

# Soft tissue management in implant dentistry and peri- implant disease

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# Gingival aesthetics

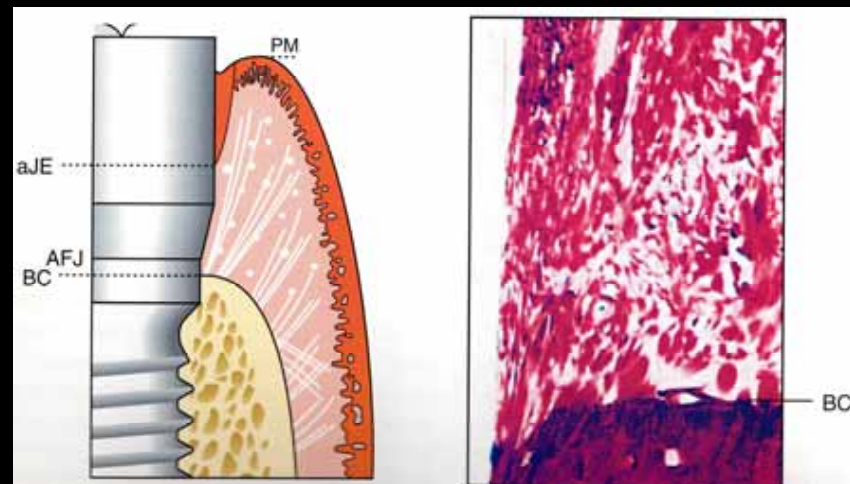
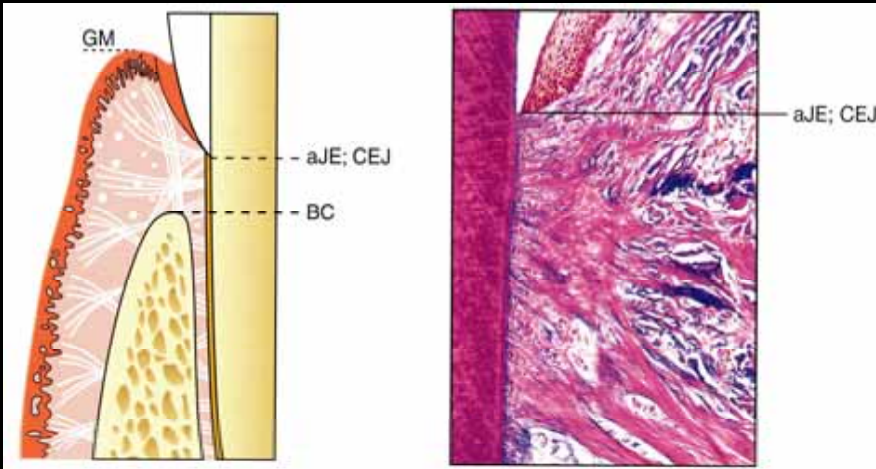


- Form
- Contour
- Absence of disease
- Harmony with dentition

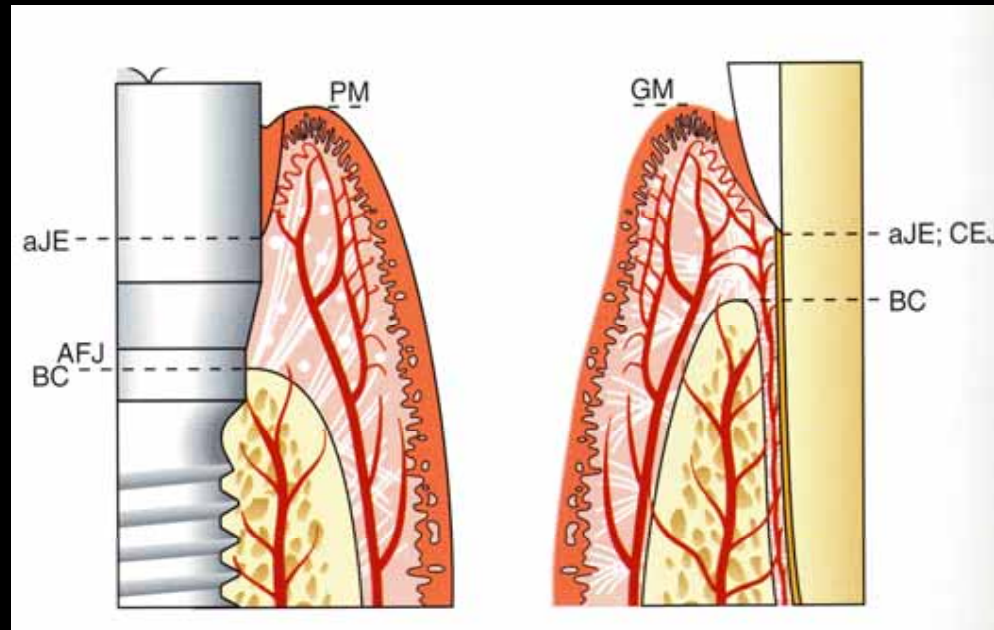
## Healthy peri-implant mucosa

- Absence of cementum
- Collagen fibres
  - run parallel to the implant surface
  - originate from the bone surface
- Supracrestal connective tissue ~2mm

Berglundh et al 1991



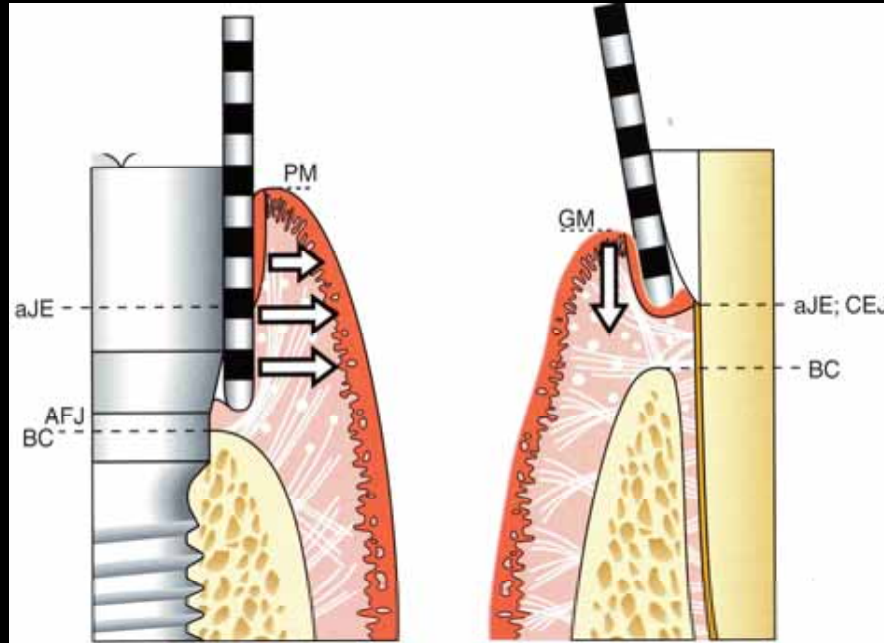
## Vasculature



- Periodontal ligament vessels
- Crevicular plexus in both

Burglundh et al 1994

# Probing



- Clinically healthy implants probed ~2mm
- Clinically healthy teeth probed ~0.7mm

Ericsson & Lindhe 1993

## Effects of plaque

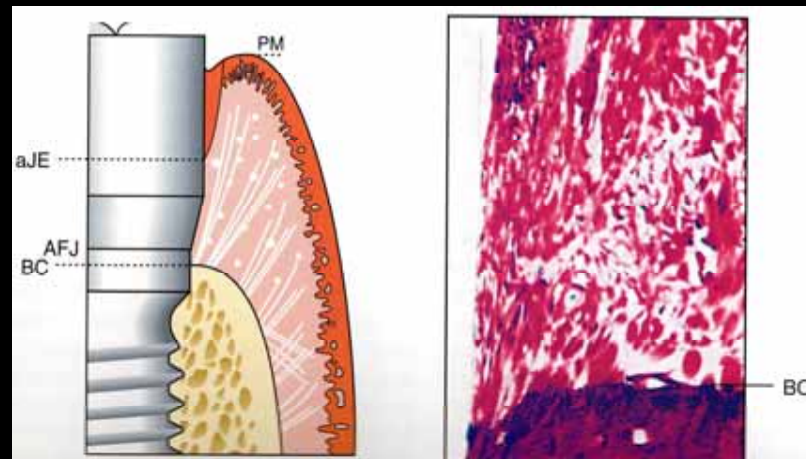
- Teeth and implants have similar response to de novo plaque Berglundh et al 1992
- Pontoriero 1994 clinical trial: the need for ST at implant sites as important as teeth
- Apical extent of inflammatory infiltrate is > in implant sites

## Ridge mucosa

- 2-4mm thick, keratinised epithelium
- Lined by CT rich in fibroblasts, collagen fibres and vascular structures
- CT is continuous with the cortical bone crest, via the periosteum
- Few scattered inflammatory cells adjacent to basement membrane, in CT papillae between rete pegs of epithelium

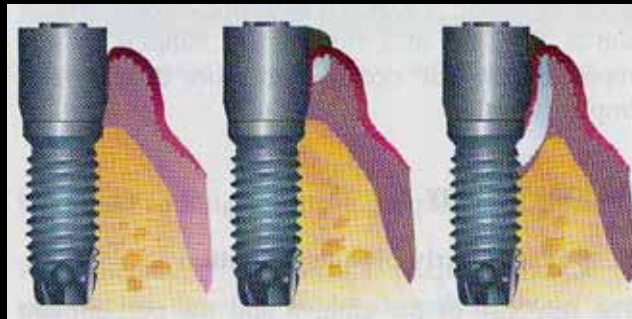
# Peri-implant mucosa

- Transmucosal passage around the abutment
- Tissue adapts to the functional demands



## Peri-implant disease

- Inflammatory process in the tissues surrounding an implant (Albrektsson & Isidor 1994)
- Peri-implant mucositis- reversible in soft tissues
- Peri-implantitis- additionally, bone loss



# Peri-implant mucositis

- Clinical features
  - Similar to gingivitis around teeth
  - Morphology of mucosa and lack of light transmission through implant may mask inflammation
  - Assessment should include bleeding on probing

## Prevalence

- BOP good indicator of peri-implant mucositis
- BOP data are infrequently reported
- Studies range from 73-90% of implant sites

# Histopathology

- Zitzmann et al 2001
  - 12 subjects with periodontal and implant sites
  - Stopped plaque control for 3/52
  - Clinical exam & soft tissue biopsies
  - Size of lesion: 0.3mm<sup>2</sup> teeth and 0.2mm<sup>2</sup> implants
  - More B cells and neutrophils in gingiva than peri-implant mucosa

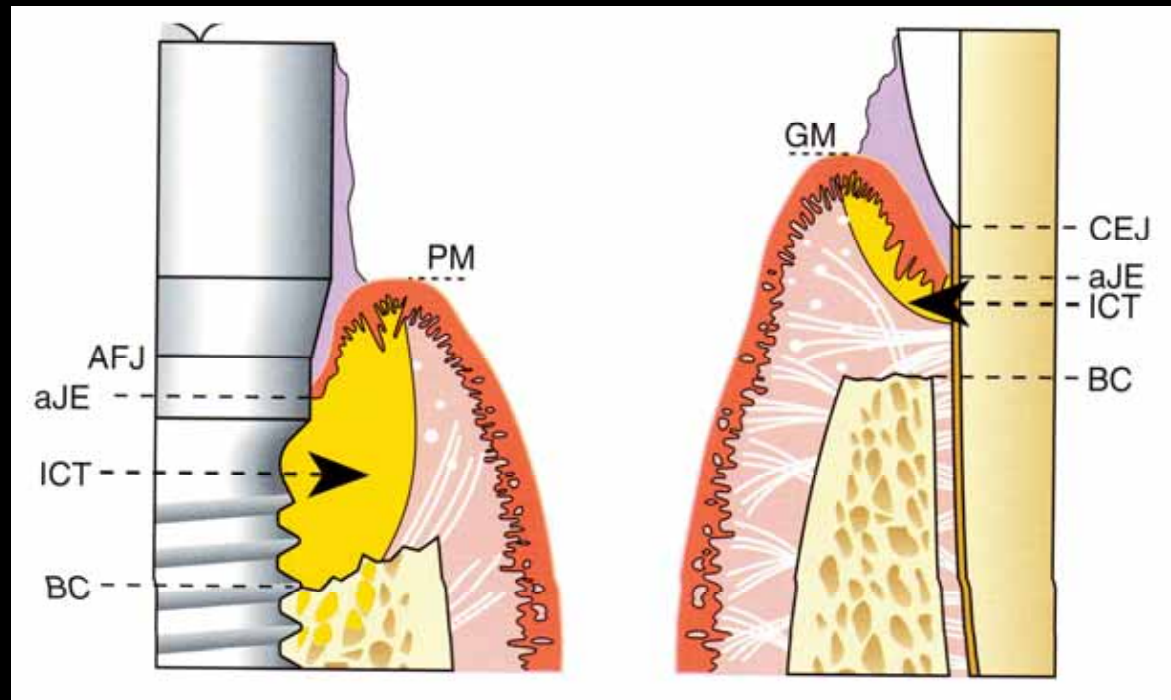
## Histopathology

- Berglundh et al 1992
  - Dog model implant vs teeth, 3/52 of plaque accumulation
  - Plaque accumulation teeth = implants
  - Inflammatory lesions teeth = implants
- Ericsson et al 1992
  - After 3/12, implants sites progressed further apically than tooth sites

## Response to long-standing plaque accumulation

- Ericsson et al 1992
  - Peri-implant mucosa- less fibroblasts than teeth
  - Teeth: breakdown is compensated for by repair
  - Implants: breakdown not compensated for by repair

# Peri-implantitis



## Clinical features

1. Inflammatory lesion in the peri-implant mucosa and
2. Loss of peri-implant bone
  - BoP + bone loss on radiographs
  - Probing essential to assess implant tissues

# Prevalence

- Initially thought to be 5-10%
- Lack of data from studies
  - Most studies report implant failure
- Fransson et al 2005
  - 662 subject based study- 5yrs function of implants
  - 1346 radiographs from Brånemark clinic

## Fransson et al 2005 & 2007

- Bone loss (between 1- >5yrs) defined as  $\geq 3$  threads
- 27.8% (184) of 662 had bone loss
- Out of 3412 implants, 423 had progressive bone loss (12.4%)
- Concluded: subject based data revealed higher prevalence of bone loss
- 94% of implants with bone loss had BoP

## Roos-Jansåker et al 2006

- 216 pts, after 9-14yrs of function
- 16% of subjects had peri-implantitis, however if same criteria as Fransson used (bone loss + BoP), prevalence would be >43%

## Conclusion: prevalence

- Recent studies
  - Prevalence of peri-implantitis: 25-45%

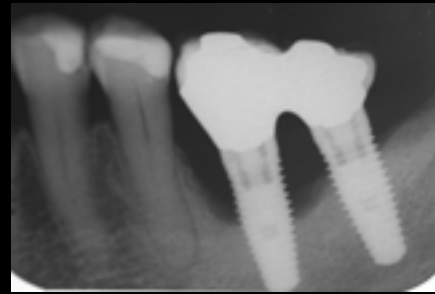
## Histopathology

- 65% of CT portion occupied with inflammatory lesion
- Macrophages, lymphocytes and plasma cells and also polymorphs
- Lesions extended into alveolar bone

## Conclusion

- Peri-implantitis lesions are poorly encapsulated and extend into the marginal bone tissue
- Absence of epithelial lining between plaque biofilm and lesion
- More pronounced on rough surfaces

## Deep pockets

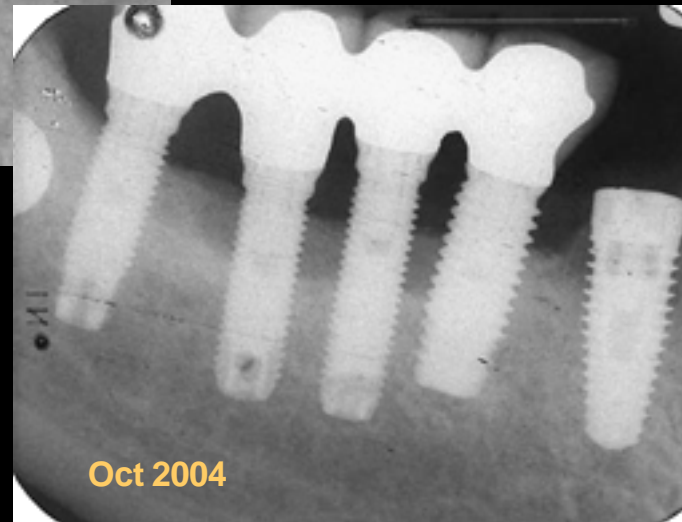
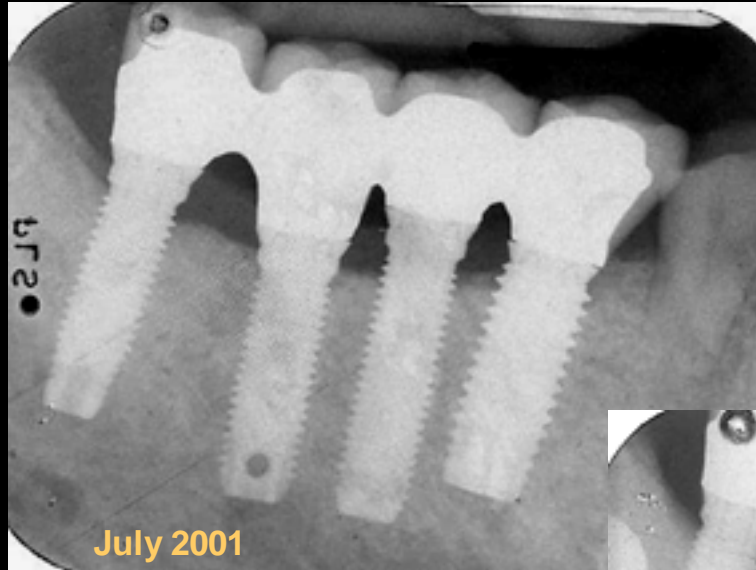




Bone loss vs fibrous  
encapsulation



# Longitudinal monitoring of bone levels



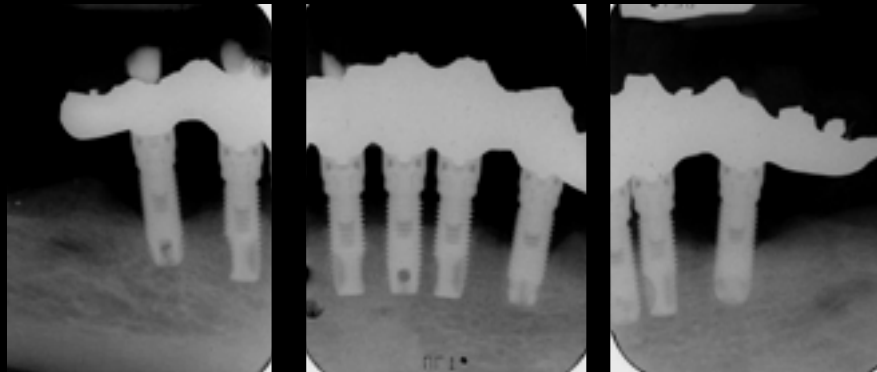
Peri-implantitis

# Bone loss

2001



2004



## Implants vs teeth

1. Tissue destruction: implants > teeth
2. Soft tissue lesion: implants > teeth
3. Peri-implant lesion often extended into the bone marrow, but not at teeth

Lindhe et al 1992

- The defence mechanism in the gingiva appears better than peri-implant mucosa

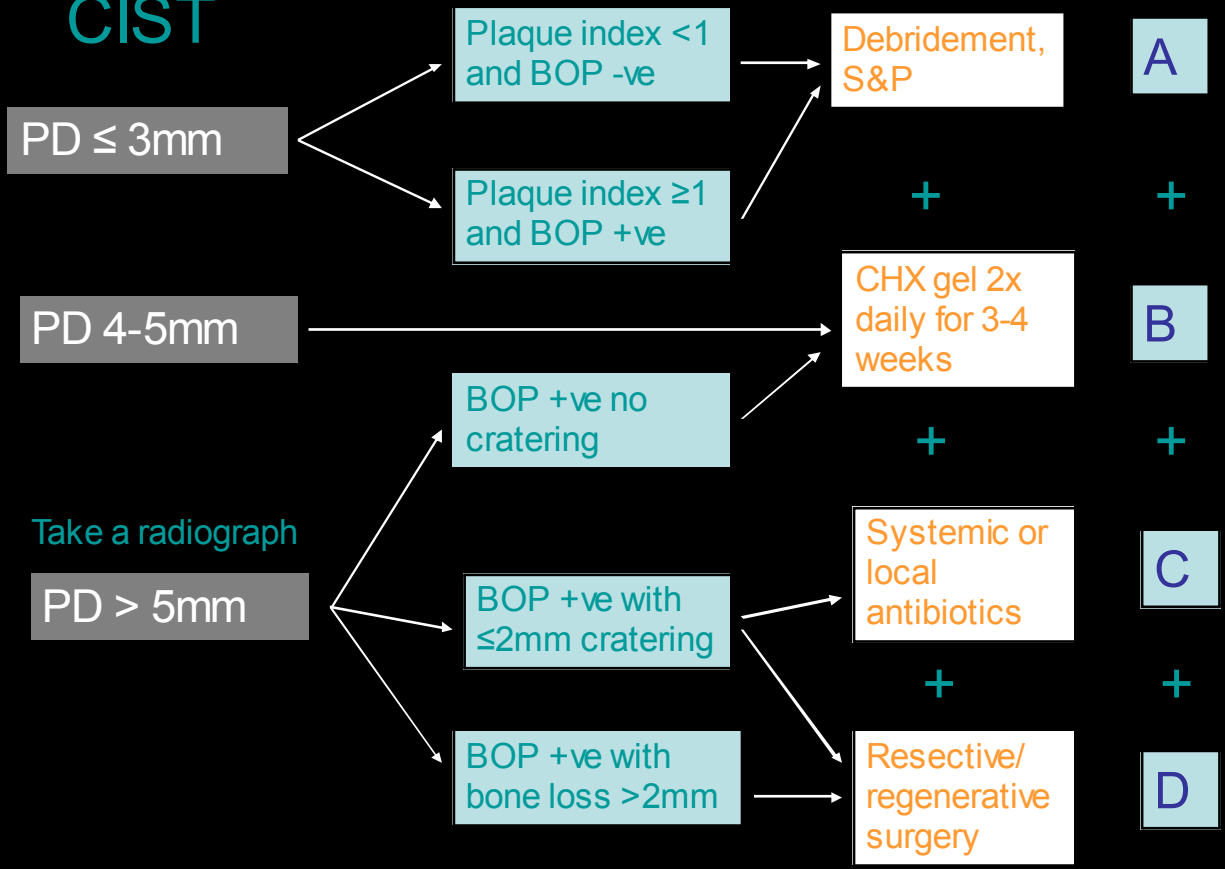
## Conclusions

- Implants:
  - 2mm junctional epithelium
  - 1mm CT attachment
- Soft tissue protection zone as with teeth
  - different CT composition
  - Different vascular supply apical to epithelium

# Treatment of Peri-implant lesions

- Diagnostic process
  - BoP
  - Suppuration
  - PD (4 surfaces)
  - Radiographic bone loss
  - Implant mobility

# CIST



## Soft tissue

- Level of tissue
- Thickness
- Bone
- Implant position
- Prosthetic emergence

# Tissue level prior to extraction

- Same, incisal or apical



# Tissue thickness

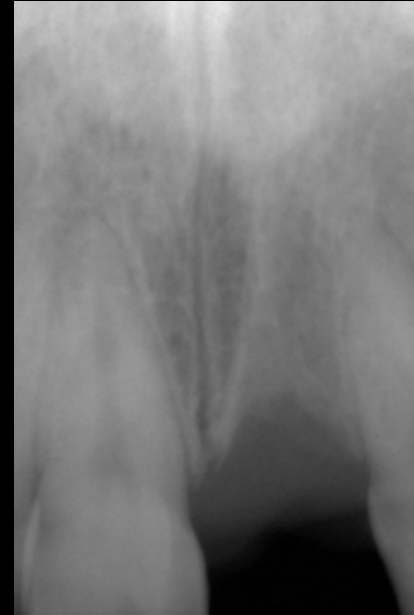
- Kan 2000 JOMI



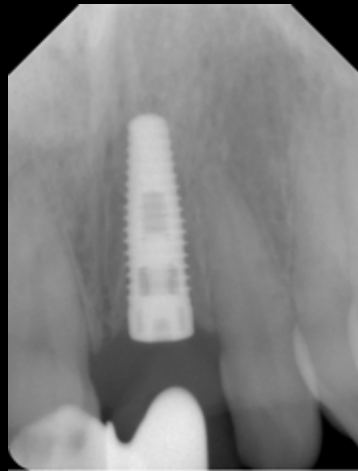
## Bone levels

- 3mm from FGM 85%
- <3mm 10%
- >3mm 5%

Kois 1994

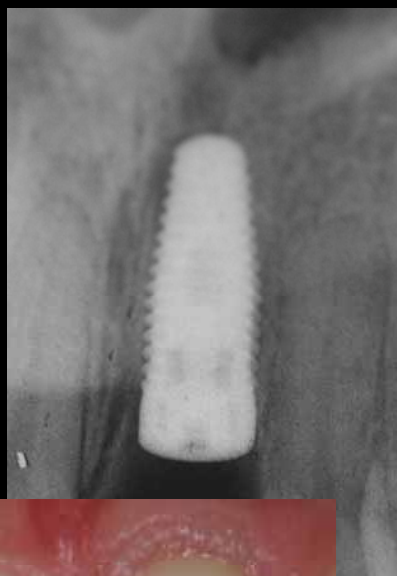


# Implant position



# Prosthetic emergence







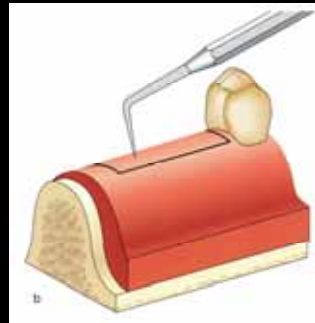
## Bone loss

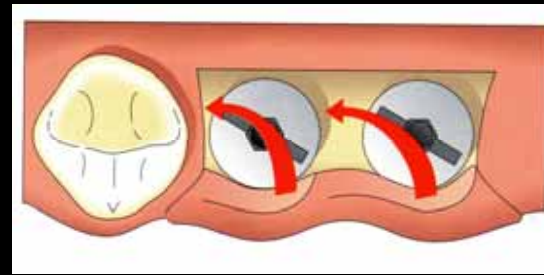
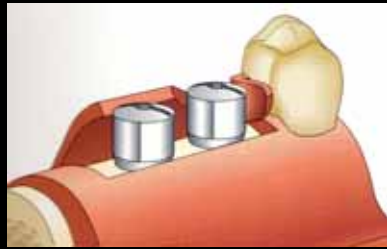
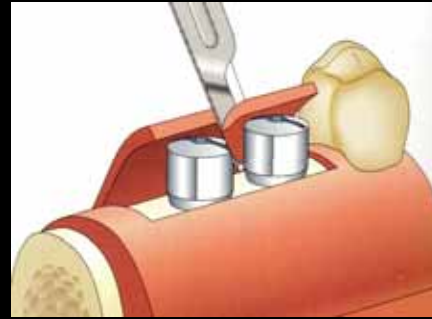
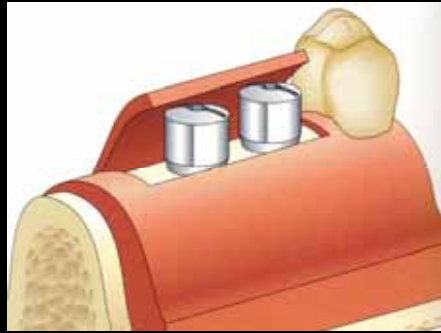
- Low bone is unpredictable ( $>3\text{mm}$ )
- Bone loss in “V” shape: more predictable
- Bone loss in “U” shape: less predictable

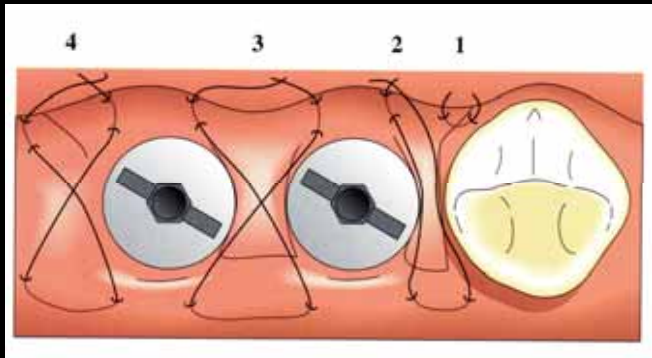
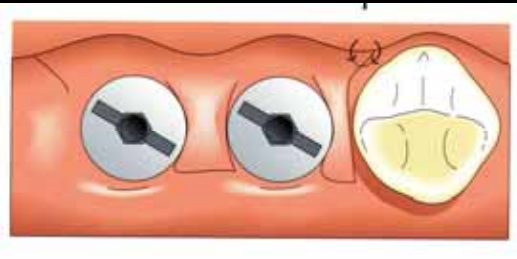
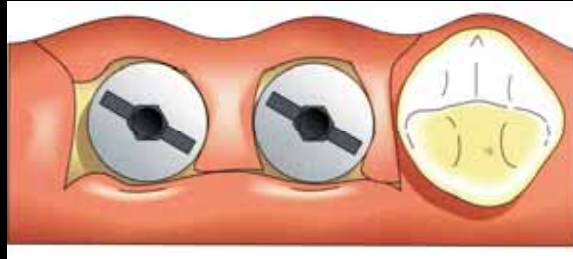
## Orthodontic extrusion

1. Vertical bone is good
2. Proclined bone is poor (bone doesn't come down easily)

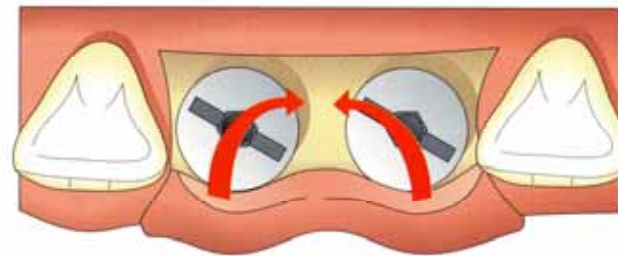
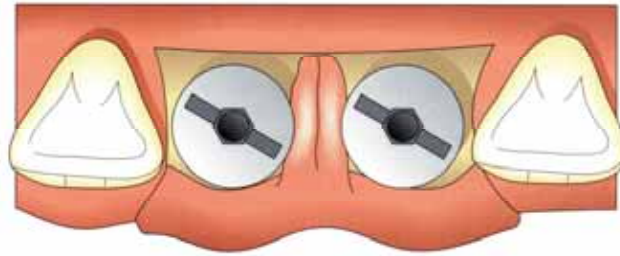
# Multiple implants



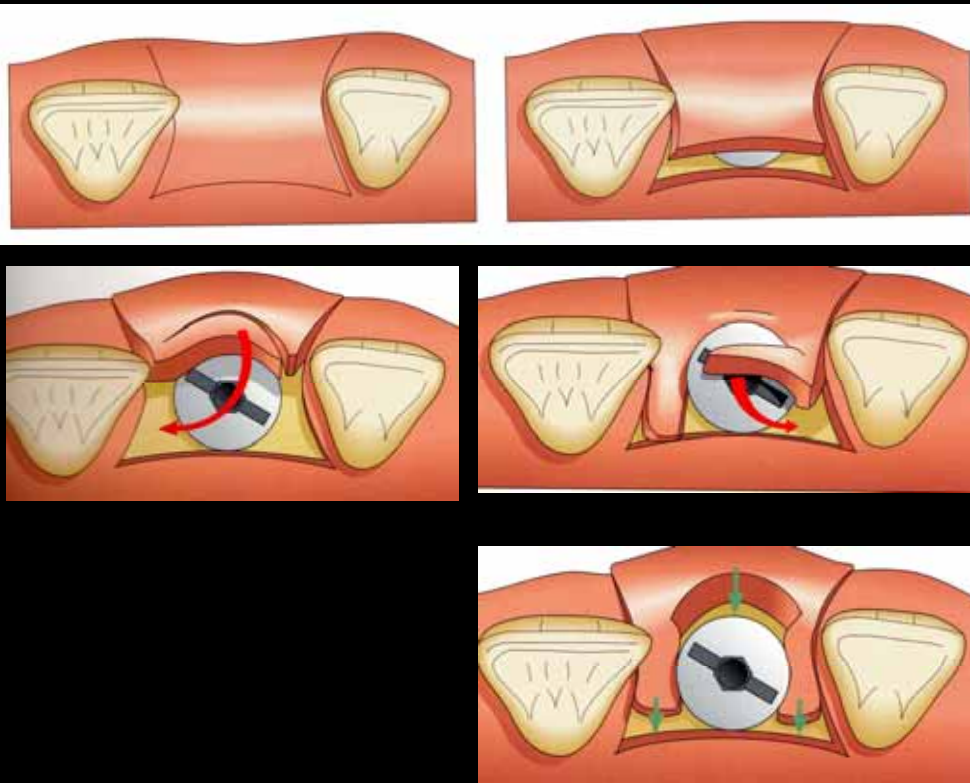


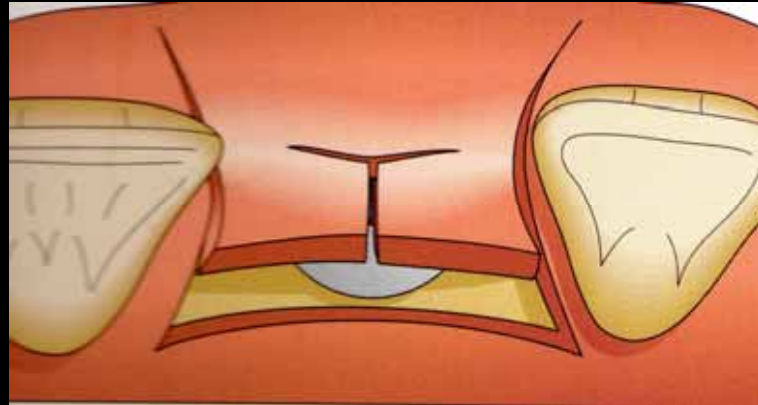


## Multiple implants

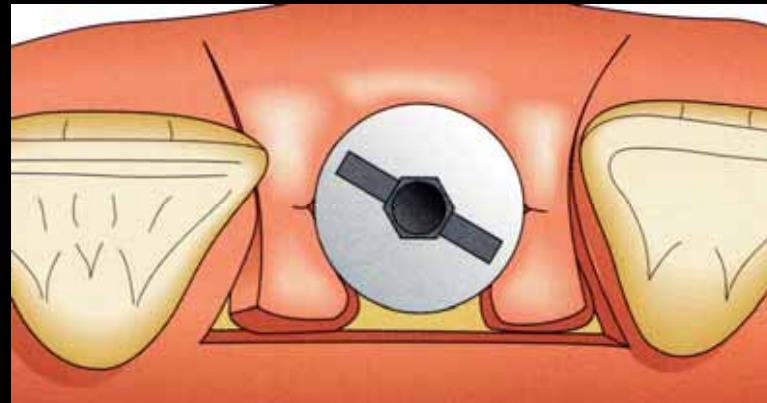


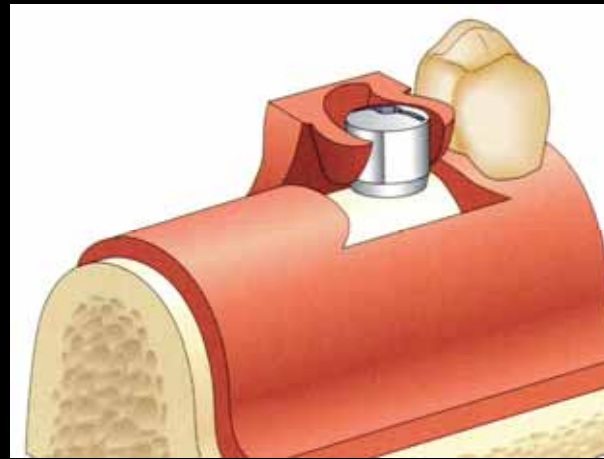
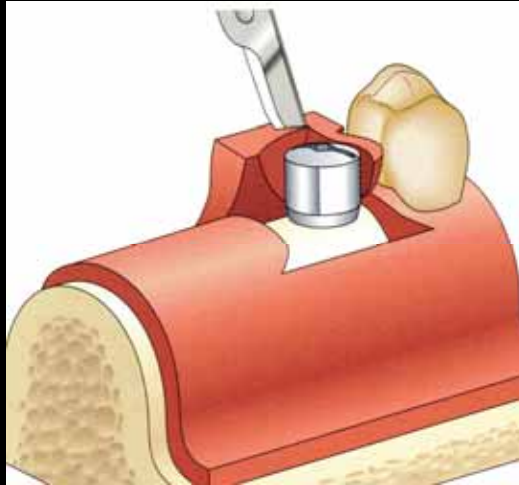
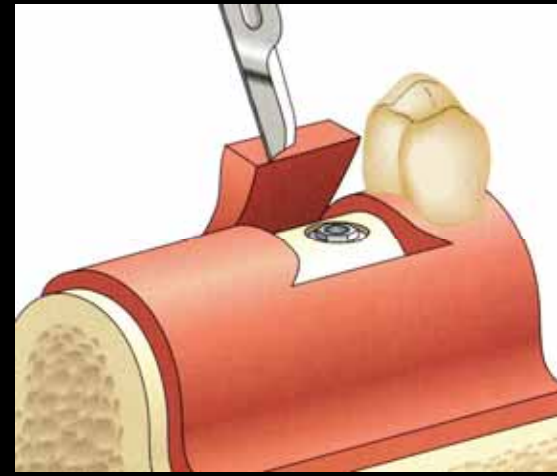
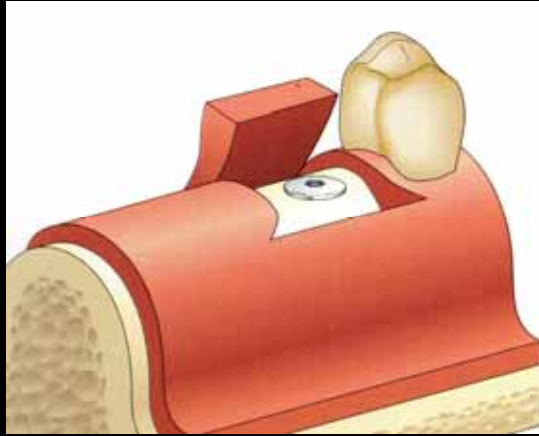
# Single implants





Single  
implants





## Papillae between teeth

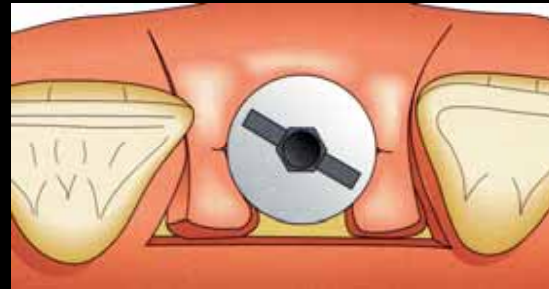
- When crest to contact point is  $\leq 5$ mm  
papilla present 100% of the time
- When crest to contact point is 6mm papilla  
present 56% of the time
- When crest to contact point is  $\geq 7$ mm  
papilla present  $\leq 27\%$  of the time

*Tarnow et al 1992*



# Papillae

- No inserted supracrestal fibres
- Shape differential
- Coronal extent of alveolar crest
- Contact point to crest distance



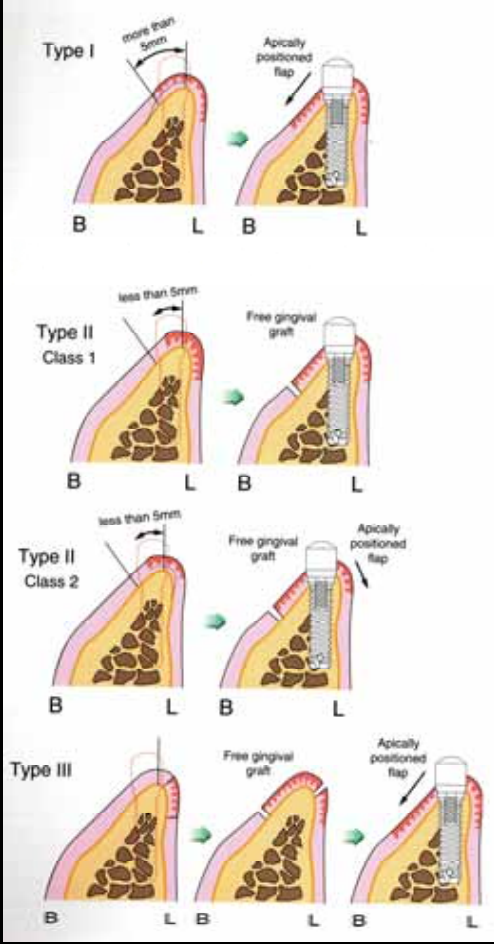
## Distance between implants

- Lateral bone loss 1.34-1.4mm
- Implants <3mm apart will loose 1.04mm of crestal bone ht
- Implants >3mm apart will loose 0.45mm of crestal bone ht

*Tarnow et al 2000*

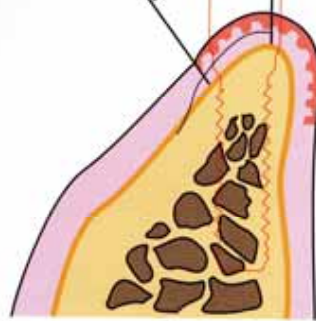
## Buccal recession

- After abutment connection, ~1mm tissue loss over 12m
- Most recession occurs in 3m
- Therefore wait  $\geq 3m$  before impression or final abutment



Type I

more than  
5mm

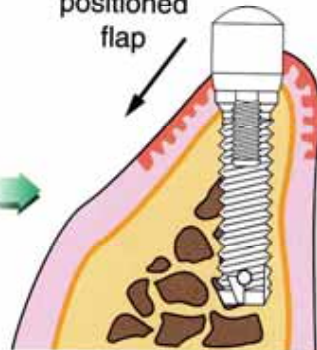


B

L

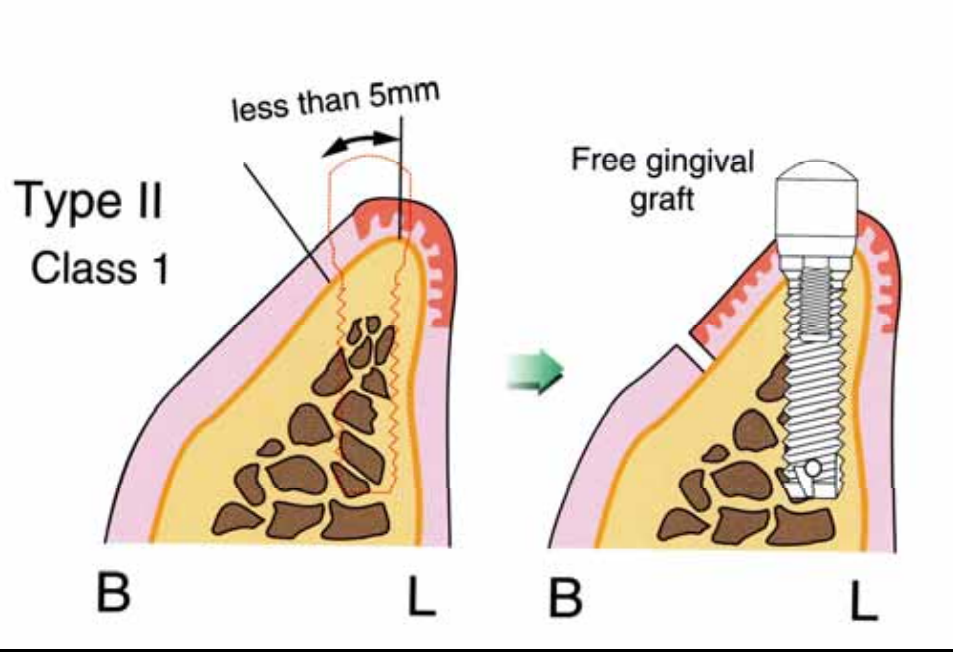


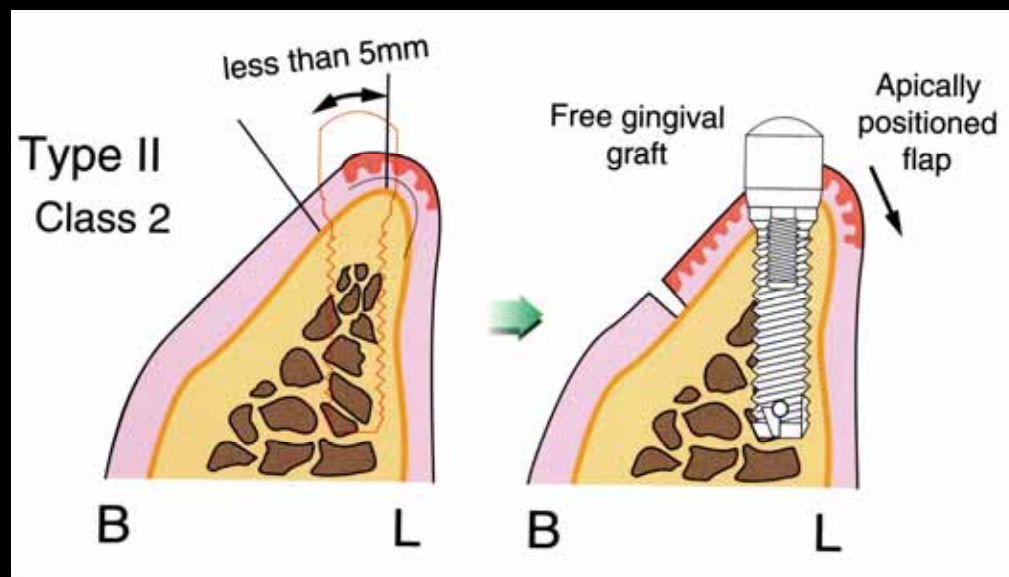
Apically  
positioned  
flap

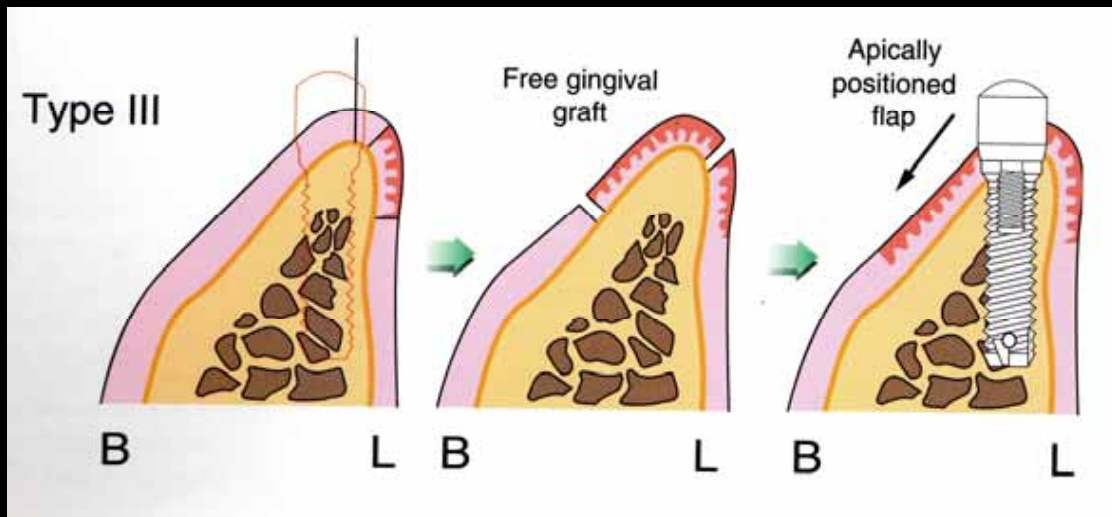


B

L







Any questions?

