

Current Concepts in

Periodontal

Diagnosis and Therapy

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Overall Course Objectives

By the end of today's session, you should be able to:

- diagnose the most common periodontal diseases and conditions likely to be encountered
- come up with a treatment plan for initial therapy to manage these cases
- evaluate the results of initial therapy, and where to go from there

Overview



1. Introduction
2. Diagnosis and Initial Therapy
3. Evaluation of Initial Therapy

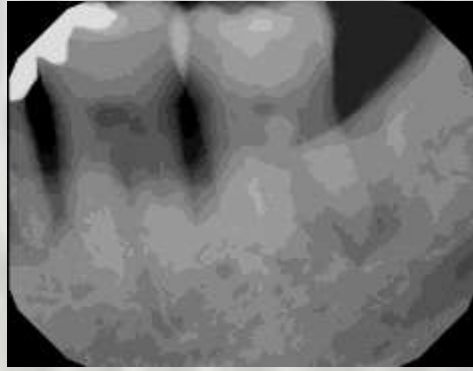
Overview

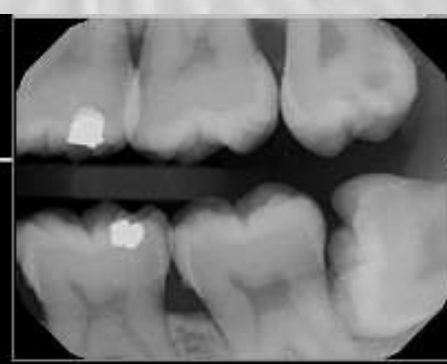


I. Introduction





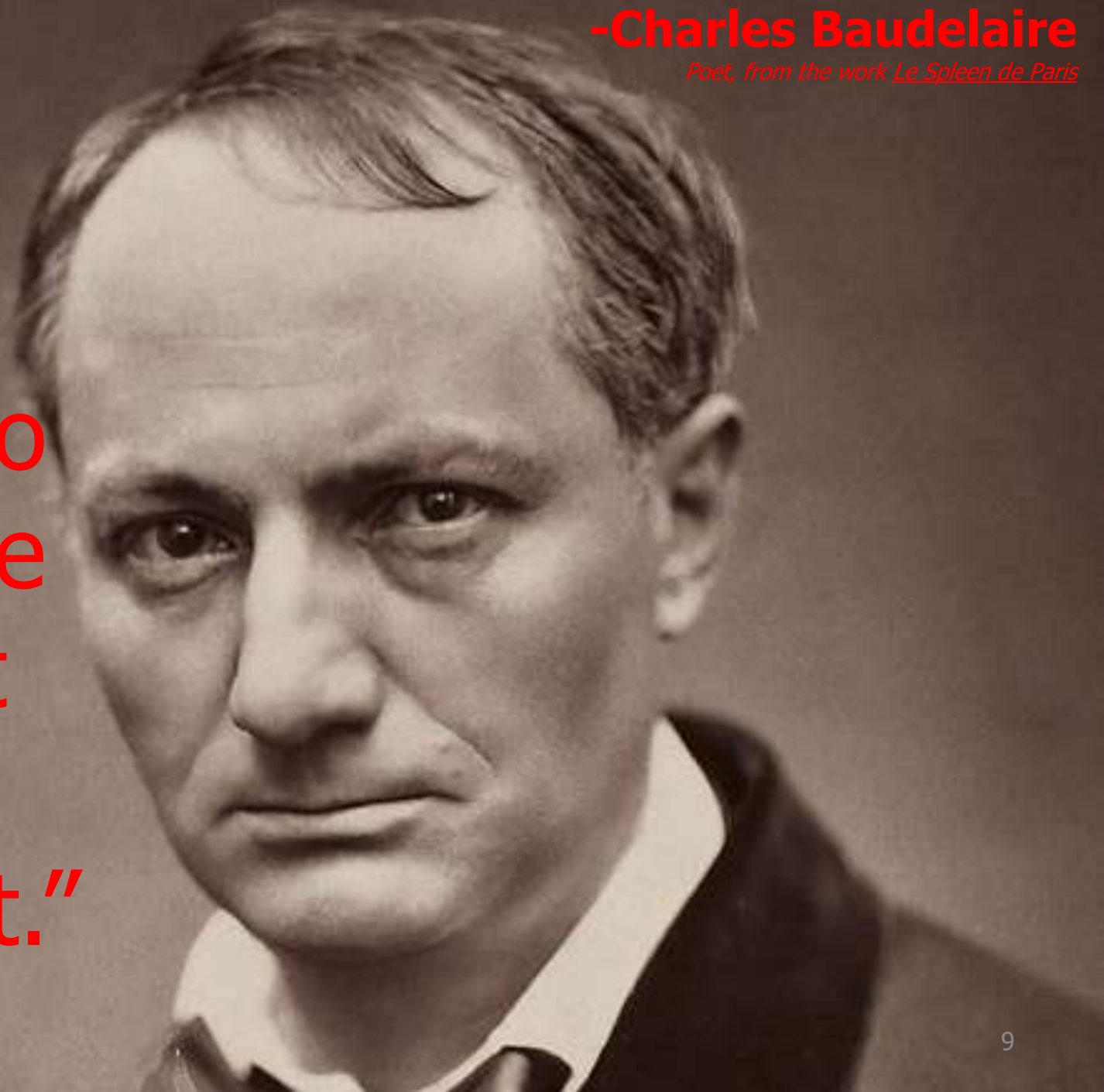




-Charles Baudelaire

Poet, from the work Le Spleen de Paris

“The
devil’s
finest
trick is to
persuade
you that
he does
not exist.”



Overview



1. Introduction
2. Diagnosis and Initial Therapy

Overview



1. Medical History
2. PSR
3. The Odontogram
4. Radiographs

Overview



I. Medical History

Medical History

- A detailed and comprehensive review of medical history is beyond the scope of this course
- However, there are three areas commonly picked up in a medical history which can aid significantly in diagnosis and initial management of periodontal disease

Diabetes Mellitus

- If your patient reports diabetes, you **MUST** find out how well that diabetes is controlled
- It is not enough for a patient to tell you, “Oh yeah, I take my meds, so it must be under control.”
- What is the most objective measure physicians use to monitor metabolic control?

НБА I с!

HbA1_c

- HbA1_c is a more chronic measure of glycemic control than is plasma glucose, which gives information about a snapshot in time.
- HbA1_c values are generally able to give information regarding a patient's level of metabolic control over the previous 3 months

HbA_{1c}

- HbA_{1c} is a more chronic measure of glycemic control than is plasma glucose, which gives information about a snapshot in time.
- HbA_{1c} values are generally able to give information regarding a patient's level of metabolic control over the previous 3 months

HbA1_c

- For HbA1_c:
 - <6% is considered normal
 - Between 6% and 7% is considered good control
 - Between 7% and 8% is considered acceptable control
 - >8% is considered unacceptable control

Why?

Uncontrolled Diabetes Mellitus

- Because uncontrolled DM leads to:
 1. More gingival inflammation¹
 2. A higher prevalence of periodontitis^{2,3,4,5,6,7}
 3. An up-regulation of pro-inflammatory cytokines⁸
 4. An increase in the activity of connective-tissue-eating enzymes⁹

Cigarette Smoking

- Ask your patients if they smoke, and if yes:
 - What do they smoke?
 - How many of those do they smoke per day?
 - For how many years have they been doing this?

Cigarette Smoking

- Patients who smoke:
 1. Have more plaque accumulation¹⁰
 2. Have more bone loss, and this is dose- dependant¹¹
 3. Have more attachment loss, and this is dose-dependant¹²
 4. Have a worse response to scaling and root planing compared to non-smokers^{13,14,15}
 5. Have a worse response to surgical therapy compared to non-smokers^{14,15,16}

Medications

- Certain classes of
 - Ca^{++} -channel blockers (think nifedipine)
 - Anticonvulsants (think phenytoin)
 - Immunosuppressants (think cyclosporine)can lead to a diagnosis of gingival overgrowth¹⁷

Overview



1. Medical History
2. PSR

PSR

- PSR stands for Periodontal Screening and Recording
- It is a **SCREENING TOOL, NOT** a diagnostic tool
- Every patient gets a PSR at their initial visit, and those who do not need complete periodontal charting get one at every recall appointment

PSR

- The purpose of the PSR is to see if a patient warrants further investigation into their periodontal status
- As a screening tool, a PSR is quick to use and interpret
- It **does not** tell you if a patient has periodontitis

WHO Probe

- A PSR is performed with a WHO probe



- The markings are:
 - Ball at the end: 0.5mm
 - Beginning of black area: 3.5mm
 - End of black area: 5.5mm

Performing a PSR

- The mouth is divided into sextants

Teeth 18-14
Sextant 1

Teeth 13-23
Sextant 2

Teeth 24-28
Sextant 3

Teeth 48-44
Sextant 6

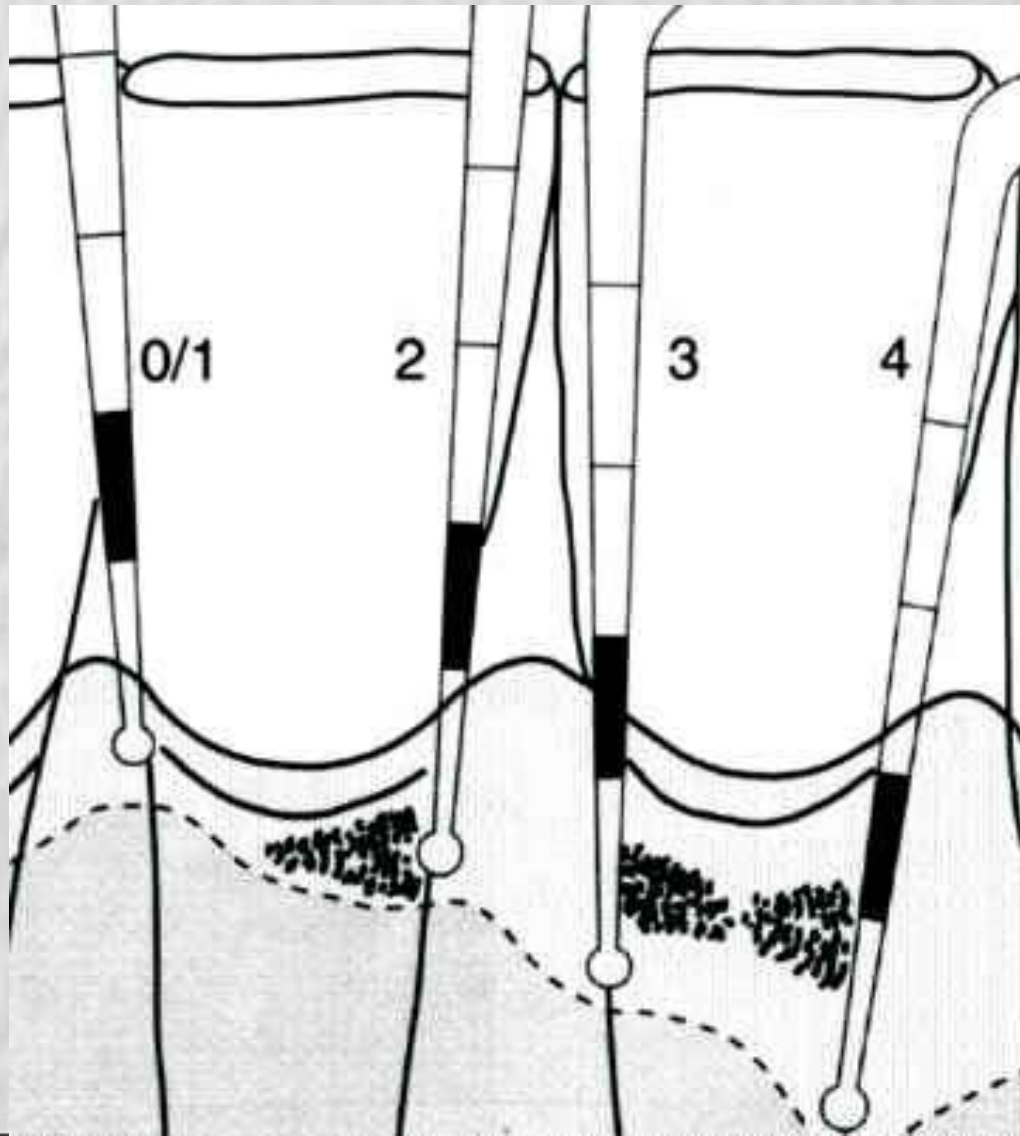
Teeth 43-33
Sextant 5

Teeth 34-38
Sextant 4

Scoring a PSR

- At each location that you probe:
 - 0** if there is no bleeding, no calculus, and you are not yet into the black area
 - 1** if there is bleeding, but no calculus and you are not yet into the black area
 - 2** if there is bleeding and calculus, but you are not yet into the black area
 - 3** if you are into, but not completely covering the black area
 - 4** if you are completely covering the black area

Scoring a PSR



Scoring a PSR



Scoring a PSR

- The WHO probe is walked circumferentially around each tooth in the mouth
- The highest score of any tooth surface in a given sextant is the score for that sextant

Scoring a PSR*

- The presence of:
 - positive recessions >3.5mm,
 - inadequate attached gingiva,
 - mobility, and
 - furcation involvements

that are picked up during a PSR are denoted by adding an asterisk* after the score for a particular sextant

Examples

3	2	3
3	2*	3

4*	1	2
2	1	2

2	2	2
2	2	3*

1	0	2
2	0	1

Decision Time!

- If you have:
 - Two or more scores of 3, or
 - One or more scores of 4

then you **MUST** do complete periodontal charting

- If you have one score of 3, or an *, you can do periodontal charting in that sextant alone
- Scores of 2 or less do not require periodontal charting

Examples

3	2	3
3	2*	3

Needs complete periodontal charting

4*	1	2
2	1	2

Needs complete periodontal charting

2	2	2
2	2	3*

Needs periodontal charting in the 4th sextant

1	0	2
2	0	1

Does not need periodontal charting

The Complete Periodontal Chart

- Patients who need all measures recorded **must** have them recorded:
 1. At the initial periodontal examination
 2. At the evaluation of initial therapy (after initial therapy has been completed)
 3. At every maintenance appointment, along with the scaling/oral hygiene instruction/appropriate radiographs (which are supposed to be done at every recall appointment)

Overview



1. Medical History
2. PSR
3. The Odontogram

The Odontogram

- Patients who need a complete periodontal charting need an odontogram to be filled out
- This can be done by hand on paper, or on a computer program

The Odontogram

- The important clinical measures to note are:
 - Probing depth
 - Recession
 - Attachment level
 - Bleeding on probing
 - Plaque and calculus
 - Furcation involvement
 - Mobility
 - Attached gingiva

A Note on Recession

- Recession is a measure of the distance between the free gingival margin and the CEJ
- It is measured at the same six sites per tooth that probing depth is measured
- It can be positive, negative, or 0
- It is called Recession on the odontogram

Measuring Recession

- A recession value can be:
 - A. 0 if the free gingival margin is at the level of the CEJ
 - B. - if the free gingival margin is coronal to the CEJ
 - C. + if the free gingival margin is apical to the CEJ



Obviously Positive Recession



Obviously Negative Recession



When Recession is Negative....

- When the CEJ is covered by the free gingival margin, there is a strong temptation to record the recession as 0 or -1 automatically, rather than actually checking for the location of the CEJ with the tip of the probe
- This is inappropriate, and can lead to over-diagnosis of periodontal disease, and by extension, over-treatment

What if there's no CEJ?

- What happens if the CEJ has been obliterated by a restorative margin?
- Use the restorative margin as the CEJ
- The idea is to have a fixed reference point



A Note on Clinical Attachment Loss

- All six sites per tooth once again have an associated attachment loss
- It is called Attach on the odontogram

Clinical Attachment Loss

- It is **CALCULATED** based on the following simple formula:

$$\mathbf{PD + Rec = LOA}$$

where PD is probing depth of a site, Rec is recession of a site, and LOA is loss of attachment at that site

Clinical Attachment Loss

- So if a particular site:
 - Probes 5mm and has 2mm of recession, then the loss of attachment would be $5 + 2 = 7\text{mm}$
 - Probes 5mm and has -2mm of recession (as in the free gingival margin is covering the CEJ by 2mm), then the loss of attachment would be $5 + (-2) = 3\text{mm}$

Furcation Involvement

- Furcations are pesky things
- Once a furcation involvement begins, it is very difficult to reverse
- They must be monitored very closely



The Nabers Probe

- This probe is curved to allow examination of furcations, which are often subgingival and in hard to reach places
- The markings are at 3mm



Measuring Furcations

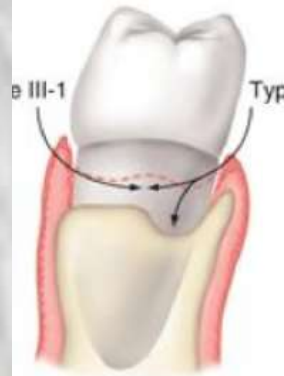
- The Glickman Furcation Classification System is the most commonly used:

Class 1 – vertical but not horizontal exposure of furcation

Class 2 – vertical and horizontal exposure of furcation

Class 3 – complete exposure of both sides of furcation

Class 4 – a Class 3 that is visible



Charting Furcations

Class 1 – is written as an open angle in the furcation on the odontogram

Class 2 – is written as a closed triangle in the furcation on the odontogram

Class 3 – is written as a filled-in triangle in the furcation on the odontogram

Class 4 – same as Class 3

Attached Gingiva

- Measure the distance from the free gingival margin to the mucogingival junction with your probe, then subtract from that your mid-buccal/mid-lingual probing depth
- An X is placed if there is ≤ 1 mm of AG
- It is called MAG on the odontogram (minimal attached gingiva)



Attached Gingiva



Attached Gingiva



**What a beautifully filled-out
odontogram!**

Overview



1. Medical History
2. PSR
3. Probing Depth, Recession, Attachment Level
4. Bleeding on Probing, Plaque, Calculus
5. Mobility and Furcation Involvement
6. Radiographs

Radiographs

- Radiographs should only be taken **AFTER** your clinical exam has been performed
- All the indications for radiographs should come from your patient history/clinical exam

Perio Indications for Radiographs

- Probing depth $\geq 4\text{mm}$
- Any + recession
- Mobility
- Furcation involvement
- Gingival pain/discomfort

Which Radiographs do I Take?

- A panoramic radiograph is a **SCREENING** tool
- It is not to be used for diagnostic purposes





K2

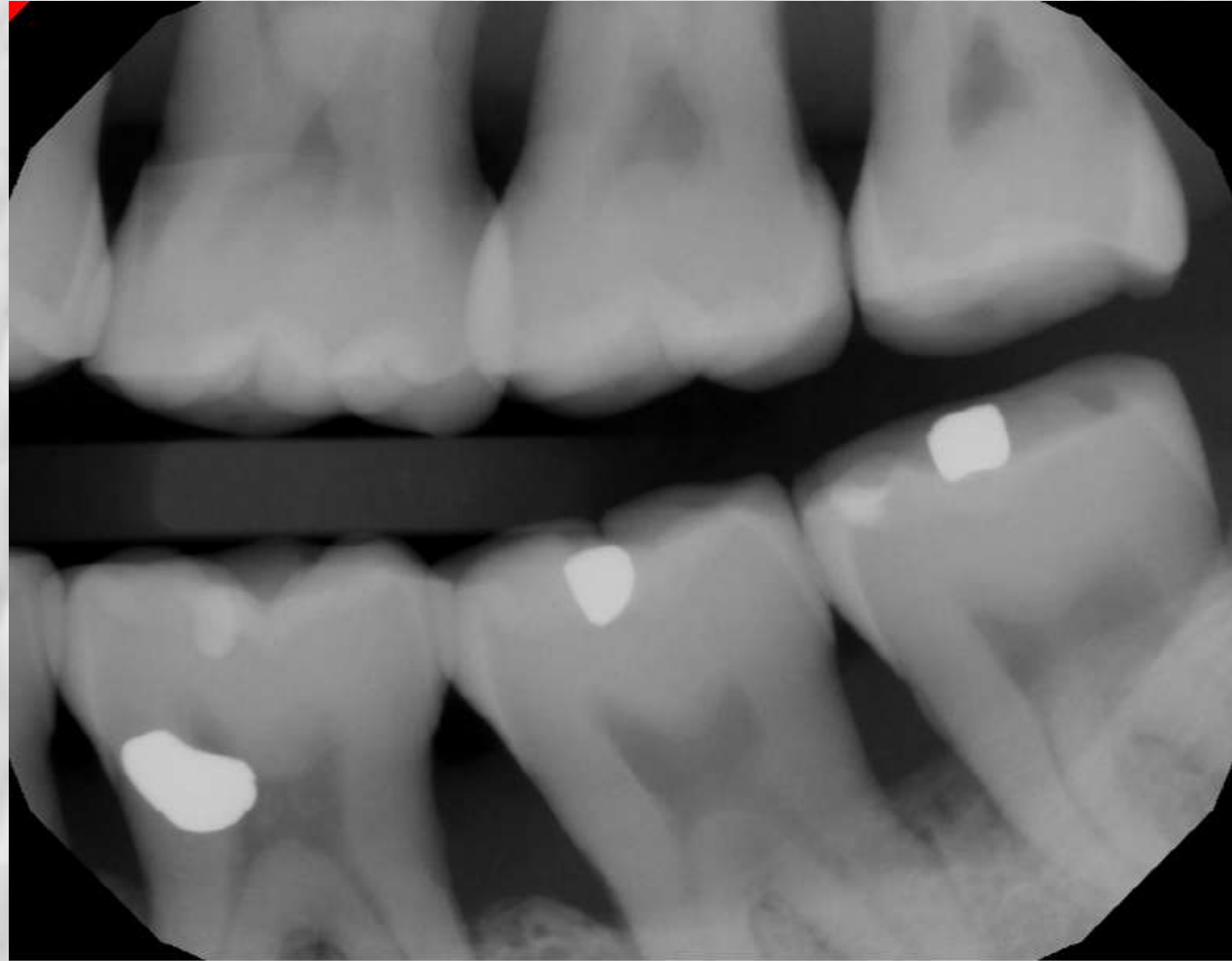
Which Radiographs do I Take?

- In the anterior, periapicals are most appropriate
- In the posterior, it depends on what you are looking at/for:
 - Bitewings give the best representation of the position of the crestal bone levels
 - Periapicals show the entire root length

– MOST OF THE TIME, YOU WILL NEED BOTH

Bitewings

- What's wrong with this bitewing?



Vertical Bitewings 😊

- Much better!



Periapicals



- What's wrong with these periapicals?

Periapicals 😊



- Much better!

What do I Look for in Radiographs?

- Radiographs should be assessed for:
 1. Presence/amount of horizontal bone loss
 2. Presence of vertical bone loss
 3. Crown:root ratio
 4. Suggestion of furcation involvement/arrows
 5. Widening of periodontal ligament space
 6. Presence of calculus
 7. Open contacts between teeth
 8. Root proximity
 9. Presence of interproximal craters

Presence/Amount of Horizontal Bone Loss

- Crestal bone is normally 0.4-1.9mm apical to the CEJ of the teeth which it supports¹⁸



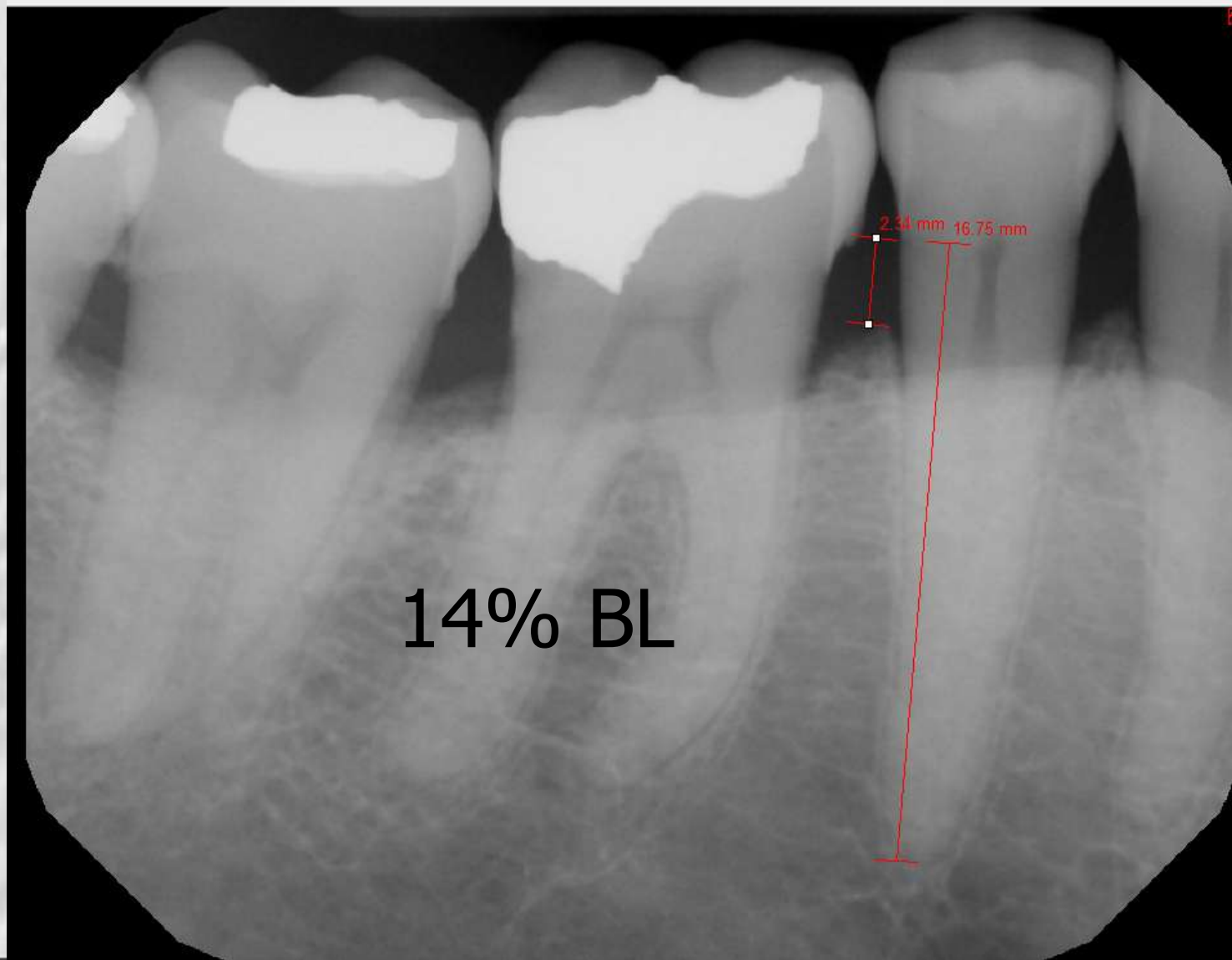
Presence/Amount of Horizontal Bone Loss

- If bone has been lost, and is parallel to this line, it is horizontal bone loss, and can be:
 - Mild: up to 15% of the root length **or** 2-3mm from CEJ
 - Moderate: 16-30% of the root length **or** 3-5mm from CEJ
 - Severe: >30% of the root length **or** >5mm from CEJ

Presence/Amount of Horizontal Bone Loss

- If doing a % measurement, use a periapical
- If doing a distance measurement, use a bitewing in the posterior, and a periapical in the anterior

Mild Bone Loss - %



Mild Bone Loss - Distance



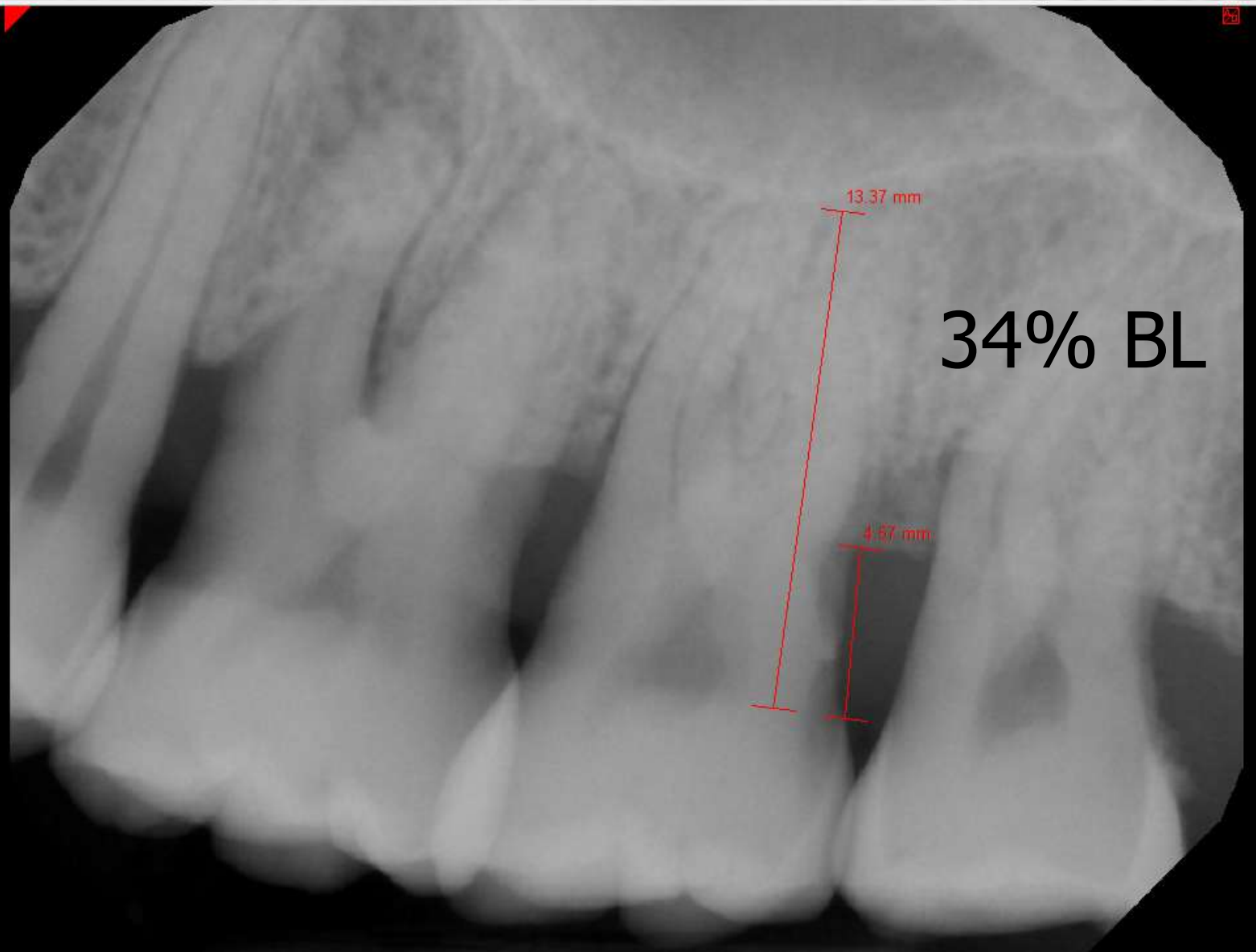
Moderate Bone Loss - %



Moderate Bone Loss - Distance



Severe Bone Loss - %



Severe Bone Loss - Distance



Why do we need both % and distance measurements?

It's confusing 😞

% vs. distance

- Because there's a lot of variation in root structure
- Very short roots might have 30% bone loss...but only because they're very short



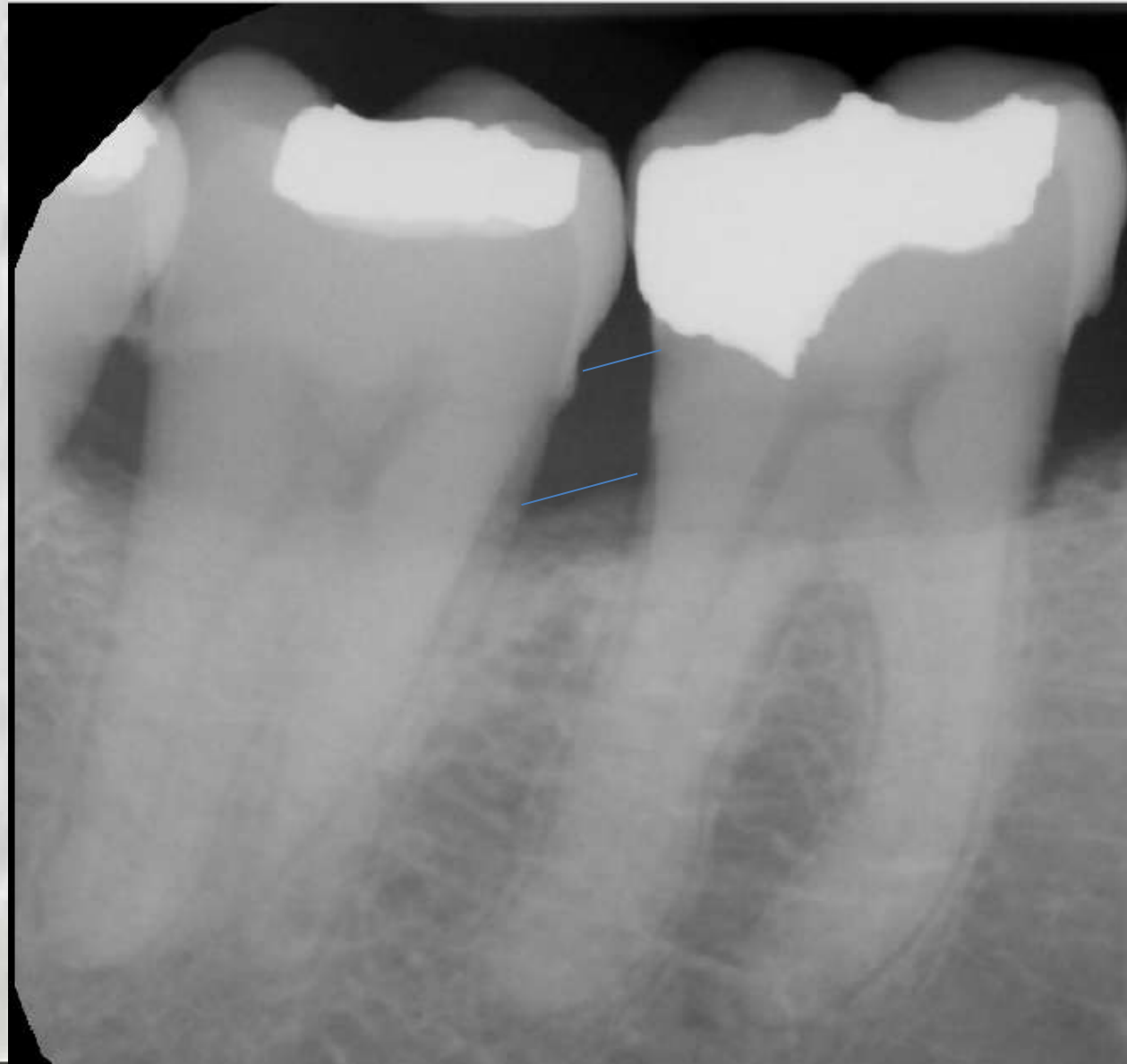
% vs. distance

- Very long roots might have 15% bone loss, but the bone loss is 6mm
- You have to use your brain



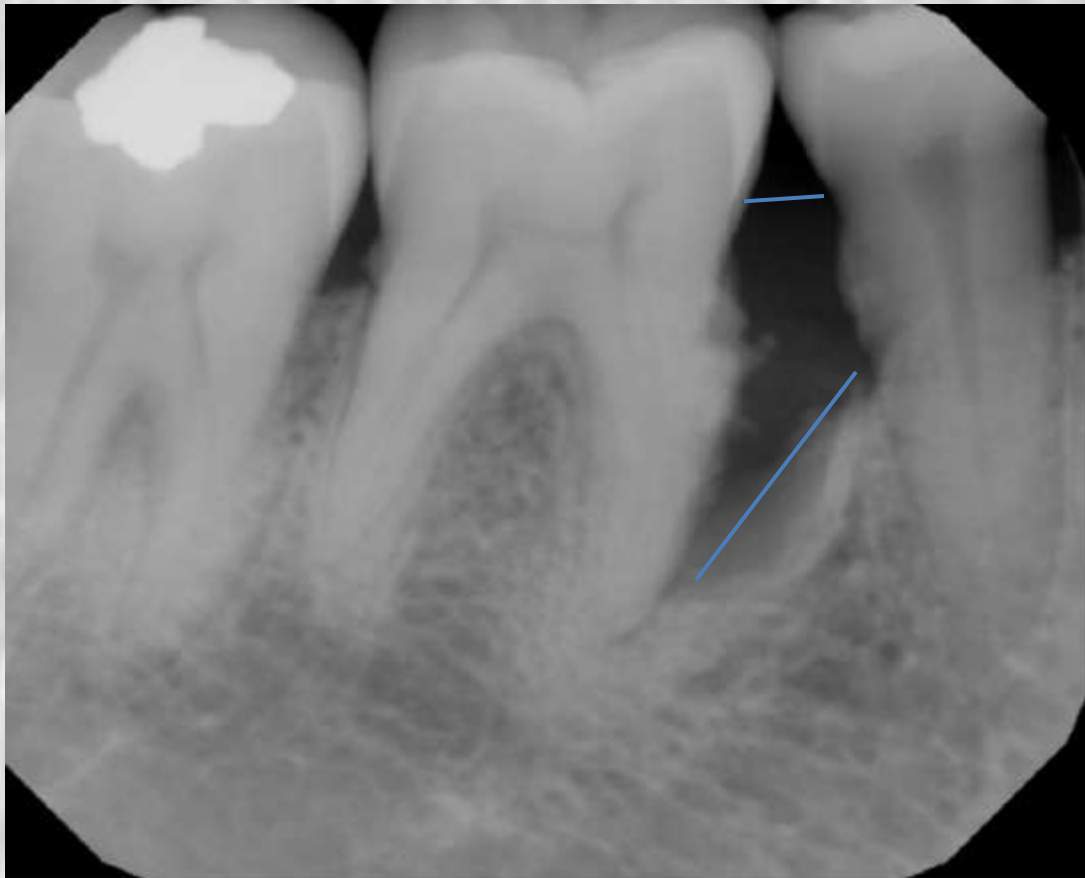
Horizontal vs. Vertical Bone Loss

- Normally, crestal bone follows an imaginary line between the CEJs of two adjacent teeth¹⁹



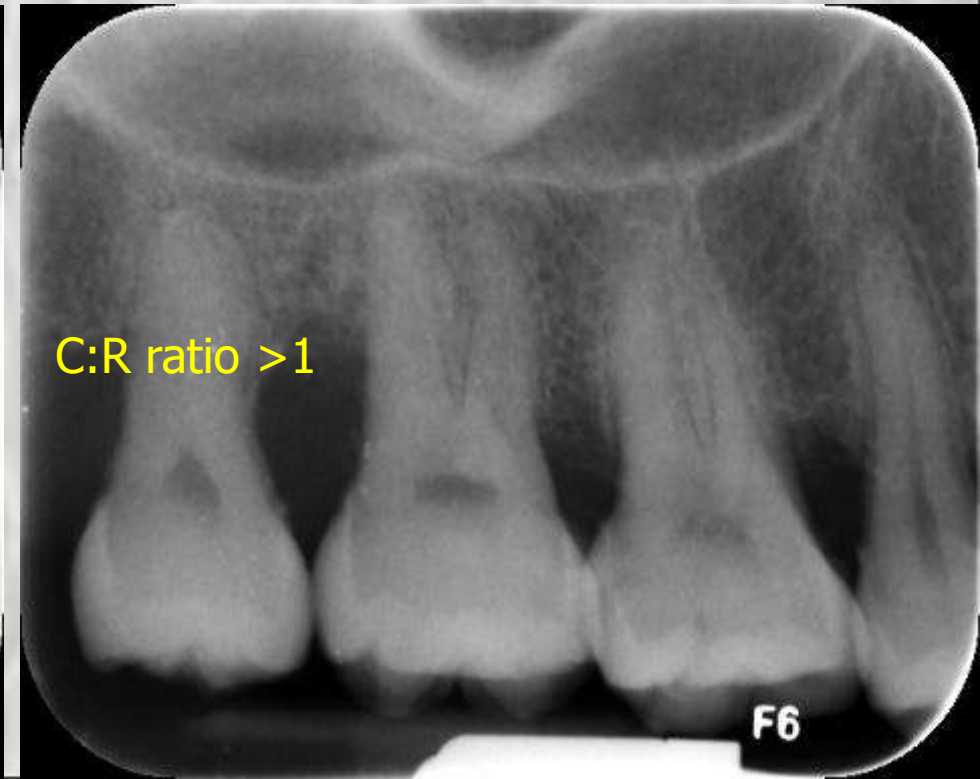
Presence of Vertical Bone Loss

- Bone loss which is **not** parallel to the CEJ-CEJ line is considered vertical bone loss



Crown:Root Ratio

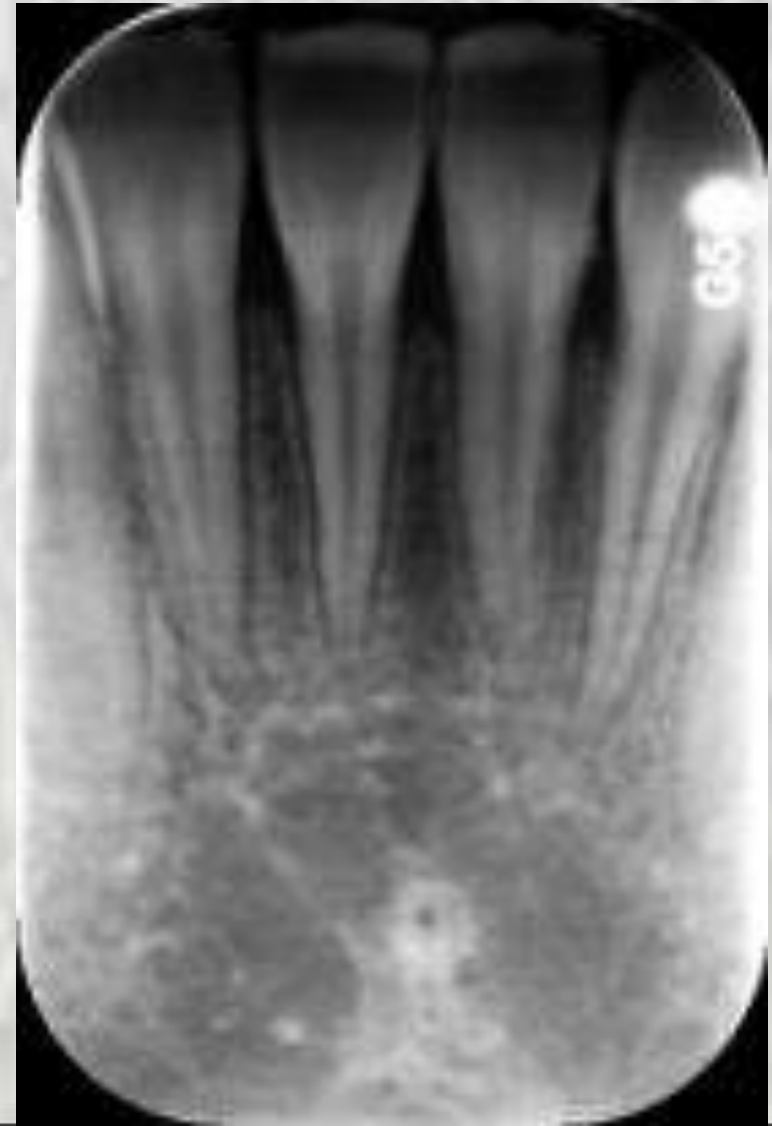
- The crown:root ratio is measured as what proportion of the tooth is coronal to the bone:what proportion is apical to the bone



Suggestion of Furcation Involvement/Arrows²⁰



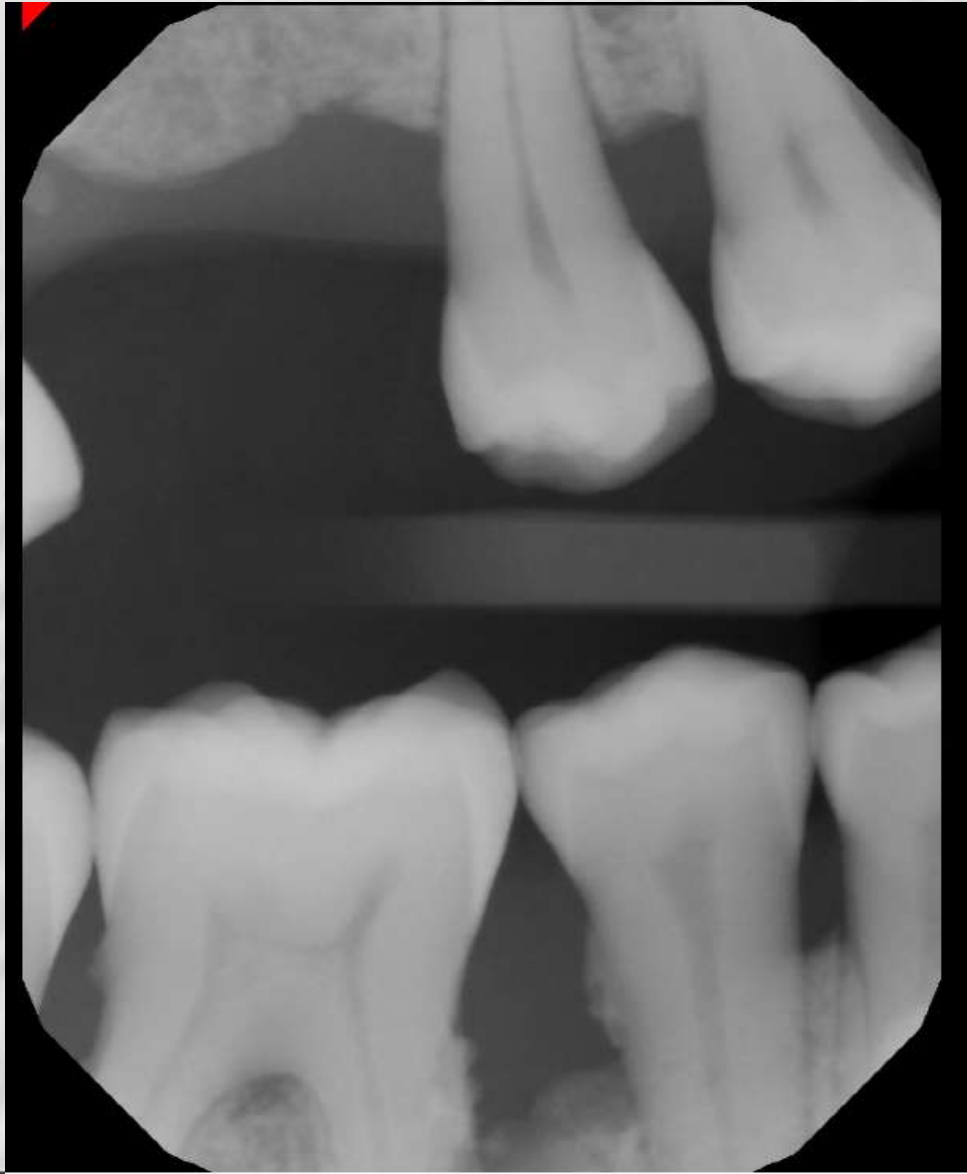
Widening of Periodontal Ligament Space



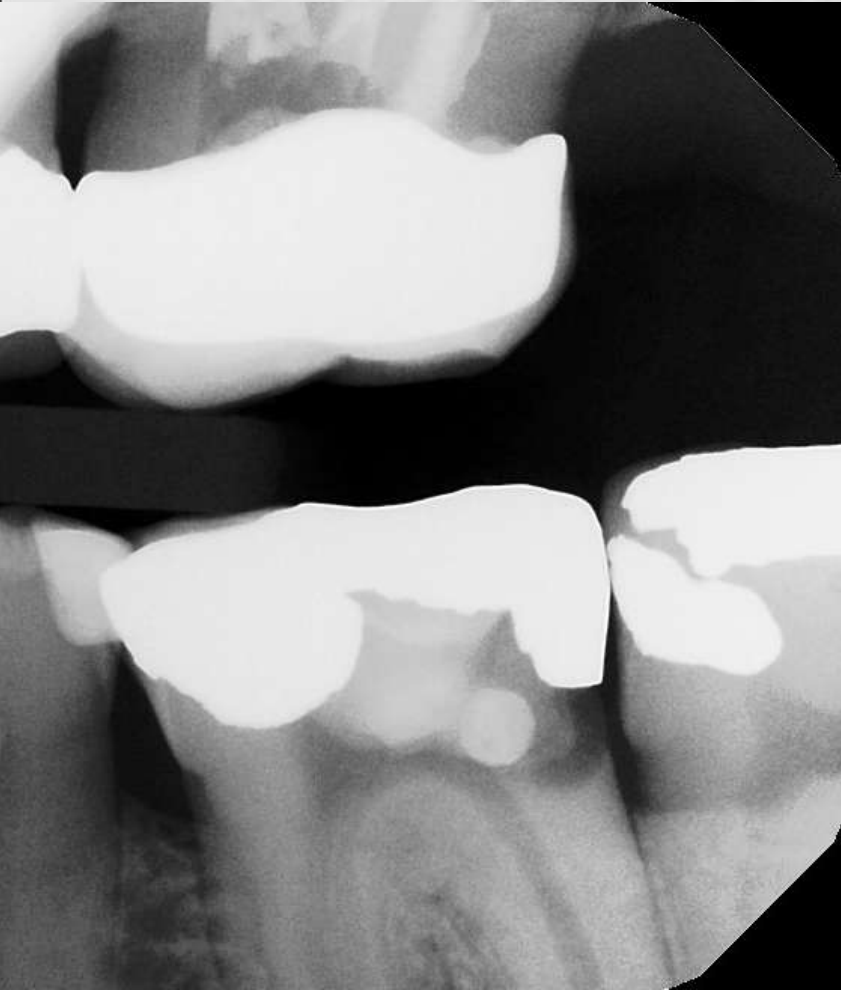
Presence of Calculus



Open Contacts



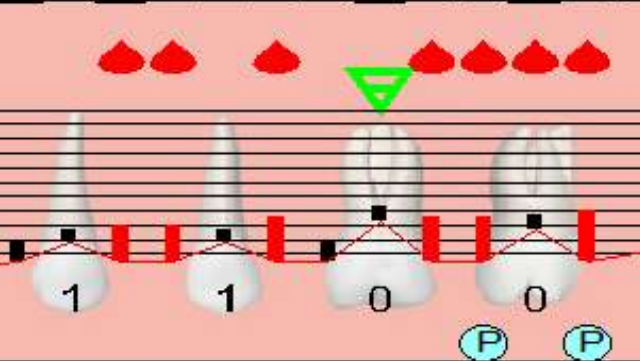
Root Proximity



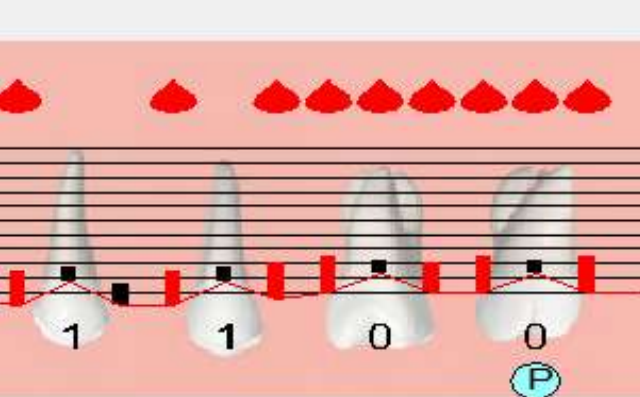
Craters

Let's look at Pt. X's left posterior
sextants

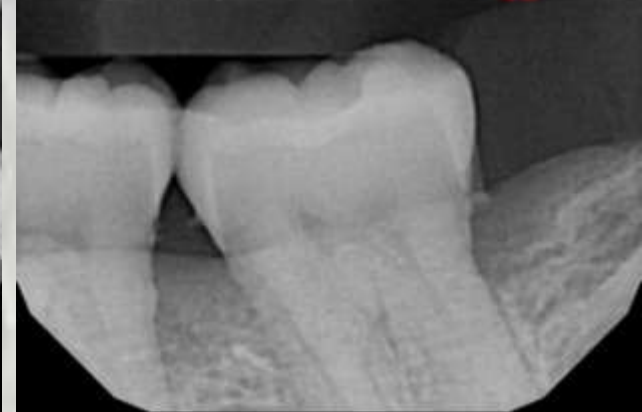
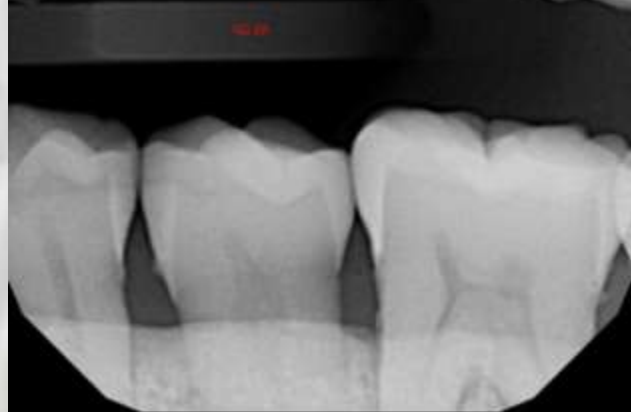
-1	-1	-1	-1	-1	-1	-1	2	-1	-1	1	-1	
3	2	5	5	2	6	3	2	6	6	2	7	
2	1	4	4	1	5	2	4	5	5	3	6	
0	0						2			0		



24	25	26	27
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-2	-1	-2	-2	-1	-1	0	0	0	0	0	0	
5	2	3	5	2	5	5	2	4	5	2	5	
3	1	1	3	1	4	5	2	4	5	2	5	
0	0					0		0	0			

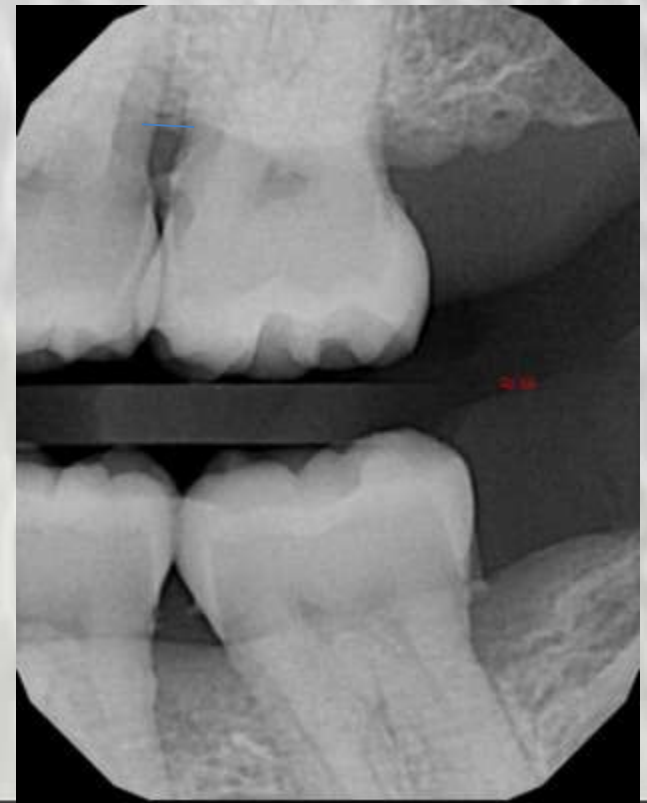


Inconsistency

- The previous sextants have something in common
- The clinical charting is showing severe attachment loss, but the radiographs are showing mild and moderate bone loss, and sometimes no bone loss
- Further, the bone levels in the radiographs are sometimes hard to identify

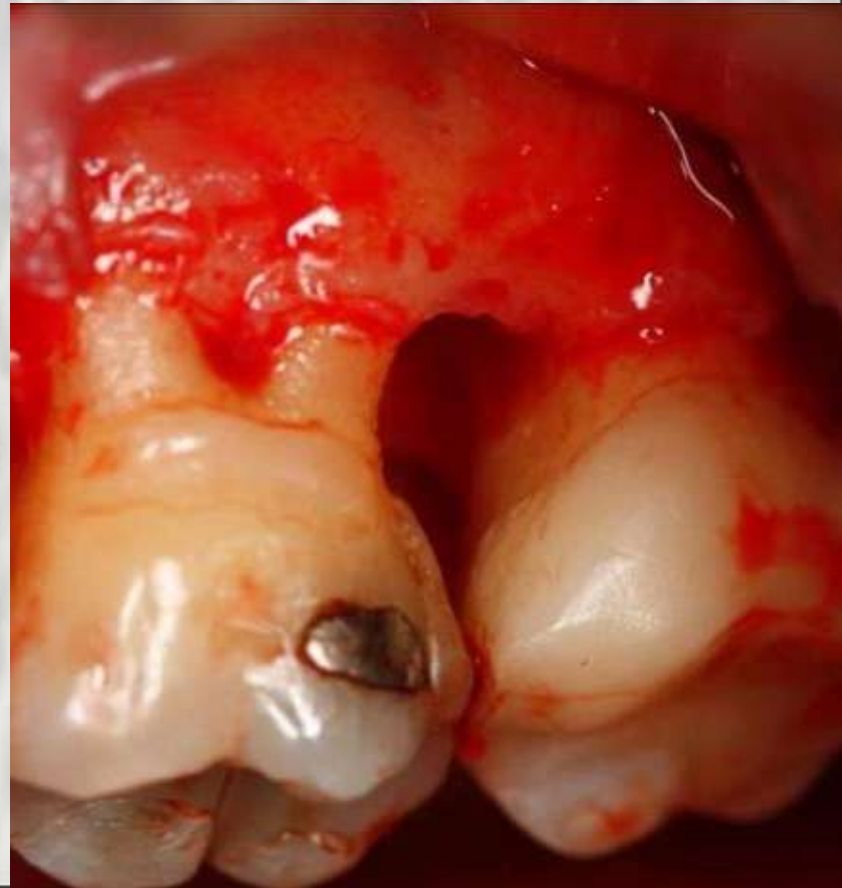
Craters

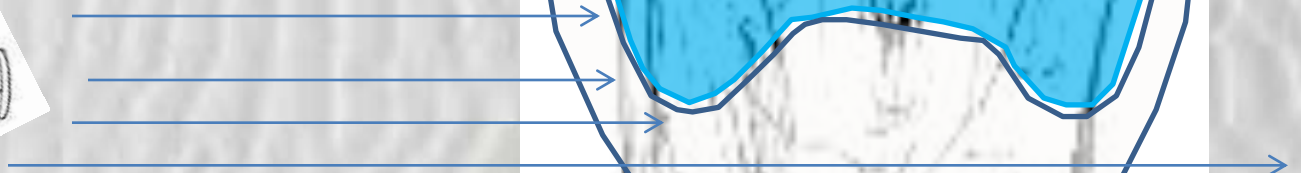
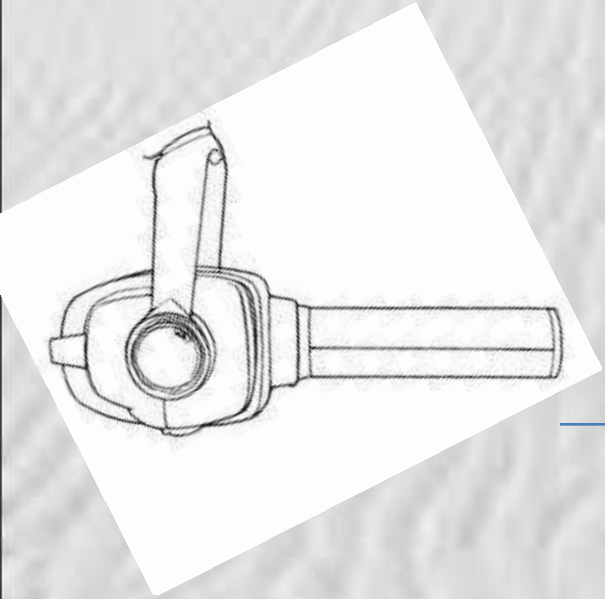
- Look at these radiographs:
- Can you see the two levels of bone between 26 and 27 in the periapical?
- Can you see how those two levels become one in the bitewing?

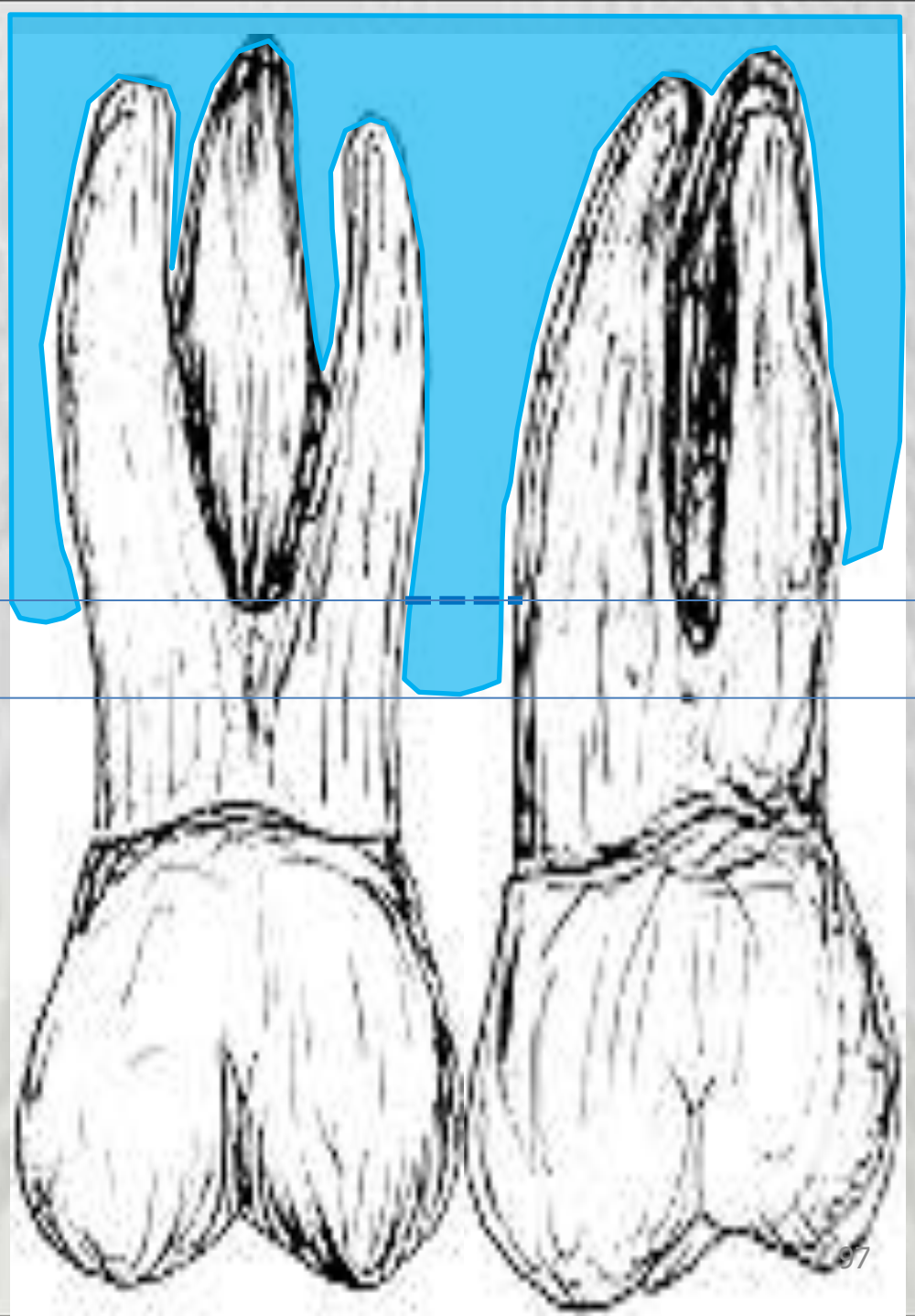
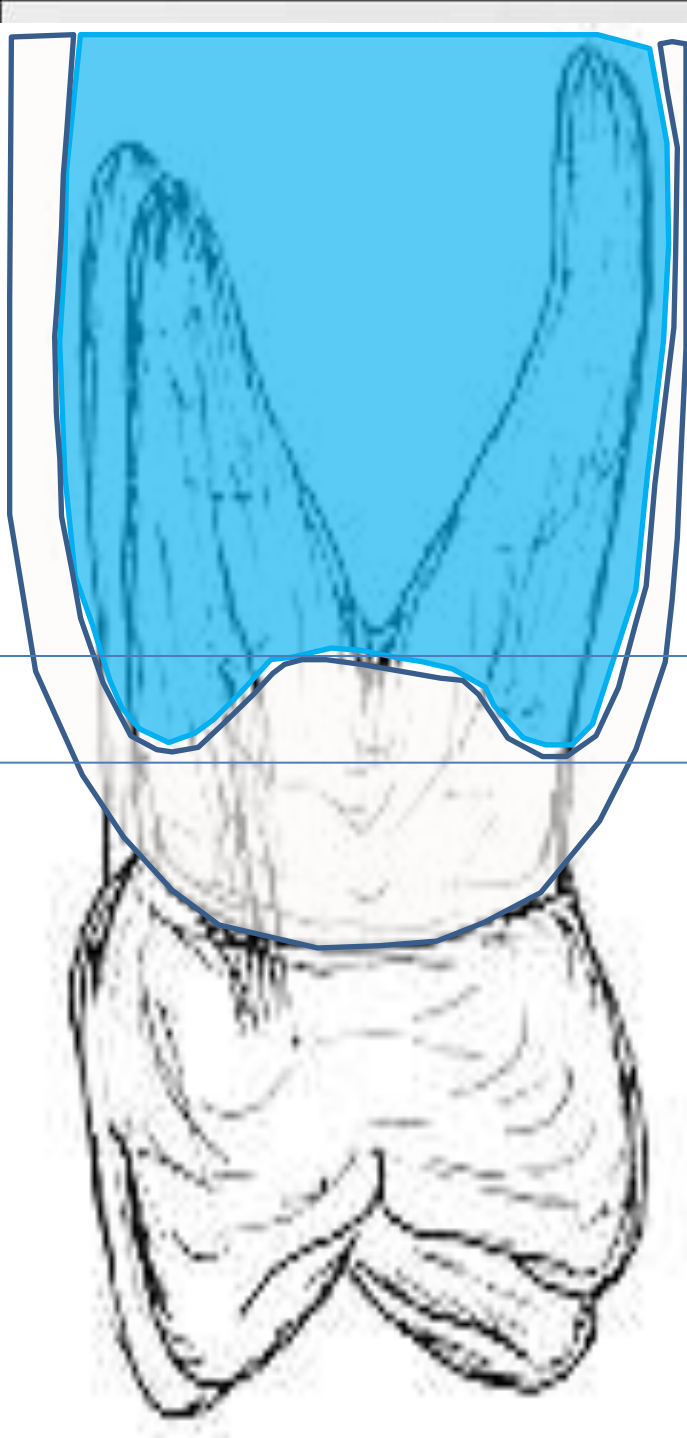


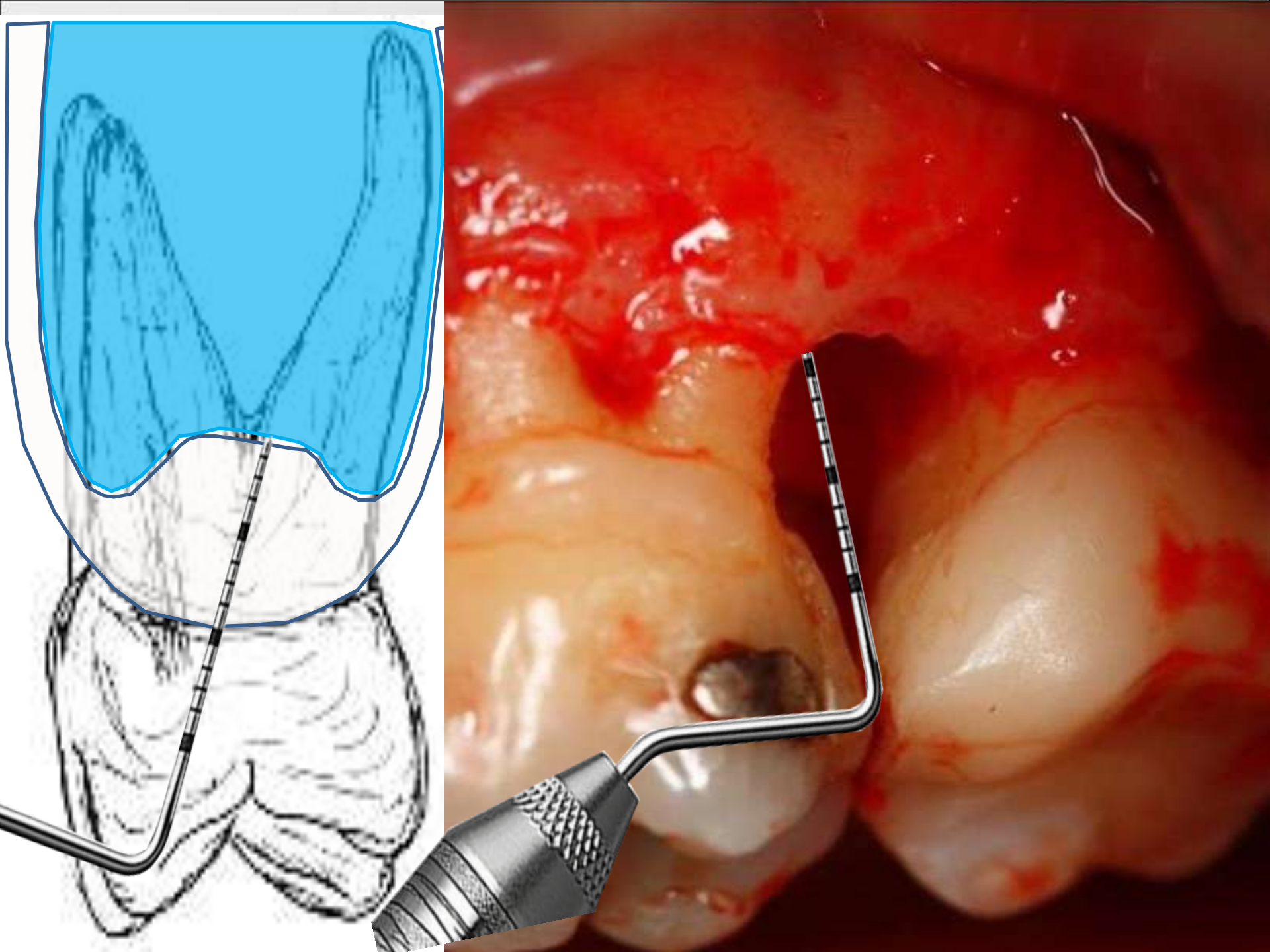
Craters

- A crater is an interproximal osseous defect consisting of a buccal and lingual or buccal and palatal wall
- It is the most common^{21,22,23} osseous defect present in periodontitis









Inconsistency

- So when your clinical attachment levels and your radiographic bone levels are disagreeing with each other... **TRUST YOUR CHARTING!**
- Otherwise you run the risk of missing disease that is present

How to Report Radiographic Findings

- Give a short, point-form narrative:
 - Generalized moderate horizontal bone loss with localized severe horizontal bone loss on teeth x, y, z etc.
 - Vertical bone loss on teeth aM, bD, cMD etc.
 - Calculus present on teeth dM, eD, fMD etc.
 - Widened pdl on teeth m, n, o etc.
 - Suggestion of furcation involvement on teeth r, s, t etc.
 - etc.

Questions?



References

1. Ervasti T, Knuuttila M, Pohjamo L, Haukipuro K. Relation between control of diabetes and gingival bleeding. *J Periodontol.* 1985 Mar;56(3):154-7.
2. Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes). *J Am Dent Assoc.* 1982 May;104(5):653-60.
3. Shlossman M, Knowler WC, Pettitt DJ, Genco RJ. Type 2 diabetes mellitus and periodontal disease. *J Am Dent Assoc.* 1990 Oct;121(4):532-6.
4. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. *J Periodontol.* 1991 Feb;62(2):123-31.
5. Safkan-Seppälä B, Ainamo J. Periodontal conditions in insulin-dependent diabetes mellitus. *J Clin Periodontol.* 1992 Jan;19(1):24-9.
6. Tervonen T, Oliver RC. Long-term control of diabetes mellitus and periodontitis. *J Clin Periodontol.* 1993 Jul;20(6):431-5.
7. Mealey BL, Oates TW; American Academy of Periodontology. Diabetes mellitus and periodontal diseases. *J Periodontol.* 2006 Aug;77(8):1289-303.
8. Salvi GE, Beck JD, Offenbacher S. PGE2, IL-1 beta, and TNF-alpha responses in diabetics as modifiers of periodontal disease expression. *Ann Periodontol.* 1998 Jul;3(1):40-50.
9. Golub LM, Lee HM, Lehrer G, Nemiroff A, McNamara TF, Kaplan R, Ramamurthy NS. Minocycline reduces gingival collagenolytic activity during diabetes. Preliminary observations and a proposed new mechanism of action. *J Periodontal Res.* 1983 Sep;18(5):516-26.
10. Ismail AI, Burt BA, Eklund SA. Epidemiologic patterns of smoking and periodontal disease in the United States. *J Am Dent Assoc.* 1983 May;106(5):617-21.
11. Bergström J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. *J Periodontol.* 1991 Apr;62(4):242-6.
12. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, Norderyd OM, Genco RJ. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol.* 1994 Mar;65(3):260-7.

References

13. Preber H, Linder L, Bergström J. Periodontal healing and periopathogenic microflora in smokers and non-smokers. *J Clin Periodontol.* 1995 Dec;22(12):946-52.
14. Ah MK, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. The effect of smoking on the response to periodontal therapy. *J Clin Periodontol.* 1994 Feb;21(2):91-7.
15. Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. *J Periodontol.* 1996 Jul;67(7):675-81.
16. Tonetti MS, Pini-Prato G, Cortellini P. Effect of cigarette smoking on periodontal healing following GTR in infrabony defects. A preliminary retrospective study. *J Clin Periodontol.* 1995 Mar;22(3):229-34.
17. Butler RT, Kalkwarf KL, Kaldahl WB. Drug-induced gingival hyperplasia: phenytoin, cyclosporine, and nifedipine. *J Am Dent Assoc.* 1987 Jan;114(1):56-60.
18. Hausmann E, Allen K, Clerehugh V. What alveolar crest level on a bite-wing radiograph represents bone loss? *J Periodontol.* 1991 Sep;62(9):570-2.
19. Ritchey B, Orban B. The crest of the interdental alveolar septa. *J Periodontol.* 1953 24:75-87.
20. Hardekopf JD, Dunlap RM, Ahl DR, Pelleu GB Jr. The "furcation arrow". A reliable radiographic image? *J Periodontol.* 1987 Apr;58(4):258-61.
21. Manson JD. Bone morphology and bone loss in periodontal disease. *J Clin Periodontol.* 1976 Feb;3(1):14-22.
22. Tal H. The prevalence and distribution of intrabony defects in dry mandibles. *J Periodontol.* 1984 Mar;55(3):149-54.
23. Vrotsos JA, Parashis AO, Theofanatos GD, Smulow JB. Prevalence and distribution of bone defects in moderate and advanced adult periodontitis. *J Clin Periodontol.* 1999 Jan;26(1):44-8.

Mini-Objectives

By the end of this part, you should:

- know what clinical and radiographic parameters are used to diagnose inflammatory periodontal disease
- know how to differentiate chronic vs. aggressive vs. “other” periodontitis
- know how to properly write a diagnosis of periodontitis

Overview



1. Introduction
2. Establishing the presence of inflammation
3. Understanding attachment loss and bone loss
4. What type is it?
5. Describing the severity and extent
6. Treatment planning initial therapy

Overview



I. Introduction

Diagnosis

- The bible¹ of periodontal diagnoses was compiled by Gary Armitage, and adopted in 1999
- An update¹³ was presented in 2015

Armitage Classification

- I. Gingival Diseases
 - A. Dental plaque-induced gingival diseases*
 - 1. Gingivitis associated with dental plaque only
 - a. without other local contributing factors
 - b. with local contributing factors (See VIII A)
 - 2. Gingival diseases modified by systemic factors
 - a. associated with the endocrine system
 - 1) puberty-associated gingivitis
 - 2) menstrual cycle-associated gingivitis
 - 3) pregnancy-associated
 - a) gingivitis
 - b) pyogenic granuloma
 - 4) diabetes mellitus-associated gingivitis
 - b. associated with blood dyscrasias
 - 1) leukemia-associated gingivitis
 - 2) other
 - 3. Gingival diseases modified by medications
 - a. drug-influenced gingival diseases
 - 1) drug-influenced gingival enlargements
 - 2) drug-influenced gingivitis
 - a) oral contraceptive-associated gingivitis
 - b) other
 - 4. Gingival diseases modified by malnutrition
 - a. ascorbic acid-deficiency gingivitis
 - b. other
 - B. Non-plaque-induced gingival lesions
 - 1. Gingival diseases of specific bacterial origin
 - a. *Neisseria gonorrhoea*-associated lesions
 - b. *Treponema pallidum*-associated lesions
 - c. streptococcal species-associated lesions
 - d. other
 - 2. Gingival diseases of viral origin
 - a. herpesvirus infections
 - 1) primary herpetic gingivostomatitis
 - 2) recurrent oral herpes
 - 3) varicella-zoster infections
 - b. other
 - 3. Gingival diseases of fungal origin
 - a. *Candida*-species infections
 - 1) generalized gingival candidosis
 - b. linear gingival erythema
 - c. histoplasmosis
 - d. other
 - 4. Gingival lesions of genetic origin
 - a. hereditary gingival fibromatosis
 - b. other
 - 5. Gingival manifestations of systemic conditions
 - a. mucocutaneous disorders
 - 1) lichen planus
 - 2) pemphigoid
 - 3) pemphigus vulgaris
 - 4) erythema multiforme
 - 5) lupus erythematosus
 - 6) drug-induced
 - 7) other
 - b. allergic reactions
 - 1) dental restorative materials
 - a) mercury
 - b) nickel
 - c) acrylic
 - d) other
 - 2) reactions attributable to
 - a) toothpastes/dentifrices
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 - 6. Traumatic lesions (factitious, iatrogenic, accidental)
 - a. chemical injury
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 - 8. Not otherwise specified (NOS)
 - II. Chronic Periodontitis†
 - A. Localized
 - B. Generalized
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 - A. Localized
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 - IV. Periodontitis as a Manifestation of Systemic Diseases
 - A. Associated with hematological disorders
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 - 3. Decreased vestibular depth
 - 4. Aberrant frenum/muscle position
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Figure 1.

Classification of periodontal diseases and conditions.

* Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing.

Figure 1. (Continued)

† Can be further classified on the basis of extent and severity. As a general guide, extent can be characterized as Localized = $\leq 30\%$ of sites involved and Generalized = $>30\%$ of sites involved. Severity can be characterized on the basis of the amount of clinical attachment loss (CAL) as follows: Slight = 1 or 2 mm CAL, Moderate = 3 or 4 mm CAL, and Severe = ≥ 5 mm CAL.

Armitage Classification

- I. Gingival Diseases
 - A. Dental plaque-induced gingival diseases*
 - 1. Gingivitis associated with dental plaque only
 - a. without other local contributing factors
 - b. with local contributing factors (See VIII A)
 - 2. Gingival diseases modified by systemic factors
 - a. associated with the endocrine system
 - 1) puberty-associated gingivitis
 - 2) menstrual cycle-associated gingivitis
 - 3) pregnancy-associated
 - a) gingivitis
 - b) pyogenic granuloma
 - 4) diabetes mellitus-associated gingivitis
 - b. associated with blood dyscrasias
 - 1) leukemia-associated gingivitis
 - 2) other
 - 3. Gingival diseases modified by medications
 - a. drug-influenced gingival diseases
 - 1) drug-influenced gingival enlargements
 - 2) drug-influenced gingivitis
 - a) oral contraceptive-associated gingivitis
 - b) other
 - 4. Gingival diseases modified by malnutrition
 - a. ascorbic acid-deficiency gingivitis
 - b. other
 - B. Non-plaque-induced gingival lesions
 - 1. Gingival diseases of specific bacterial origin
 - a. *Neisseria gonorrhoea*-associated lesions
 - b. *Treponema pallidum*-associated lesions
 - c. streptococcal species-associated lesions
 - d. other
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Classification of periodontal diseases and conditions.

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Periodontitis

- *“periodontitis is defined as inflammation of the gingiva and the adjacent attachment apparatus. The disease is characterized by loss of clinical attachment due to destruction of the periodontal ligament and loss of the adjacent supporting bone.”^{2,3}*

Periodontitis

- To oversimplify matters significantly:
 1. A bacterial biofilm initiates an inflammatory reaction in the periodontal soft tissues
 2. The inflammatory reaction produces tissue-destructive cells and enzymes
 3. A vicious pro-inflammatory circle results in progressive attachment loss
 4. The amount of destruction is potentiated by a bunch of innate and environmental variables

Diagnosis of Periodontitis

- Let's take two sample diagnoses:
 1. Generalized moderate chronic periodontitis
 2. Localized severe aggressive periodontitis
- Each of these can be broken down into components

Diagnosis of Periodontitis

1. Generalized moderate chronic periodontitis
 2. Localized severe aggressive periodontitis
- Working backwards, diagnoses have:
 - something reminding us that inflammation is present
 - something telling us that attachment loss is present
 - something describing the type of attachment loss
 - something describing the severity of attachment loss
 - something describing the extent of attachment loss

Overview



1. Introduction
2. Establishing the presence of inflammation

Inflammation

- Inflammation puts the **itis** into **gingivitis** and **periodontitis**
- The cardinal signs of inflammation are:
 1. ***Rubor*** (redness)
 2. ***Calor*** (heat)
 3. ***Tumor*** (swelling)
 4. ***Dolor*** (pain)



-Aulus Cornelius Celsus
De Medicina, 1st century AD

A Different Way to Think about *Rubor*

- While it is true that inflamed gingiva sometimes appear more red, a more objective measure is bleeding on probing



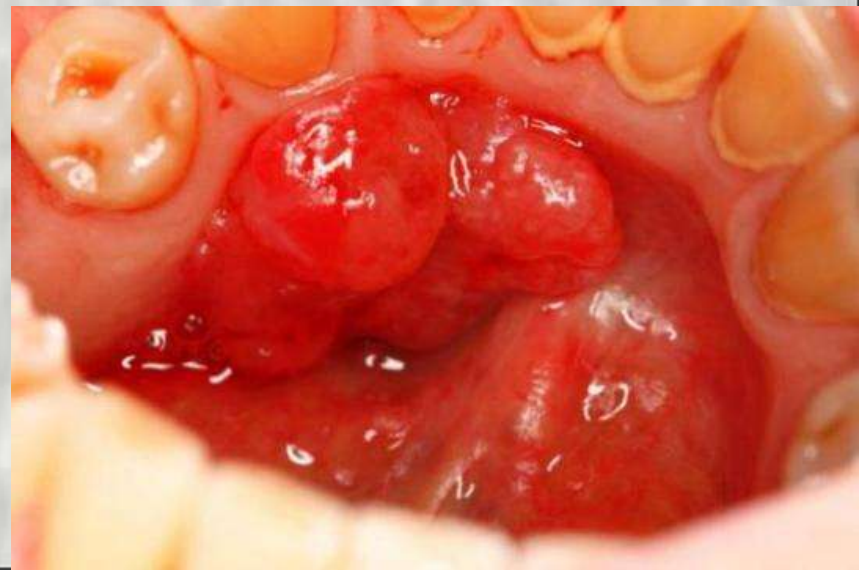
Bleeding on Probing

- Highly correlated with histological signs of gingival inflammation⁴
- May be an earlier and more sensitive sign of inflammation than redness or swelling⁵



A Different Way to Think about *Tumor*

- Recall that *tumor*, or swelling, is one of the cardinal signs of inflammation
- We tend to think of the word tumor as a “bump” of various size and shape, where said “bump” should not be



A Different Way to Think about *Tumor*

- Periodontists think about *tumor* slightly differently
- In our world, as the gingiva becomes inflamed, it swells

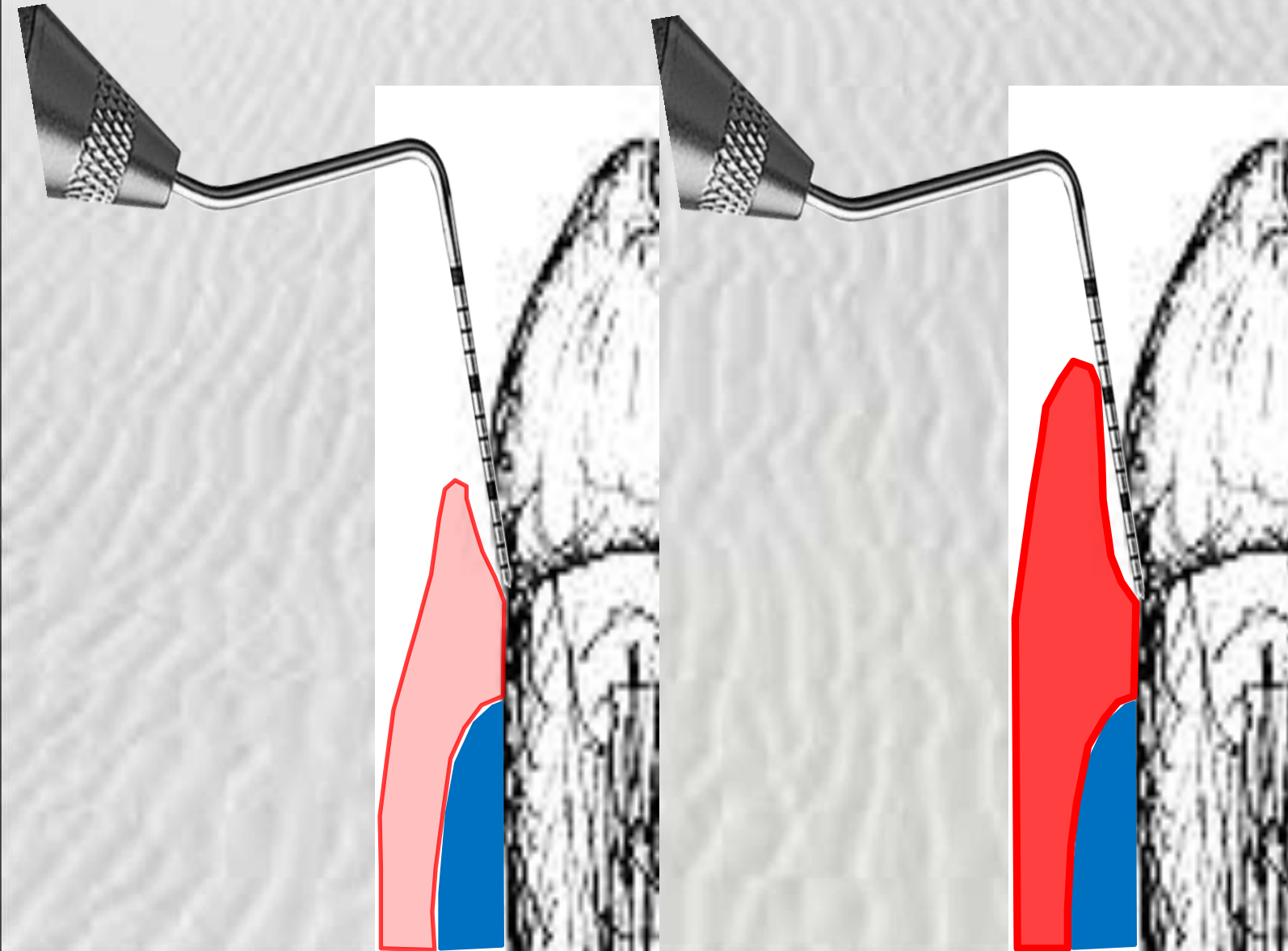


A Different Way to Think about *Tumor*

- While it is true that we can visually assess the swelling present, a more objective means of describing it involves using probing depth



Probing Depth and Inflammation



Probing Depth $\geq 4\text{mm}$

- By convention, a healthy *sulcus* is $\leq 3\text{mm}$, while a periodontal *pocket* is $\geq 4\text{mm}$
- Why is $\geq 4\text{mm}$ our cut-off for “pathological”?

Probing Depth $\geq 4\text{mm}$

- Pockets of $\geq 4\text{mm}$:
 - are more difficult to instrument, and have less calculus removed from them⁶
 - gained clinical attachment following therapy, reducing pocket depth⁷
 - are more likely to experience disease recurrence/periodontal breakdown following therapy⁸

Clinical Inflammation

- So the clinical signs to use to identify inflammation that may lead to progressive disease⁹ are:
 1. Probing depth $\geq 4\text{mm}$
 2. Bleeding on probing
- This gives you your **itis**

Important Note

- When you are dealing with a new patient who has never undergone any form of perio treatment, sites which have:
 1. Probing depth $\geq 4\text{mm}$, or
 2. Bleeding on probing, or
 3. A combination of bothqualify as **inflamed**

Overview

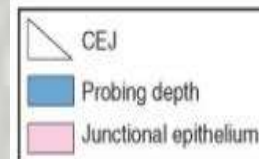
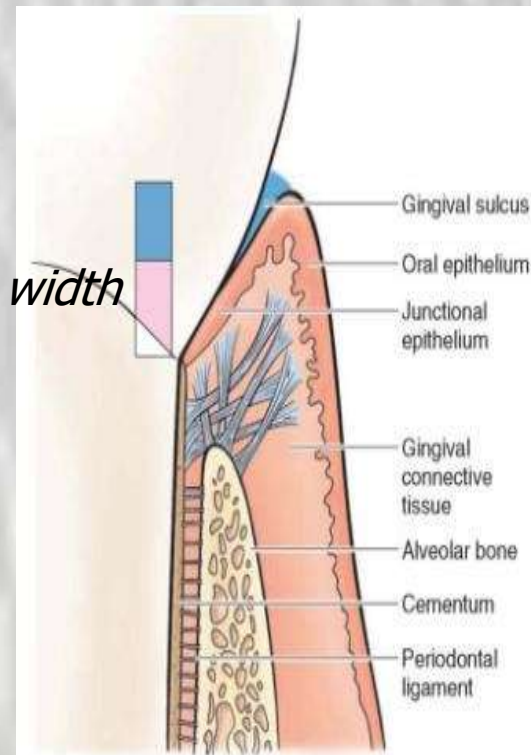


1. Introduction
2. Establishing the presence of inflammation
3. Understanding attachment loss and bone loss

What is “Attachment?”

- Attachment can refer to anything which retains the tooth in the mouth, such as:
 - The epithelial attachment of the gingiva
 - The connective tissue attachment of the gingiva
 - The periodontal ligament

Biologic width

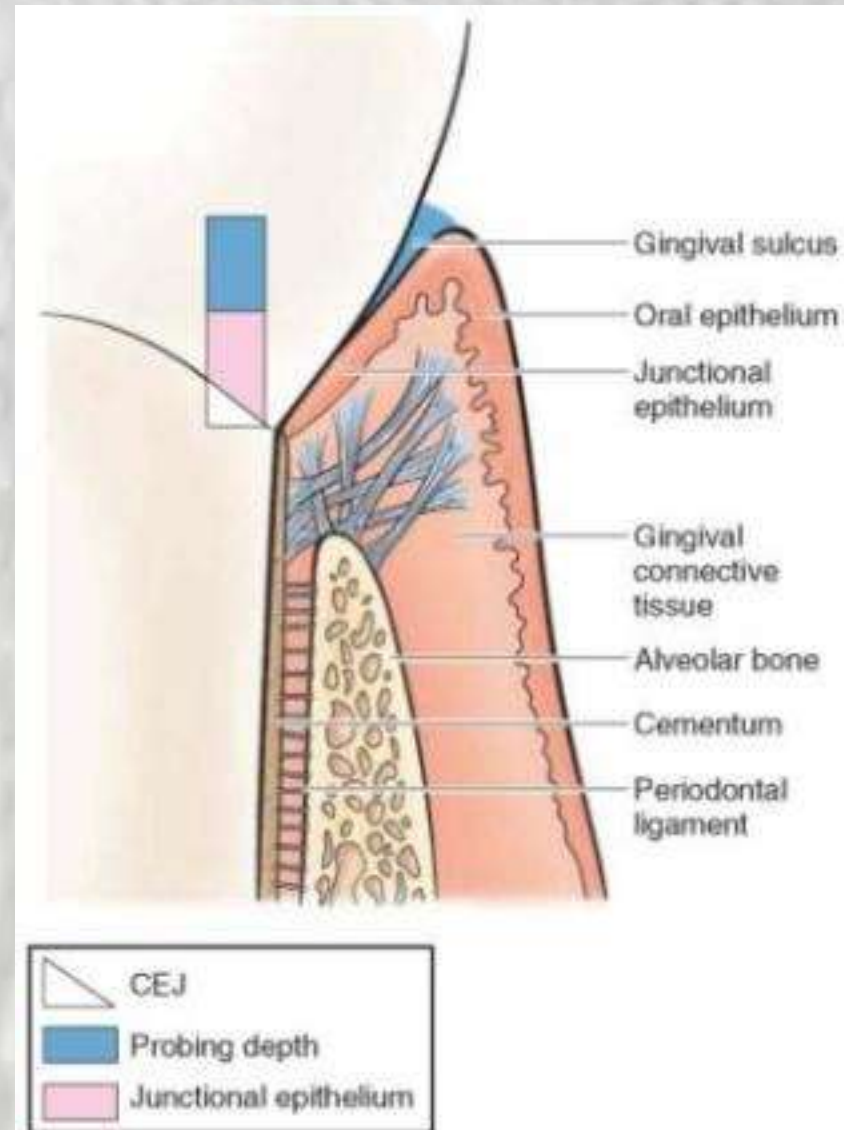


Attachment Loss

- Recall that what differentiates periodontitis from gingivitis is the presence of attachment loss

Attachment Loss

- Attachment loss proceeds in an apical direction¹⁰:
 - The inflammatory infiltrate unzippers the junctional epithelium (2-4 days)
 - The collagen of the connective tissue is lysed and lost (4-10 days)
 - Bone is resorbed (years)



Are attachment loss and bone loss the same thing?

Bone Loss

- When you see bone loss on a radiograph, by definition there has been attachment loss
- Does this mean when you detect attachment loss clinically, there is automatically bone loss?

Not Always....

- There are two situations where you may have clinical attachment loss, but will not see any bone loss on radiographs:
 1. In the early stages of periodontitis
 2. When periodontitis has created a very common osseous defect...a **crater**

Early Periodontitis

- Recall that the early stages of periodontitis involve soft tissue changes at the junctional epithelium and connective tissue attachment
- These changes precede the development of radiographic bone loss by 6-8 months¹¹

Attachment Loss/Bone Loss

- So based on the fact that we have evidence of inflammation, and attachment loss and/or bone loss, we have a **periodontitis**, not a **gingivitis**

Overview



1. Introduction
2. Establishing the presence of inflammation
3. Understanding attachment loss and bone loss
4. What type is it?

Chronic Periodontitis

- The overwhelming majority of periodontitis you will see will be chronic periodontitis
- Any other types of periodontitis have specific features, which if are present would lead you to modify your diagnosis of chronic into something else

Aggressive Periodontitis

- Recall Armitage's paper
- By definition, aggressive periodontitis refers to attachment loss that happens rapidly

Ann Periodontol Armitage

<ul style="list-style-type: none">II. Chronic Periodontitis[†]<ul style="list-style-type: none">A. LocalizedB. GeneralizedIII. Aggressive Periodontitis[†]<ul style="list-style-type: none">A. LocalizedB. GeneralizedIV. Periodontitis as a Manifestation of Systemic Diseases<ul style="list-style-type: none">A. Associated with hematological disorders<ul style="list-style-type: none">1. Acquired neutropenia2. Leukemias3. OtherB. Associated with genetic disorders<ul style="list-style-type: none">1. Familial and cyclic neutropenia2. Down syndrome3. Leukocyte adhesion deficiency syndromes4. Papillon-Lefevre syndrome5. Chediak-Higashi syndrome6. Histiocytosis syndromes7. Glycogen storage disease8. Infantile genetic agranulocytosis9. Cohen syndrome10. Ehlers-Danlos syndrome (Types IV and VIII)11. Hypophosphatasia12. OtherC. Not otherwise specified (NOS)V. Necrotizing Periodontal Diseases<ul style="list-style-type: none">A. Necrotizing ulcerative gingivitis (NUG)B. Necrotizing ulcerative periodontitis (NUP)VI. Abscesses of the Periodontium<ul style="list-style-type: none">A. Gingival abscessB. Periodontal abscessC. Pericoronal abscess	<ul style="list-style-type: none">VII. Periodontitis Associated With Endodontic Lesions<ul style="list-style-type: none">A. Combined periodontic-endodontic lesionsVIII. Developmental or Acquired Deformities and Conditions<ul style="list-style-type: none">A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis<ul style="list-style-type: none">1. Tooth anatomic factors2. Dental restorations/appliances3. Root fractures4. Cervical root resorption and cemental tearsB. Mucogingival deformities and conditions around teeth<ul style="list-style-type: none">1. Gingival/soft tissue recession<ul style="list-style-type: none">a. facial or lingual surfacesb. interproximal (papillary)2. Lack of keratinized gingiva3. Decreased vestibular depth4. Aberrant frenum/muscle position5. Gingival excess<ul style="list-style-type: none">a. pseudopocketb. inconsistent gingival marginc. excessive gingival displayd. gingival enlargement (See IA.3. and IB.4.)6. Abnormal colorC. Mucogingival deformities and conditions on edentulous ridges<ul style="list-style-type: none">1. Vertical and/or horizontal ridge deficiency2. Lack of gingiva/keratinized tissue3. Gingival/soft tissue enlargement4. Aberrant frenum/muscle position5. Decreased vestibular depth6. Abnormal colorD. Occlusal trauma<ul style="list-style-type: none">1. Primary occlusal trauma2. Secondary occlusal trauma
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Aggressive Periodontitis

- Because we do not see our patients every day (or week, or month, or even year sometimes), or we see patients for the first time after they have already been affected by the disease, we need other features to help us identify the problem

Primary Features of Aggressive

- Attachment loss which affects first molars and incisors¹²
- Patient age <25 years¹³
- Familial history of the disease¹⁴

Secondary Features of Aggressive¹³

- Amounts of microbial deposits may be inconsistent with amount of destruction
- Progression may be self-limiting (burn-out)
 - May be associated with *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis*
 - May be associated with neutrophil defects

Age and Aggressive Periodontitis

- NOTE: A “younger” person can have a chronic periodontitis
- Inflammation and attachment loss/bone loss in someone under the age of 25 does NOT automatically diagnose an aggressive periodontitis
- Other features need to be present as well

Type of Periodontitis

- So we either have a **chronic periodontitis**, or the features are telling us that we have an **aggressive periodontitis**

Overview



1. Introduction
2. Establishing the presence of inflammation
3. Understanding attachment loss and bone loss
4. What type is it?
5. Describing the severity and extent

Severity

Guidelines for Determining Severity of Periodontitis

	Slight (Mild)	Moderate	Severe (Advanced)
Probing depths	>3 & <5 mm	≥5 & <7 mm	≥7 mm
Bleeding on probing	Yes	Yes	Yes
Radiographic bone loss	Up to 15% of root length or ≥2 mm & ≤3 mm	16% to 30% or >3 mm & ≤5 mm	>30% or >5 mm
Clinical attachment loss ¹	1 to 2 mm	3 to 4 mm	≥5 mm

Extent of Chronic Periodontitis

- The extent of chronic periodontitis refers to the number of teeth affected:
 - Localized is $<30\%$
 - Generalized is $\geq 30\%$

Extent of Chronic Periodontitis

- More severe forms trump less severe forms
- In other words, you can have a:
 - generalized mild with a localized moderate and/or severe

but **NOT** a

- generalized severe with a localized mild and/or moderate

Extent of Aggressive

- The extent of aggressive periodontitis refers to the number of teeth affected:
 - Localized is incisors and first molars
 - Generalized is incisors and first molars plus other teeth, which may follow no clear pattern at all, and often affects most or all the other teeth¹³

Overview



1. Introduction
2. Establishing the presence of inflammation
3. Understanding attachment loss and bone loss
4. What type is it?
5. Describing the severity and extent
6. Treatment planning initial therapy

Initial Therapy

- Initial therapy \neq just Sc/RP
- Initial therapy = removing the primary etiology of inflammatory periodontal disease – **plaque** – and addressing as many secondary etiologies as possible

Possible Secondary Etiologies

- Calculus
- Smoking
- Uncontrolled diabetes
- “Hopeless” teeth
- Mal-posed teeth
- Impacted teeth
- Caries
- Overcontoured restorations
- Open margins
- Overhangs
- Occlusal trauma
- Ill-fitting prostheses
- Narrow embrasures
- Open contacts
- Tissue-invasive bacteria
- Medications

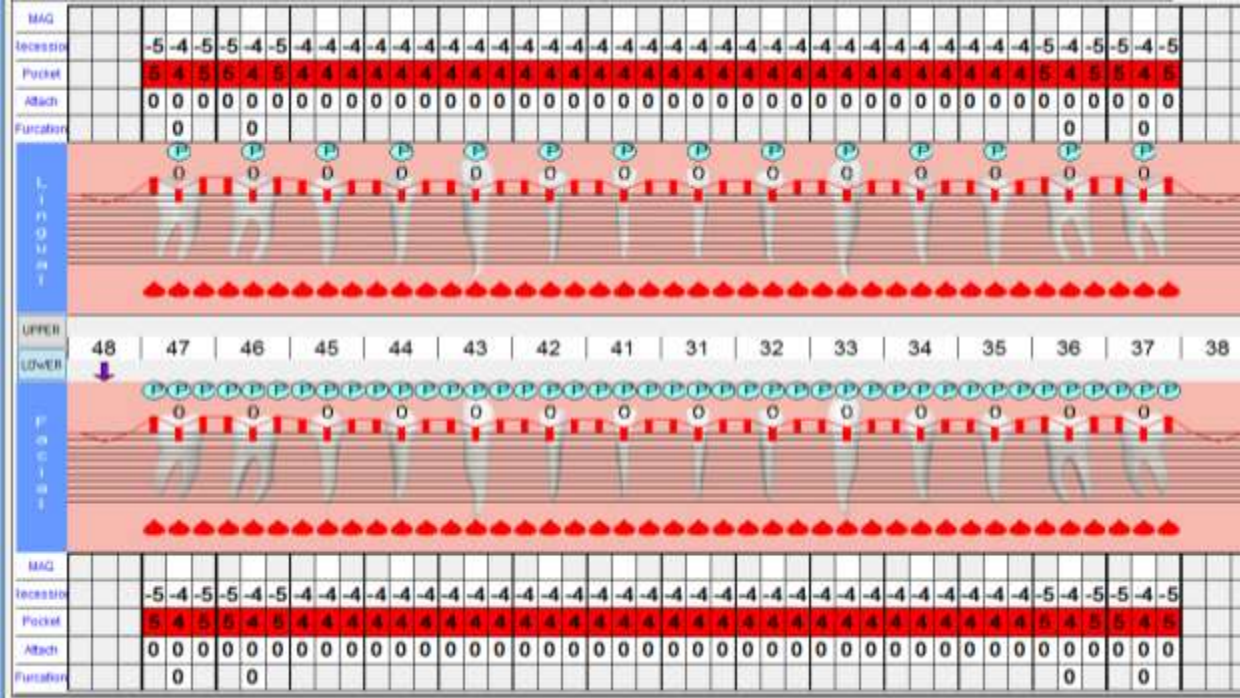
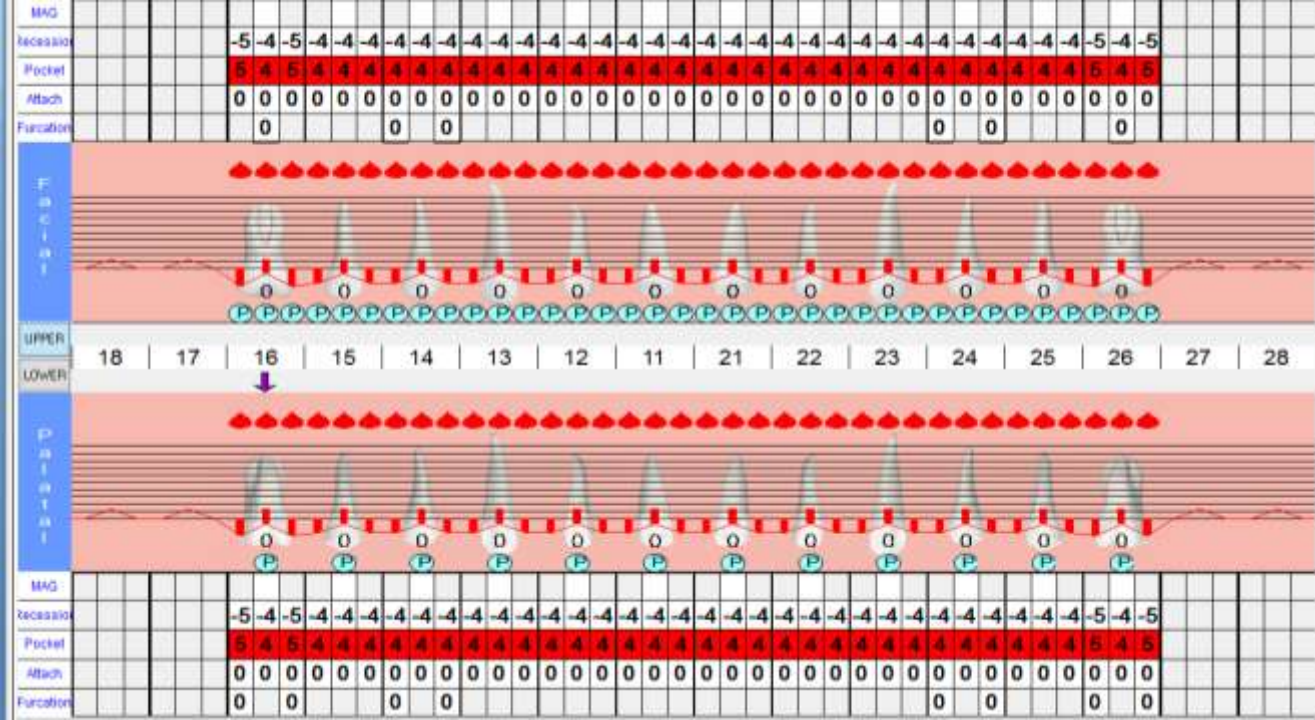
Initial Therapy

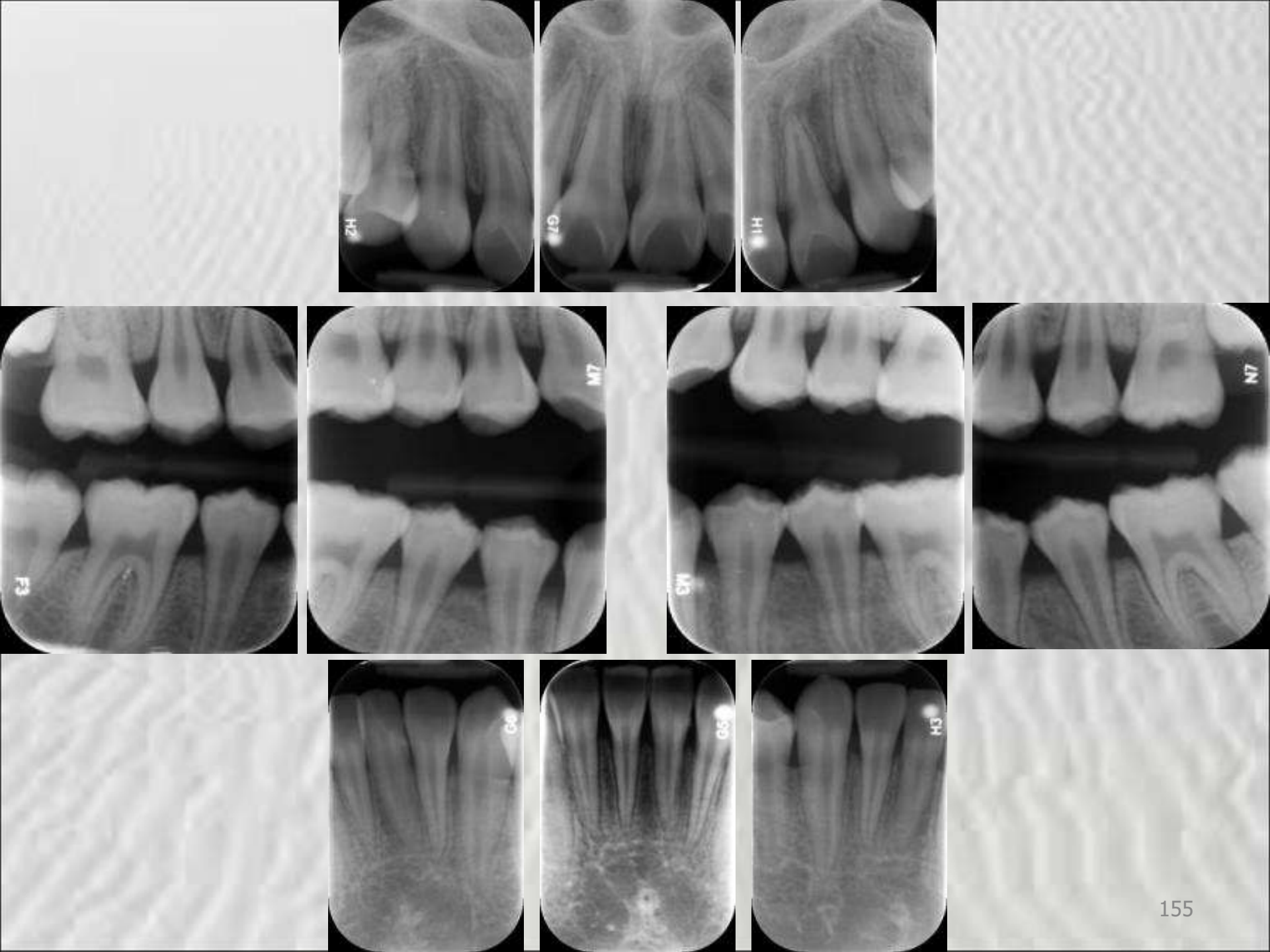
- Scaling and root planing
- Smoking cessation aids
- Medical consultation
- Select extractions
- Caries control
- Contouring restorations
- Patching/replacing margins
- Removing overhangs
- Adjusting/replacing prostheses
- Occlusal adjustment/fabrication of occlusal guard
- Creation of physiologic embrasures
- Closing contacts
- Systemic chemotherapeutics *(specifically for cases of aggressive)*

Let's go through some cases....

Case I – Pt.A

- 12 year old healthy ♀ presents for an initial exam
- No family history of tooth loss
- No history of previous periodontal treatment
- Pt. does not brush or floss at all





Case I

- Pt. A has:
 - PD \geq 4mm and BoP
 - No attachment loss (AL) and no bone loss (BL)
 - LOADS of plaque everywhere
 - No family history (or other aggressive features)

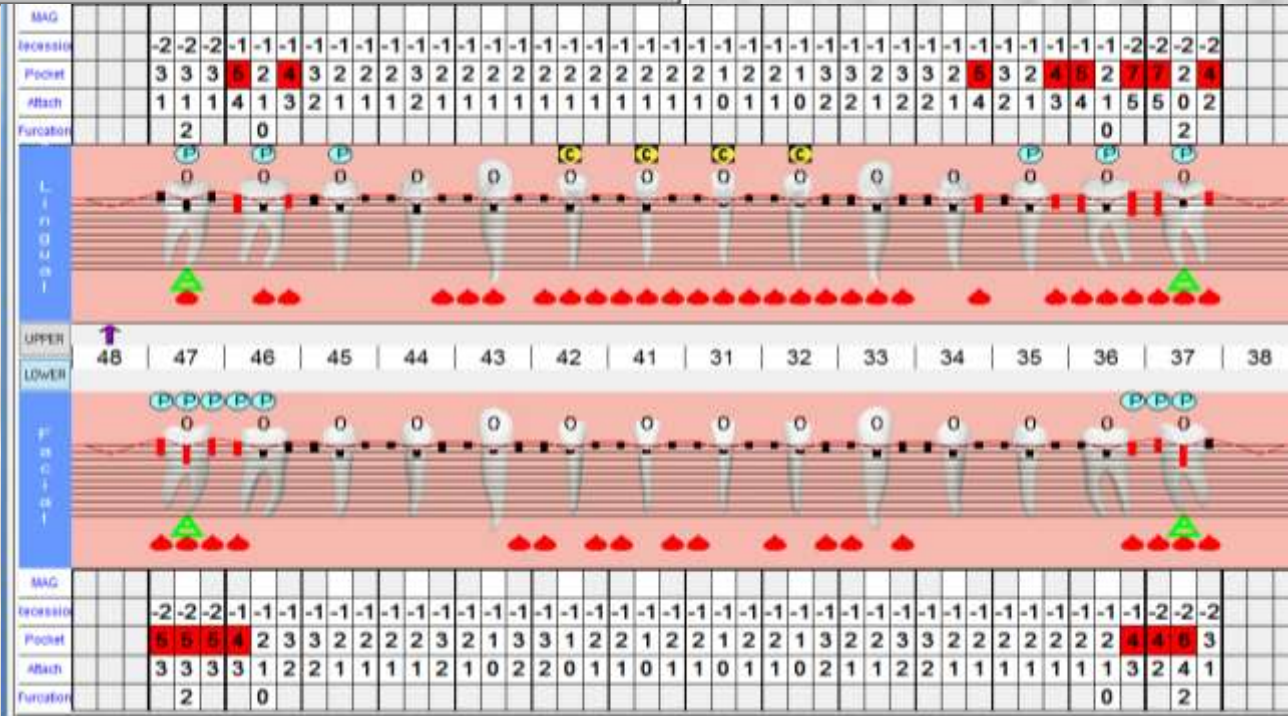
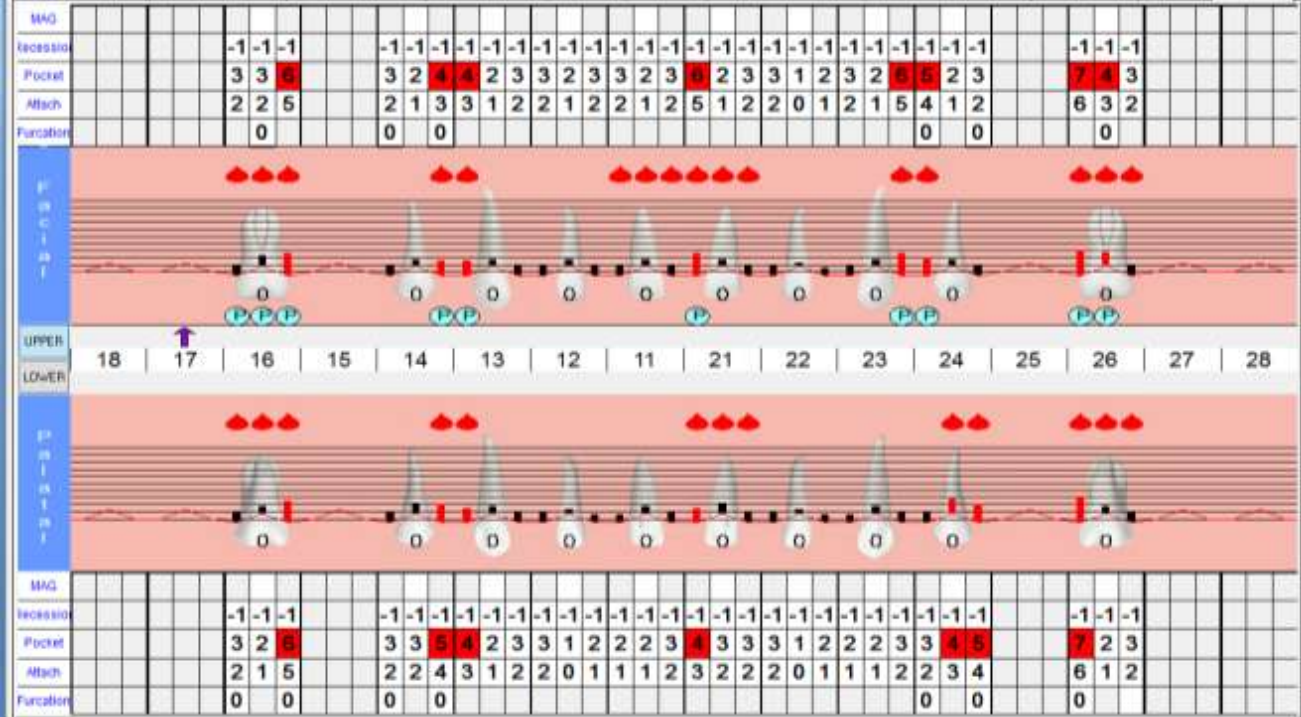
Dx: **Gingivitis**

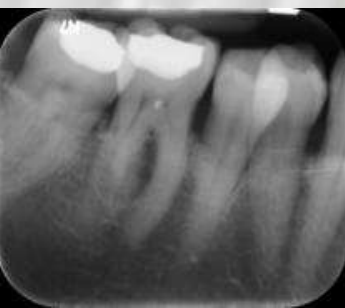
Case I

- Tx plan:
 - Full mouth scaling of affected teeth
 - Oral hygiene instruction
 - EIT after 6 weeks to see if BoP and PD have resolved, to see if OH has improved, and to decide on maintenance schedule

Case 2 – Pt. B

- 50 year old ♀ with Type 2 diabetes mellitus (unknown level of metabolic control) presents for initial exam
- No history of periodontal treatment
- Brushes and flosses 1x/day
- A previous dentist, with OMFS, had treatment planned bilateral sinus lifts and implants 15 and 25





Case 2

- Pt. B has:

- PD \geq 4mm and BoP

- 25% of teeth with severe AL/BL, 29% of teeth with moderate AL/BL, and 46% of teeth with mild AL/BL

- No family history (or other aggressive features)

Dx: Generalized mild chronic periodontitis with localized moderate chronic periodontitis on teeth I4, I3, 24, 35, 34, 46, 47 and localized severe chronic periodontitis on teeth I6, 23, 26, 37, 36

Case 2

- Tx Plan:
 - **STOP THE IMPLANT RESTORATIVE TX PLAN!**
 - Medical consult to assess patient's HbA1c
 - Sc/RP of affected teeth
 - Oral hygiene instruction
 - (endo consult 24)
 - Consider closing contacts 14/13, 21/22
 - EIT after 6 weeks etc.

Case 3 – Pt. C

- 14 year old healthy ♂ presents for initial exam, upon referral from pediatric dentist
- No previous periodontal treatment
- Mother and aunt have a history of “tooth loss at a young age.”
- No meds, no allergies



Case 3

- Pt. C has:
 - ☑ PD \geq 4mm and BoP
 - ☑ Severe attachment loss around incisors and first molars
 - ☑ An age of onset, a family history and relatively little plaque and calculus, which argues for aggressive

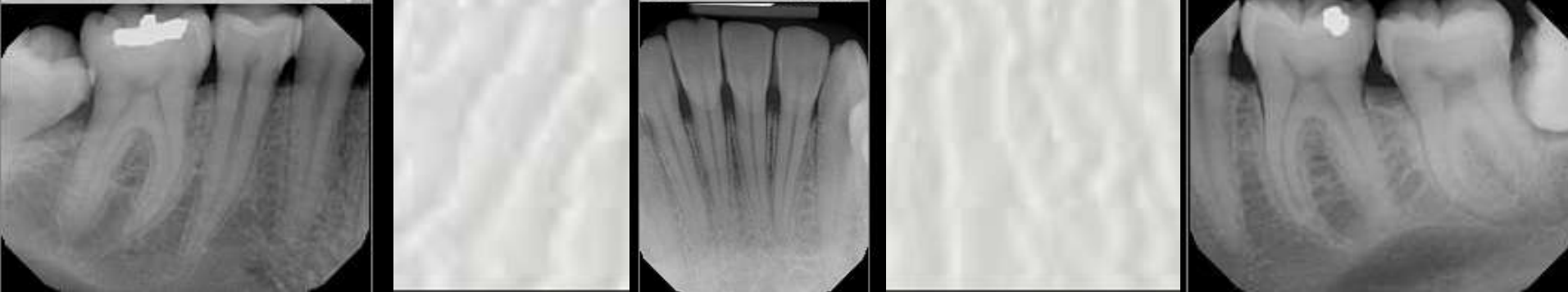
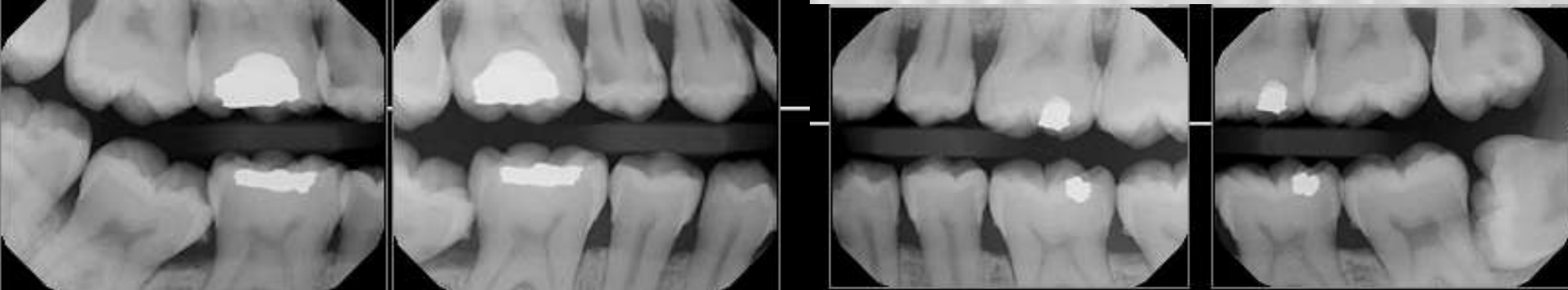
Dx: Localized severe aggressive
periodontitis

Case 3

- Tx plan:
 - Scaling and root planing of affected teeth
 - Antibacterial medication
 - Oral hygiene instruction
 - Advise parents to bring other siblings/cousins in to be screened
 - EIT after 6 weeks etc.

Case 4 – Pt. D

- 24 year old Hep C+ ♀ presents for initial exam
- Smokes 1/2 pack of cigarettes a day, and has for the last 7 years
- No history of periodontal treatment
- No family history of periodontal problems
- Brushes 2x/day but does not floss
- Feels that her “gums bleed very easily”



Case 4

- Pt. E has:

- PD \geq 4mm and BoP

- 16% of teeth with severe AL, and 60% of teeth with moderate AL, even though the radiographs don't show it

- No family history (or other aggressive features, despite her young age) NOR predisposing systemic conditions

Dx: Generalized moderate chronic periodontitis and localized severe chronic periodontitis on teeth 17, 14, 27, 33, 41

The most important case of the morning

The Little Details

- Anyone can pick up on periodontal disease when it's severe enough
- It takes a sharp eye and a sophisticated mind to pick up on it when it's in its mild stage
- Who cares, by the way?

We Do...

- ...because periodontitis is **MUCH** easier to treat when it's in its early stages

Case 4

- Tx plan:
 - Medical consult for liver function
 - Exo of third molars
 - Scaling and root planing of affected teeth
 - Oral hygiene instruction
 - Smoking cessation counseling
 - Caries control I5MD, I4D, 34D, 45D (and closure of contact on M)
 - EIT after 6 weeks etc.

Questions?



References

1. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol*. 1999 Dec;4(1):1-6.
2. Parameter on chronic periodontitis with slight to moderate loss of periodontal support. American Academy of Periodontology. *J Periodontol*. 2000 May;71(5 Suppl):853-5.
3. Parameter on chronic periodontitis with advanced loss of periodontal support. American Academy of Periodontology. *J Periodontol*. 2000 May;71(5 Suppl):856-8.
4. Oliver RC, Holm-Pedersen P, Loe H. The correlation between clinical scoring, exudate measurements and microscopic evaluation of inflammation in the gingiva. *J Periodontol*. 1969 Apr;40(4):201-9.
5. Greenstein G, Caton J, Polson AM. Histologic characteristics associated with bleeding after probing and visual signs of inflammation. *J Periodontol*. 1981 Aug;52(8):420-5.
6. Caffesse RG, Sweeney PL, Smith BA. Scaling and root planing with and without periodontal flap surgery. *J Clin Periodontol*. 1986 Mar;13(3):205-10.
7. Lindhe J, Socransky SS, Nyman S, Haffajee A, Westfelt E. "Critical probing depths" in periodontal therapy. *J Clin Periodontol*. 1982 Jul;9(4):323-36.
8. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: II. Incidence of sites breaking down. *J Periodontol*. 1996 Feb;67(2):103-8.

References

9. Claffey N, Nylund K, Kiger R, Garrett S, Egelberg J. Diagnostic predictability of scores of plaque, bleeding, suppuration and probing depth for probing attachment loss. 3 1/2 years of observation following initial periodontal therapy. *J Clin Periodontol.* 1990 Feb;17(2):108-14.
10. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest.* 1976 Mar;34(3):235-49.
11. Goodson JM, Haffajee AD, Socransky SS. The relationship between attachment level loss and alveolar bone loss. *J Clin Periodontol.* 1984 May;11(5):348-59.
12. Parameter on aggressive periodontitis. American Academy of Periodontology. *J Periodontol.* 2000 May;71(5 Suppl):867-9.
13. American Academy of Periodontology Task Force Report on the Update to the 1999 Classification of Periodontal Diseases and Conditions. *J Periodontol.* 2015 Jul;86(7):835-8.
14. Consensus Report: Aggressive Periodontitis. 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. *Ann Periodontol* 1999;4:53.

Overview



1. Introduction
2. Periodontal Examination and Charting
3. Diagnosis and Initial Therapy
4. Evaluation of Initial Therapy

Mini-Objectives

By the end of this part you will:

- know when to do and what to do during an evaluation of initial therapy appointment
- know what critical decisions have to be made at this appointment
- know how to make those decisions

Overview



1. Introduction
2. When and what to do during evaluation of initial therapy
3. Big decisions!
4. Where to go from here....

Overview



I. Introduction

**Recall our patient from Case 2,
Pt. B**

Case 2

- 50 year old ♀ with Type 2 diabetes mellitus (unknown level of metabolic control) presents for initial exam
- No history of periodontal treatment
- Brushes and flosses 1x/day
- A previous dentist, with OMFS, had treatment planned bilateral sinus lifts and implants 15 and 25

Case 2

- Pt. B has:

- ☑ PD \geq 4mm and BoP
- ☑ 25% of teeth with severe AL/BL, 29% of teeth with moderate AL/BL, and 46% of teeth with mild AL/BL
- ☑ No family history (or other aggressive features) nor predisposing systemic conditions (remember DM Type 2 doesn't count)

Dx: Generalized mild chronic periodontitis with localized moderate chronic periodontitis on teeth I4, I3, 24, 35, 34, 46, 47 and localized severe chronic periodontitis on teeth I6, 23, 26, 37, 36

Case 2

- Tx Plan:
 - **STOP THE RESTORATIVE TX PLAN!**
 - Medical consult to assess patient's HbA1c
 - Sc/RP of affected teeth
 - Oral hygiene instruction
 - (endo consult 24)
 - Consider closing contacts 14/13, 21/22
 - EIT after 6 weeks etc.

Treatment Sequence Up Until Now

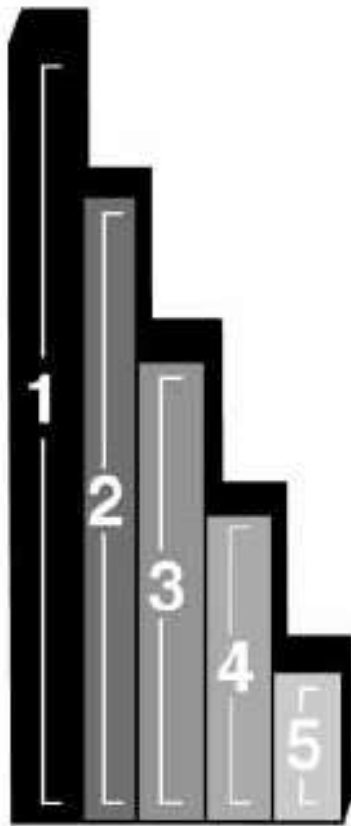
- If you have done things properly, you have:
 1. Performed an initial complete exam and taken appropriate radiographs
 2. Made a diagnosis
 3. Formulated a treatment plan
 4. Received informed consent from the patient
 5. Executed initial therapy

Evaluation

- When you do therapy, there has to be a measurable outcome by which to evaluate its effectiveness
- Evaluation of Initial Therapy aka EIT, aka Re-evaluation, is when this happens

The “Evaluation Criteria Staircase”¹

The “evaluation criteria staircase”



L e v e l

Probing pocket depth ≤ 4 mm (a)

No clinical signs of gingival inflammation (b)

No bleeding on pocket probing (c)

No further loss of clinical attachment (d)

No further loss of alveolar bone (e)

Overview



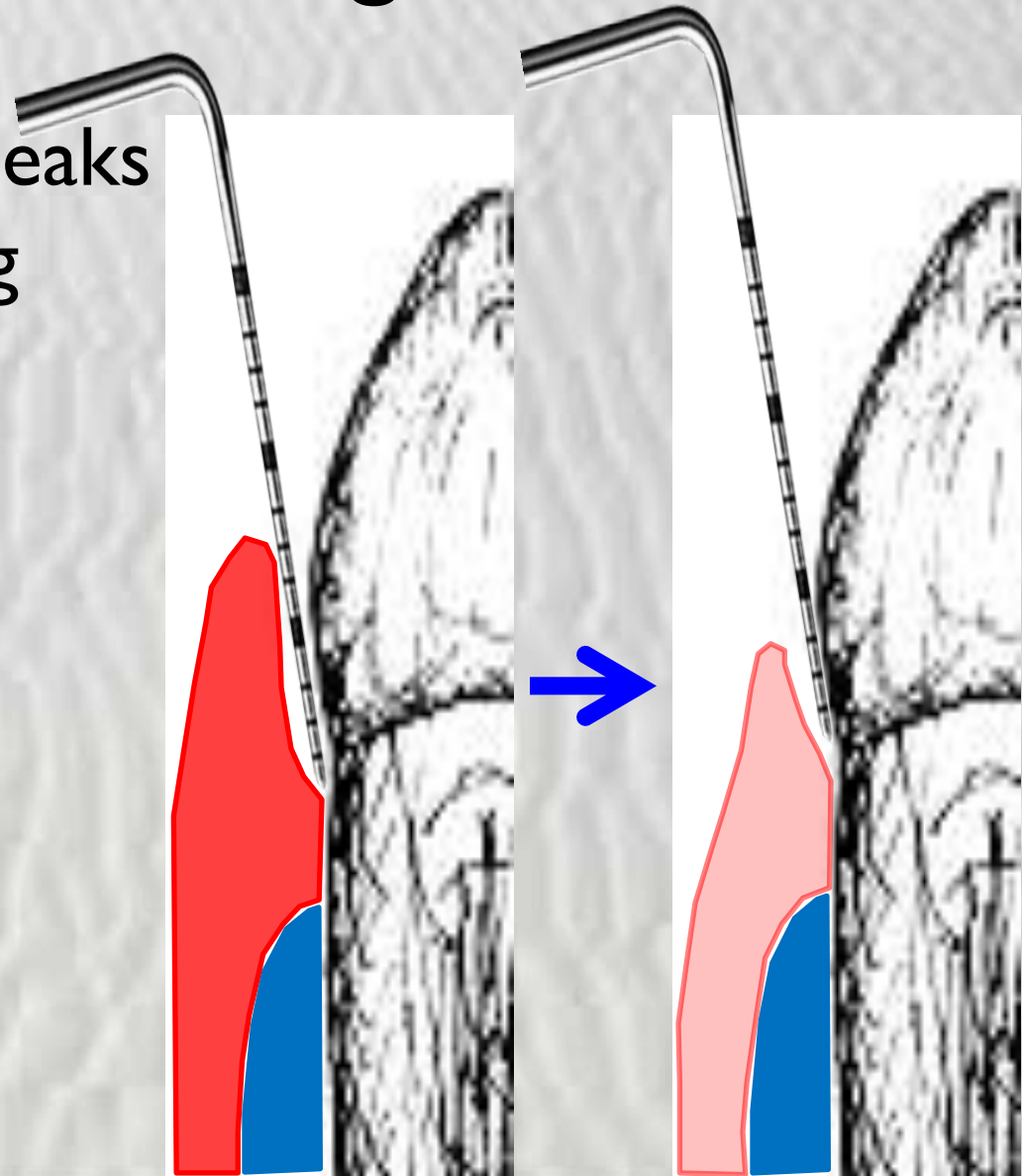
1. Introduction
2. When and what to do during evaluation of initial therapy

Timing of EIT

- When is it appropriate to evaluate one's initial therapy?
- The timing is measured from the Sc/RP, which while not the only component of initial therapy, should be performed first* (with the possible exception of select extractions in certain circumstances)

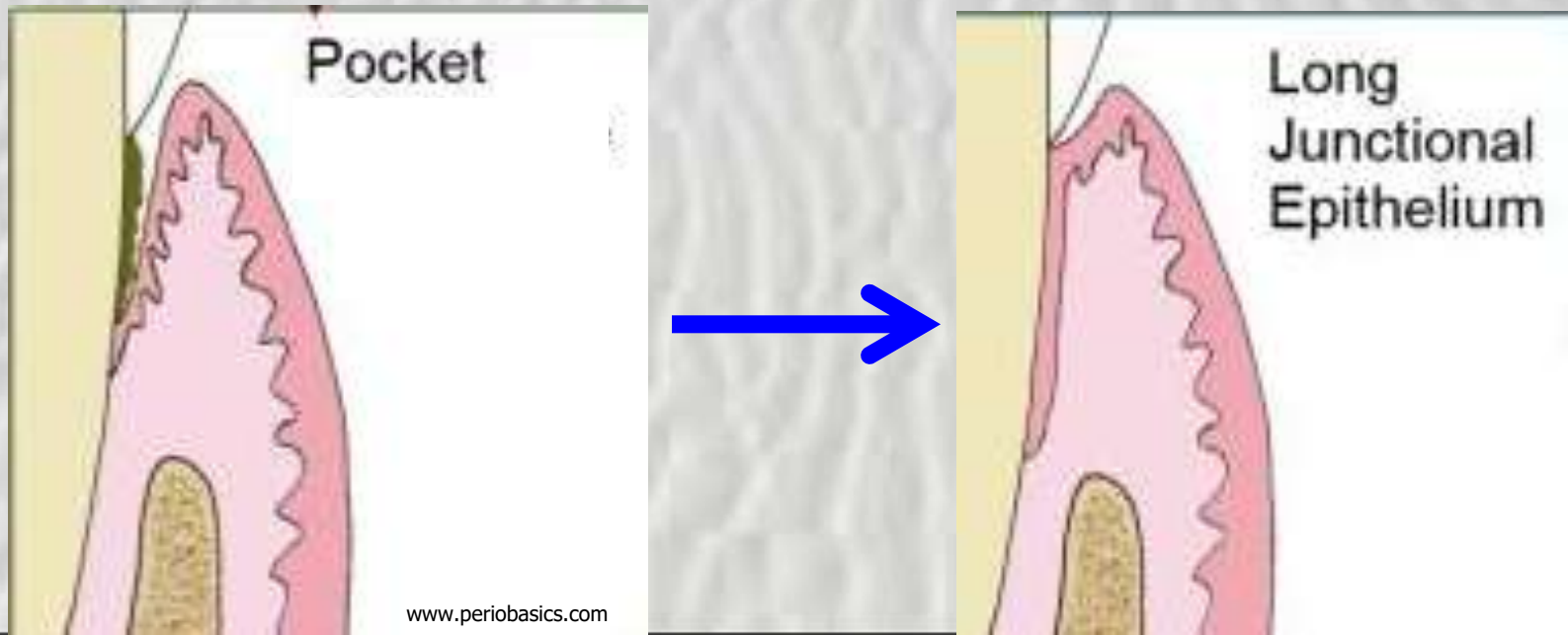
Recession following Sc/RP

- Gingival recession peaks at 8 weeks following Sc/RP²



Histological Healing following Sc/RP

- The pocket heals by **repair** in the form of a long junctional epithelium
- This begins as early as 2 weeks after Sc/RP³



Reduction in BoP following Sc/RP

- HIGHLY variable
- If you average the results of all the relevant studies, you get a 57% reduction in BoP following Sc/RP⁴
- However, this is dependant on the patient's level of oral hygiene

Probing Depth Reduction following Sc/RP

- HIGHLY variable
- The greatest reductions in PD were found by 8 weeks after Sc/RP²
- One can expect an average PD reduction of⁴:
 - 1.29 mm in pockets 4-6 mm deep
 - 2.16 mm in pockets ≥ 7 mm deep

By the way....

- What are the mechanisms of probing depth reduction following Sc/RP?

1. Recession²

2. Gain in clinical attachment via formation of long junctional epithelium⁵
3. Reconstitution of the integrity of the junctional epithelium⁶

So....

- ...what's too early?
 - Less than 4 weeks
- ...what's too late?
 - More than 8 weeks

Aim for 6 weeks

(following Sc/RP)

What to do during EIT?

- During an EIT appointment, you should:
 - Perform a complete exam
 - Make future treatment decisions with your patient
 - Scale teeth as necessary (and smooth out the roots if you see/feel any rough areas)
 - Re-iterate oral hygiene instructions as necessary

Overview



1. Introduction
2. When and what to do during evaluation of initial therapy
3. **Big decisions!**

Decisions

- Two decisions have to be made at EIT:
 1. Decision about a periodontal maintenance schedule
 2. Decision about what further therapy is indicated

Maintenance

- **PATIENTS WITH DIAGNOSES OF PERIODONTITIS NEED MAINTENANCE APPOINTMENTS q3-4 MONTHS⁷**

Future Therapy

- That leaves a decision about future periodontal therapy as the most important thing to do at an EIT appointment
- This is based on the patient's EIT charting

Step 1. Comparison

- The first thing to do when making future treatment decisions is to compare the EIT charting with the initial charting

Step 1. Comparison

- So for all the areas that on the initial exam had:
 - deep probing depths...did PD decrease?
 - bleeding on probing...is it still there?
 - visual inflammation...is it still there?
 - plaque and calculus accumulation...are they still present?

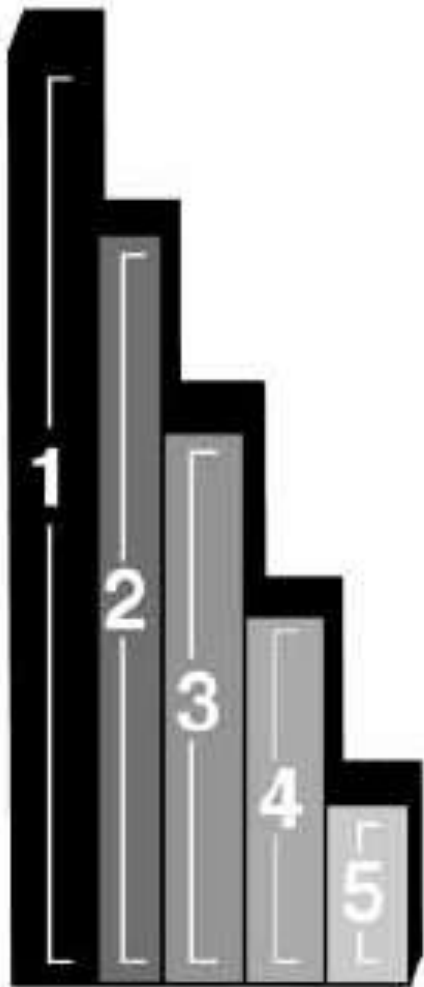
Comparison

- If you got an improvement, good for you, and for your patient 😊
- **HOWEVER**, an improvement alone does not mean the patient does not need further treatment

Comparison

- The real question you should be asking is, “Did my patient’s measures improve to an acceptable enough level that no further active treatment is necessary?”

Ideal Situation



Probing pocket depth ≤ 4 mm (a)

No clinical signs of gingival inflammation (b)

No bleeding on pocket probing (c)

No further loss of clinical attachment (d)

No further loss of alveolar bone (e)

L e v e l

Ideal Situation

- Wherever you have the ideal result, the patient needs no further therapy in that region (this region is a healthy reduced periodontium), and they **must** be put on a periodontal maintenance schedule

So now 4mm is alright?

- A 4mm probing has different meanings at different stages of treatment
- While it is considered pathological in an untreated patient, following treatment, if it is not bleeding, it is often considered maintainable

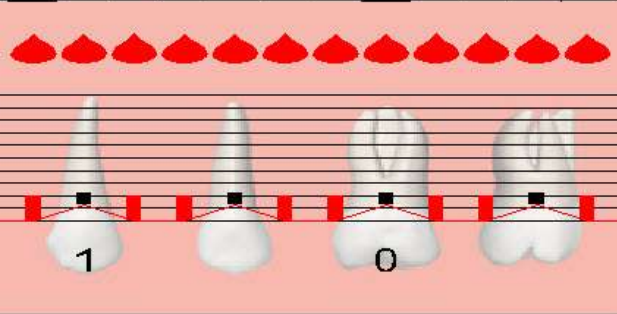
**Does that mean that no 4mm
pocket ever needs surgery?**

Exception #1

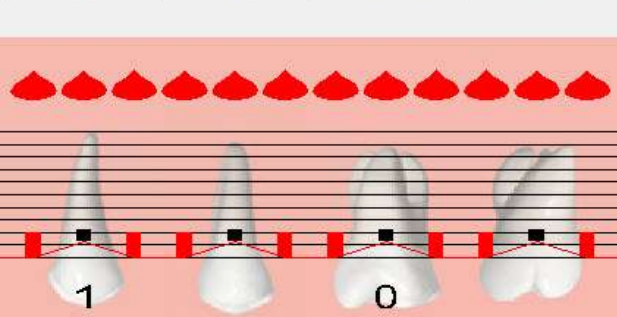
- **IF** you have a region of 4mm pockets, all of which are BoP, inflamed, and associated with radiographic evidence of osseous defects...

Exception #1

0	0	0	0	0	0	0	0	0	0	0	0
4	2	4	4	2	4	4	2	4	4	2	4
4	2	4	4	2	4	4	2	4	4	2	4
0		0				0			0		



24	25	26	27
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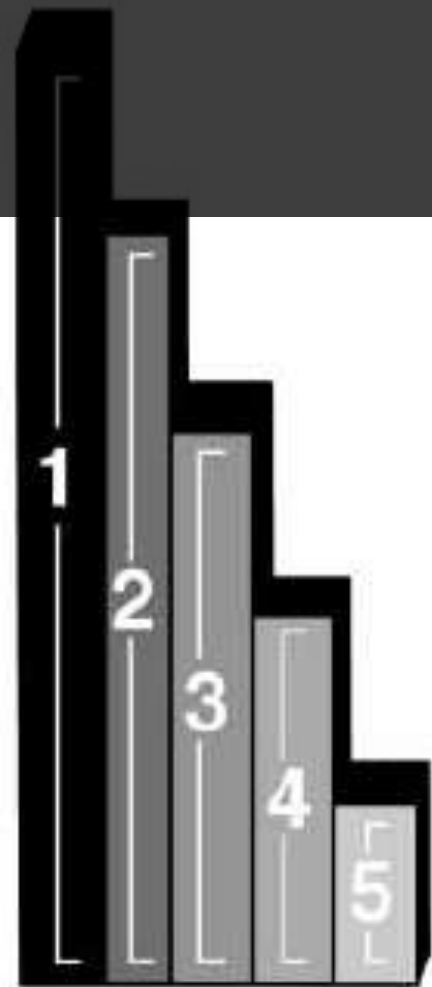


0	0	0	0	0	0	0	0	0	0	0	0
4	2	4	4	2	4	4	2	4	4	2	4
4	2	4	4	2	4	4	2	4	4	2	4
0		0			0		0	0		0	

...then surgical correction is indicated



Less than Ideal Situation



Probing pocket depth ≤ 4 mm (a)

No clinical signs of gingival inflammation (b)

No bleeding on pocket probing (c)

No further loss of clinical attachment (d)

No further loss of alveolar bone (e)

L e v e l

Less than Ideal Situation

- **POCKETS \geq 5 MM GENERALLY NEED TO BE CORRECTED SURGICALLY**, because mostly, they're unmaintainable, and very susceptible to further periodontal breakdown^{11,12}
- At the same time, the patient **must** be put on a periodontal maintenance schedule

What Happened?

- If all the inflammation resolved, why didn't the probing depth resolve with it?
- 2 reasons:
 - 1. Presence of osseous defects...whether you can see them radiographically or not!**
 2. Tissue character (fibrotic gingiva will shrink less than edematous gingiva)

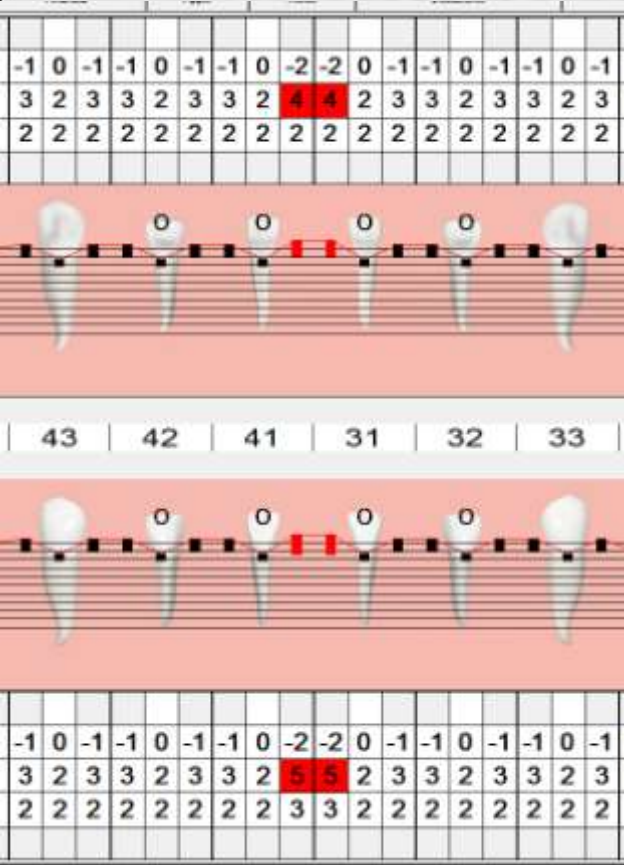
Does that mean we're going to surgerize every pocket $\geq 5\text{mm}$?

Exception #2

- **IF** you have an isolated pocket of 5mm which is
 - not bleeding and not inflamed,
 - not accumulating plaque or calculus,
 - not associated with an osseous defect,
 - in a healthy, non-smoking patient with excellent oral hygiene and maintenance compliance....

Exception #2

...then you may consider maintaining that area non-surgically



Wait a minute....

Why not re-Sc/RP everywhere?

- Why can't you just redo your Sc/RP a second time, for all the sites that don't respond, to see if you do better?

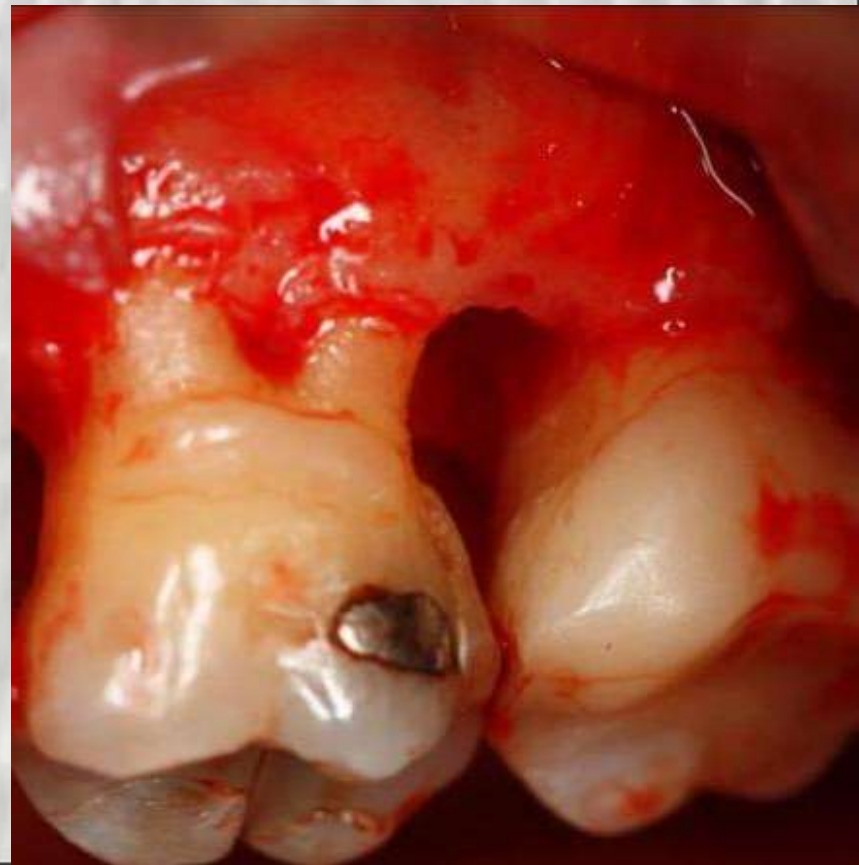


Why not re-Sc/RP everywhere?

- Studies have shown that for pockets $\geq 5\text{mm}$, surgical therapy is most predictable at reducing their depth^{8,9,10}
- Long-term studies demonstrate that sites which have only been subjected to Sc/RP are at higher risk of disease recurrence than sites which received surgery^{11,12}

Why not re-Sc/RP everywhere?

- Why?
- Because underlying osseous defects are:
 - Very common^{13,14,15}
 - Not addressed by scaling and root planing



By the way...

- ...were you all aware of the damage you can cause with too much Sc/RP?



Courtesy of Dr. Tom Koertge



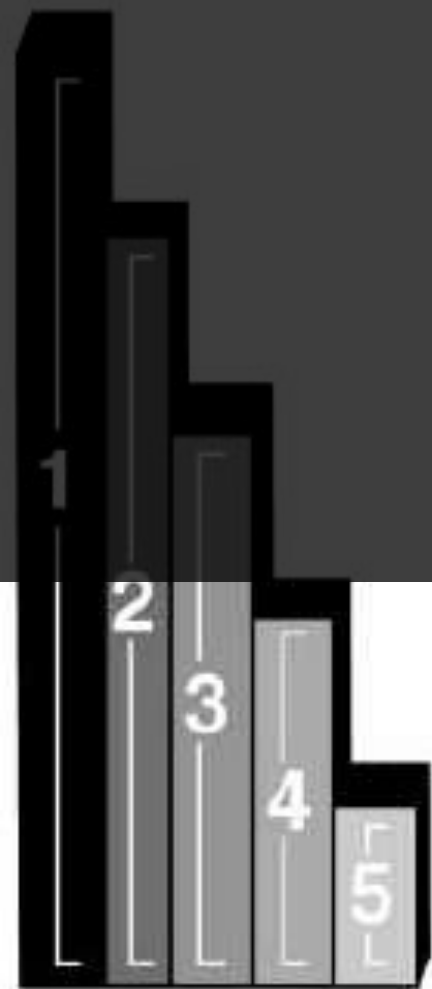
Courtesy of Dr. Sharon Lanning

Exception #3

- **IF** you have an isolated pocket of 4-6mm which
 - has been Sc/RP,
 - **still has clinically detectable subgingival calculus,**
 - is not associated with an osseous defect,

then you may consider redoing the Sc/RP at that site

Not so Great Situation



Probing pocket depth ≤ 4 mm (a)

No clinical signs of gingival inflammation (b)

No bleeding on pocket probing (c)

No further loss of clinical attachment (d)

No further loss of alveolar bone (e)

L e v e l

Not so Great Situation

- In this case, you still have problems with pocket depth and inflammation, and surgical therapy is indicated, **IF** the cause of the inflammation is not poor OH in the form of significant plaque and calculus accumulation
- The patient **must** be put on a periodontal maintenance schedule

Not so Great Situation

- If the reason for the non-resolution of inflammation is that the patient's OH is inadequate, then prior to surgical therapy, OH must be improved¹⁶
- The patient is put onto a periodontal maintenance schedule, and as soon as OH improves adequately, you can do surgery

Bad Situation



Probing pocket depth ≤ 4 mm (a)

No clinical signs of gingival inflammation (b)

No bleeding on pocket probing (c)

No further loss of clinical attachment (d)

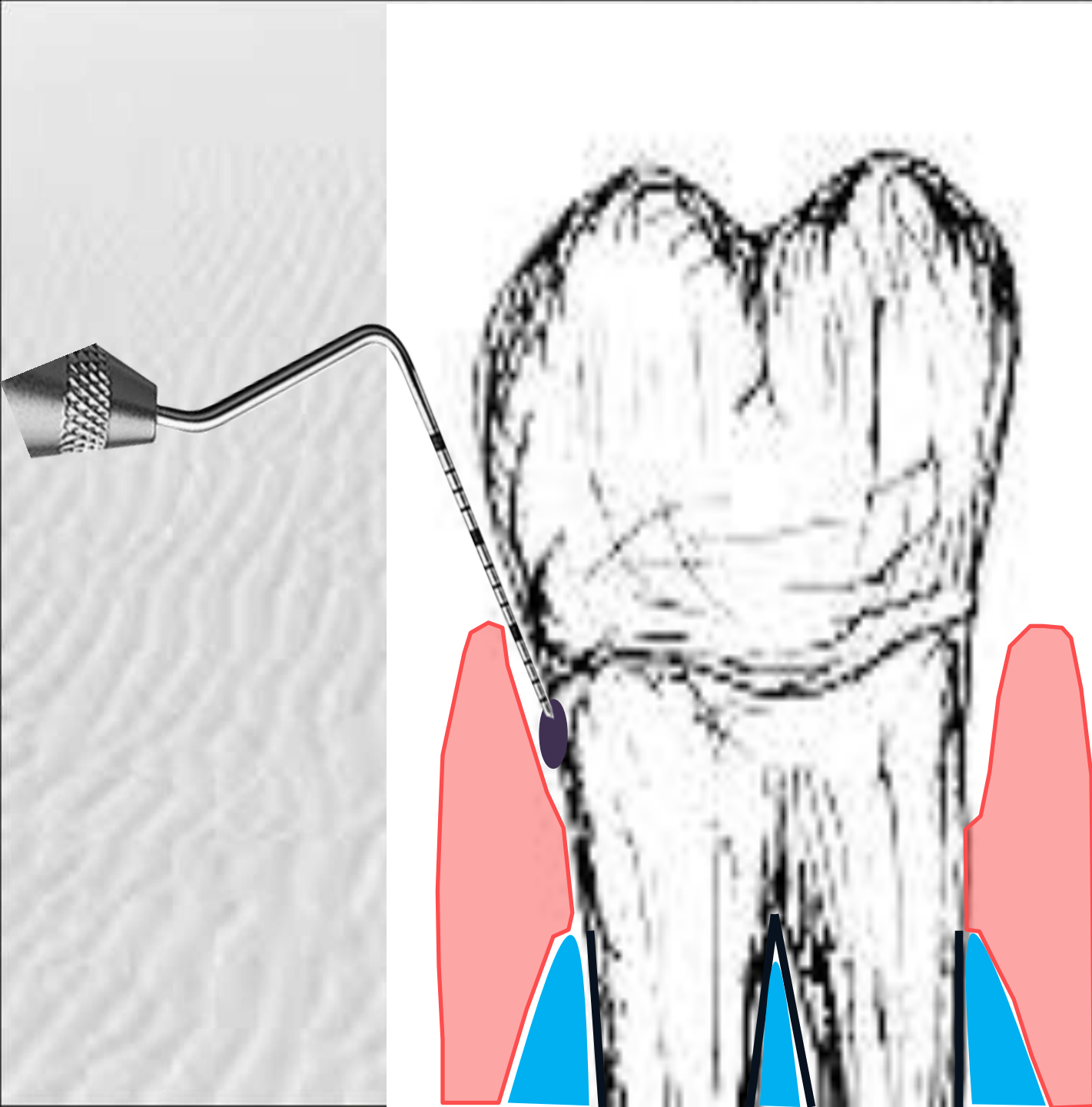
No further loss of alveolar bone (e)

Bad Situation

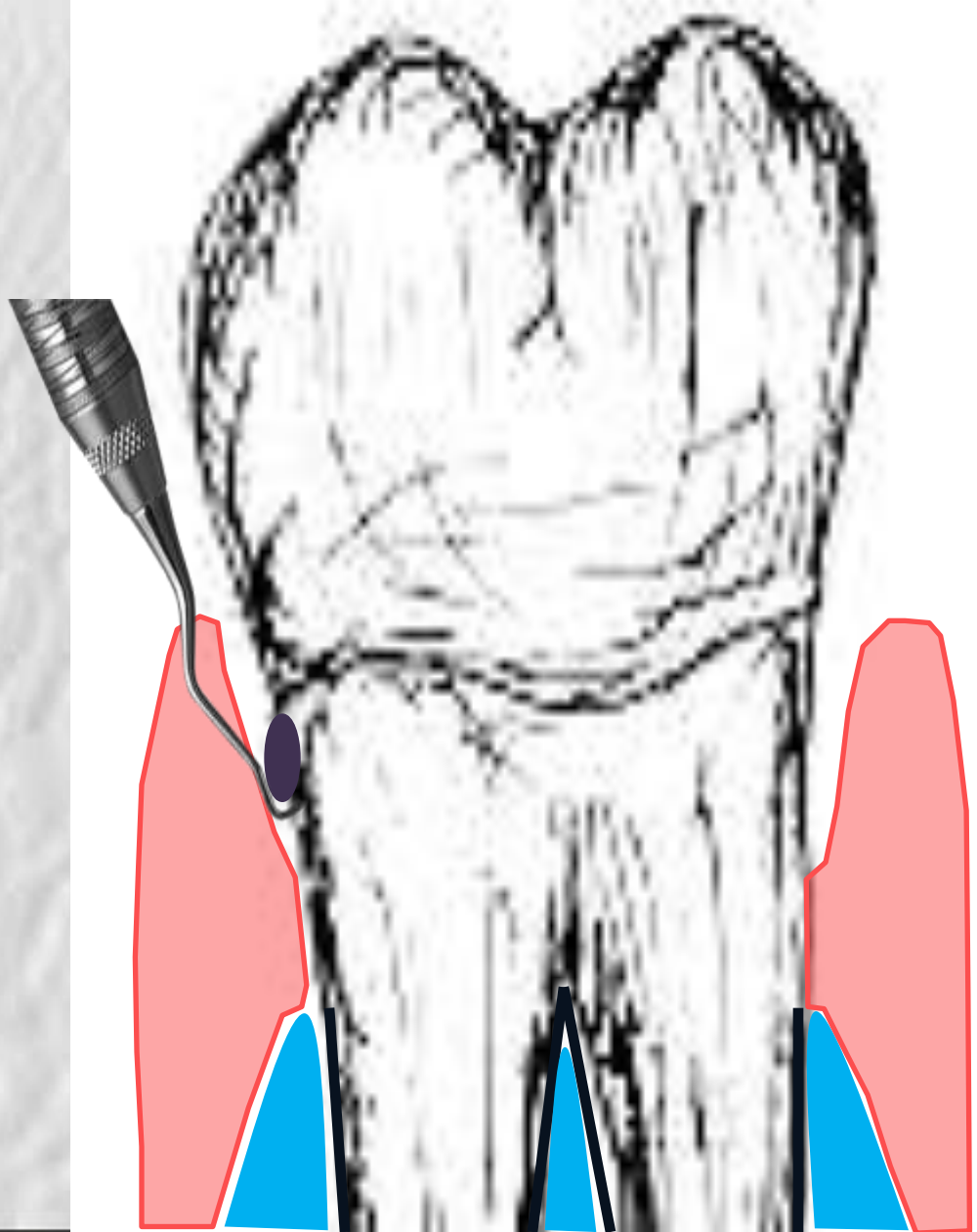
- What happens, if at the EIT appointment, in addition to persistent PD $\geq 5\text{mm}$ and persistence of BoP and inflammation, you have progressing attachment and bone loss?

Well, hold on a minute....

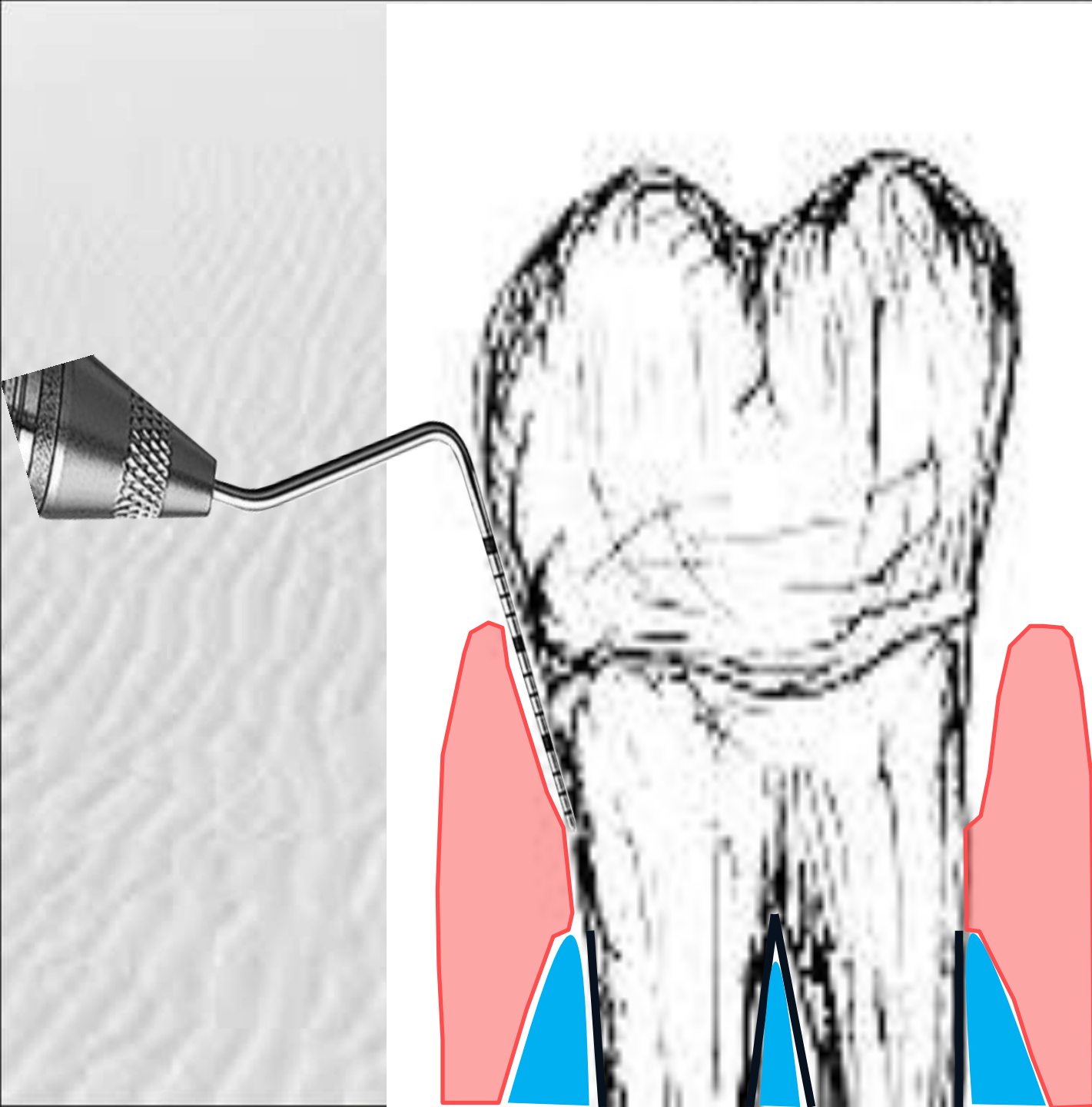
- If you have probing depth, and thus by extension attachment loss, which is **WORSE** at EIT than it was initially, then there is a strong possibility your initial probing was wrong
- Why?



Initial exam



Sc/RP



Evaluation
of initial
therapy
exam

Progression?

- So if your clinical measurements are implying progression, take another radiograph to confirm

Progression!

- If your radiographs are demonstrating additional bone loss, clearly identifiable, in such a short time period, then you are dealing with an aggressive periodontitis, or a periodontitis as a manifestation of systemic disease, and immediate referral to a periodontist is advised

Overview



1. Introduction
2. When and what to do during evaluation of initial therapy
3. Big decisions!
4. Where to go from here....

Chronic Disease

- The nature of a **chronic** disease is that its effects are long-lasting, if not everlasting
- Many **chronic** diseases can't be cured, but instead can be treated or managed
- **Periodontitis** falls into this category

Chronic Disease

- Since the attachment loss seen in **periodontitis** most often can not be regenerated fully, this brings up the question, ‘How do we diagnose a patient who has been successfully treated for **periodontitis**?’
- So back to Case 2....

Case 2

- Tx Plan:
 - **STOP THE RESTORATIVE TX PLAN!**
 - Medical consult to assess patient's HbA1c
 - Sc/RP of affected teeth
 - Oral hygiene instruction
 - (endo consult 24)
 - Consider closing contacts 14/13, 21/22
 - EIT after 6 weeks etc.

Case 2

- Pt. B got her medical consult, which showed her HbA_{1c} to be at 8.5%. Her physician adjusted her meds, and it came down to 6.5%
- Sc/RP was performed, and OH was STRESSED
- 6 weeks later, at EIT....

EIT Update

Updated Diagnosis: **Localized severe chronic periodontitis** on 16, 23, 26, 36, 37 and **localized moderate chronic periodontitis** on 24 on a **healthy reduced periodontium**

- Treatment Plan:

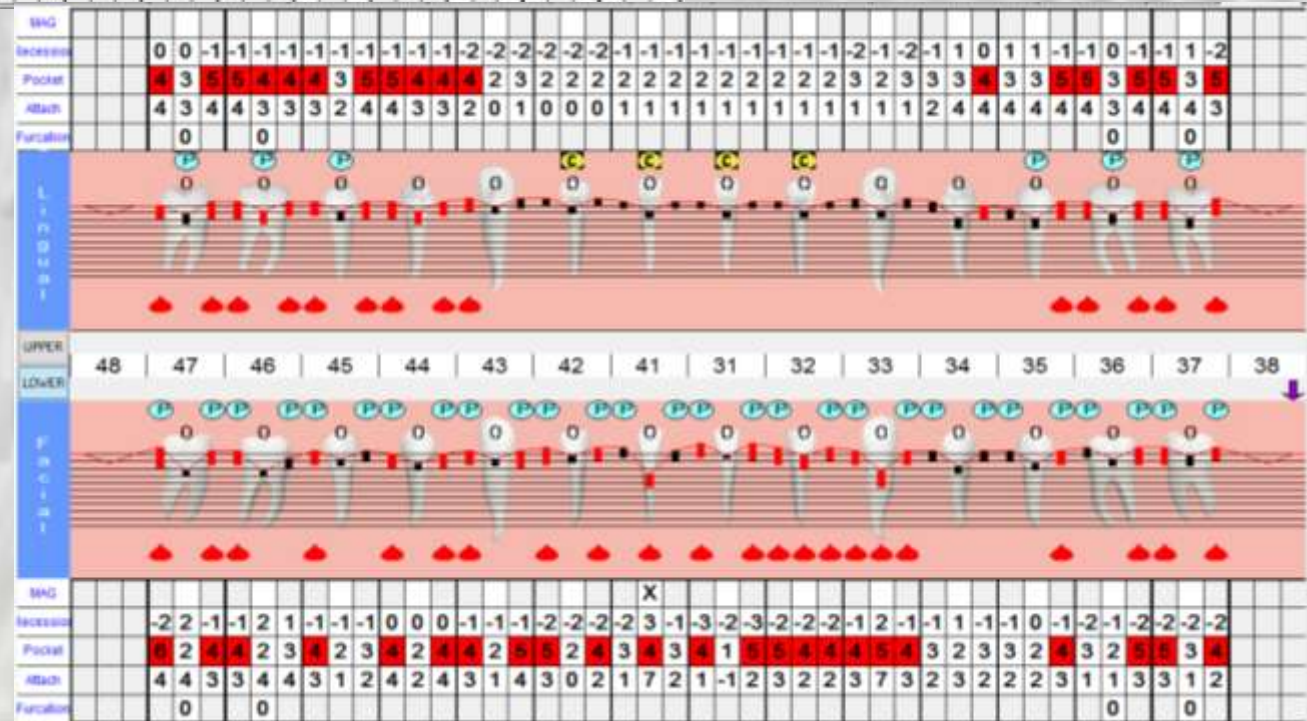
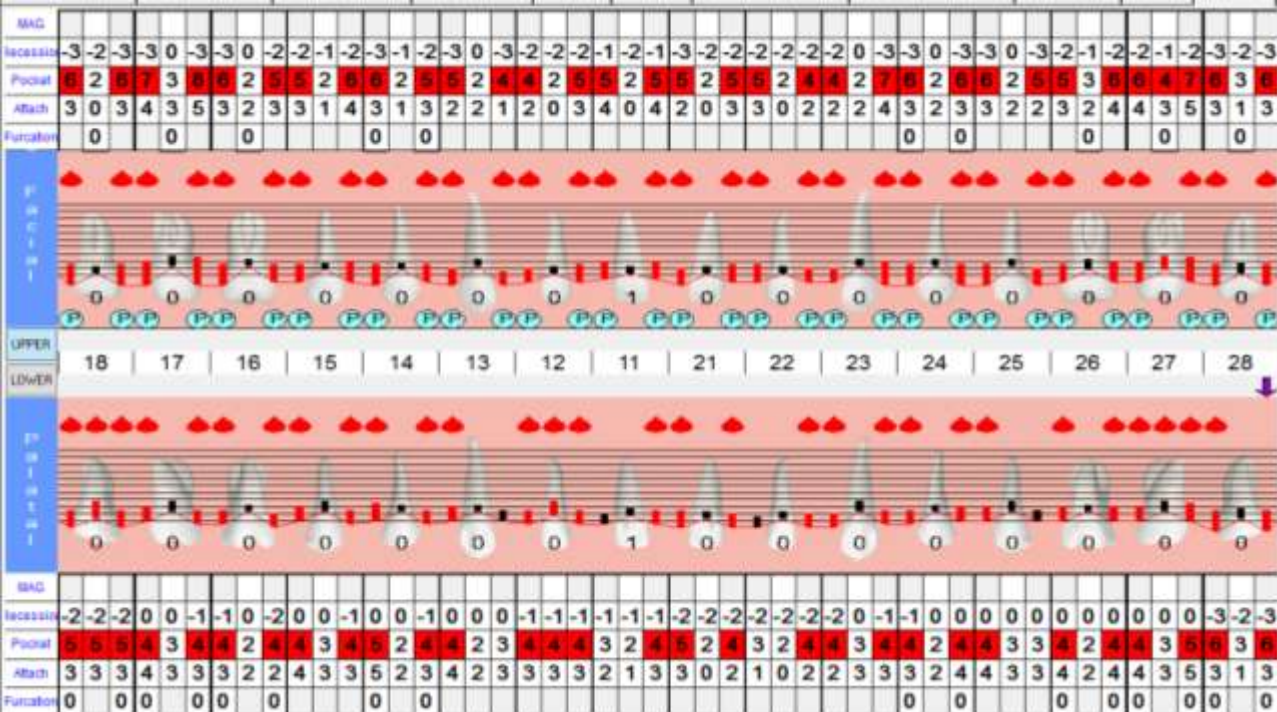
Sextant 1 Surgical therapy	Sextant 2 No sx	Sextant 3 Surgical therapy
Sextant 6 No sx	Sextant 5 No sx	Sextant 4 Surgical therapy

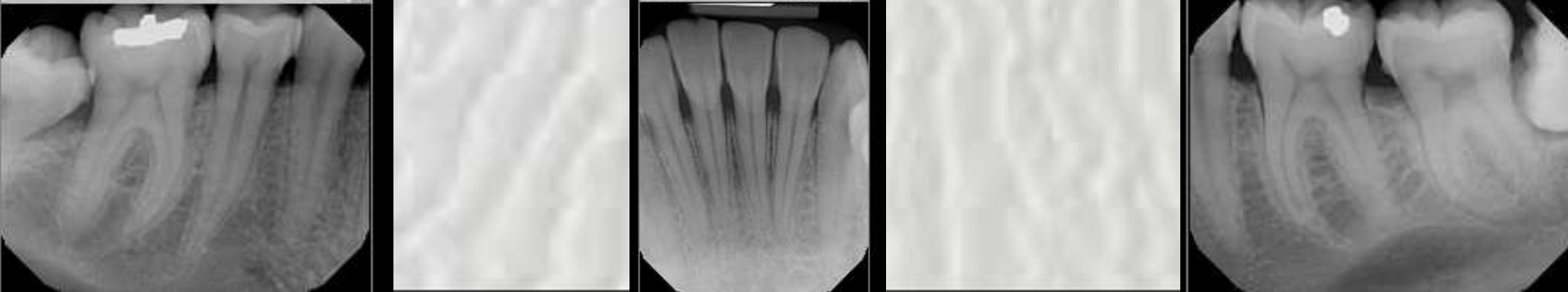
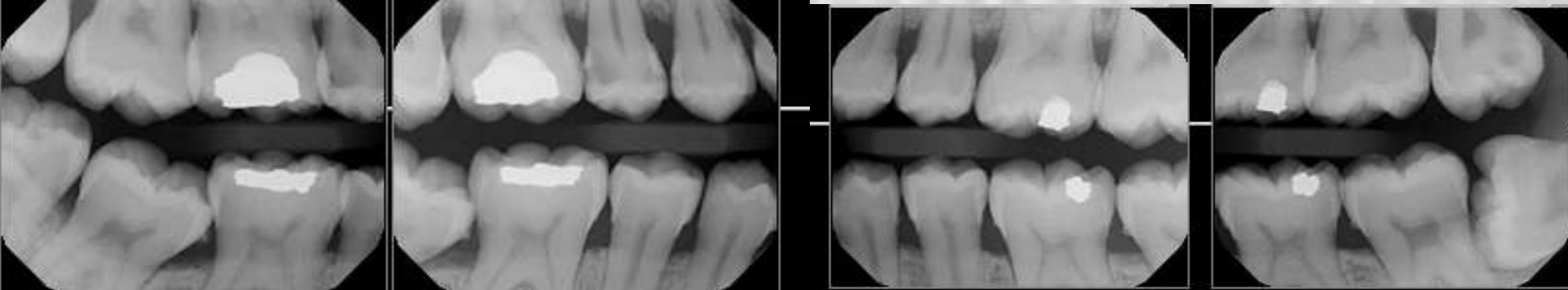
- Maintenance: q3months

The Healthy Reduced Periodontium

- In this case, the diagnosis of localized blah blah blah **on a healthy reduced periodontium** reflects the fact that while there are still inflammatory issues in some areas, others have been treated and are stable, but are at reduced attachment levels

Back to Case 4....





Case 4

- Pt. D has:

- ☑ PD \geq 4mm and BoP
- ☑ 16% of teeth with severe AL, and 60% of teeth with moderate AL, even though the radiographs don't show it
- ☑ No family history (or other aggressive features, despite her young age) nor predisposing systemic conditions

Dx: Generalized moderate chronic periodontitis
and localized severe chronic periodontitis on
teeth 17, 14, 27, 33, 41

Case 4

- Tx plan:
 - Medical consult for liver function
 - Exo of third molars
 - Scaling and root planing of affected teeth
 - Oral hygiene instruction
 - Smoking cessation counseling
 - Caries control I5MD, I4D, 34D, 45D (and closure of contact on M)
 - EIT after 6 weeks etc.

EIT Update

- Updated Diagnosis: **Generalized moderate chronic periodontitis** and **localized severe chronic periodontitis** on teeth 17, 14, 27, 33; inadequate attached gingiva 33, 41
- Treatment Plan:

Sextant 1 Surgical therapy	Sextant 2 No sx	Sextant 3 Surgical therapy
Sextant 6 Surgical therapy	Sextant 5 Mucogingival surgical therapy	Sextant 4 Surgical therapy

- Maintenance: q3months

“Why do I need surgery? I thought the deep cleaning was the treatment for periodontitis.”

Patient Communication

- If your patient says this to you, it means you screwed up



Remember this Slide?

- If you have done things properly, you have:
 1. Performed an initial complete exam and taken appropriate radiographs
 2. Made a diagnosis
 3. Formulated a treatment plan
 4. **Received informed consent from the patient**
 5. Executed initial therapy

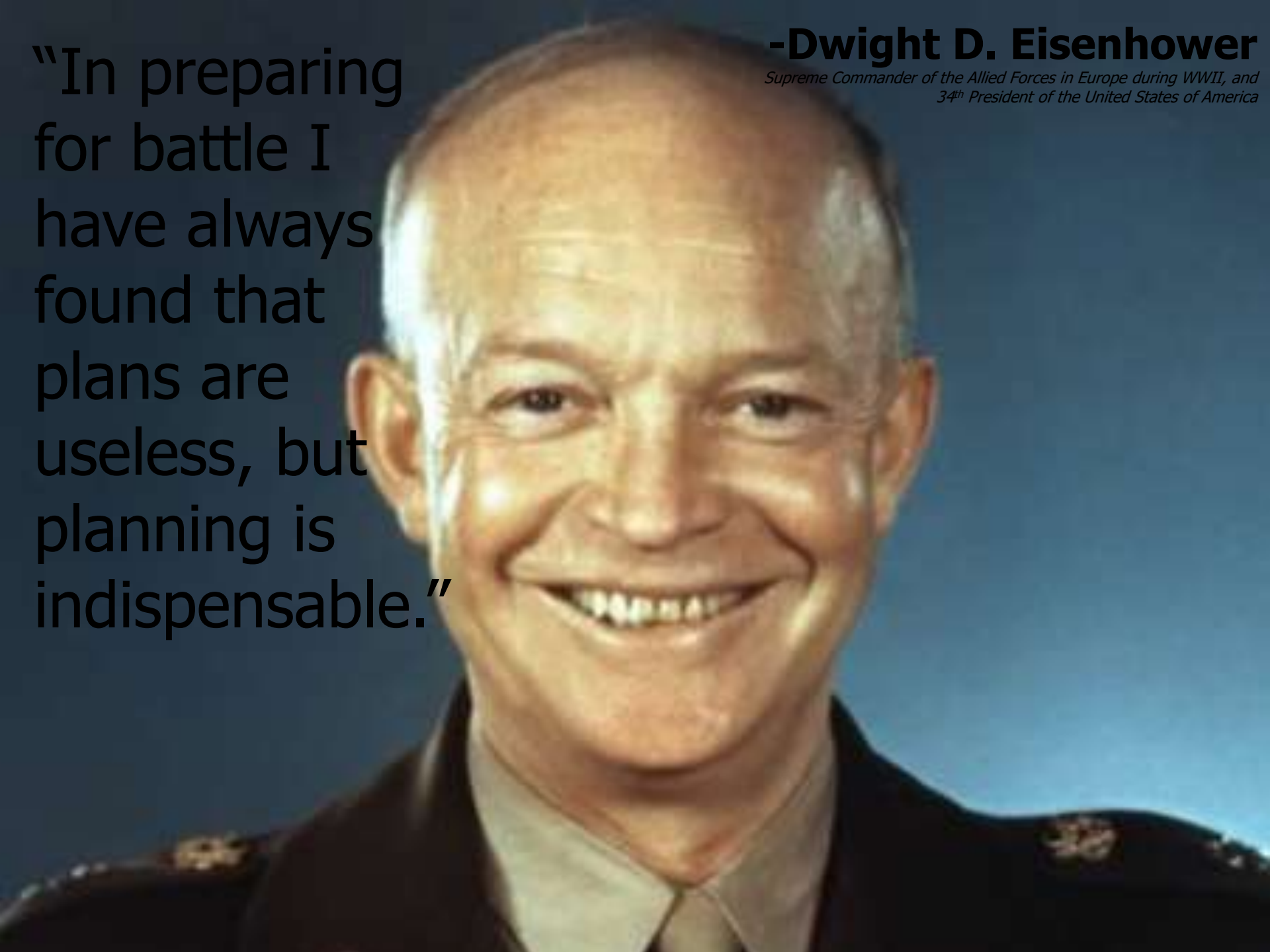
Patient Communication

- You should **NOT** present initial therapy to your patient as definitive treatment for periodontitis
- Rather, you should present it as just that...initial therapy, the first step, phase one of treatment, etc.

“In preparing for battle I have always found that plans are useless, but planning is indispensable.”

-Dwight D. Eisenhower

*Supreme Commander of the Allied Forces in Europe during WWII, and
34th President of the United States of America*



Questions?



Thanks for being a wonderful audience!

References

1. Lundgren D, Asklöv B, Thorstensson H, Härefeldt AM. Success rates in periodontal treatment as related to choice of evaluation criteria. Presentation of an evaluation criteria staircase for cost-benefit use. *J Clin Periodontol.* 2001 Jan;28(1):23-30.
2. Badersten A, Nilvéus R, Egelberg J. Effect of nonsurgical periodontal therapy. I. Moderately advanced periodontitis. *J Clin Periodontol.* 1981 Feb;8(1):57-72.
3. Waerhaug J. Healing of the dento-epithelial junction following subgingival plaque control. I. As observed in human biopsy material. *J Periodontol.* 1978 Jan;49(1):1-8.
4. Cobb CM. Non-surgical pocket therapy: mechanical. *Ann Periodontol.* 1996 Nov;1(1):443-90.
5. Tagge DL, O'Leary TJ, El-Kafrawy AH. The clinical and histological response of periodontal pockets to root planing and oral hygiene. *J Periodontol.* 1975 Sep;46(9):527-33.
6. Fowler C, Garrett S, Crigger M, Egelberg J. Histologic probe position in treated and untreated human periodontal tissues. *J Clin Periodontol.* 1982 Sep;9(5):373-85.
7. Rosling B, Serino G, Hellström MK, Socransky SS, Lindhe J. Longitudinal periodontal tissue alterations during supportive therapy. Findings from subjects with normal and high susceptibility to periodontal disease. *J Clin Periodontol.* 2001 Mar;28(3):241-9.
8. Antczak-Bouckoms A, Joshipura K, Burdick E, Tulloch JFC. Meta-analysis of surgical versus non-surgical methods of treatment for periodontal disease. *J Clin Periodontol* 1993;20: 259–268.
9. Heitz-Mayfield L, Trombelli L, Heitz F, Needleman I, Moles D. A systematic review of the effect of surgical debridement vs. non-surgical debridement for the treatment of chronic periodontitis. *J Clin Periodontol* 2002; 29 (Suppl. 3): 92–102.
10. Hung H-C, Douglass CW. Meta-analysis of the effect of scaling, root planing, surgical treatment and antibiotic therapies on periodontal probing depth and attachment loss. *J Clin Periodontol* 2002; 29: 975–986.

References

11. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: I. Response to 4 therapeutic modalities. *J Periodontol.* 1996 Feb;67(2):93-102.
12. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: II. Incidence of sites breaking down. *J Periodontol.* 1996 Feb;67(2):103-8.
13. Manson JD. Bone morphology and bone loss in periodontal disease. *J Clin Periodontol.* 1976 Feb;3(1):14-22.
14. Tal H. The prevalence and distribution of intrabony defects in dry mandibles. *J Periodontol.* 1984 Mar;55(3):149-54.
15. Vrotsos JA, Parashis AO, Theofanatos GD, Smulow JB. Prevalence and distribution of bone defects in moderate and advanced adult periodontitis. *J Clin Periodontol.* 1999 Jan;26(1):44-8.
16. Nyman S, Lindhe J, Rosling B. Periodontal surgery in plaque-infected dentitions. *J Clin Periodontol.* 1977 Nov;4(4):240-9.