

ENDO II

(FALL JR. YEAR)

TO SAVE SOME TIME:

- My notes from lectures that there were no powerpoint / handouts from...
- REMEMBER THE FINAL IS CUMULATIVE FROM BOTH SOPH. + JR. YEARS!!!

LEC. 2Traumatic Injury to Teeth

9/5/06

Subluxation - ^{abnormal} loosening of tooth w/in socket - NOT displaced.
Luxation - ^{physical} displacement of tooth w/in socket

Types of Luxation

- 1) Lateral - B or L displacement
- 2) Extrusive - out of socket
- 3) Intusoria - into socket * Bad to have
 ⇒ complete destruction to PDL in this injury.

Complications of Luxation Injuries (LORN)

- 1) Necrosis
- 2) Obliteration - injury causes pulp canal to become calc.
- 3) Root Resorp. (not a problem if ^{root} fx) - luxation inj. = inj. to PDL + causes resorp
- 4) Loss of Marginal Bone - due to crushing of bone

Clinical Signs

- ⊖ vitality
- discoloration

Radiographic

- ⊕ or ⊖ radiographic lesion
- arrest of root formation (immature)
- external inflam. root resorp. ⇒ sign of necrosis

Root Resorp.

- VITAL 1) Internal - inside tooth ⇒ pulp responsible for resorption ^{2 vital tooth.}
 2) External - outside tooth ⇒ from external tissue
 (91b) ⇒ Nonvital tooth; has to do w/ PDL

→

Diff. Dx. b/w Internal + External Root Resorp.

Radiographic

Canal

- can follow canal thru lesion = Ext.
- cannot follow canal, well-defined margins of lesion = Int.

* can only get resorption if it is alive hence

Internal - pulp vital (cannot follow canal)

External - pulp non-vital (can follow canal thru lesion)

Internal Resorp.

Tx: RET b/c vital pulp

- NaOCl (5.25%) (Full strength bleach) ^{antibacterial}
- CaOH (necrotizing effect) "Intracanal medicament" ^{antibact.}
- Change CaOH every 7-10 days
- obturation w gutta percha (after all tissue has been removed)

Full strength NaOCl = 5.25%
internal root resorp.
(strength for approximation)

External Resorp.

* 99% known usually ext. resorp.

Types:

- ① surface
- * ② Inflamm.
- * ③ Replacement
- ④ Cervical

S
-
R
C

A) * ⇒ Inflammatory root resorp.

- injury to PDL + cementum
- significant inflamm. of PDL
- dentinal tubules exposed
- infected necrotic pulp tissue present
- progressive if endodontics not instituted.

Tx: Endodontics



Ext. Root Resorp.

- * ① Inflammatory
- * ② Replacement
- ③ Surface
- ④ Cervical

Tx: Endodontics (For external root resorp. due to inflamm.)

- pulp is removed
- CaOH placed (antibact.)
- CaOH placed every 3 mo.
- Final obturation $\bar{c}$ gutta percha @ 1 yr.

B) * Replacement Root Resorp. = Ankylosis (Ext - Root Resorp.)

- Injury to PDL causes some PDL cells to die and body no longer recognizes tooth and starts to replace tooth $\bar{c}$ bone.
- Doesn't matter if endo. performed - it's over
- loss of PDL cells and bone touches tooth and they become one.
 - Absence of vital PDL
 - Fusion alw. bone $\bar{c}$ root surface
 - Replacement tooth substance $\bar{c}$ bone.

Dx:

Radiographic $\Rightarrow$ disappearance of PDL

Clinical $\Rightarrow$ percussion = metallic sound / dull
NO MOBILITY.

Tx:

No known Tx!

Avulsion - tooth knocked out of socket

- must respect integrity of surviving PDL cells

Tx: Immediate Reimplantation

- want to maintain vitality of PDL b/c will die when exposed to air.
- look for a more serious injury
- As little tx. of tooth as possible should occur as reimplantation $\Rightarrow$ Rinse $\bar{c}$ saline, anesthetic, H₂O
 - * No chemicals, No scraping, No apicoectomy.

Avulsion, Tx. in office:

- Rinse $\bar{c}$ Sterile saline
- Immediate Replacement
- Splint 7-14 days (max - usu. 10 days)
- Endo. therapy if necessary (before splint removed)
 $\Rightarrow$ if apex open, leave alone
- Tetanus prophylaxis
- ABs

Closed Pulp:

- Endo. tx after 7-14 days.
- CaOH placed
- CaOH replaced every 3 mo.

Open Pulp:

- recall every month for development
- tx. accordingly.

Splinting Technique:

- do not traumatize tissues
- allow proper OT
- allow physiologic mv. (luxation + avulsion)
- Peridex month since given to pt.

* Longer tooth out of mouth = $\downarrow$ success rate

Avulsion Storage medium:

- Best - Hanks Balanced Salt Soln. (HBSS)
- milk
 - saliva
 - tap H_2O - undesirable
 - dry storage - "

⇒ HBSS + Milk

- essential nutrients
- neutral pH + physiologic osmolality
- no bacteria
- lack of toxic components.

Adjunctive Tx. to Avuls.

- Tetanus Booster
- AB.

PA granuloma - chronic, low grade, ASX

Radio: PA radiolucency

Dx: Necrotic

Tx: RCT

S26 PA granuloma
Radiculocyst
122 Apical Scar

differentiate via Bx.

Radicular Cyst - chronic, ASX

Radio: PA radiolucency (cortical bone)

Dx: Necrotic

Tx: RCT

* Rudin's
hyaline
Bodies

AAA - symp.

SAA - surre-inflamm., Symp.

Radio: NONE

Dx: Necrotic

Tx: I+D

Ab
Accen + Debride
RCT

Phoenix A. - acute inflam of chronic, Symp.
(sim. SAA)

Radio: PA radiolucency

Dx: Necrotic.

Tx: I+D

Ab
A&D
RCT

Supp. Apical Abs. (ovx.)

AAA (Supp. abs.)

- parulis - ASX

Radio: PA radiolucency / bone loss @ apices of teeth.

Dx: Necrotic

Apical Scar

- ASX
dense fibrous CT @ end of endo. tx tooth.
from post. cortical plate

Dx: necrotic RCT already done.

Radio: PA radiolucency

Tx: NONE

Condensing osteitis - ASX < vital
non-vital

Radio: ↑ bone density
caries

Tx: Reversible pulpitis - caries control
irrev - RCT
necrotic - RCT.

PA Lesions of Endodontic Origin

Lec. 3

(ASX)

Periapical Granuloma - chronic inflamm.

PA granuloma
Radicular Cyst
Apical Scar

Low grade, long standing response to
Canal bacterial irritants.

~57% all cases
*most common root path. 9/12/06

Periapical Granuloma

- most common root path (~57% of all cases)

Periapical Granuloma

- 1) Vascular Elements
- 2) Inflamm. Cells
- 3) Epithelial Cells
- 4) Collagen Granules

Forms via:

Microbial

Mechanical

Chemical

Dx: Clinical, non-vital
Radio: PA radiolucency

Tx: RCT

① PA granuloma (57%)

② Radicular cyst

③ ~~PA~~ Apical Scar (12%)

(Asx)

Radicular Cyst

2nd most common pathology of root-end para radiolucency
70% are true cysts

Defn: Chronic inflamm. response of periapex developing from chronic lesions to previous granulomatous tissue.

Etiology:

- ① Microbial (Caries / bacteria)
- ② Mechanical (preparations)
- ③ Chemical (dentin desensitizing agents)

Histo: PA granuloma char. by central fluid-filled and epithelial lined cavity.

• Lining:

- stratified sq. epithelium

- Rushton Hyaline bodies (*characteristic of Radicular Cyst)

• CT Wall: MICE

- cholest. slits

- multinucleate giant cells

- Inflammatory filtrate adjacent to epi

- Collagen, Fibrin, etc.

Dx: Clinical → Non-Vital

Radio → pa radiolucency = a distinct thin layer of cortication (not always)

Rx → only definitive dx.

Tx: RCT

PA gran. vico
Vessels
inflam. cells
collagen
epithel cell

(Symptomatic)

Acute Apical Abscess

Defn: Severe inflamm. response of the pa connective tissue caused by contaminants from pulpal canal

Etiology = anything that causes pulp to become necrotic

Dx: Clinical → (⊕ percuss. + palp.)
Radio → Symptomatic, Non-vital
NONE . pain + swelling.

Clinically

- Swelling
- ↑ Temp.
- Malaise
- Tooth feels elevated out of socket

Histo:

WBCs
microorgs.

Tx:

- I + D - If drainage not performed, infec. may spread thru fascial planes of H + N.
- Access and debridement.
- Ab
- RCT (final treatment)

Symptomatic

Phoenix Abscess

Defn: Acute exacerbation of an existing chronic inflammation

Dx: Clinical

↳ same as acute apical abscess

- non-vital
- symptomatic

- pain
- swelling
- temp.
- malaise
- & tswllng

Radio

↳ pa radiolucency + pain + swelling - malocclusion

取:

- I + D
- Access + Debridement
- Ab
- RCT

same as
acute
apical
abscess

Suppurative Apical Periodontitis (Fistula)

Defn ¹⁴⁶ Formation of a parulis and active pus formation draining thru the stoma of a sinus tract.

dx: clinically - looks like a pimple
in sinus tract

Radio - PA radiolucency.

method $\Rightarrow$ tracing fistula tract $\approx$ GP

Apical Scar

→ 3rd most common (12% of cases)
per pathology

Defn: Dense mass of fibrous tissue @ the apex of an endodontically tx. tooth.

Etiology:

perforation of cortical plate(s)

Dx: Clinically → Asx to percuss. + palp.
Radiographic → root canal (2 or 2/0
pa surgery)

Tx: If dx. apical scar made, NO TX. NEEC.
(it is a healing process + is not pathologic)

Condensing Osteitis

→ in 4.5% of patients

Defn: Very low-grade ^{subclinical} inflamm. response, that may lead to an ↑ in bone density which creates a barrier against the irritant

Etiology: Long standing, low grade irritation of pulp tissue such as decay, occlusal trauma, etc.

*would happen before a RCT performed.

Dx:

Clinical:

Radiographic:

Asx. → vital / non-vital
→ Irritant

Decay (vital tooth)
Increased bone density
= Condensing osteitis

Histo: ↑ proliferation vs. destruction
Thickening of Trabeculae (↑ osteoblasts vs. osteoclasts)
↑ size of marrow space

condensing osteitis
Tx:

- If Reversible pulpitis → Caries control + restoration
- If Vital Inflamed tissue → RCT
- If Necrotic → RCT

-
- ① Periapical Granuloma (59%)
 - ② ~~Acute~~ Apical Abscess Rad. cyst.
 - ③ Apical Abscess (12%)

PA gran. 59
Radicular Cyst
Apical abscess 12

Endo

Problems c (VCCR pulpotomy

(sim. to apexogenesis)

= "calcsific metamorphosis"

"Bridge formation adjacent to $\text{Ca}(\text{OH})_2$ "

10/3/06

IF PULP TISSUE REMOVED or BECOMES NON-VITAL, APEXOGENESIS NOT an option.

Apexogenesis: (vital tooth)

2-3 mm $\text{Ca}(\text{OH})_2$

3+ mm IRM

Semi-permanent resto. (re-eval. 1 wk - / 3 mo. for $\text{Ca}(\text{OH})_2$)

Cvek Pulpotomy - ^{90% remain vital.} when trauma - NOT caries.

A variation of apexogenesis - it endo. tx. will NOT be done.

Apexification (non-vital)
non-vital tooth

- root formation will not continue

- goal: get rid of any necrotic debris

Thin walls
Short roots
open apex.

(NaOCl dilute / instrumentation / $\text{Ca}(\text{OH})_2 \Rightarrow$ re-eval 1, 3, 6)

Be careful w/ apex locators - may give false reading.

Intracanal Medicament

$\text{Ca}(\text{OH})_2 \Rightarrow$ Fill entire canal.

placement of $\text{Ca}(\text{OH})_2$

- ① glass slab - mix anesthetic w/ $\text{Ca}(\text{OH})_2$ and mix to get thick paste material.
- ② premixed $\Rightarrow$ may have concern about cross contamination

Intentional Reimplantation

Removing and replacing a tooth to do therapy such as an apicectomy.

Indications:

ex: lower molar $\bar{c}$ nearby nerve - do not want
to hit nerve

* Do not dry out tooth, do not traumatize tooth

- Handle tooth by crown only.
- if have to handle tooth, do $\bar{c}$ wet gauze

- Place retrofill in apex = MTA

Endo

(skip)

Lec. 9 Mgt. and Tx. of perforations +
other procedural accidents

10/24/06

Perforation

- 1) Chamber access - Floor + roof of pulp chamber may be close together.
- 2) Canal access
- 3) filling/instrumenting canal
- 4) root preparation

When see pulp chamber narrow on pre-op, pay extra attention to prevent perforation thru floor.

Chamber access

- Pulp recession
- misjudgement of long axis of tooth.
- location
- orifice opening $\Rightarrow$ Gates Glidden - if too big may perf.
- Furcation wall

maxilla - pm + m

mandible - m

* $\Rightarrow$ The D wall of m canal was found to be less thick after use of Gates Glidden.

- Internal furcation wall most likely to perforate.
- Failure to maintain a curve
- Attempt to overcome a ledge or sep. instrument
- Attempt to overcome a calcified canal
- Root preparation $\Rightarrow$ Furcation walls = DANGER ZONE
 - mistake τ site + direction of root in tooth.

max. perf: 74%
mand. perf: 26%
RCT perf: 47%
post perf: 53%

* perf. away from perio
and near apex = good prog

10/24/06.

Root Perforation

- 55 cases of root perf.
 - max. 74% } diff. b/c using a mirror and difficult to see placement + depth of bur.
 - mand. 26%
 - During RCT 47%
 - During post prep. 53% } post prep perf. > RCT perf
- ↳ perf. here almost equal to chance of perf. when performing RCT.

Root Preparation

- Furcation walls are DANGER ZONE
- Root too big for size of canal
- Post space prep. not w/in long axis of tooth.

Dx. Perforations

- Direct visualization - microscope
- Tight canal that became "very loose"
- Bleeding - paper point (will see blood not @ tip)
- Apex Locators

Histological Considerations

- Cementum Damage
- Perio. damage
- Wound Healing

⇒ perf. away and near apex (away from ^{perio.}) = good prognosis
⇒ perf. higher and near perio. attachment = poor prognosis.

* POST PLACEMENT → most posts 65-100

D - mand. molar
L - max. molar

Tx. of Perforation

BEFORE

• Prevention:

- Eval. radiograph size chamber + canals
orientation
long axis of tooth.

- stay away from danger zone (FURCATION)

- Curve stainless steel instruments

(EXCEPT for max. central incisor)

- Take X-ray when in doubt

- match post size to size of canal.

* most posts are size 65-100 ∴ be careful!

⇒ posts usually go into D mand. molar

L canal max. molar.

AFTER

• Don't panic

- do not tell patient while its happening

- close tooth, sit patient up and explain.

• Non-Surgical ⇒ seal

• Surgical → Lift flap to fix

• Extraction

MATERIALS

- GP

- GI

- Cavit

- Amal.

- $\text{Ca}(\text{OH})_2$

* MTA ⇒ most used material today
to seal perforations.

"medical grade concrete"

* Achieving Hermetic Seal = MOST DIFFICULT

Seal perf. via Internal matrix concept

- MTA / $\text{Ca}(\text{OH})_2$ seal perf.
- MTA / GI intor

→ 6-12 mo. til see healing 10/24/06.

Seal Perf:

"Internal Matrix Concept"

- place MTA / $\text{Ca}(\text{OH})_2$ into perf. area and then seal w/ GI or MTA
- leave space to place GP

* need 6-12 mo. after placement of sealer to see any type of healing.

→ Use of ultrasonic to vibrate sealer into canal to seal perf. area.

Prognosis of Root Perf.

Time related to repair:

Long - poor
Short - good

If apical $\frac{1}{3}$ perf. = good (away from perio. attach apparatus)
middle $\frac{1}{3}$ = fair
coronal $\frac{1}{3}$ = poor
→ B/c becomes a perio problem instead of endo.