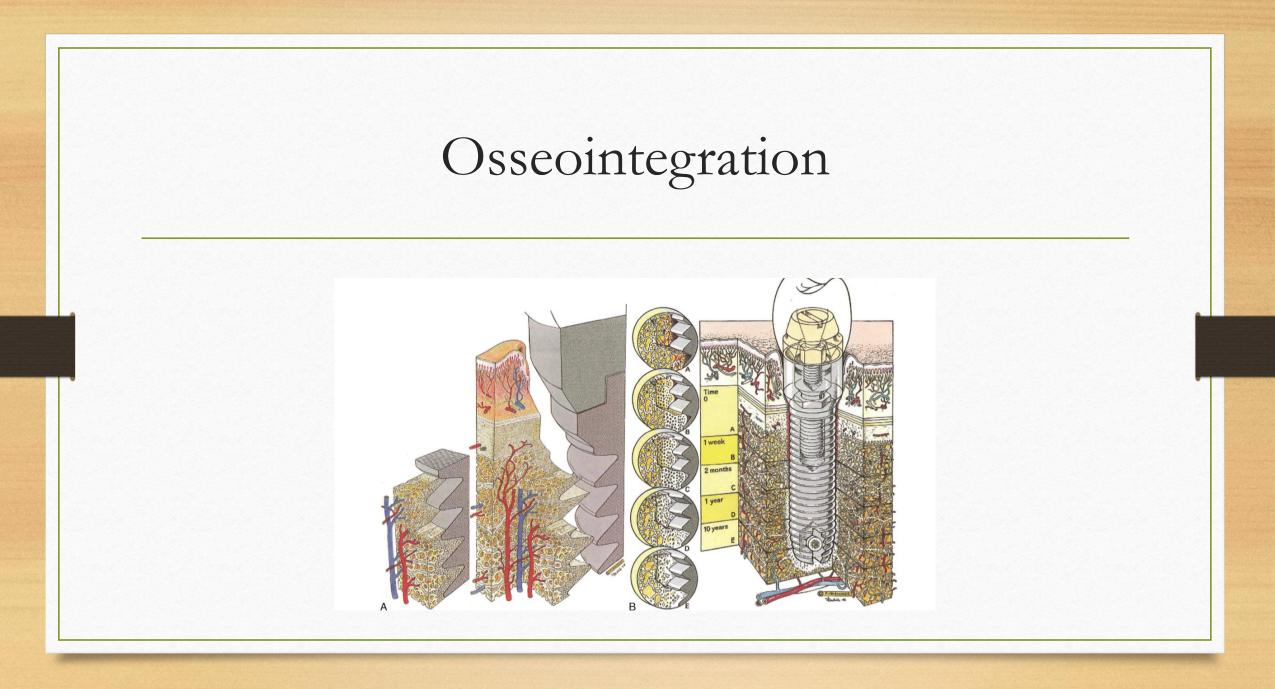
Introduction to periimplant diseases

Dr. Omar Karadsheh

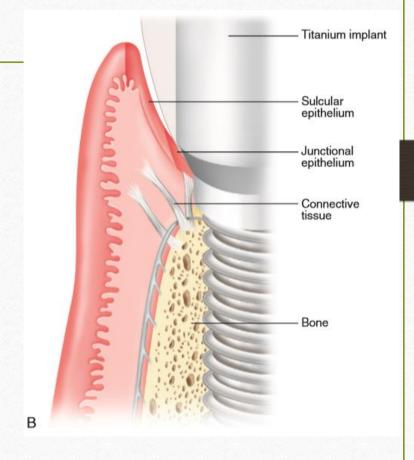
Introduction

- Osseointegration
- Tooth Vs. Implant
- Implant examination
- Peri-implant disease: Diagnosis and Treatment



Mucosa around implant

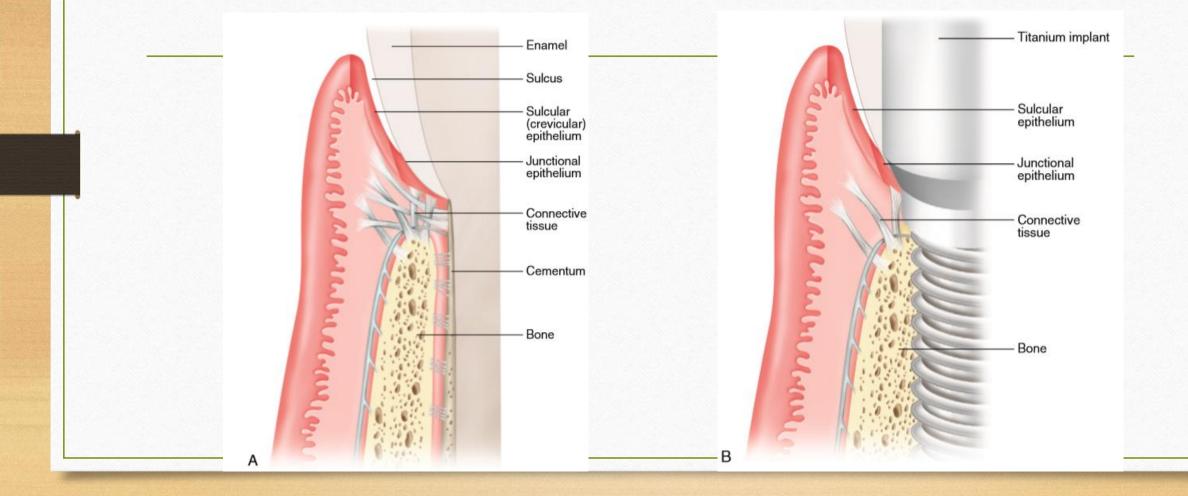
- Periimplant soft tissues are similar in appearance and structure to periodontal soft tissues.
- The soft tissues consist of:
 - ✓ connective tissue covered by epithelium.
 - ✓ a gingival/mucosal sulcus,
 - ✓ a long junctional epithelial attachment,
 - \checkmark a zone of connective tissue above the supporting bone

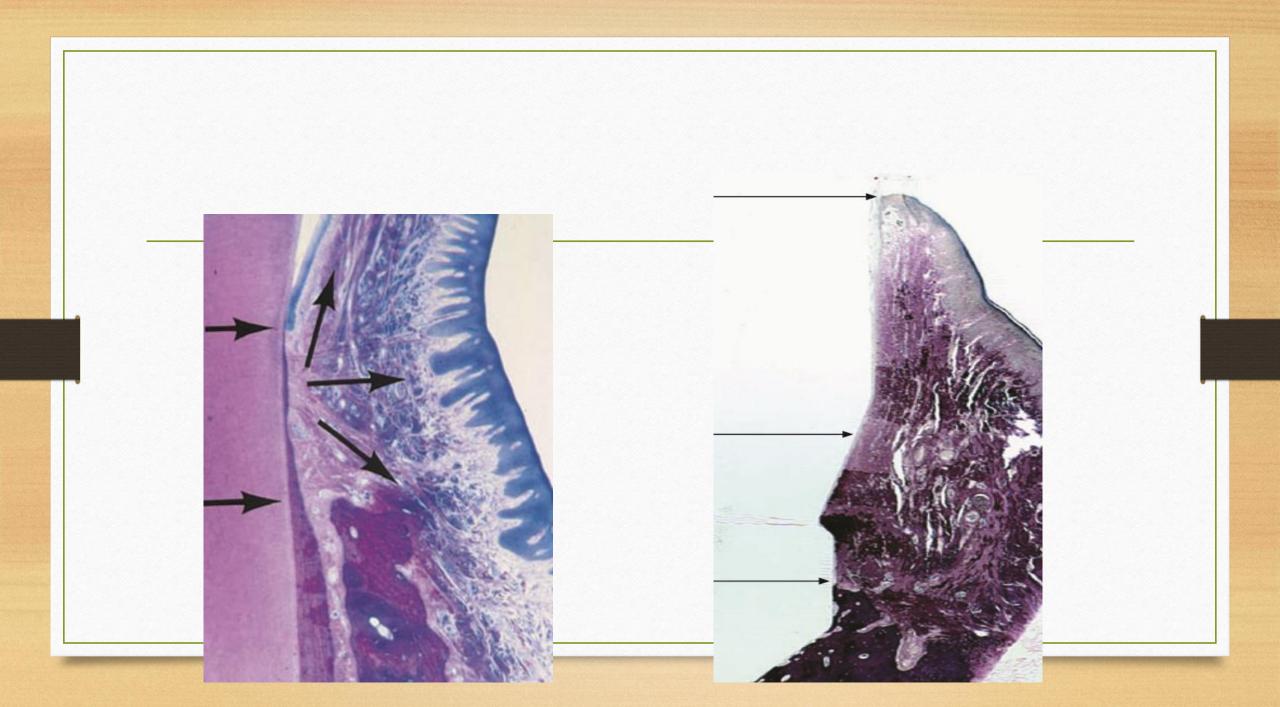


Implant Vs. Teeth

- Biological width?
- Collagen/Fibroblasts?
- Vascularity?
- Resistance to infection?

Implant Vs. Teeth





Differences?

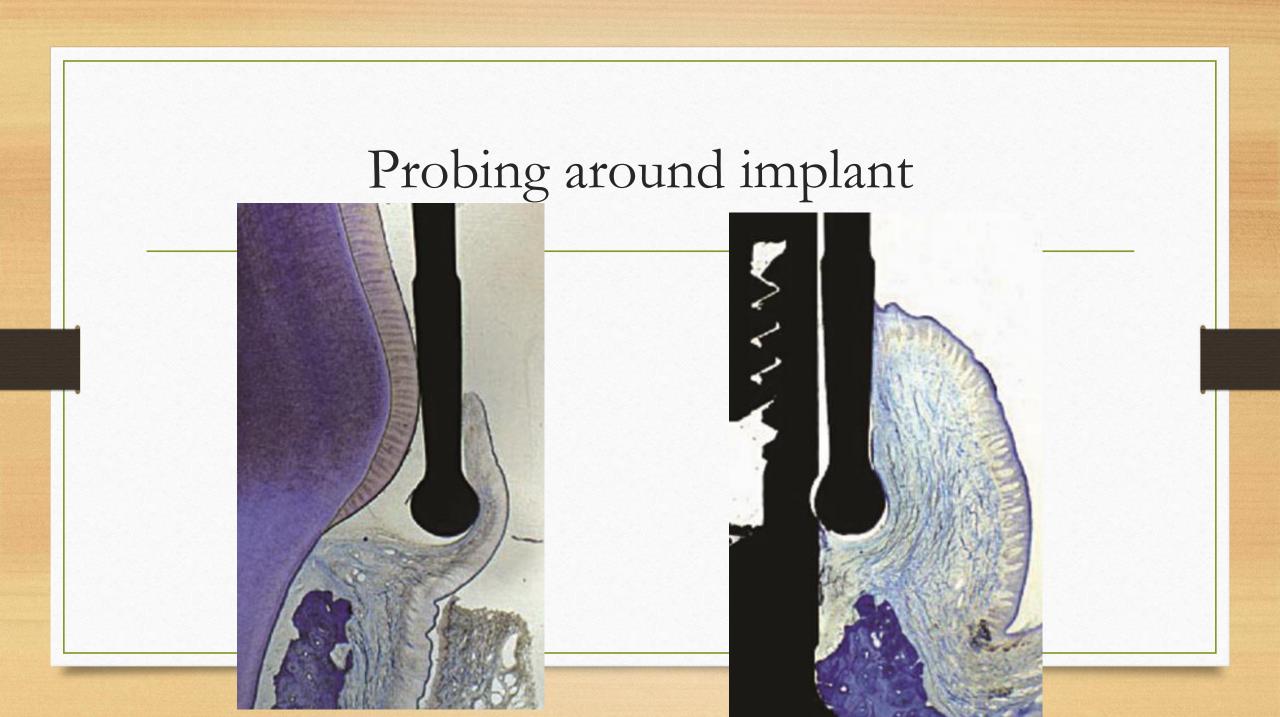
Implant	Tooth
Epithelial barrier (JE) = $2ml$	JE: 0.97mm
No PDL	PDL
No cementum	Cementum with inserting fibers
Collagen fibers rub parallel to implant, never insert into implant surface	Collagen fibers radiate from tooth surface to all directions.
Less vascularised	More vascularised
More collagen fibers, less fibroblasts	less Collagen, more fibroblasts
No proprioception, bears entire occlusal load	PDL act as shock absorbant, migrate under heavy occlusion

Soft tissue interface

• Biologic width:

teeth 2.04 mm (0.97 JE, 1.06 CT) Vs. Implants 4.5 mm (2mm JE,1-2 CT)

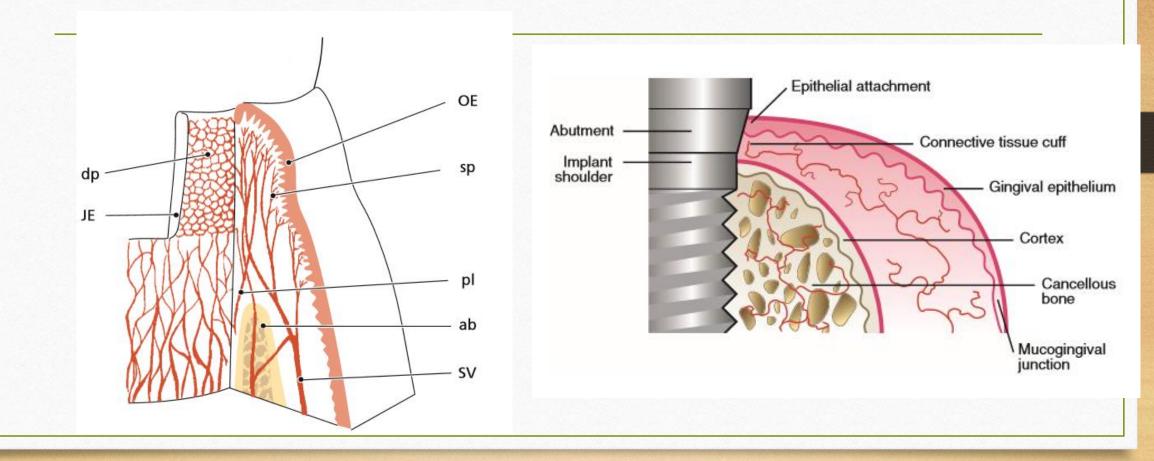
- The connective fibers are parallel to the implant surface without attachment to the metal body (adhesion). Consequently, the resistance to probing around implants is decreased as compared to that around teeth.
- However, when probing in healthy tissues, the tip of the probe seems to reach similar levels at the implant and tooth sites. Marginal inflammation around implants is associated with a deeper probe penetration as compared to that around teeth.



Soft tissue healing and vascular supply

- Due to the lack of the vascular plexus of the periodontal ligament, the implant blood supply comes from two sources:
 - \checkmark the peri-implant mucosa
 - \checkmark the supraperiosteal blood vessels.

Vascular supply



- The potential for repair is limited due to the:
 - A. Lack of periodontal ligament
 - B. Reduction of the cellular components of the mucosa
 - C. Reduced vascularization.

Soft tissue interface

- The peri-implant mucosa is sealed, and not attached to the implant.
- A biological width is maintained, whatever the thickness of the mucosa.
- Compared to the gingiva, the peri-implant mucosa is a scar-like tissue, rich in collagen fibers, poor in fibroblasts, and with limited blood supply.
- The potential for repair is more limited than with gingival tissue.

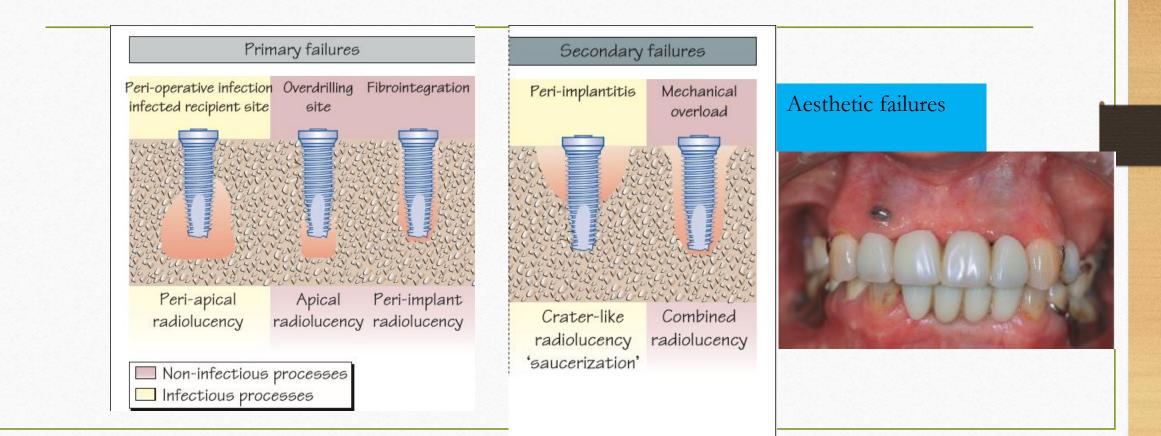


Implant complications

• Early Complications

• Late complications

Complications



Biologic complications

• Involve pathology of the surrounding peri-implant hard and soft tissues.

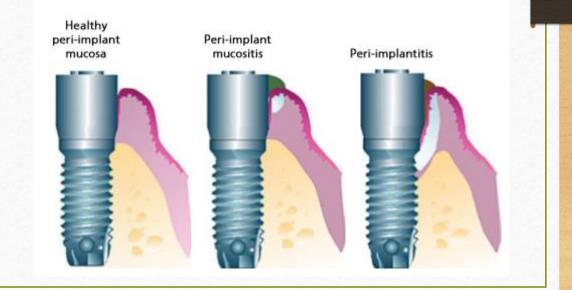
• Frequently, soft-tissue problems are an inflammatory response to bacterial accumulation.

• The cause of bacterial accumulation around implants is key to understanding the problem.

Peri-implant disease

- Peri-implant disease is a collective term used to describe inflammatory processes in tissues that surround implant(s). (Albrektsson & Isidor 1994).
- Peri-implant Mucositis

• Peri-implantitis

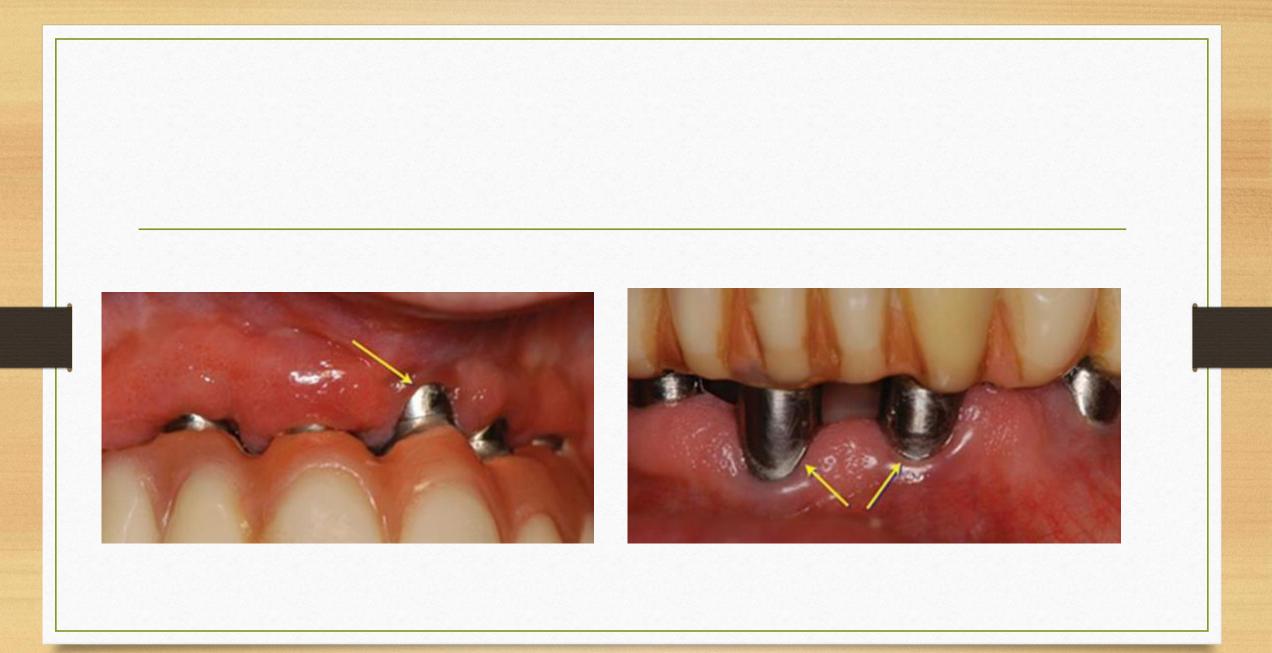


Peri-implant mucositis

- Defined as an inflammatory lesion that resides in the mucosa, not involving bone.
- Caused by bacterial plaque
- Local factors:
 - ✓ poor OH,
 - \checkmark the junction of an ill-fitting implant-abutment or abutment-crown connection,
 - \checkmark highly textured, macroscopically rough implant surfaces
 - ✓ trapped excess submucosal cement
 - ✓ large bulbous crowns

Clinical features of PI mucositis

- Similar to gingivitis
- Erythema, edema, and swelling
- Bleeding on probing
- Occasionally, however, the reaction of peri-implant soft tissues to bacterial accumulation is profound, almost unusual, with a dramatic inflammatory proliferation





Inflammatory proliferation caused by a loose-fitting connection between the abutment and the implant.





A, Clinical photograph of abscess caused by excess cement trapped within the soft tissues.B, Radiograph of implant with cemented crown (same patient as in A). Notice the subgingival depth of the crown-abutment (cement line) junction, which is below the level of the adjacent interproximal bone and therefore impossible to adequately access with explorer to remove excess cement.

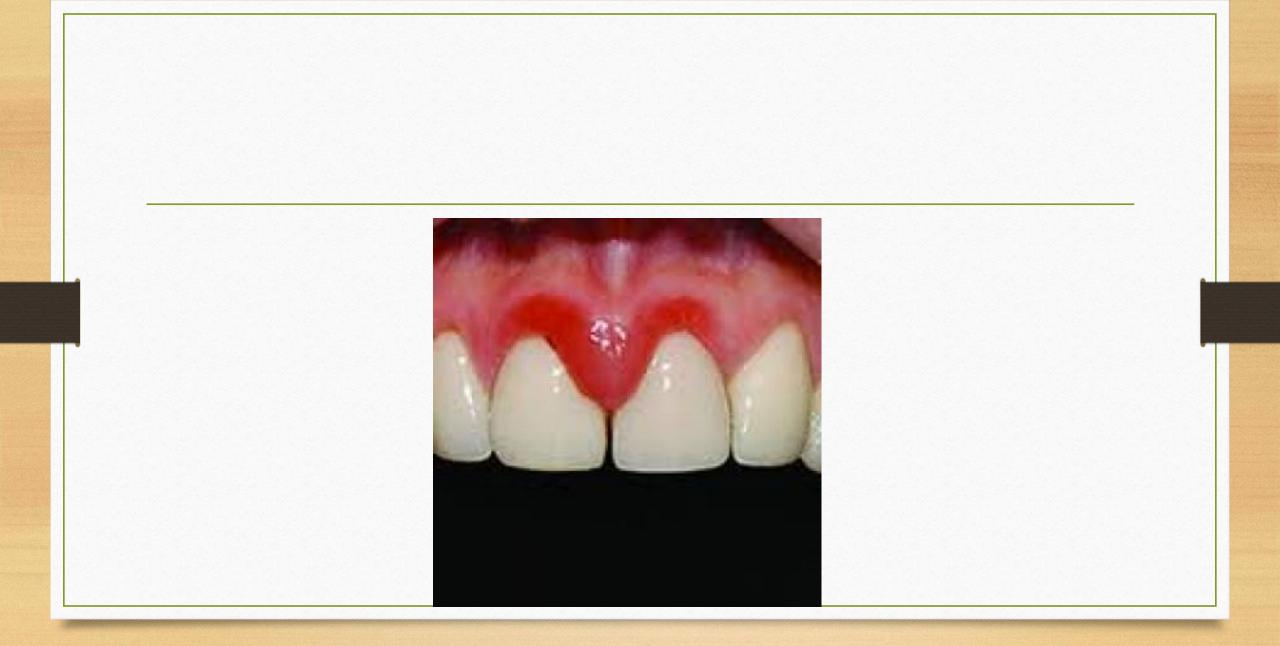
Human clinical studies



Animal clinical studies

Clinical photograph showing 5 months of undisturbed plaque formation on three different types of implants in a Beagle dog.





Histopathology & Microbiology

Histopathology

• Similar to Inflammatory cell infiltrate seen in gingivitis but with a larger extent

Microbiology

• Similar to gingivitis

Conclusion:

- Peri-implant mucositis and gingivitis have many features in common.
- The host response to bacterial challenge at teeth and implants includes the development of clinical signs of inflammation and the establishment of inflammatory lesions in the mucosal/ gingival connective tissues.
- Since peri-implant mucositis represents the obvious precursor of peri-implantitis, as does gingivitis for periodontitis, treatment of mucositis appears to be an important prerequisite for the prevention of peri-implantitis

(Lang et al. 2011)

Peri-implantitis

 "Changes at the level of crestal bone, presence of bleeding on probing and/or suppuration; with or without concomitant deepening of peri-implant pockets"

(Lang and Berglundh, 2011)

Peri-implantitis

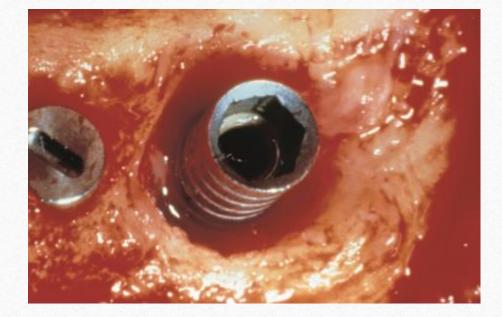
- Presence of an inflammatory lesion in the peri-implant mucosa and loss of peri-implant bone.
- Diagnosis requires detection of both (BoP) and bone loss on radiographs.
- Peri-implantitis initially affects the marginal part of the peri-implant tissues and the implant may remain stable and in function for varying periods of time.
- Implant mobility is therefore not an essential symptom for peri-implantitis, but may occur in the final stage of disease progression and indicates complete loss of integration.

- Swelling and redness of the mucosa
- Bleeding on gentle probing.
- Suppuration from the "pocket".
- Mobility ??



- Radiographic bone loss: → symmetrical loss (Similar loss M, D, B, L)
 → circumferential, trough-like, saucer-shaped bone loss
- The clinical appearance of peri-implantitis may vary and may not always be associated with overt signs of pathology.

Moderately advanced bone loss around an implant with the typical circumferential trough type of boney defect

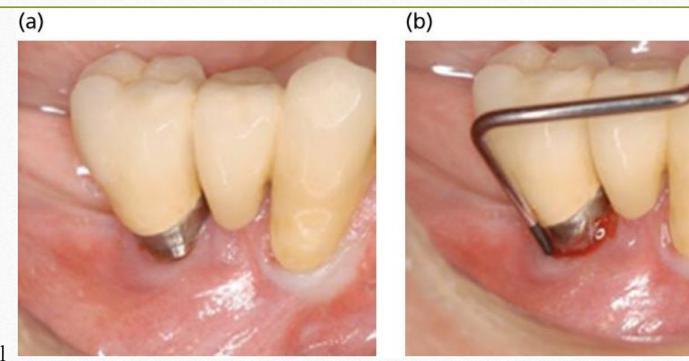




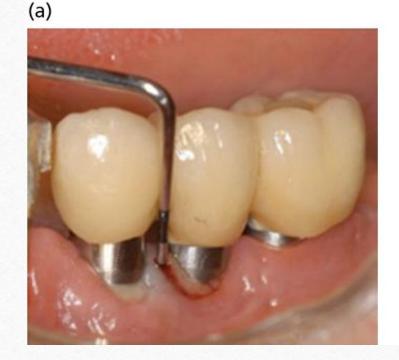
Clinical symptoms of peri-implantitis. Note the large amounts of plaque and calculus and visible signs of inflammation in the peri-implant mucosa.

Clinical photographs from an implant-supported crown in the premolar position in the left side of the mandible.

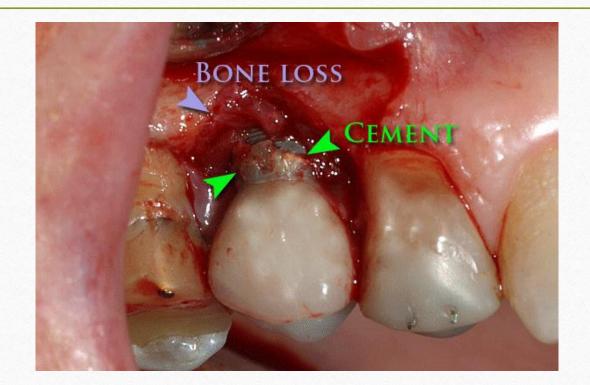
- (a) No or minor signs of inflammation in the surrounding mucosa.
- (b) Probing resulted in bleeding and suppuration from the implant site in the lateral incisor position.



Clinical (a) and radiographic (b) characteristics of three implant sites with peri-implantitis in the left side of the mandible. Note the presence of swelling and suppuration in the peri-implant mucosa (a) and the pronounced bone loss around the implants in the radiograph (b).







Experimental peri-implantitis

- Teeth Vs. implant
- Experimental periodontitis/periimplantitis model:
- Beagle dogs
- implants placed at PM region in one side, teeth on the other
- Ligature pushed into sulcus to induce disease and allowed plaque accumulation and formation of submucosal/subgingival biofilm
- Biopsies and radiographs from around teeth and implants

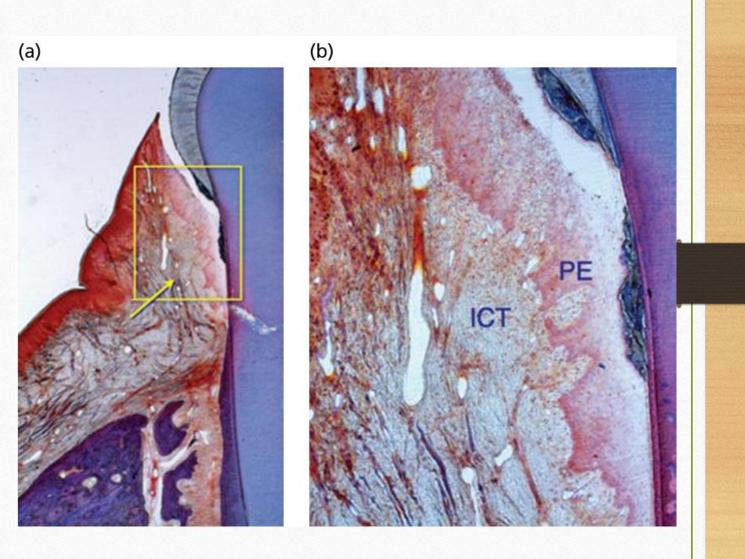
Microbiology

• Implants and teeth that are surrounded by healthy soft tissues are associated with biofilms with small numbers of Gram-positive coccoid cells and rods.

• Sites with extensive periodontal and periimplant inflammation harbor biofilms with large numbers of Gram-negative anaerobic bacteria

Periodontitis

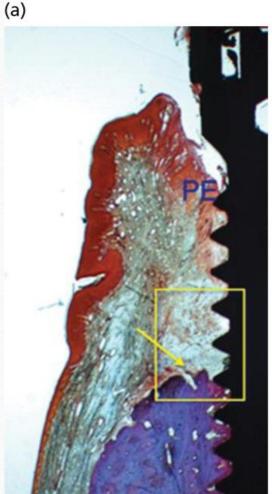
(a) Microphotograph of a buccolingual ground section showing a periodontitis lesion. Note the apical extension of the infiltrate (arrow), but also the presence of a zone of normal connective tissue between the infiltrate and the bone crest. (b) Larger magnification of outlined area in (a). Note the calculus on the tooth surface, the pocket epithelium (PE), and the infiltrate (ICT).

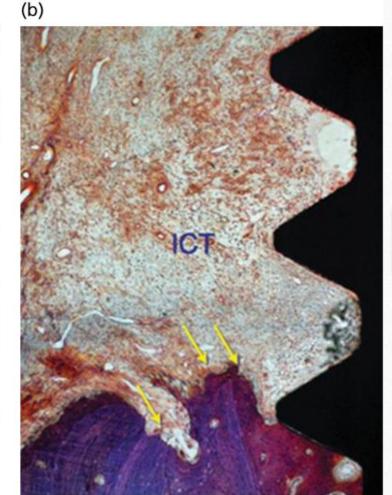


Periimplantitis

(a) Microphotograph of a buccolingual ground section showing a peri-implantitis lesion. The apical portions of the infiltrate (arrow) extend into contact with the bone.

(b) Close-up of outlined area in (a) showing the large infiltrate (ICT) apical of the pocket epithelium and in direct contact with the biofilm on the implant surface. Osteoclasts (arrows) are present on the bone surface. (PE, pocket epithelium.)



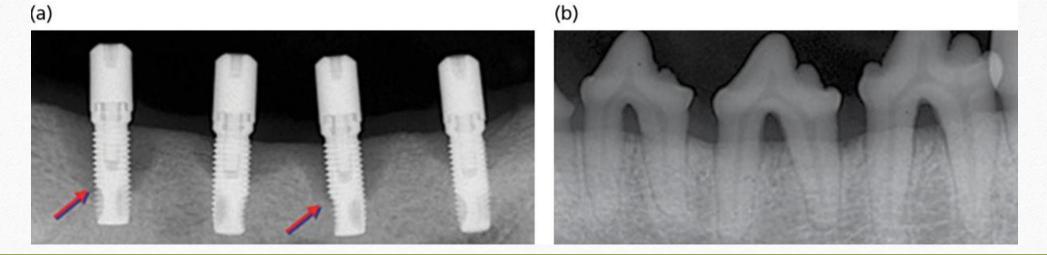


Peri-implantitis sites exhibited:

- Inflammatory lesions larger and extended closer to the bone crest than those in periodontitis
- While the lesions in the plaque-associated periodontal sites were consistently separated from the alveolar bone by a 1-mm wide zone of non-inflamed connective tissue, the lesion in the peri-implant tissue in most situations extended to the alveolar bone.
- Contained larger proportions of neutrophil granulocytes and osteoclasts than lesions in periodontitis.

Albouy et al.

• the amount of bone loss that occurred following ligature removal was significantly larger at implants with a modified surface than at implants with a turned surface and at teeth



Conclusion

- Peri-implantitis lesions are poorly encapsulated, extend to the marginal bone tissue, and may, if allowed to progress, lead to the loss of the implant.
- The large numbers of neutrophils in the peri-implantitis lesion and the absence of an epithelial lining between the lesion and the biofilm, indicate that the peri-implantitis lesions have features that are different from those of periodontitis lesions.
- Progression of peri-implantitis is more pronounced at implants with rough than at those with smooth surfaces.
- The plaque that had formed in the deep "pockets" was similar at tooth and implant sites, and was dominated by Gram-negative and anaerobic species

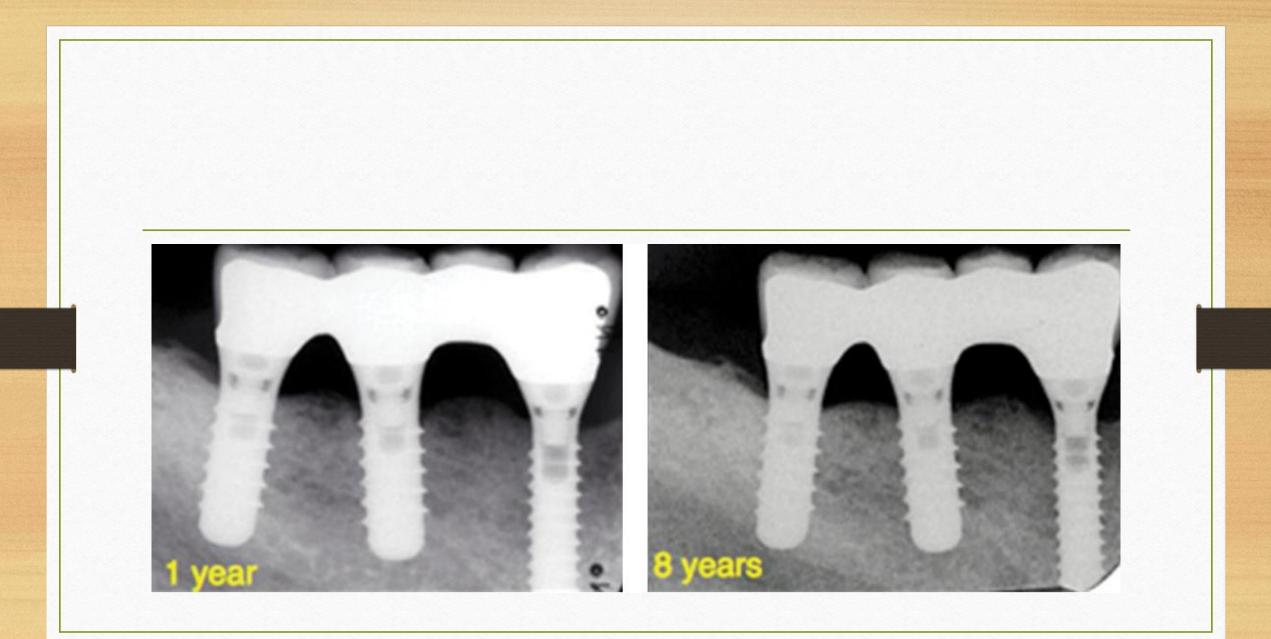
Prevalence of Peri-implantitis

- Variable values
 - ✓ Threshold level for bone loss & PD
 - ✓ Follow-up protocols
- Peri-implant Mucositis: 50% of implants
- Peri-implantitis: 12% and 43% of implant sites 10% of implants

(Zitzmann and Burglundh, 2008)

(Zitzmann and Berglundh, 2008)

(Mombelli et l. 2012)







- Systematic monitoring of peri-implant tissues is recommended over time.
- Clinical parameters can be easily used to assess peri-implant condition.
- The follow-up measurements must be compared to baseline values at the time of prosthesis placement.



- Plaque assessment
- Bleeding on probing (dichotomic measurement)
- Probing depth (Light forces (0.2–0.25N)
 - PD from 2-4mm but deeper baseline values may be found
- Suppuration
- Implant mobility: indicates failure, no predictive value
- Radiographic evaluation
 - Long-cone paralleling technique
 - At baseline and 1 year follow-up, then adapted to individual clinical assessment

Radiographic evaluation

- To verify the lack of complications during the bone healing process,
- To better understand the reasons for the pathological condition,
- For follow-up of the periimplant bone level.

Risk indicators

Consensus Report of the Sixth European Workshop on Periodontology concluded that risk indicators for PI:
(1) poor oral hygiene, or unhygienic suprastructure
(2) a history of periodontitis,
(3) diabetes,

- (4) cigarette smoking,
- (5) alcohol consumption
- (6) implant surface.

Most of these risk factors (1 to 4) have been recognized and reported in the literature.

The report suggests that although data for the latter two risk factors (5 and 6) are limited, they appear to be relevant to peri-implantitis

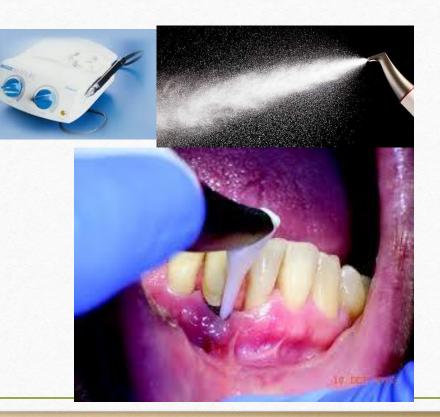
Treatment of Peri-implant mucositis

Non-surgical therapy

- Mechanical debridement
 - ✓ Manual plastic/titanium scalers
 - \checkmark Ultrasonic scalers with carbon fiber tip
- Polishing
- Antiseptics
- Er:YAG laser

Treatment of Peri-implant mucositis

- Non-surgical therapy
 - Polishing :
 - ✓ Rubber cup with toothpaste or polishing paste
 - Airpolishing with sodium
 bicarbonate or hydroxyapatite
 particle



Treatment of Peri-implant mucositis

- Non-Surgical treatment
 - Mechanical debridement
 - Polishing
 - Antiseptics
 - \checkmark Mouth rinses
 - \checkmark Subgingival irrigation with 0.2% chlorhexidine irrigation/gel
 - ✓ Chlorhexidine chips

Treatment of Peri-implantitis

- Non-surgical therapy + local antibiotics
- Surgical therapy + systemic antibiotics
- Surgical treatment includes full-thickness flap elevation for access followed by degranulation, surface debridement by laser or mechanical instruments, surface decontamination with laser or antimicrobials, and bone augmentation.
- Implantoplasty



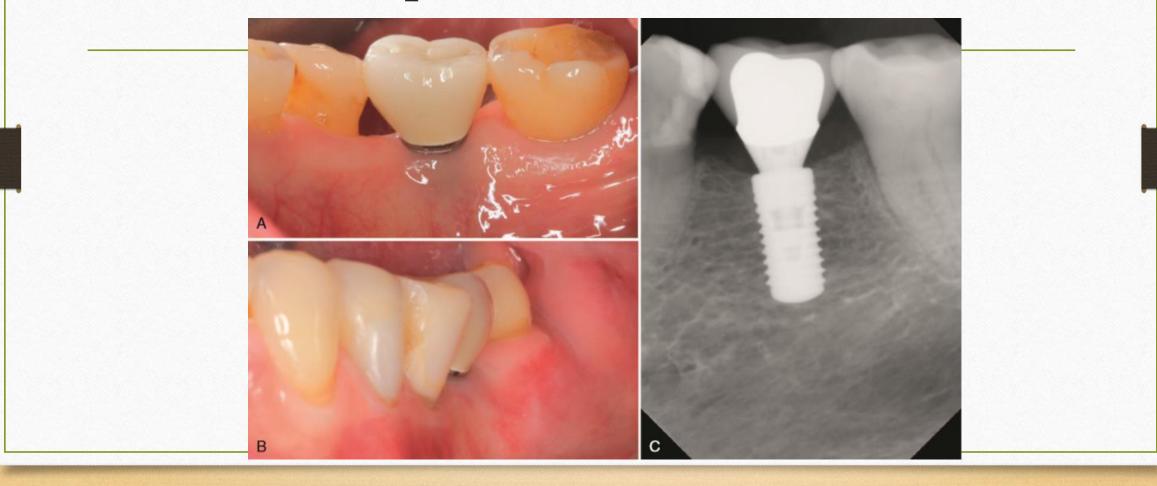
Surgical treatment of peri-implantitis

- Currently available scientific data is insufficient to suggest which treatment intervention of periimplantitis is most effective and to allow any specific recommendations for the use of locally or systematically administered antibiotic.
- Implant surface decontamination/disinfection remains challenging especially for implants with roughened surfaces.
- For some treatment modalities, recurrence of periimplantitis appears to be high (up to 100%) after 1 or more years of treatment and retreatment may be necessary.
- Surgical access appears to be necessary to arrest periimplant bone loss.
- Surgical treatment may result in gingival recession and compromised esthetics.
- At sites with high esthetic demands, definitive treatment of periimplantitis may include the removal of the implant, grafting of the site and placement of another implant.

Implant maintenance



Implant Maintenance



Implant maintenance



Buccal probing of implant impeded by implant restoration.