ENDODONTICS

- EPT – stimulates nerve endings with *low current* and *high potential difference* in voltage; stimulates A delta fibers; no gloves should be used because causes false negative.
  - Results from EPT: -*chronic pulpitis* = higher current than normal
    -*acute pulpitis* = lower current than normal (acute inflammation mediators lower the pain threshold).
    -*hyperemia* = lower current than normal, but higher than acute pulpitis.
  - False positives – pus-filled canal or nervous patient.
  - False negatives – trauma, insulating restoration, or wearing gloves.
- Trauma causing deep intrusion to a permanent tooth causes pulp necrosis and conventional RCT.
- SLOB Rule – root farther (buccal) from film will move to same direction cone is directed; lingual surface is always closest to the cone so buccal is always farthest.
- Referred Pain - Forehead: max. incisors
  Nasolabial: max. canines and PMs.
  Temporal: max. 2nd PM.
  Ear: mand. molars
  Mentalis: mand. Incisors, canines, and PMs.
- Hemophilia is NOT a contraindication to endo.
- Special case – trauma with pulp obliteration but PDL normal; asymptomatic and no EPT response; TX= observe as long as tooth asymptomatic and no PA changes.

ACCESS:

- mand. molar = trapezoidal, most common tooth for RCT; tipped ML so overprepared ML access; in 40% of cases, may have 2 canals in distal root;
- max molar = triangular, highest RCT failure, MB root is most complex of all teeth, because under MB cusp and must be accessed from DL position; M→P line is longest; 59% have MB2; the most common curvature of the palatal root is toward the facial.
- Lingual wall of mandibular teeth most often perforated.
- U-shaped radiopacity overly apex of palatal root of max 1st molar is zygomatic process.
- Facial access on primary max incisors recommended.
- Mand. incisors and max 1st PMs most cautious for access because common in perforations.
- Perforations into furcations of multi-rooted teeth have the poorest prognosis.

TEETH CHARACTERISTICS:

- Max. 1st PM – lingual root may be wider; 2 roots=60%; thin oval access, common perf on mesial concavity.
- Max. 2nd PM – more accessory canals than 1st pm; thin oval access; 85% has 1 root; overfilling either max. PMs will enter the maxillary sinus.
- Mand. 1st PM – 25% have 2 canals and 2 foramen.
- Mand. 2nd PM – 97% have 1 canal.
- Mand. Canine – slight labial incline so access toward lingual; thin MD, wide BL; access opening is a large oval with greatest width placed incisogingivally.
- Max. Canine – longest tooth.
- Max. lateral incisor – 55% has distal/lingual root curvature.
- Max. Anterior - teeth have slight distal inclines; all max. anteriors ALWAYS have 1 root!
- Mand. Incisors – may have 2 canals with the labial being the straighter one; may have distal/lingual curvature.
- Vital Teeth that don't need RCT: 1. Cementoma, 2. Traumatic bone cyst, 3. Globulomaxillary cyst.
- Pulp capping: only most successful with pinpoint exposures;

- Poorest prognosis when perforation into furcation of multi-rooted tooth.
- Recapitulation: using MAF after each increase in file size to remove any dentin filling not removed by irrigation.
- Obturation only 2nd to canal debridgement.

ENDO Liquids:

- Sodium Hypochlorite (1%, 2.6%, or 5.25%) – germicidal solvent and antimicrobial; GP points can be disinfected in 5% NaOCl for 1 minute; toxic to vital tissues; 3 roles: 1) good tissue solvent.
  2) antimicrobial effects
  3) lubricant
- Hydrogen Peroxide (3%) – bubbly solution removes debris b/c certain chemicals physically foams debris from canal (effervescent effect) and liberates oxygen so destroys anaerobes.
- Urea Peroxide (Gly-Oxide) – decomposition; better than hydrogen peroxide and for narrow/curved canals for slippery effect of glycerol; better tolerated by tissue than NaOCl and more germicidal than H2O2 so EXCELLENT for tx of canals with normal PA tissue and wide apices.
- Chloroform – the vapor is very dangerous and used to dissolve gutta percha.
- Glass Bead Sterilizer – sterilized endo files in 15 sec at 220°C.
- EDTA (17%)– ethylene diamine tetra-acetic acid; not good irrigation solution; decalcifying process is self-limiting and stops as soon as chelator is used up; can remain active up to 5 days so must irrigate/inactivate with NaOCl at the end of the appt; chelating agents – calcify tissues in order to clean root surface for gutta percha and sealer to adapt;
  - chelating agent acts by substituting sodium ions that combine with dentin to form soluble salts for calcium ions that are bound in less soluble combination creating softer canal edges to facilitate canal enlargement.
  - EDTA removes the mineralized portion (decalcify) of the smear layer.
- EDTAC – EDTA and cetavlon; greater antimicrobial action but greater inflammatory potential; inactivator – NaOCl.
- RCPrep – EDTA and urea peroxide so BOTH chelation and irrigation; for adequate RC debridgement, must achieve glassy smooth walls of canal; foamy solution with natural effervescence.
- Most common cause of RCT failure is inadequate disinfected RC; 2nd most common cause is poorly filled canals.
- MTA – mineral trioxide aggregate; calcium and phosphorus; long setting time and difficult to manipulate; increase pH; most superior retro-filling/retrograde material.
  - Mta seals apical portion of root canal and is always after apicoectomy alone will not yield a good result.
  - Advantages: 1)RO
    2) hydrophilic
    3) biocompatible
    4) induces hard tissue formation.

BROKEN FILES:

- If broken file past apex, surgery is performed.
- If broken file in apical 1/3 and no RL, then no surgery is needed but recall is a must.
- If broken file in apical 1/3 but RL is present, then surgery is performed; prepare and obturate to the point of blockage and then perform an apicoectomy without! retrofilling.
- Best prognosis if vital and no PA lesion.
- Easier to retrieve an instrument if it wedged coronal or at the curvature of the canal but very difficult if instrument has past canal curvature.

INSTRUMENTATION:

- 3 types of Instrumentation: 1. Filing (push&pull) – produces irregular shaped canals.

2. Reaming (repeated rotation) – produces round shaped canals.
3. Circumferential Filling (push and pull with emphasis on scraping canal walls) – enhances preparation for flaring.

- Narrowest diameter at DCJ (.5-1.0mm from apex); widest diameter = orifice.
- Broaches – for pulp tissue and soft material removal not for canal enlargement.
- Files (stainless steel) – cut COUNTERCLOCKWISE; strongest of file but cut the least aggressively.
- K-File – most useful instruments for removing hard tissue to enlarge canal; clockwise-counterclockwise motion while pressure placed apically; K-flex file = modified K type file.
- Reamers – fewer flutes than files and removes debris CLOCKWISE but places material COUNTERCLOCKWISE; shave dentin using only a reaming action to enlarge canals.
- Hedstrom stainless steel files – for filing action only and much faster than other files because sharp edge but must be careful; modification is S-file.
- Very light apical pressure is applied when using nickel titanium rotary files.
- Rotary instruments work faster and improve access early in tx compared to heated instruments.
- Endo first then perio, unless the case is of a primary periodontal lesion; common clinical finding of periodontal problem is pain to lateral percussion on a tooth with a wide sulcular pocket.
- Tooth must be asymptomatic and DRY at the time of obturation.
- Debridement is the most crucial aspect of RC tx; want glassy, smooth canal walls.
- Most common cause of RCT failure is inadequate disinfecting of RC system; 2nd most common cause of failures (40%) is leakage from poorly filled canals.

OBTURATION:

- If an accessory canal isn't totally filled during obturation, then observe and evaluate every 3 mo.
- Main fct of RC sealer is to fill discrepancies between core filling material and dentin walls.
- ZOE Based Sealer – lubricant, bonding agent, and antimicrobial activity; disadv: staining, slow setting, non-adhesion, and solubility;
- All sealers are radiopaque from metallic salts in sealer.
- If GP past apex, file used beyond apex to avoid breaking cone; a broken cone in PA area can cause orthograde retx failure.
- How to remove GP: 1) rotary
  2) ultrasonic
  3) heat
  4) heat and instrument;
  5) file and chemical.
- Indications for using solvent-softened custom gutta percha:
  1. Lack of apical stop
  2. Abnormally large apical portion of the canal.
  3. Irregular apical portion of the canal.
     - Don't use if tugback is <1mm and DOESN'T produce better apical seal than normal GP.

VERTICAL FRACTURES:

- diffuse RL/halo surrounding root due to bony attachment apparatus; most common cause is due to too much condensation; inlays have been show to cause vertical fractures.
- Diagnostic aids: 1) fiberoptic light
  2) wedging the tooth
  3) persistent periodontal defects.
  4) patient bite on bite stick.
- An additional radiograph taken with steep 45° vertical angulation in addition to conventional 90°.
- Vertical fractures thru root has poor almost hopeless prognosis.
- Anterior tooth root fractures usually in HORIZONTAL plane and may be visible in xray.

BLEACHING:

- Superoxol: most common bleaching agent for RCT teeth; 30% solution of hydrogen peroxide and distilled water; apply to heat to superoxol cotton til tooth lightens; heat liberates oxygen.
  - bleaching effect is due to direct oxidation of stain-producing substances.
  - Complications: cervical root resorption, acute apical periodontitis (#1 complication), and enamel and dentin color changes.
  - Bleaching causes color change in enamel and dentin.
- Walking Bleach Technique – Sodium Perborate and 2-3 drops of superoxol in tooth chamber for 4-7 days and repeat prn.
- Hydrogen Peroxide (30-50%) – most effective bleaching agent, in alkaline medium.

FLAPS:

- Submarginal Curved Flap (semilunar flap) – not used for anterior root end surgery;
  - Disadvantages: 1) limited access & visibility
    2) tearing of incision corners
    3) if large lesion, then incision occurs over defect and scarring occurs.
    4) incision extent is limited by attachments.
- Submarginal triangular and rectangular flap – requires 4 mm of attached gingiva and healthy periodontium; flap is raised by scalloped incision in attached gingiva with 1 or 2 vertical incisions; scarring but access is good; not as much recession.
- Full Mucoperiosteal Flap – maximal access and visibility so most ideal flap; raised from gingival sulcus; difficult to reposition and suture and may have recession.
- Indications for Periradicular Surgery: 1) non-negotiable canal, blockage, curvature.
  2) complications from procedural accidents.
  3) failed tx from irretrievable posts/root fillings.
  4) horizontal apical fractures causing apical necrosis.
  5)biopsy.

LESIONS & BACTERIA:

- Blow-out Lesions (non-vital) – all probing normal until swelling probed and suddenly drops; tx = RCT.
- Narrow Sinus Tract Lesions (non-vital) – Probing normal except 1 narrow area; tx = RCT
- Periodontal Lesions – probing defect is *conical* shape and needs RCT and perio if needed; pain to lateral percussion; eventhough perio lesion, must do endo first then perio.
- Pulp-chamber retained amalgam must be 3 mm into each canal for retention;
- RCT teeth have more fractures because loss of structural integrity.
- Bacteria infected in root canals:1) eubacterium.
  2) fusobacterium
  3) porphyromonas
  4) peptostreptococcus
  5) prevotella
- Streptococcus initiates lesion to pulp exposure but STRICT ANAEROBES play role in periapical pathoses.
- Virulence Factors involved in periradicular pathosis:
  1. Lipopolysaccharide- found on gram negative surface.
  2. Enzymes – neutralize antibodies and complement components.
  3. Extracellular vesicles – involved in bacterial adhesion, proteolytic activities, hemaglutination & hemolysis.
  4. Fatty acids – affect chemotaxis and phagocytosis.
- CYSTs – inflammatory response with epithelial lining; well-defined RL limited by continuous RO sclerotic border of bone; associated with chronic infected and sometimes mobile teeth.

    - Central, fluid-filled, epithelium-lined cavity, surrounded by a granulomatous tissue and & peripheral fibrous encapsulation.
- Osteomyelitis – From PA infection with diffuse spread into medullary spaces with necrosis of bone; tx= drainage and antibiotic;
    - acute max = well localized infection; acute mand = more diffuse & widespread infection;
    - severe pain, fever, and lymphadenopathy with loose and sore teeth.
    - Progresses rapidly and little radiographic evident until 1-2 weeks and then appears "MOTH-EATEN" radiolucency.
    - Tx = drainage and antibiotics.
- Periodontal Abscess – positive for palpation and percussion and response from EPT;
    - Gram neg. rods like Capnocytophagia, Vibrio-corroding, and Fusobacterium.
- Gingival Abscess – from mastication; tx = OH and dental tx.
- Apical Scar – PA granuloma, cyst or abscess that heals with scar tissue; well-circumscribed RL & non-vital; tooth is non-vital, so needs RCT.
- Radicular Cyst – pre-exiting granuloma; NON-VITAL so needs RCT.
- THREE VITAL TEETH LESIONS so NO RCT! –
    1) *Cementoma* – anterior area of mandible; RL lesion that calcifies; disorder of production of bone and cementum-like tissue in tooth areas of jaw.
    2) *Traumatic Bone Cyst* – no epithelia lining; asymptomatic and RL appears scalloped around roots of teeth; intramedullary hemorhage, blood clot liquefies and leave empty space;
    3) *Globulomaxillary Cyst* – jct. of globular and maxillary processes of maxilla; pear-shaped RL btw L.I. and canine roots; may be fissural cyst or OKC.
- Phoenix Abscess (recrudescent abscess) – develops as granulomatous zone; diagnose with percussion and xray; large PA RL and is an acute exacerbation of chronic apical periodontitis.
- Granuloma – granulomatous tissue with PDL due to pulp death so RCT needed; no symptoms; can result in abscess and only differs from cyst by histologic examination; well-defined RL.

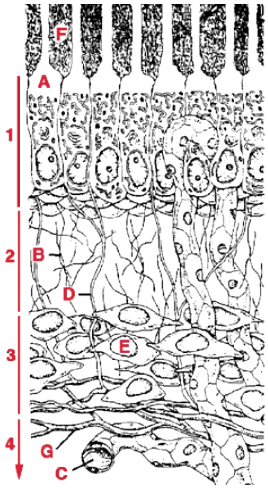
RADIOLOGY:

5. Faster film (E-speed) requires less radiation but quality image.
6. Increase kVp causes decreased patient's skin dose; needs to be 70kVp or higher.
7. Collimation – restriction of x-ray beam size so doesn't exceed 2.5 in at pt's skin.
8. Max radiation does – 50 mSv per yr/whole body.
9. Stand at least 6' away in area that lies b/w 90-135^{o} to the x-ray beam (an area of minimum scatter radiation).

PULP & DENTIN:

- Decreases with age – size of pulp and # of reticular fibers (less cellular and more fibrous).
- Increases with age - # of collagen fibers and calcifications; apical portion of pulp contains more collagen than the coronal portion.
- Pulp stones are associated with chronic pulp disease from advanced carious lesions and large restorations.
- Pulp has myelinated (sensory) and unmyelinated (motor) nerve fibers – they are afferent and sympathetic; no proprioceptors!
- Pulp only free nerve ending with *only receptor for pain*!
- Predentin – adjacent to odontoblast layer of pulp, 10-47 μm of dentin remains unmineralized; if layer lost, predisposes to internal resorption by odontoclasts.
- Mantle Dentin – 1st formed dentin because odontoblast layer gets organized.
- Circumpulpal Dentin – Most dentin formed.
- Secondary Dentin – forms after tooth eruption and during life.
- Tertiary/Reparative Dentin – irregular dentin formed in response to injury.

- Primary function of pulp is dentin formation! Also nutrition for dentin and induction (forms dentin to enamel)
- In pulp, type 1: type 3 collagen ratio is 55:45%; type 5 collagen in small amts.
- Type 1 collagen predominates in dentin; odontoblasts make type 1 and fibroblasts in pulp make type 1 and 2.
- 4 Pulp Zones: 1) Odontoblastic layer – outermost layer w/ odontoblasts(A); adjacent to predentin & mature dentin (F).
  2) Cell-Free Zone (zone of weil) – rich in nerves (D) (incl nerve plexus of raschkow) & capillaries (C);
  3) Cell-Rich Zone – innermost pulp layer with fibroblasts (E).
  4) Central zone (pulp proper) – larger nerves and blood vessels;



- Cells in pulp – fibroblasts, odontoblasts, histiocytes and lymphocytes.
- Cells in diseased pulp – PMNs (after pulp exposure), plasma cells, basophils, eosinophils, lymphocytes and mast cells.
- Pulpal inflammation is a chronic cellular response with plasma cells, macrophages, and lymphocytes; After pulpal exposure, PMNs (acute inflammatory cells) are attracted to the area.
- Vital Pulp is resistant but non-vital pulp is fertile ground for micro-organisms.
- Pulp Nerve Fibers: 1) *A – delta Fibers* = large, myelinated nerves that perceive quick, sharp, momentary pain and dissipates quickly.
  2) *C Fibers* = small, unmyelinated nerves that perceive dull throbbing ache with diffuse pain and can be referred pain; not easily provoked but signifies irreversible local tissue damage;
- Unmyelinated fibers regulate the lumen size of blood vessels.

PULPAL DIAGNOSIS:

- Best method to elicit the most accurate thermal response is to individually isolate the suspected teeth with a rubber dam and then bathe each tooth in hot or cold water.
- Irreversible Pulpitis – bending over/lying down intensifies pain; often no PA lesion;
  - SPONTANEOUS, diffuse pain; intensifies with heat and relief with cold; tender to percussion.
  - Thermal test are the best aid to diagnose an irreversible pulpitis.
- Reversible Pulpitis – requires irritant to evoke pain and pain removed when stimulus is removed; NOT SPONTANEOUS; pain with cold not hot; usually sedative filling or new restoration is enough tx; most common cause is bacteria.
  - Pulpal hyperemia is an excessive accumulation of blood in the pulp due to vascular congestion.
  - **most effective way to reduce pulp injury during tooth preparation is to minimize dehydration of dentin!!!
- Necrotic Pulp – no symptoms but may sometimes respond to heat; EPT is valuable b/c there will be no response at any current level;
- Chronic Apical Abscess (suppurative apical periodontitis) – long-standing, low-grade infection of PA bone from RC; painless; may follow an acute alveolar abscess or unsatisfactory RCT.
  - diffuse RL (unlike cysts and granulomas which are well-defined RL) and PDL widening; slightly loose, tender to percussion;
  - often cause of sinus tract in gingival tissue of kids; tooth pain stops upon drainage;
  - NON-VITAL so RCT.
- 30-50% of bone calcium must be altered before RL presents; the alteration occurs at the jct between the cortical and cancellous bone.
- Periapical abscess is the most common of the all dental abscesses.
- Acute Apical Abscess – pus collection in alveolar bone; sequence of symptoms: tender tooth to severe throbbing pain to percussion with swelling; loose tooth, fever; no response to EPT or cold but may

respond to heat; tx = drainage and debride the canals and then at a later date perform RCT and give PCN but if not pcn then clindamycin = increases bone levels but chance of pseudomembranous colitis.
    - ER Tx = drainage, antibiotics and analgesiccs and then RCT at a later date.
- Hyperplastic Pulpitis – red/cauliflower growth of pulp in and around carious exposure caused by chronic irritation and vascular supply.
- Caries spread laterally at DEJ to increase organic content and involve many dentinal tubules, Tomes fiber-react causing fatty degeneration and later decalcification(sclerosis); once odontoblasts are involved, pulpal changes occur;
- Only reliable clinical evidence that secondary dentin as formed is decreased tooth sensitivity.

RESORPTION:
- Pulpal inflammation often causes internal resorption when dentinoclasts (undifferentiated connective tissue cells) resorb the tooth structure in contact with the pulp.
- External Resorption – always with bone resorption;
    - Etiology: 1) trauma
      2) pulp inflammation
      3) ortho
      4) impacted teeth
      5) bleaching
      6) non-vital teeth
- Bowl-Shaped Resorption (inflammatory resorption) – involves dentin and cementum; tx – immediate RCT; CaOH every 3 mo and after 1 yr, obturate with CaOH sealer;
- Pulp doesn't play a role in cervical root resorption.
- Surface Resorption – acute injury to PDL and root surface; heals itself.
- Replacement Resorption – resorption of root surface and bone causing ankylosis; often seen in replant cases; accompanies dento-alveolar ankylosis, characterized by progressive replacement of root by bone (no pdl); *signs*: no mobility, metallic percussion sound, and infraocclusion.
- Bowl-shaped, surface, and replacement resorption all can be caused by replantation! All 3 are types of external root resorption.
- Internal Resorption – asymptomatic but seen in xrays as irregular RL anywhere along the canal; once pulp is removed, resorption ceases; may respond to pulp vitality tests; Tx = pulpectomy;
    - Undifferentiated connective tissue pulp cells are activated to form dentinoclasts that resorb the tooth structure in contact with the pulp.
    - Etiology: 1) trauma
      2) caries
      3) pulp capping with CaOH
      4) cracked tooth – pink tooth
      5) partial removal of pulp (pulpotomy)
- *Pink tooth syndrome* is often a sign of internal resorption and cervical root resorption; characterized by pinkish tooth due to granulation growth undermining the coronal dentin.

PULP TX:
- Apexification – induce further root development in PULPLESS tooth by stimulating formation of hard substance at apex → CaOH creates alkaline env't to promote hard tissue deposition;
    - *Procedure* – access tooth, remove pulp tissue, CaOH-methylcellulose paste injected into the canal to cervical level; double seal cement to close cavity and recall after 3 mo.; if apex forms then RCT.
    - May be required after pulpectomy;
- Apexogenesis – maintain pulp VITALity during pulp tx to allow root development; for immature teeth with incomplete root formation with damaged coronal pulp but healthy radicular pulp.
    - Place CaOH/MTA over radicular pulp and recall every 3 mo til root forms then complete RCT;

- Root Submersion – resection of a tooth's root 3 mm below alveolar crest; prevents resorption and maintain better proprioception; Indications: 1) rampant caries.
  2) periodontal conditions
  3) failure of prosthetic cases
  4) requiring better denture control.
- Crown lengthening indications – sub G caries, perforations, and resorptions.
- Pulp Capping: success is recognized by formation of complete barrier of dentin at exposure site;
  - Dycal = $CaOH_2$
  - If pulp capping fails and tooth becomes symptomatic, it maybe impossible to treat with routine endo due to severe calcifications in the root canal; perforations more common in the RCT.
  - IPC – wait 3-4 before tooth is reopened and decay is removed;
  - DPC – very successful in immature teeth; perform if small exposure (<1mm) and if exposure was <24hrs; perform partial pulpotomy if >1mm and >24 hr.
- Pulpectomy – removal of pulp and fill with ZOE if want roots to resorb or place temporary until RCT can be completed.
- Pulpotomy - Uncontrolled bleeding with pulpotomy – perform pulp amputation at a more apical level.
  - Indications for pulpotomy: 1) carious primary tooth (healthy radicular pulps)
    2) carious perm tooth with underdeveloped roots.
    3) if RCT isnt available.
    3) ER tx for perm. tooth with acute pulpitis.
  - Only temporary procedure for perm teeth.
- Apicoectomy – obliquely resecting most apical portion of root with buccal bone around apex removed; retrograde amalgam filling; common reason for apicoectomy and retrofilling is tooth with post and needs to be retreated;
  - indications: reverse filling, gain access to pathosis, poorly filled apex;
  - Retx for post, core, and crown requires curretage, apicoectomy and retrofill;
- Periapical Curretage – same as apicoectomy but doesn't remove apex; removal and examination of diseased tissue and determining extent of lesion are objectives of curretage.

AVULSION:

- 5 Factors: 1) Time: w/in 30min, little resorption vs over 2 hrs which increases the failure rate.
  2) Storage Media: influence viability of PDL cells; milk best b/c pH =6.5-6.8; saline and saliva is ok.
  3) Tooth Socket: no curettage or forced replantation.
  4) Root Surface: no scrapping, dried, or added chemicals.
  5) Splint Stabilization: splint for maximum of 2 wks for initial PDL attachment.
- *Intentional Replantation:* extract and do RCT and replant; not a substitute for endo surgery.
  - Indications: 1) cant do normal RCT
    2) obstruction of canal.
    3) Perforating internal and external resorption
    4) Previous tx failed.
- If REPLANT w/in 2 hrs: RCT 10-14 days after with CaOH; replace every 3 mo and then obturate after 1 year.
- If REPLANT after 2 hours: RCT before replant, soak in 2.4% fluoride (fluoride slows the resorptive process) at pH 5.5 for 20 min, currette blood clot and irrigate with saline, wash tooth with saline, replant and splint for 4-6 weeks.
- Main cause of failure of replanted teeth is external root resorption; ankylosis can also cause failure but better prognosis than external root resorption.
- After 60 min of dry storage (or water) of an avulsed tooth, few PDL cells survive.
- Saliva can be storage up to 2 hrs but milk can store up to 6 hrs.

- Transplantation – transfer of a tooth from one alveolar socket to another in the same person or into another person; transplanting partially developed root teeth has better prognosis;

POSTS:

- Major disadv of posts is they weaken tooth structure.
- Need at least 4mm of GP to preserve apical seal.
- Threaded posts increase chance of fracture while parallel/tapered posts are preferred.
- Pins increase stresses and microfractures in dentin.
- Cusps adjacent to lost marginal ridges should be restored with onlay.
- RCT cause destruction of coronal tooth structure and reduce structural integrity; minimum preparation of RCT tooth is ONLAY.

OPERATIVE

CARIES:

- Main cause of caries is bacteria or plaque formation; following cleansing of tooth, new plaque growth accumulates mainly on *interproximal surfaces*;
- Rate at which carious destruction of dentin progresses is slower in adults than in young people, due to generalized **dentinal sclerosis** which occurs w/ aging;
- *Zones of Dentin Lesion*: from innermost to outermost layers;
    1. Zone 1 – **normal dentin** w/ no bacteria;
    2. Zone 2 – **subtransparent dentin –** zone of demineralization but capable of remineralization and no bacteria;
    3. Zone 3 – **transparent dentin –** softer than normal dentin & same as zone 2;
    4. Zone 4 – **turbid dentin** – bacterial invasion & not able to remineralize;
    5. Zone 5 – **infected dentin –** decomposed dentin filled w/ bacteria;
- *Zones of Enamel Lesion:*
    1. **Translucent Zone –** deepest zone;
    2. **Dark Zone** – no polarized light; demineralization;
    3. **Body of Lesion** – largest part of lesion which also has demineralization;
    4. **Surface Zone** – unaffected by caries;
- Root Surface Caries = **senile caries**; spreads on surface rather than depth; use GI;
- Secondary Caries = **recurrent caries**; margins of existing filling;
- Defense mechanisms of pulp to irritation:
    1. **Sclerotic Dentin** – peritubular dentin formation; INITIAL defense;
    2. **Reparative Dentin** – irritation dentin fromation;
    3. **Vascularity -** inflammation
- *Lactobacillus* produce levan (polymer of fructose) not dextran;
- *Strep Mutans, Mitis, Sanguis, & Salivarious* initiate decay; they produce **dextran sucrase** (glucosyltranferase) – catalyzes formation of glucans from dietary sucrose;
    → Glucans = dextrans & mutans; Glucan forms plaque which hold lactic acid, produced from strep, against tooth;
- *Strep Mutans* produces great amounts of lactic acid & stimulated by sucrose;
- **Cariostatic** – stops caries; **Cariogenic** – causes caries, like bacteria *Strep mutans & Lactobacilli casei*;
- **Cariogenic Bacteria** must be acidogenic (produce acid) & aciduric (tolerate acid environment) & ability to form protective matrix (dextran);
    → **Strep Mutans & Sobrinus** are two most common cariogenic bacteria found in man;
- Predominant Bacteria Found in Plaque:
    1) *Strep Sanguis* – found earliest but NOT primary etiological agent in caries;
    2) *Actinomyces viscosus* & *naeslundii*
    3) *Strep mutans* (primary etiological agent), *mitis*, & *salivarious*
    4) *Veillonella*, *Lactobacilli casie*, & *Fusobacterium*
- Demineralization pH = 5.5; Remineralization pH > 5.5;
- Saliva helps prevent caries by: 1) diluting acid
  2) reservoir for Ca & PO_4 ions for remineralization
  3) reservoir for Ca, PO_4, & Fluoride ions & other ions for hypermineralization of enamel;
- Pit & fissures cares are most susceptible areas on tooth for plaque retention so highest prevalence of all caries; smooth surface areas are 2nd most susceptible;
- Acute Caries/Rampant Caries – rapid progressing, mostly children, lesion has small entrance but deep & narrow large lesion; may have pain;
- Chronic Caries – slow progression; mostly adults; dark pigment w/ leathery dentin & shallow lesion;
- Root Surface/Senile Caries – older patients & that attack cementum & radicular dentin; spreads more on surface rather than depth; **best prevention is to maintain periodontal attachement**;

→ Gingival recession is most related to initiation of caries in elderly;

- An incipient carious lesion on interproximal surface is usually located GINGIVAL to the contact area;
- Residual Caries – caries that remains in completed prep either by dentist's intension or accident;
- Secondary/Recurrent Caries – decay appearing at & under restoration margins;
- **Maxillary 1st Molar is tooth most likely to benefit from occlusal sealant placement;**
- Least likely microbial species found in dental plaque is *Staph Aureus*;

OPERATIVE:

- BW is best xray for diagnosing lesion of DL of canine; can use wedge to diagnose also;
- *Kissing Lesions* – prepare larger 1st & fill smaller 1st; access & shade better when done in both appts;
- Occlusal Reduction:

	Amalgam	**Gold**	**PFM**
○ Working cusps:	2.5-3mm	1.5mm	1.5-2mm
○ Non-working cusps:	2mm	1mm	1.5-2mm

- Direct Pulp Cap – CaOH hopefully stimulates reparative dentin bridge;
- Indirect Pulp Cap – waiting time after placing CaOH & IRM = 3-4 months hoping for secondary dentin formation;
- Class 1 carious lesions are least likely to occur on lingual surfaces of mandibular incisors;

INSTRUMENTS:

- Carbide Burs – slight (-) rake angle & edge angle of 90°; rotate rapidly before contacting tooth; used for cavity preps & best at HIGH SPEEDS;
  → **the greater # of blades causes less efficient cutting but smoother surface;**
- Steel Burs – used mainly for finishing procedures;
- Rotary instrument that produces roughest tooth surface after use is **cross-cut tapered fissure bur** at slow speed;
- **Bur blades** – each bur blade has 2 sides & 3 important angles;
- Rake face – faces direction of bur rotation; **Clearance face** – faces away from bur rotation;
- Edge Angle **–** angle formed b/w rake face & clearance face;
- Rake Angle of Bur – angle b/w line connecting edge of blade to axis of bur & rake face; **most important design characteristic of a bur blade;**
  → (-) angle = when rakeface ahead of radius; minimizes fractures; for hard materials like amalgam;
  → (+) angle = when radius ahead of rakeface; for soft materials, like acrylic;
- More cutting blades causes less efficiency but smoother surface & vice versa;
- Bur Formula = 10 – 85 – 8 – 14
  =blade width(1.0mm) cutting edge angle(85°) blade length(8mm) blade angle(14°)
- **Nib –** working end of non-cutting instrument (ball burnisher, condenser, etc.)
- Angling the shank of instrument so cutting edge of blade w/in 2mm of long axis of handle;
- Files are used to trim excess filling material, especially at gingival margins;
- The # of bevels that make up cutting edge can classify hand cutting instruments:
  → hatchets & chisels have single bevel while excavators are 2 beveled;
- Excavators: remove caries & refine internal parts of preparation;
  1. *Hatchet Excavator* – cutting edge of blade in same plane as handle; primarily for anterior teeth for preparing retentive areas;
  2. *Hoe Excavator* – cutting edge of blade perpendicular to axis of the handle;
  3. *Angle Former* – cutting edge at an angle other than 90 degrees to blade;
  4. *Spoon Excavator* – can be sharpend w/ **handpiece stones;**
- Chisels: used mainly to cut enamel;
  1. *Stright, Slightly curved, or Bin-angle* – primarily used for planing/cleaving enamel;
  2. *Enamel Hatchets* – chisel bladed instrument w/ cutting edge in plane of handle;
  3. *GMTs* – similar to enamel hatchet but has curve blade & angled cutting edge;
- Hand instruments transferred to dentist held by assistant b/w thumb & forefinger;

AMALGAM:

- The most frequent cause of failure of dental amalgam restoration is **improper cavity design;**
- Amalgam coefficient of thermal expansion 2x that of teeth;
- Amalgam tensile strength 1/5 to 1/8 it's compressive strength; more abrasion resistant than composite; Most amalgam restorations show slight setting expansion;
- If amalgam chips during carving, it's b/c amalgam was condense AFTEr its working time elapsed;
- Vaporization of amalgam during condensation of amalgam; greatest potential hazard of chronic mercury toxicity come from inhaling mercury vapor;
- Amount of mercury after condensation affects:
  1) Porosity of restoration
  2) Compressive strength of restoration
  3) Corrosive resistance of restoration
  4) Surface finish
- Amount of mercuring in set amalgam related to how much mercury-rich matrix is left after condensation; most important consideration of amalgam's **strength** is MERCURY CONTENT;
- The smaller the condenser point, the greater pressure exerted on the amalgam;
- High mercury content (if >55%) shows severe marginal breakdown; ideally = 43-50%;
- Moisture contamination of amalgam results in severe expansion of amalgam & corrosion; If amalgam w/ moisture, the zinc forms hydrogen gas; also ↓ compressive strength;
- Amalgam contaminated by moisture during trituration & condensation are the MAIN CAUSE of fractures; amalgam compressive strength greatly reduced when contaminated w/ moisture;
- For Amalgam → ↑ trituration time = ↓ setting expansion; correct trituration, ↑ strength but inadequate titration, ↑ corrosion; **better to overtitrate than undertitrate**;
  - → Properly triturated amalgam is shiny, wet, smooth, & homogenous;
  - → Purpose of trituration is coat the alloy particles w/ mercury; objective of trituration is to bring about an amalgamation of the mercuyr & alloy;
  - → During tritration, oxide film is rubbed off and clean metal is readily attacked by mercury;
- AMALGAM: ↓ setting expansion = ↓ free mercury & partical size
  ↑ trituration time & condensation pressure
  ↑ strength = ↑ condensation pressure & trituration time
  ↓ voids & partical size
- Amalgam RXN = Silver-tin Alloy + Mercury → Silver-tin Alloy + Silver-Mercury + Tin Mercury
  Ag_3Sn (gamma) Ag_3Sn(gamma) Ag_2Hg_3(gamma-1) Sn_3Hg (gamma-2)
  - → **Gamma** (30%) – unreacted alloy; STRONGEST & LEAST CORROSION; **Silver-Tin**;
  - → **Gamma-1** (60%) – matrix of unreacted alloy; 2nd strongest; **Silver Mercury;**
  - → **Gamma-2** (10%) – WEAKEST & softest phase; most corrosion; **Tin-Mercury;** add copper to reduce gamma-2; copper reacts w/ tin to prevent gamma-2;
- Components:
  1) *Silver* – **40-70%;** ↓ setting time, ↑ expansion & strength
  2) *Tin* (opposite of Silver) – **25-27%;** ↓ expansion & strength, ↑ setting time; component in amalgam that causes CONTRACTION;
  3) *Copper* – **6%/less;** ↓ creep & corrision & gamma-2 formation, ↑ strength & less marginal breakdown;
  4) *Mercury* – **3%/less**; initiates & activates reaction w/ alloys;
  5) *Zinc* – **1%/less;** ↓ oxidation of other elements;
  6) *Palladium* - **1%/less;** ↓ corrosion
  7) *Indium* – **1%/less**; ↓ surface tension
- Factors that influence final mercury content of a restoration:
  1. Original Mercury-alloy ratio
  2. Amount of trituration
  3. Condensation pressure & time

- **Creep** – deformation w/ time in response to stress; one of the main cause of marginal fractures of amalgam; overtrituration & undertrituration can cause ↑ creep; **time-dependent;**
  - → High copper & low mercury content & ↑ condensation pressure all ↓ creep;
  - → Creep of metal indicates that the metal will deform under *static load.*
- Marginal leakage of amalgam restorations ↓ w/ age;
- Discolored, corroded, superficial layer of amalgam is SULFIDE;
- Amalgam is BRITTLE but posses good compressive strength; brittleness of amalgam is why the occlusal margins aren't beveled;
- Class V Amalgam - 1) Retentive grooves on **gingivoaxial & incisoaxial line angles;**
  2) Outline deformed trapezoide or *kidney shaped;* parallel arcs if possible;
  3) NON-PARALLEL MD walls but PARALLEL OG walls;
  4) All walls DIVERGE;
  5) MD walls PARALLEL to transisional line angles but never beyond line angles; direction of MD walls determined by direction of enamel rods;
  6) axial wall should be uniformly deep into dentin & CONVEX to conserve tooth structure & minimize pulp irritation;
- 2mm b/w pulp & amalgam pulpal floor;
- MD walls of Class 1 amalgam diverge (same as direct gold & gold inlays) to prevent unsupported enamel at MD marginal ridges; width of marginal ridges for PMs = 1.6mm, for Molars = 2.0mm;
- Extend outline form before excavating any caries;
- Reverse "S" curve is curve put into B or L walls so wall meets external tooth surface at 90° angle;
- All walls meet tooth surface at 90° angle/butt joint;
- For class 2, B & L walls of proximal section converge occlusally but is determined primarily by position of adjacent teeth in relation to tooth being restored;
- When prepping class 2 on mand. 1st PM, bur tilted lingual to prevent hitting **facial pulp horn** & maintain dentinal support of lingual cusp;
- Gingival cavosurface margin beveled only if it is placed in enamel; bevel is no wider than enamel;
- Convenience Form – form of cavity prep takes to aid the operator in preparing, placing, or finishing the restoration;
- Retention Form – resist dislodgement or displacement of the restoration; B & L walls of Class 2 prep CONVERGE occlusally to prevent amalgam dislodgement;
  - → Occlusal dovetail & retention grooves in proximoaxial line angles provide resistance to dislodgement; grooves placed in axiobuccal & axiolingual line angels & extend axial wall height;
- Resistance Form - take to resist forces of mastication to prevent fracture of restoration & tooth; flat walls at right angles of tooth's long axis help achieve resistance form;
  - → When restoring cusp w/ amalgam, requires at least 2 mm of cusp be removed to provide resistance form;
- For Class 2 prep, should have independent retention & resistance form for both proximal & occlusal portions;
- **Most detrimental to strength of posterior tooth in a cavity prep is ↑ in F-L width;**
- *Matrix band* removed PRIOR to final carving; most difficult tooth to adapt matrix band is mesial of maxillary 1st PM; matrix band thickness = 0.002 inches;
  - → wedging action b/w teeth should provide enough separation to compensate for thickness of matrix band;
  - → proper proximal contour is provided by carving restoration & adapting contoured matrix;
  - → primary function of matrix is to restore anatomical contours & contact areas;
- Amalgam restorations should be finished & polished to reduce marginal discrepancies which reduces chance of recurrent decay; heat generation during polishing should be avoided;
- Amalgam is POOR THERMAL INSULATOR so explains why cold sensitivity is most common problem encountered after placing amalgam restoration;

PINS:

- Pins – 1-1.5mm inside cavosurface margin; >.5mm inside DEJ; 2mm into dentin & 2mm into amalgam;
  - → Should be inserted into DENTIN ONLY; they are retained by dentin's leasticity;
  - → **should be placed PARALLEL to external surface of tooth**;
  - → Function to retain restorative material; retention of pin ↑ as the diameter ↑;
  - → One pin per missing axial line angle is used; pins can WEAKEN restorative material when used;
  - → Optimum pin placement is at the ling angles of the tooth where tooth-to-root mass is greatest & risk of perforation is minimal;
  - → Threaded pins used to retain amalgam should NOT BE PARALLEL to each other or long axis of tooth;
- Indications for Pins:
  1) Class II amalgam prep where 1/more cusps have been lost
  2) very large class III amalgam prep
  3) Class V amalgam prep that far exceeds minimal dimensions
  4) prep for amalgam build-up over which a crown will be placed
- Contraindicated for young teeth w/ large pulps & teeth w/ reversible pulpitis;
- If pulp is hit when drill pin hole, obtain hemostatis, dry w/ paper point, place CaOH and find better pin hole location;
- Pins Types:
  1) **Cemented** – pinhole > pin
  2**) Friction Lock** – NOT RCT TEETH; pinhole < pin;
  3) **Self-Threading** – most common & most retentive; hole size just under screw diameter;
- TMS system has 4 pin sizes (regular, minim, minikin, & minuta) which are availabe in titanium or stainless steel plated gold;

GOLD:

- Most ductile & malleable metal;
- Chamfer bevel = hollow ground bevel; scooped out bevel to create more bulk of restoration material;
- Gold – retention from design of prep & friction b/w cavity wall & casting;
  - → Retention directly proportional to length (3mm) & parallelism of axial wall (6° taper);
- Gold Constituents:
  1) Gold - ↓ corrosion, ↑ ductility & malleability
  2) Copper - ↑ hardness; orange color; ranks 3rd in malleability;
  3) Silver – modified red color; ↓ temp, ↑ ductility & 2nd in malleability
  4) Platinum - ↑ temp, ↑ tensile strength, ↓ coefficient of thermal expansion
  5) Palladium - ↑ temp & hardness; absorbs hydrogen gas; whitening effect;
  6) Zinc – prevents oxidation
  7) Iridium – grain refiner; ↑ tensile strength & hardness;
- High Gold Alloys: 1) **Type 1** = 83% noble metal; soft & easily burnished b/c ↑ ductility; for **inlays**;
  2) **Type 2** = 78% noble metal; medium, for **onlays**;
  3) **Type 3** = 75% noble metal; hard, for **crowns;** when heated to cherry red color & quenched immediately, ↑ in malleability & ductility but ↓hardness & strength;
  4) **Type 4** = 75% noble metal; **bridges & RPDs;**
- Medium Gold = 25-75% gold/noble metals; Low Gold = 25% or less gold;
- Gold Substitute Alloys – do not contain gold, but called PASSIVE b/c they form protective surface oxide film layer that provides maximum corrosion resistance;
- **Karat –** the number of pure gold parts of a gold alloy, based on 24 parts (100% gold) as unit;
- Pure gold is only used in gold foil;
- **Fineness** – measured based on parts of pure gold per 1,000 = pure gold;
- Class V Prep for Gold:

1. Sharp internal line angles & small retentive undercuts at axio-occlusal & axio-gingival line angles; this is main characteristic in proper RETENTION;
2. M & D walls flare & meet the cavosurface at 90°; M & D walls placed at line angles; M & D walls diverge facially;
3. Conves axial wall w/ .5mm into dentin; occlusal wall slightly deeper than gingival wall b/c there is a thicker layer of enamel in occlusal wall;

- **Class V Gold** – Retention form → sharp internal line & point angles;
  Resistance form → flat MD walls & convex axial walls;
- Both Retention & Resistance form of Class V gold is SAME for Direct Gold;
- Gold Foil – oldest type of gold formed by rolling & beating gold into thin sheats, this causes elongation which give fibrous appearance; available in sheets, cylinders, & pellets;
  - → used for bulk filling & finishing veneer for mat gold;
  - → always microscopic voids due to improper condensing & using oversized pellets;
  - → surface hardness, tensile strength, & yield strenght are all increased during condensation of gold foil; good condensation with less force is accomplished w/ small point/condenser;
  - → direct gold is heated prior to condensation to drive off moisture & volatile compounds;
  - → Indications: 1) Ideal Lesion – no greater than 1-2mm into dentin
    2) Ideal Pulp – at least 2mm of dentin b/w restoration & pulp
    3) Ideal Periodontium – no tooth mobility
- **Direct Gold** - ↑ coefficient of thermal conductivity (12x amalgam); #1 indication for direct gold is small class 3 lesion; most important in adaptation of gold is direction force is applied;
- Class III Cavity Prep for Direct Gold: (use LINGUAL approach)
  1) Outline form is horizontal slot positioned gingival to contact area;
  2) Retention form from sharp internal anatomy
  3) Resistance form is provided by flat walls
- Material of choice for class III on distal of canine is amalgam or direct gold;
- *Disadvantages* of Gold: 1) ↑ thermal conductivity (12x that of amalgam)
  2) expesive & non-esthetic
  3) time consuming & technique senstive
  4) need to use cement which is weakest part of cast gold restoration
- Onlays – inferior retention than full crowns due to crown's greater axial surface area; restores large lesions that involve more than 1/3 intercuspal dimension & at least 50% of crown remains or loss of cusps w/ at least 1mm dentin supporting remaining cusps;
  - → Parallelism of axial walls is primary retentive feature in only prep; sharp point & line angles increase onlay retention;
  - → **Shoeing** a functional cusp is NEVER INDICATED; it is minimal/partial cusp coverage via a finishing bevel on cusp crest; **Cap** a cusp is preferred b/c complete coverage of cusp;
  - → From facial to lingual, the axiopulpal line angle of an onlay prep is longer than the axiogingival line angle;
- Always bevel/plane margins or wall junctions of onlay cavity to remove unsupported enamel AND compensate for casting inaccuracies; bevel DOESN'T minimize need for gingival extension;
  - → Bevel used mainly to improve marginal adaptation;
  - → 3 types of bevels: 1) short bevel – cuts only external 1/3 of enamel prisms
    2) full bevel – involves entire thickness of enamel
    3) wide bevel – involves full thickness of enamel & some dentin
- Most effective means for verifiying enough occlusal clearance is **wax bite chew-in**;
- Inlay – lack of undercuts is the characteristic common to all class II gold inlay preps; an occlusal lock/dovetail should be done to prevent proximal dislodgement; marginal ridges need to be rounded;
  - → All margins are beveled resulting in 40° marginal metal;

- **Crystalline Gold/Mat Gold** – formed by electrolytic precipitation yielding a crystalline structure resembling trees/links of chain; used for bulk fillings; flow & adaptation not as good as other gold;
- **Powdered Gold** – formed by atomizing; granules in this materal have spherical shape; can be placed in very short time period; denser than foil thuse easier to manipulate & condense;
- Cohesion of gold at room temperature is example of ATOMIC ATTRACTION;

COMPOSITE:

- Dental adhesion = dental bonding;
- **Adhesive joint** – adhesion of intermediate material w/ 2 surfaces;
- **Adhesive Potential** – smaller the angle, the greater the wetting & potential for adhesion;
- Composite < Amalgam for compressive strength & occlusal wear; serious limitation is polymerization shrinkage;
- Amount of stress for composite depends on **C-Factor** = ratio of bonded:unbonded areas;
- Composite: ↓ wear resistance is primary cause of failure of class II composite restorations; **difficulty in finishing these restoration is the softness of the resin & hardness of the filler**;
  - → Contraindicated in pts w/ heavy occlusion or bruxism;
  - → The most desirable finished surface for composite is obtained w/ aluminum oxide disks;
- Composite Resins – are dimethacrylate monomers & polymerize by addition mechanism initiated by free radicals, which generate by chemical activation or external energy;
- Disadvantage of Methyl Methacrylate - ↓ resistance to abrasion & ↑ thermal coefficient of expansion;
- In comparison to poly (methyl-methacrylate acrylic), composite has ↓ coefficent of thermal expansion, ↓polymerization shrinkage, ↑ compressive strength, & ↑ stiffness;
- **Biphenol A-glycidyl methacrylate** – component common to most composite resins, sealants, bonding & glazing agents, & resin cements for ortho bands;
- **Chemical Activated** (self-cure) **Resins**: 2 pastes = benzoyl peroxide(initiator) + tertiary amine (activator);
- **Light Activated Resin –** (VLC) → diketone photoinitiator (camphoroquinone) & amine activator;
- Visible Light Cure Composites (VLC)- have α-diketone initiator which absorbs energy from visible light (peak intensity = 474 nm; blue light) and then ketone reacts w/ amine to produce free radicals;
  - → **Increment thickness** most affects curing a light-activated composite resin;
  - → Most popular way to polymerize matrix monomers using an external energy source to activate polymerization process; VLC have completely displaced UV light systems;
  - → Light energy range = 410-500nm; **curing light is used at wavelengths 400-500nm**;
  - → Light needs to be held w/in 2mm of resin to be effective; provides DENSER restorations than self-cure resins b/c no mixing required so no air bubbles;
  - → Most hazardous to retina so can cause retinal damage; Must have protection w/ pts who had recent cataract removed; **with darker resin shades, cure a little longer;**
  - → Most serious limitation is POLYMERIZATION SHRINKAGE;
  - → Advantages: 1) greater depth of resin can be cured
    2) Resin can be polymerized thru enamel
    3) intensity of visible light remains relative constant;
- The light source affects ther perception of color b/c the light source must contain the color's wavelength to be matched in order to see that color;
- **Composite Components**:
  1. *Filler* – barilium silica glass/quartz/zirconium silica; combined w/ 5-10% weight of colloidal silica; reduces polymerization shrinkage & increases hardness;
  2. *Matrix* – difunctional monomers;
     i. Bis-GMA – highly viscous (**Sealants are generally comprised of Bis-GMA**)
     ii. Urethane Dimethacrylate (UEDMA)
     iii. Tri-ethylene Glycol Dimethacrylate (TEGDMA) → added to reduce viscosity;

3. *Coupling Agent* – silane provides adhesive b/w inert filler & organic matrix;

- **Composite Fillers** - 1) Macrofill = 10-100 microns; first composite resins made;
  2) Midifill (small particle) = 1-10 microns;
  3) Minifill = .1-1 micron
  4) Microfill (fine particle) = .01-1 micron; SMOOTHEST FINISH & greatest resistance to occlusal wear;
  5) Hybrid = mixture, usually MIDIFILL or MINIFILL w/ MICROFILL;
- Hybrid Resin Composites – highly filled w/ glass & SiO_2; good esthetics; use silica fillers to ↑ hardness & wear resistance but highly polishable;
- ↑ filler in restorative composites & ↓ filler in flowable composites;
- the higher filler & BIS-GMA, the greatly reduced coefficient of thermal expansion;
- Only advantage of unfilled resins = ↓ coefficient of thermal conductivity; common cement bases; unfilled resins have high coefficient of thermal expansion = 7-8x that of tooth;
- Unfilled resins are the SOFTEST of all restorative materials; also lower modulus of elasticity;
- Unfilled resins have the greates extent of marginal leakage related to temperature change;
- **Dentin Conditioner** – primarily removes the smear layer of dentin & etch the intertubular dentin to produce microspaces w/in dentin surface; placed after enamel is etched
- **Primer** – hydrophilic monomer (ie→ hydroxyethyl methacrylate – HEMA); penetrates smear layer & fills intertubular dentin;
- **Bonding Agent** – unfilled resin adhesive (BIS-GMA, HEMA);
- Generations of Adhesives: -- 4th Generation – 3 step etch & rinse adhesives
  -- 5th Generation – 2 step etch & rinse adhesives
  -- 6th Generation – Type 1 → 2 step w/ primer & adhesive separate; Type 2 → 1 step
  -- 7th Generation – 1 step
- Bonding of composite to dentin depends on **difunctional coupling agents**;
- Acid Etch – when used, all enamel margins should be beveld for more surface area and to enhance the seal & retention to reduce microleakage; purpose of acid etch is more surface area & roughen surface;
  → the acid cleans surface debris so better wetting of enamel by resin;
  → acid-etch composites have best initial seal but over time seal weakens so AMALGAM has best seal over time;
  → it increases retention & adaptation by: 1) ↑ surface area;
  2) conditioning surface for better wetting;
  3) creating surface irregularities for better mechanical locking;
- In class 3 composite prep, retention points should be placed ENTIRELY in dentin w/ grooves placed along **gingivo-axial & inciso-axial line angles**; small rounded retentive areas are preferred;
- Outline for of composite class V resembles amalgam class V except that the composite internal angles are much more ROUNDED;
- Whenever possible, used composite syringe to place composite to reduce trapping air in restoration;
- Most important factor in preparing & restoring Class II composite is MOISTURE CONTROL;
- Material most likely to cause an adverse pulpal reaction whe placed directly in a deep cavity prep!
- Normal wear mechanism of the resins is best explained by abrasion of matrix, , exposure of filler, & dislodgement of filler particles;

CEMENTS:

- Chelation of Calcium ions on tooth by ionized Polyacrylic acid side-groups is principal mechanism of chemical adhesion to tooth structure;
- Solubility of Cements → Zinc Polycarboxylate > Zinc Phosphate > GI Cement;
- Cements main function in cast restorations is seal the cavity, NOT retention;

- Low coefficient of thermal conductivity is property most characteristic of curren available cements;
- Glass Ionomer Cement – good thermal indicators; disadv – higher film thickness; limited strength and wear resistance but ↓ strength; often used for root surface cavities; doesn't polish as well;
  - → Powder= **fluoroalumino-silicate glass**; Liquid= **Polyacrylic Acid**(adhesive & biocompatible);
  - → ↑ solubility when first mixed so very technique sensitive;
  - → micromechanical bond w/ composite resins; also for Class V restorations w/ composite "sandwich technique"; only GIC used as cement & permanent restoration;
  - → good thermal insulator (so no pulpal protection needed);
  - → "**fluoride-sponge**" – b/c can absorb fluoride when local ionic concentrations are high, then slowly release fluoride when the environment concentration decreases;
  - → ↓ compressive strength, tensile strength, & hardness compared with composite;
  - → 3 Types: 1) Conventional GIC – luting agent
    2) Light-cured GIC – liner or base; preferred b/c of extended working time;
    3) Resin-modified Light Cured GIC - Fuji
- Zinc Phosphate – Powder = **Zinc Oxide**; Liquid = **orthophosphoric acid**; acidic (pH = 3.5) & can cause irreversible pulpal damage; shrinks slightly upon setting; oldest luting agent;
  - → Retention = mechanical interlocking; SUPERIOR STRENGTH;
  - → ↓ compressive strength when mixed faster; cold slab - ↑ working time & ↓ setting time;
  - → setting time ↑ when less water; provides an anti-bacterial effect;
  - → can be used as base or liner if HIGH COMPRESSIVE strength is needed;
  - → if zinc phosphate cement based used w/ restoration, varnish is applied PRIOR to placing base;
  - → CAN be used under composite;
  - → if high powder-liquid ratio, ↓ viscosity, stronger final set & ↓ solubility; **powder-liquid ratio is most important variable of cement's STRENGTH (the more powder, the stronger);**
- Zinc Polycarboxylate Cement – chelation of calcium ions provides chemical adhesion; NOT irritating to pulp; thick & short working time; first cement developed for adhesion to tooth structure;
  - → Powder = Zinc Oxide + Magnesium Oxide; Liquid = Polyacrylic acid & copolymers;
  - → Compressive strength less than $ZnPO_4$ but tensile strength greater than $ZnPO_4$;
- ZOE/IRM: ↑ strength & abrasion resistance; ↓ solubility; pH of ZOE = 7 so least irritating of cements; Powder = Zinc Oxide & Liquid = Eugenol; provisionals are usually cemented w/ ZOE cement!
  - → eugenol has palliative effect on pulp but not a thermal insulator;
  - → placed on dentin/enamel prior to bonding b/c it compromises bonding;
  - → retains about 20% by weight of **polymethyl methacrylate** in powder component;
  - → pts may be allergic to oil of cloves in eugenol; not for DPC b/c can irritate pulp;
  - → **Carboxylic acid** is the component that could replace eugenol in a zinc oxide paste;
  - → inhibits composite polymerization setting rxn b/c of eugenol; *used for*:
    1. Intermediate Restorations
    2. Base under non resin restorations
    3. Deciduous teeth restorations
    4. Restorative emergencies
  - → 4 Types of ZOE: 1) Type I = temporary cement
    2) Type II = permanent cement
    3) Type III = reinforced ZOE for temporary filling & thermal insulating base
    4) Type IV = cavity liner

LINERS & BASES:

- Bases – material 1-2mm thick that function as barrier agains pulpally irritating agents, provide thermal insulation, & provide adequate resistance to compressive forces of mastication;
  → Serve as replacement or substitute for protective dentin destroyed by caries & cavity preparation;
- Primary base under amalgam/composite is CaOH but under gold is $ZnPO_4$/ZnPolycarboxylate/GI;

- Primary base not used under polycarboxylate cements b/c doesn't irritate the pulp;
- Most common used secondary base is placing ZINC PHOSPHATE over CaOH base that has been placed over pulpal exposure (DPC);
- Cements used as bases should be mechanically stronger so mixed with maximum powder content;
- Only distinction b/w base, cement, & cavity liner is final thickness:
  - → Cement = 15-25 microns, Liners = 5 microns, Base = 1-2 millimeters;
- The most important consideration for pulp protection in restorative techniques is the **thickness of remaining dentin**; Selecting the approriate base or liner to restore the axial wall of a Class II restoration depends on the biological effect required & thickness of remaining dentin;
- Cavity varnish reduces initial microleakage of amalgam restoration;
- Cavity Liners → used to seal dentin tubules; 3 types:
  1. *Copalite* (cavity VARNISH)→ not good under resin; cavity varnish; **solution liner** = 1-5 microns;
     a. Cavity Varnish Functions: reduce marginal leakage, prevent acid penetration, protect pulp tissues, & prevent mercury penetration;
  2. *Dycal* (CaOH) → **suspension liner** = 20-25 microns;
  3. *ZOE* → **suspension liner**; prevents thermal shock;
- Suspension liners are thicker than solution liners;
- **CaOH** → ability to stimulate formation of secondary dentin; RADIOLUCENT; most commonly used suspension liner that prevents thermal shock;
- When using acid-etch to restore class IV fracture, exposed dentin should be covered w/ CaOH liner;

MATERIAL'S TRAITS & INVESTING:

- Brittle materials have high compressive strength but low tensile strength;
- **Alloy –** mixture of 2/more materials mutually soluble in the liquid state; solidifies thru a range of temperatures;
- Modulus of Elasticity – measures stiffness or rigidity of material; Modulus of elasticity is the ratio of stress to strain;
- Ductility – ability of metal to easily be worked into desired shapes; expressed in percent elongation; it depends on plasticity & tensile strength; ductility↓ with temp ↑;
- Malleable – metal being able to be hammered into a thin sheed w/o rupture; depends on plasticity; malleability ↑ w/ ↑ temperature;
- Coefficient of Thermal Expansion: tendency of material to change shape w/ temp. changes;
  - → Tooth = 11.4 ppm/^{o}C
  - → Gold = 14.4 ppm/^{o}C
  - → Amalgam = 22-28 ppm/^{o}C
  - → Composite = 28-35 ppm/^{o}C
  - → Unfilled Resins = 81-92 ppm/^{o}C
- Consequence of thermal expansion & contraction differences b/w restorative material & adjacent tooth structure is **percolation;**
- Perculation – cyclic ingress & egress of fluids @ restoration margins;
  - → ↑percolation = ↑recurrent decay;
- Elastic Limit – greatest stress a material can be subjected to and still return to its original dimensions when the forces are released;
- Proportional Limit – the greatest stress produced in a material such that the stress is directly proportional to the strain; ↑proportional limit = more resistance to permanent deformation;
  - → Similar to elastic limit; can interchange the terms;
- Adhesive Potential – predicted by measuring the spreading/wetting of the adhesive over a substrate surface; done by determining contact angle of drop of adhesive as it spreads out;
  - → Smaller the angle, the greater wetting & potential for adhesion
  - → 2 types of adhesion: physical forces (van der Waals) & chemical forces (chemisorption)

- when a liquid wets a solid completely, the contact angle b/w the liquid & sold is 0^o;
- Toughness – total energy absorbed to the point of fracture; it is affected by yield strength, tensile, strength, percent elongation, & modulus of elasticity; brittleness is opposite of toughness;
- Resilience – energy that a material can absorb before the onset of plastic deformation;
- Percent elongation of metal is measure of ductility & is related to permanent strain at fracture;
  - → Property that most closely describes ability of cast gold inlay to be burnished is percentage elongation;
- **Yield Strength > Proportional Limit > Elastic Limit**
- **Quenching** advantages → 1) Maintains Castings malleability & ductility
  2) casting easier to clean
- Annealing – soften material by heating; metal becomes tough & less brittle;
  - → 3 stages – recovery, recrystallizaiton, & grain growth;
- Tempering – hardening by heat treatment;
- Gypsum Investment Expansion ↓ when: 1) older investment
  2) ↑ water:powder ratio
  3) ↓ spatulation
  4) ↑ time b/w mix & water bath
- Components of Gypsum Investments:
  1. **Refractory Filler** – silicon dioxide like *quartz* or *cristobalite* (60-65%); provides thermal expansion for investment;
  2. **Binder** - α-calcium hemihydrate (30-35%); adds strength;
  3. **Modifiers** – like magnisium oxide, NaCl, boric acid, graphite, or potassium sulfate
- Thermal expansion is the main cause of mold expansion which compensates for solification shrinkage of specific alloy; *Variables that Influence Gypsum Expansion*:
  → Older investment = ↓ expansion
  → ↑ water powder ration = ↓ expansion
  → ↑ spatulation time = ↑ expansion
  → ↑ time b/w mixing & immersion in water bath = ↓ expansion
- Thinner mix of gypsum investment causes ↓ setting expanion, ↓ strength, ↑ setting time, & ↑ porosity;
- **Sprue** – diameter > 1.5mm; diameter of sprue should be >/= to the thickest part of patter; sprue attached at 45^o angle to thickest part of pattern;
- Invest wax pattern immediately to avoid shape changes due to relaxation of internal stresses in wax;
- Types of Inlay Wax → **Type A** (hard, low flow), **Type B** (medium flow), **Type C** (soft, high flow; for crowns or onlays);
  → Contains: *Paraffin wax*(soft & **main ingredient**), *Gum Dammar*(medium), *& Carnauba wax* (hard);
- Zones of Flame - from inner to outer zones → *mixing zone* (cool & colorless)
  → *combustion zone* (green/blue & surrounds inner core)
  → *reducing zone* (hottest zone & only part of flame that should be used to heat the alloy)
  → *oxidizing zone* (if contacts metal, a dull film of **dross –scum on molten metal,** develops over metal surface)
- *Example* - Porcelain at of PFM is separated at porcelain-metal interface, separation may be caused by **degassing metal at too low temp** or **fusing opaque coat of porcelain at too low a temp;**
- Properties usually found in materials consisting of *ionic bonds* are brittleness & high melting point (not weakness);

FLUORIDE & SEALANTS:

- Fluoride concentration in community water depends on air temperature & water consumption;
- Forms of fluoride in water: 1) Sodium Silicofluoride

2) Hydrofluorosilic Acid (well water)
3) Sodium Fluoride

- Fluoride supplements recommended if communal fluoride water conc < .7ppm for up to 13 yo;

Recommended Dosages of Supplemental Fluoride:			
AGE	**<0.3ppm**	**0.3-0.7ppm**	**>0.7ppm**
6mo-3yrs	.25mg	none	none
3-6yrs	.5mg	.25mg	none
6-16 yrs	1mg	.5mg	none

- FLUORIDE: 1) Creates Fluoroapatite
  2) Inhibits acid production that causes decay
  3) ↑ enamel remineralization
  4) Inhibits production of glucosyltransferase (dextran sucrase)
  5) Bacteriocidal Action
- Fluoride ion easily exchanged for hydroxyl ion in enamel b/c fluoride is slightly smaller than hydroxyl ion & fluoride ion has greater affinity for hydroxylapatite crystal than hydroxy ion;
- Fluorosis is **enamel hypoplasia;** IRREVERSIBLE; doesn't occur after most teeth erupted but can occur in primary or permanent teeth;
- Fluoride conc > 4mg/L = toxic; convert ounces to grams = 8.2 ounces x 28.35 (constant) = 232 grams;
- **Probable Toxic Dose** for Fluoride = > 5mgF/kg;
- Fluoride is excreted by kidneys;
- Prenatal fluoride not approved by FDA but DOESN'T cross placenta;
- Fluoride may corrode surface of titanium implants;
- Systemic distribution of fluoride may affect tooth morphology;
- Daily Use Fluoride Gel = 0.4% Stannous Fluoride & 1% neutral NaFl; used for root caries, xerostomia, radiation therapy, & teeth for overdenture;
- 3 Types of Topical Fluoride:
  a. **Acidulated Phosphate Fluoride -** 1.23% NaFl + 1M orthophosphoric acid; **pH = 3-3.5**; most common in practice; may affect existing restoration by removing the glaze;
  b. **Sodium Fluoride** – 2%; over the counter 0.05% recommended; **pH = 9.2**;
  c. **Stannous Fluoride** – 8%; poor taste & may cause staining; **pH = 2.1-2.3;**
- Daily application of 1.23% Acidulated Fluoride in fitted trays for 4 min is MOST EFFECTIVE way to increase the fluoride content in the external layers of teeth;
- Low viscosity sealants wet acid etch teeth best (30-50% Phosphoric Acid);
- Retention of fissue sealants is chiefly the result of *mechanical microretention;*
- Fluoride therapy & occlusal sealants modify the HOST the most;
- Sealant Properties closer to Unfilled Resins; Components:
  → Monomer → Bis-GMA
  → Initiator → benzoyl peroxide
  → Accelerator → amine
  → Opaque Filler → titanium oxide

MISCELLANEOUS:

- *Woodburry* RD frame has more retraction but *Young's* frame (u-shaped) is more popular;
- Isolate a minimum of 3 teeth w/ RD; for tooth being clamped, hole is 1 size larger than the holes over teeth without a clamp; punching holes too close together in RD may cause damage to gingival papilla;
- When using #212 clamp for class V, punch hole larger and slightly FACIAL to other holes in arch;
- Pregnant pts have more inflamed gingiva;
- **DENTIN -** 1) *Primary Dentin* – form initial shape of tooth; deposited b/f completion of apex;
  2) *Secondary Dentin* – formed after apex completed (regular dentin – slow formation rate);
  3) *Tertiary Dentin* – aka *Reparative Dentin* – formed by replacement of odontoblasts; irregular shape & limited to site of irritation; composition same as secondary dentin;

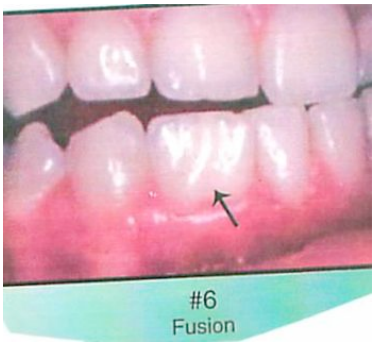
4) *Sclerotic Dentin* – when dead tracts/empty tubules calcify;

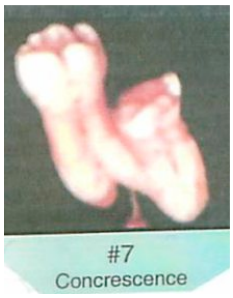
- Dentin is less dense than gold, enamel, amalgam, & porcelain;
- **Galvanic Shock** – brief & sharp electrical sensation when 2 different materials contact (like amalgam & gold); 1 microamperes = 500 mV; gradually subsides & disappears in a few days;
- PM contacts from facial view → Junction of occlusal & middle third;
- Molar contacts from faical view → Middle third;
- Posterior teeth occlusion view of contacts → slight BUCCAL of middle third; **this creates a wide lingual & narrow facial embrasure;**
- In posterior teeth, gingival tissues fill cervical embrasure; it is normally "col" shaped from F-L cross section view;
- **Height of Contour** – thickest portion or point of greatest circumference of the tooth viewed from occlusal surface;
- Bleaching – **In-Office =** 35% hydrogen peroxide (4-10 min cycles);
  - → **At-Home =** 10% carbamide peroxide;
  - → Extrinsic stains → vital bleaching; bleaching affects color change in both dentin & enamel;
  - → Best stains for bleaching: yellow > brown > orange > grey;
  - → Materials for "walking bleaching" are sodium perborate & 30% aqueous Hydrogen peroxide;
- Green & orange stains on maxillary incisors are usually attributed to poor oral hygiene;
- Anticholinergic drugs cause xerostomia b/c block receptor sites for acetylcholine;
- LA reduces saliva in mouth b/c reduces anxiety & sensitivity;
- Clinical Signs of **Occlusal Trauma**: Mobility, Thermal Sensitivity, Attrition, & Facial Recession;
- Glycerin, Kaolin, & Sodium Fluoride can all treat root sensitivity;
- **Hydrodynamic Theory** – pain results from indirect innervation caused by dentinal fluid movement in tubules, stimulating mechanoreceptors near predentin;
- **Zinc Chloride** – most likely to cause NECROSIS of the sulcular epithelium & adjacent layer of CT when impregnated into cord for gingival retraction; epi, alum sulfate or alum chloride don't cause necrosis;
- Good hygiene & fluoridation will least protect groove defects;
- Most sensitive area of tooth during cavity preparation is **DEJ;**
- a hyperemic pulp may respond to low levels of current from an EPT;
- Reversible pulpitis changes to Irreversible pulpitis primarily b/c of invasion of microorganisms;
- Drugs that act as anti-sialogogues (anti-salivary agents) – **Atropine** & **Methantheline** (Banthine);
- Use of **Propantheline Bromide** (Pro-Banthine) to control salivary secretions is contraindicated in pts w/ glaucoma or cardiovascular distress;
- Reversible Hydrocolloids have the LONGEST SHELF-LIFE;
- The syringe material that is most rigid and most difficult to remove from the mouth is POLYETHER;
- Most effective way to reduce injury to the pulp during restoration procedure is to minimize dehydration of dentinal surface;
- Dentist adjusts the shade of a restoration using a complementary color; this procedure results in a decreased value!
- **Dextranase –** the enzyme when incorporated into a mouthwash is most likely to interfere w/ microbial aggregation in the plaque mass;

ORAL PATHOLOGY

TOOTH ABNORMALITIES:

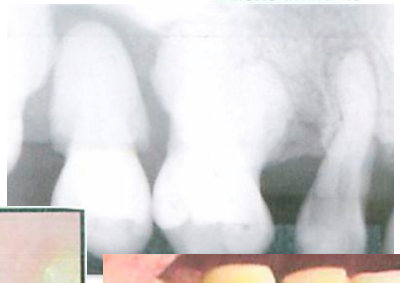
- Anodontia – missing all teeth (either prim/perm); associated w/ **Hereditary Ectodermal Dysplasia.**
- Oligodontia – congenitally abscent teeth; 6 or more.
- Hypodontia – absence of only a few teeth.
- Diphyodontia – having 2 successive set of teeth (humans);
- Polyphydontia – more than 2 sets of teeth in a lifetime.



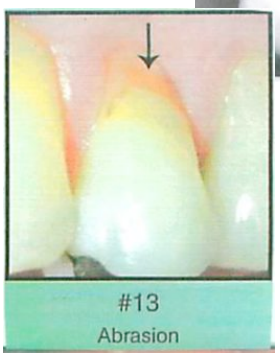


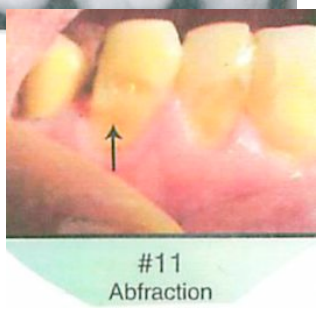
- Hypsodontia – teeth w/ high crowns/cusps; associated w/ diet of abrasive foods.
- Fusion – crown fused as 1 wide crown but 2 roots; developemental union of 2/more teeth; dentin united in both teeth; 2 germs = 1 crown; during INITIATION & PROLIFERATION; Macrodontia.
- Concrescense – only cementum of 2/more teeth become united;

- Dens in Dente - dens invaginatus; caused by deep invagination of enamel organ during formation; Taurodontims – enlarged pulp & shortened roots.
- Gemination – division of single tooth germ by invagination; incomplete formation of 2 teeth; share same root canal; during initiation & proliferation;
- Twinning – complete division of single tooth bud; completely separated & MIRROR IMAGE;

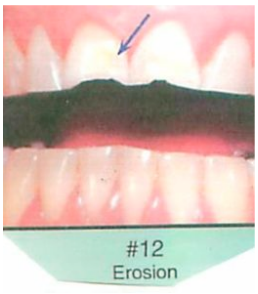


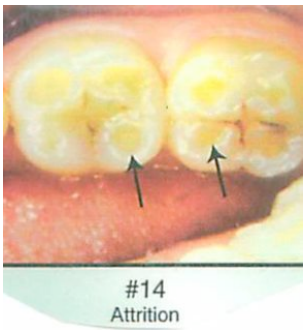
- Ankylosis – fusion of cementum/dentin to alveolar bone after loss of periodontal membrane; most common tooth – PRIMARY 2nd MOLAR, so perm 2nd PM fails to erupt.
- Hypercementosis – apical ½ of root (s-times whole root); often PREMOLARS; often vital teeth & sometimes chronic inflammation of tooth; nothing seen clinically but x-rays show bulbous enlargement that surrounds continuous & unbroken periodontal membrane space.
  → Paget's Disease Hypercementosis – absence of periodontal membrane & lamina dura surrounding hyperplastic cementum.



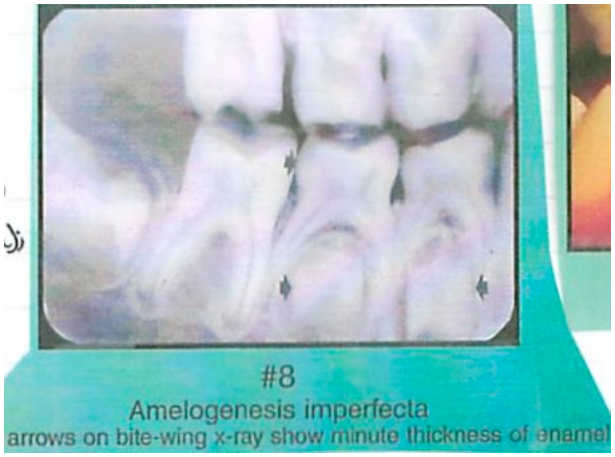


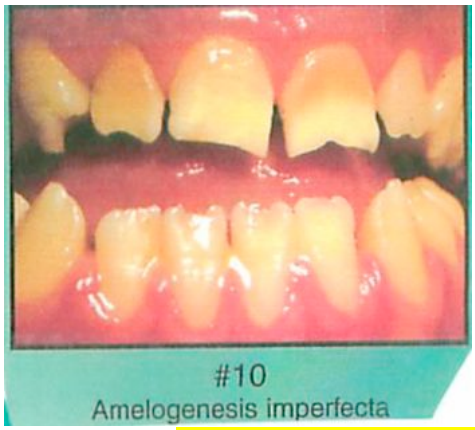
- Abfraction – cervical erosive lesion from tensile & compressive forces but cant be attributed to any cause; enamel "pops off" at base of tooth;
- Abrasion – abnormal loss of tooth structure due to non-masticatory physical friction; tooth brush abrasion causes V-shaped wedges at cervical margin in CANINES & PMs.



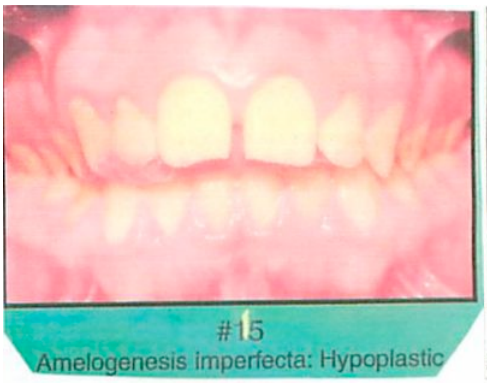


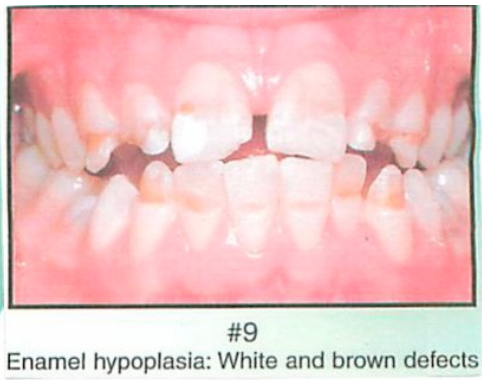
- Attrition – wearing away of enamel & dentin to normal function or bruxism; commonly exposes dentin on LINGUAL INCISAL of MAX ANT. & FACIAL INCISAL of MAND. ANT.
- Erosion – loss of tooth structure by non-mechanical means; affects smooth & occlusal surfaces; "cupping appearance."
- **Amelogenesis Imperfecta (AI)–** hereditary ECTODERMAL DEFECT; three types: 1) Hypoplastic, 2) Hypocalcified, 3) Hypomaturation;

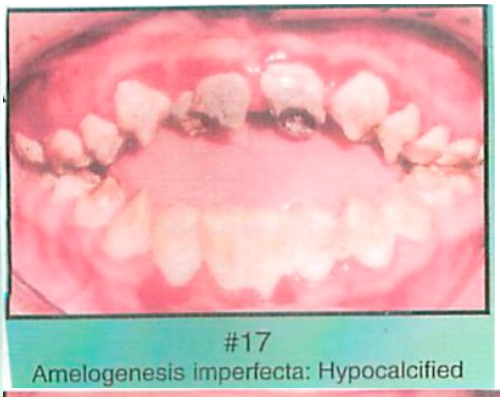

#8
Amelogenesis imperfecta
arrows on bite-wing x-ray show minute thickness of enamel


#10
Amelogenesis imperfecta

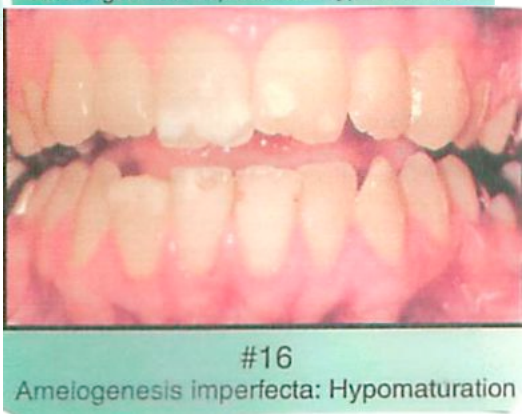
- Enamel Hypoplasia (AI Type 1)– developmental defect where enamel of teeth is hard in context but thin & deficient in amount; incomplete formation or defective formation of enamel matrix;
  → Due to illness/injury during teeth formation; common in child w/ history of genenralized growth failure in 1st 6 months of life; it is a dental manifestation of HYPOPARATHYROIDISM (tx w/ Vit.D).
  → Clinical – lack of contact, ↓ in enamel thickness; rapid breakdown of occlusal surface; pits & groves missing; yellow/brown stain when dentin exposed.

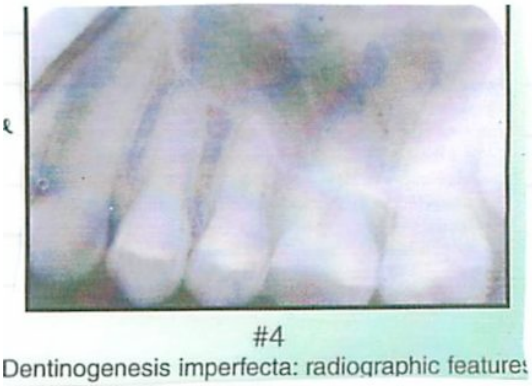

#15
Amelogenesis imperfecta: Hypoplastic


#9
Enamel hypoplasia: White and brown defects

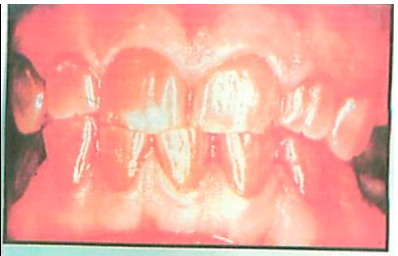

#17
Amelogenesis imperfecta: Hypocalcified

- Enamel Hypocalcification (AI Type 2) – hereditary defect in which enamel is soft & undercalcified but normal in quantity; caused by defective mineralization maturation of ameloblasts;
  → chalky & surfaces wear down easily; can be removed w/ prophy cup.
- Enamel Hypomaturation (AI Type 3) – enamel can be pierced by explorer under pressure & can be chipped away from normal dentin; from IMMATURE CRYSTALLITES;
- **Dentinogenesis Imperfecta:** hereditary mesodermal defect; autosomal dominant in 1:7000 kids;
  → teeth have amber, gray, purple opalescence/discoloration; pulp completely obliterated by continue deposition of dentin; crowns – short & bulbous & roots are narrow;
  → enamel chips w/in 2-4 yrs after eruption; either prim/perm. teeth; also presents w/ abnormal constriction at CEJ; 3 types:
  1. **Type 1** – dentin abnormality from OSTEOGENESIS IMPERFECTA; shows blue sclera or history of bone fractures;
  2. **Type 2** – MOST COMMON; dentin abnormality w/ no bone involvement;
  3. **Type 3** –BRANDYWINE type; dentin abnormalities but more variation than type 2; multiple pulp exposures in dentin;


#16
Amelogenesis imperfecta: Hypomaturation

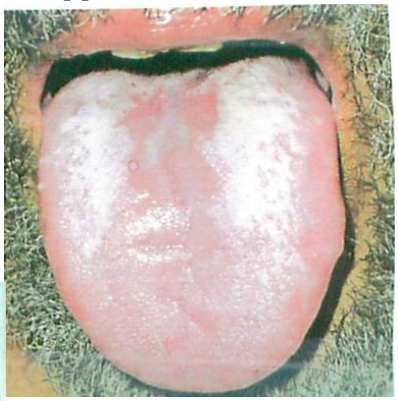

#4
Dentinogenesis imperfecta: radiographic features

CAUSE	INTRINSIC TOOTH STAIN
Dentinogenesis Imperfecta	--translucent/opalescent hue, gray to bluish-brown.
Erythroblastosis Fetalis	--bluish-black, greenish-blue, tan or brown.
Porphyria	--red/brownish.
Fluorosis	--white opacities or light bronw to brownish-black.
Pulpal Injury	--Pink & becomes orange-brown to bluish-black.
Internal Resorption	--Pink.
Tetracyclin	--Light gray, yellow, tan to darker grey.

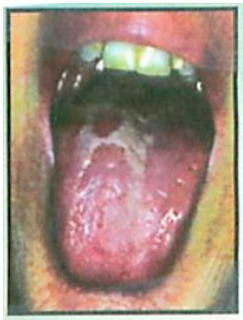

#3
Picture Illustrates the "Opalescent Hue" of Dentinogenesis imperfecta

- Dentin Dysplasia (ROOTLESS TEETH)– autosomal dominant; normal eruption but bluish cervical region; 2 types:
  1. **Type 1** – Radicular Dysplasia; Most COMMON; normal crowns & slight amber translucency; complete obliteration of pulp; abnormal spaces b/w teeth & mobility;
     → X-rays show short roots, obliterated pulp, PA radiolucencies;
  2. **Type 2** – Coronal Dysplasia; normal appearance of perm. teeth; semi-transparent opalescent primary teeth; incomplete obliteration of pulp & pulp stones;
     → X-rays reveal complete obliterated pulp after eruption, pulp stones, and no PA radiolucencies.
- Internal Resorption – nay have no trigger but may be caused by resorption of dentin by pulpal walls from inflammation of pulpal injury; pink-hued area on crown; if no perforation, perform RCT.
- Idiopathic External Resorptive Lesions – invasion of cervical region of root by fibrovascular tissue which resorbs dentin, enamel, & cementum; source of resorption is attachement apparatus;

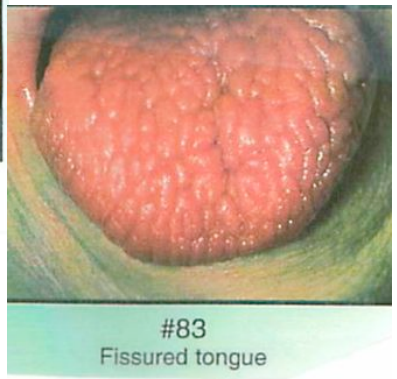
TONGUE DISEASES:



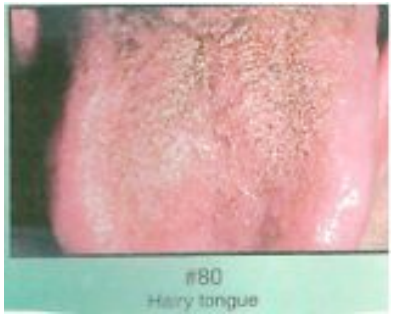
- Median Rhomboid Glossitis – asymptomatic, elongated, erythmatous patch of atrophic mucosa at mid-dorsal surface of tongue due to CHRONIC Candida INFECTION;
  → more often in diabetes & immunosuppressed pts & on prolonged antibiotic therapy;
  → smooth, denuded, beefy, red lesion devoid of filiform papillae; most common – midline of dorsum of tongue, **anterior to circumvallate papillae**.


#82
Geographic tongue

- Geographic Tongue (***Migratory Glossitis***)– desquamation of filiform papillae; white lesions surrounding atrophic red central zones; irregular shaped map-like smooth swollen patch on tongue; changes daily in size & shape;
  → slight burning; heals in one site & then found in another site; associated w/ fissured tongue.
- Fissured Tongue – deep median fissure of tongue w/ lateral symmetrical radiating grooves; found in…
  → **Melkersson-Rosenthal Syndrome** – 1) fissured tongue, 2) granulomatous cheilitis, 3) facial paralysis.
  → ↑ w/ age; asymptomatic but may be painful if infected;


#83
Fissured tongue

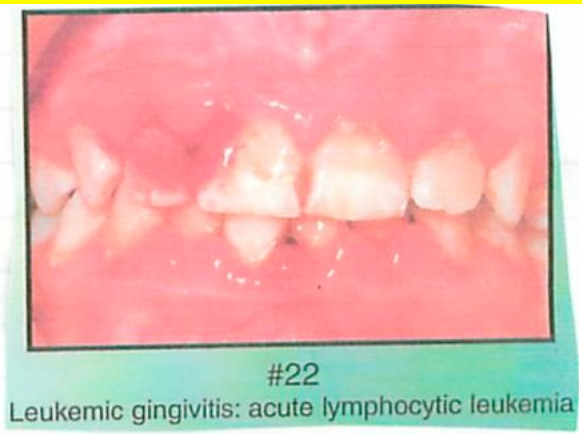
- Burning Mouth Syndrome – common condition that pts. feel like mouth has been scalded; usually affect front part of mouth but sometimes only tongue; sometimes ↓ taste sensation/alters taste; clinically mouth appears normal; common in post-menapausal women; diagnosed by elimination of other diseases.
- Hairy Tongue – benign condition of **hypertrophy of filiform papillae**; dorsum appears furry; yellow-white to brown-black; from SMOKING.
  → Midline dorsume of tongue; 4 types of *Papillae*:
  1. ***Filiform*** – most numerous; v-shaped rows; no taste buds & ↑ keratinization;

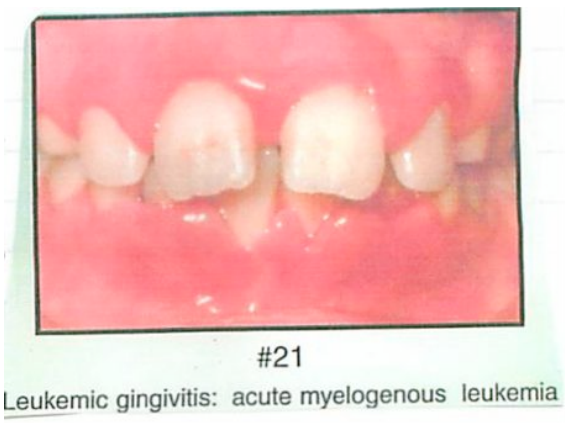

#80
Hairy tongue

2. ***Fungiform*** – flat, mushroom shaped & found at tip & lateral margins;
3. ***Circumvallate*** – largest but least numerous & circular shaped; inverted V-shape; associated w/ Von-Ebner's glands.
4. ***Foliate*** – lateral margins as 3-4 vertical folds.

LEUKEMIA:

- Form of cancer that begins in blood-forming cells of bone marrow; Can modify inflammatory rxns.
- The hematopoietic cells make leukocytes (WBC) to defend body against infections but some of the leukocytes are damaged & wont die off & multiply excessively; b/c of the WBC accumulation, there is a ↓ in production of RBC, platelets, & normal WBCs; so the damaged WBCs overwhelm bone marrow & enter blood stream and invade other organs;
- Leukemia causes – genetics, family, viruses (EBV), radiation, chemical toxins.
- **Monocytic Leukemia** – oral lesions most likely observed in this type of leukemia; rarely chronic form; gingival hemorrhage, gingival hyperplasia, petechiae, ecchymoses, & ulcerations.
- Classified by dominant cell type & duration from onset to death; 4 Types:
  1. **Acute Lymphocytic Leukemia (**ALL) **– most common type** in kids; peak age = 4 yrs; "null cells"
     → Most leukemia responsive to therapy; tx = chemo, radiation, & bone-marrow transplant.
  2. **Acute Myelogenous Leukemia** (AML) **– most malignant type**; **most common in adults**;
     → Malignant disease of bone marrow which hematopoietic precursors are arrested in early stage development; involves **granulocytes & megakaryocytes**.
     → has 30% more blasts in bone/blood; these myeloblasts contain Auer Rods;
  3. **Chronic Lymphocytic Leukemia** (CLL) – **least malignant type**.
     → Chronic leukemias are 50% of leukemias & are slow progressing but less devastating.
  4. **Chronic Myelogenous Leukemia** (CML) – characterized by overgrowth of granulocytic precursors (*Myeloblasts* & *Promyelocytes*) in bone marrow & blood;
     → common in young & middle-aged adults; more in men; FATAL; survival avg = 4 yrs;
     → 2 distinct phases; 90% of pts have *Philadelphia Chromosome* where long area of Chromosome 22 is translocated usually to chromosome 9;
     → Symptoms – spongy bleeding gums, fever, fatigue, weight loss, massive splenomegaly, joint & bone pain, repeated infections.
     → both AML & CML are most common in adults!
- Oral lesions of Myelogenous Leukemia: -gingivitis & gingival hemorrhage
  -generalized gingival hyperplasia
  -petechiae & ecchymosis
  -ulcerations.
- **Candidiasis** is common in leukemia children b/c susceptible to infections;


#22
Leukemic gingivitis: acute lymphocytic leukemia


#21
Leukemic gingivitis: acute myelogenous leukemia

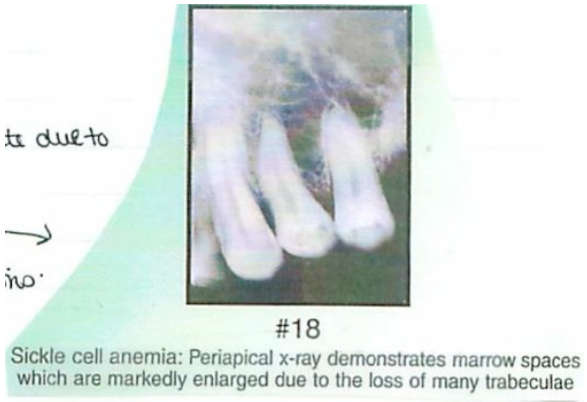
- Chronic Leukemias – characterized by proliferations of lymphoid or hematopoietic cells that are more mature than acute leukemias; insidious onset w/ weakness & weight loss; SPLENOMEGALY.
  - Petechiae, ecchymosis, hemorrhages, & bacterial infections.

- WBC>100,000/mm^3 w/ *granulocytes* & *lymphocytes* (mature forms) dominating.

- Acute Leukemias – have rapid onset & progression; abrupt onset w/ fever, weakness, malaise, anemia, & submandibular lymphadenopathy; organs involved – liver, spleen & bone marrow.
  - Petechiae, ecchymosis, hemorrhages, & bacterial infections.
  - WBC – 30,000 to 1,000,000/mm3 w/ myeloblasts & lymphoblasts (immature forms);
  - If untreated, pts will die in 6 months; high incidence in pts w/ *Down's Syndrome*.
  - 75% of cases, the lymphocytes are NEITHER B or T cells but called "null" cells.
- Stem cell Leukemia – cell too immature to classify;
- Aleukemia – leukemic cells in bone marrow but circulating WBCs are neither immature nor ↑ in number; Subleukemia – leukemic cells in blood but no increase in WBCs in blood;
- Leukemoid – increase in number of circulating granulocytes.

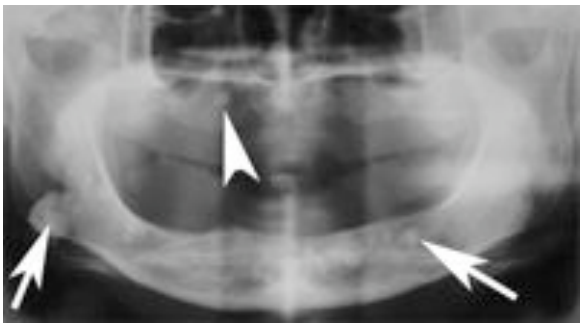
OTHER BLOOD DISORDERS:

- Aplastic Anemia – anemia where bone marrow generates defective RBCs; the most serious & life-theratening blood dyscrasia associated w/ drug toxicity; 2 types:
  1. *Primary* – fatal; unknown cause; affects young adults; Symptoms – pallor, weakness, malaise, dyspnea, headache, & vergito;
     → Oral symptoms – spontaneous bleeding, petechiae, & gingival infections.
  2. *Secondary* – caused by exposure to toxic agents; occurs at any age; good prognosis.
- Plummer-Vinson Syndrome – iron-deficiency anemia w/ atrophic changes in buccal, glossopharyngeal, & esophageal mucous membrane;
  → also koilonychia (spoon shaped nails) & dysphagia;
  → complications - carcinoma of the tongue & post-cricoid regions;
  → tx – iron, vit. B & ↑ protein.
- Sickle-cell Anemia – inherited disease (need 2 traits to get it) in which RBC's become crescent-shaped; so cause blood clots causing painful episodes called "sickle cell pain crisis"; more in females < 30 yrs;
  → produces abnormal hemoglobin (hemoglobin S); 10% of african. amer. carry trait & 0.2% have it.
  → Dental x-ray shows marrow spaces enlarge b/c loss of trabeculae but **teeth unaffected**.
  → Gene mutation is single nucleotide (T for A) of β-globin gene resulting in glutamic acid substitute by valine; life of RBC decreases from 120 to 20 days.


#18
Sickle cell anemia: Periapical x-ray demonstrates marrow spaces which are markedly enlarged due to the loss of many trabeculae

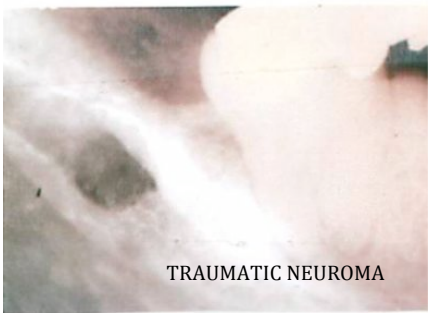
- Agranulocytosis – decrease in circulating granulocytes/neutrophils caused by antimetabolic, antibiotic, & cytotoxic & antithyroid drugs about ½ of the time;
  → cyclic neutropenia causing severe gingivitis, necrotizing ulcers of gingiva & palate, & gingival bleeding; ragged ulcers covered by gray membrane.
  → oral infection w/ rapid periodontal destruction is most characteristic feature of the disease.
  → Sudden fever, chills, jaundice, & weakness;
  → Histologically – little/no apparent inflammatory cell infiltrations.
- Pernicious Anemia – common, chronic, progressive, megaloblastic anemia b/c lack of intrinsic factor – used for absorption of Vit B_{12} which is for maturation of erythrocytes;
  → So decrease in erythrocytes; **Schilling 24-hr urine test** evaluates B_{12} absorption.
  → S&S - painful tongue (atrophic glossitis), cheilitis, tingling numbness of extremities & painful swallowing.
- Thalassemia – Major & Minor – hemolytic/bursting anemia from genetics; decreased levels of erythrocytes & abnormal hemoglobin;
  → Flaring of Max. ant. teeth w/ malocclusion; Anemic pallor of oral mucosa;

- Erythroblastosis Fetalis – when baby Rh-pos & mom Rh-neg & mom produces antibodies that cross placenta & attach & destroy fetuses RBCs causing anemia;
  - → Most common = ABO incompatibility;
  - → Least common = Rh incompatibility; most severe w/ tx of **Rhogam**.
  - → Baby may have enlarged liver/spleen, edema, jaundice, anemia;
  - → Oral – teeth appear green, blue, or brown hue & enamel hypoplasia.
- **Purpura spots** - purple on skin from small bleeding vessels near surface of skin; may be on mucous membrane or internal organs; two kinds:
  1. *Thrombocytopenic Purpura* (**Werlhof's Disease**)– bleeding diorder b/c decrease platelets causing bruising, petechiae, & hemorrhages; oral – gingival hemorrhage & petechiae on palate;
     i. TE's are contraindicated!!
  2. *Thrombotic Thrombocytopenic Purpura* – severe & fatal form from decreased platelet count due to consumption of platelets by thrombosis.
- Polycythemia – too many RBC's in circulation so blood to thick causing blockages & stroke; 2 types:
  1. **Primary** – when excess RPC produce tumorous abnormalities in tissue that produce bllod cells; 75% of pts have SPLENOMEGALY;
  2. **Secondary** – increased RBC b/c of another condition like pulmonary disease, Osker's disease (high altitude), or secretion of Erythropoietins;
     i. Oral – purplish/red mucous membranes (esp. tongue & gingiva), swollen & bleeding gingiva, submucosal petechiae, ecchymosis, & hematomas.

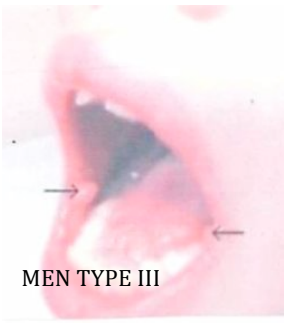


BENIGN NEOPLASMS (NON-ODONTOGENIC TUMORS):

- Osteoma – benign tumor of mature compact or cancellous bone; well-circumscribed RO masses;
  - → new bone growing on another piece of bone;
- Gardner's Syndrome (Polyposis Syndrome) - autosomal dominant disorder of multiple osteomas of the jaw, particularly @ angle of mandible;
  - → multiple polyps in GI eventually leading to colon cancer (adenocarcinoma in 4th decade).
  - → Early puberty, abnormal retina; multiple epidermal cysts of face, scalp, extremities;
  - → Oral – multiple impacted & supernumerary teeth; "cotton wool" appearance; multiple odontomas.

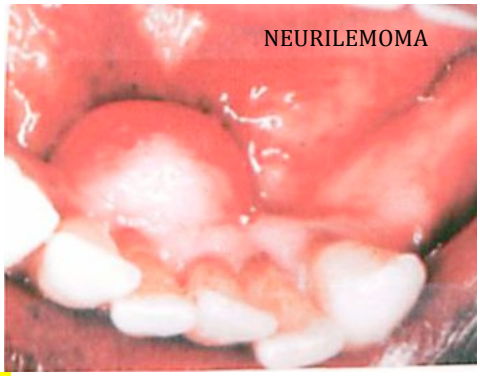


- Traumatic Neuroma – lesion caused by trauma to peripheral nerve; most commonly seen in mand. mucobuccal fold in region adjacent to mental foramen, esp in edentulous mouths; Tx = excision.
  - → Small, firm, movable, well-encapsulated nodule; pain when palpated; extraction sites are also common sites for a neuroma.
  - → Histologically – abundant nerve tissue & collagenous fibrous tissue, chronic inflammatory cell infiltrate, & SCHWANN CELLS.
  - → Multiple neuromas on lips, tongue, palate may indicate MEN III (multiple endocrine neoplasia sydrome) – autosomal dominant.

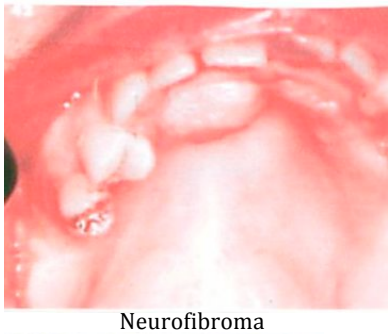


- Multiple Endocrine Neoplasia Syndrome – autosomal dominant; 3 types:
  1. *MEN I* – tumors/hyperplasia of pituitary, parathyroid, adrenal cortex & pancreatic islets;
  2. *MEN II* – (Sipple's Syndrome & Subtype A) – parathyroid hyperplasia or adenoma; these pts have pheochromocytomas of adrenal medulla & medulla carcinoma of thyroid; ability to metatasize & cause death;
  3. *MEN III* – (Subtype IIB) – mucocutaneous NEUROMAS, pheochromocytomas of adrenal medulla & medullary caracinma of the thyroid; often neuromas of oral cavity.

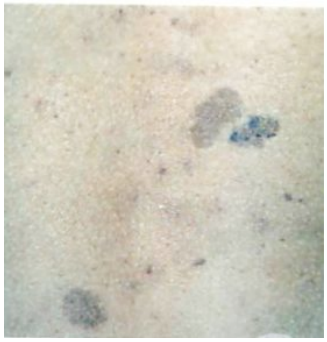
- Neurilemoma (SCHWANNOMA) – well-demarcated, benign lesion of fibroblastic proliferation of nerve sheath cell (schwann cells); often tongue;
  → Electric-shock like pain;
  → often mandibular mucobuccal fold near mental foramen.


NEURILEMOMA

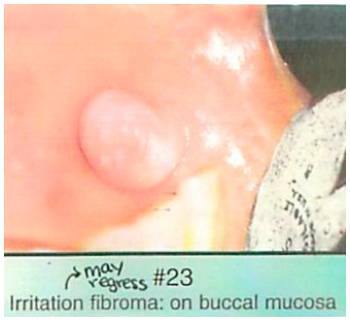
- Juvenile Nasopharyngeal Angiofibroma – rare benign neoplasm; adolescent males; mass in nasopharynx causing obstruction/epistaxis; tx = surgery.
- Lymphangioma – benign hemartomas of lymphatic channels early in life; doesn't undergo malignant changes; skin/mucous membrane; most common on dorsal & lateral tongue surface of ant. position;
  → Tx = aspiration then excision.
  → Clinically – raised, diffsue, nodules, pink, red, brown, or black; 4 types:
  1. Lymphangioma Symplex – small, thin walled lymphatics; asymptomatic.
  2. Cavernous Lymphangioma – dilated lymphatic vessels; soft, flluctuant.
  3. Cystic Lymphangioma – macroscopic lymphatic spaces; varies in sized.
  4. Benign Lymphangioendothelioma – lymphatic channels dissecting thru collagen; size varies & painless.


Neurofibroma

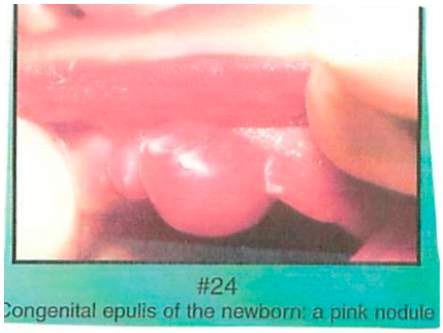
- Neurofibroma – tumor of nerve fiber itself; neoplasm of schwann cells & fibroblasts; appears as sessile, firm, pink nodule; most common benign peripheral nerve tumor;
  → often seen as part of NEUROFIBROMATOSIS;
  → Common on tongue, BM, & vestibule; 5-15% may become malignant



- Multiple Neurofibromatosis – autosomal dominant disorder associated w/ MULTIPLE NEUROFIBROMAS; Chromsome 17; 2 types:
  1. *Type 1* - **Von Recklinghausen's Disease** – 1:3000 people; mild cases w/ limited # of tumors; multiple neurofibromas on face, extremities, intraorally, & trunk; Café-au-Lait Spots on skin >1.5cm.
     → Potential disfigurement & high risk for malignant transformation;
     → Spots on Iris (Lisch nodules) & axilla (Crowe's Sign);
     → Loss of tumor suppressor gene (NF1 & NF2).
     → Xray – well demarcated unilocular/multilocular RLs, possible root disturbance, or jaw enlargement; tx = excision or do nothing;
  2. *Type 2* – more severe but 1:50,000 people.

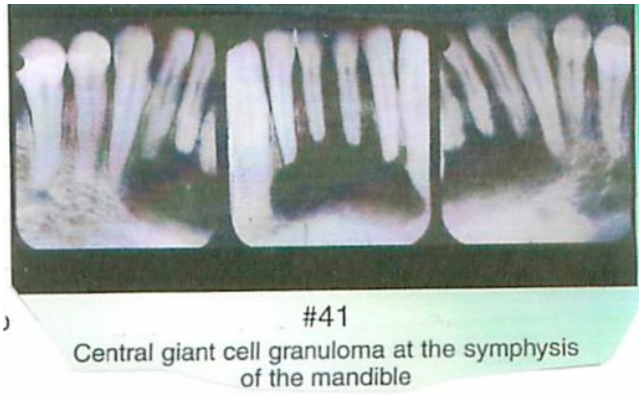


- Fibroma – most common oral benign neoplasm of connective tissue origin; on BM, lateral tongue, & lower lip; most common tumor in oral cavity.
  → Pink, painless, smooth, elevated, well-demarcated.
  → Difference b/w true fibroma & irritation fibroma is that true fibroma doesn't regress but irritation fibroma does even after excision.



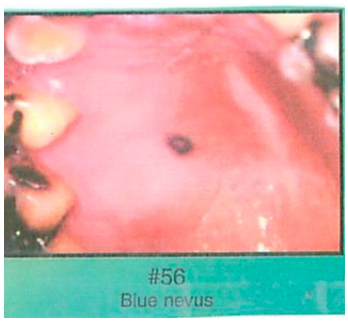
- Congenital Epulis of Newborn (Congenital Gingival Granular Cell) – often ant. maxilla & 10x more in females; lesion is pink, soft, & compressible; tx = surgical excision
- Granular Cell Myoblastoma – rare neoplasm; uninflamed, asymptomatic mass; most common on tongue;
- Both Congenital Epulis of Newborn & Granular Cell Myoblastoma are *histologically identical* w/ both having granular cells but Myoblastoma has pseudoepitheliomatous hyperplasia overlying epithelium but congenital epulis doesn't.

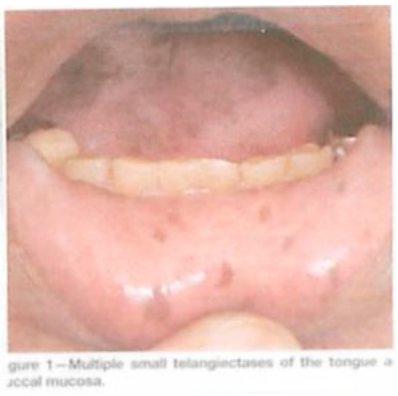
- Central Giant Cell Granuloma – intraosseus destruction lesion of ant. mand & max. where large lesions expand cortical plate causing tooth mvmt & root resorption; may be caused by trauma; can be aggressive or non-aggressive; 70% anterior to 1st mand. molar;
  - → Spindle-shaped mesenchymal cells & aggregates of multinuclated giant cells;
  - → more common in children, females, and mandible;
  - → Slow growing w/ asymptomatic swelling; rapid growth with painful & loose dentition;
  - → Similar to ameloblastoma & odontogenic keratocyst.



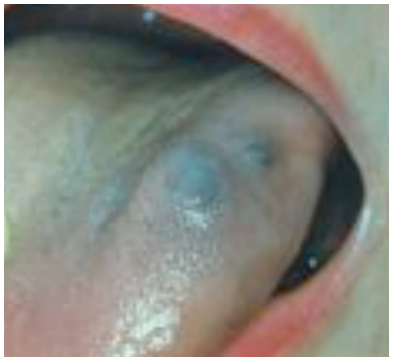
RED/BLUE/BLACK ORAL LESIONS:

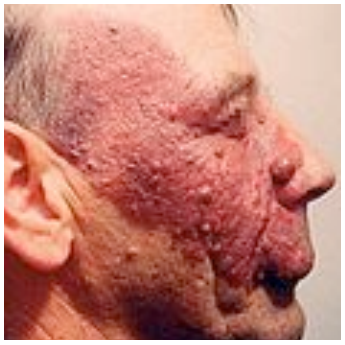
- Amalgam Tatoo – oral pigmented lesion; on gingiva, BM, or alveolar mucosa;
- Bismuth – heavy metal used in dermatology; "bismuth line" – think, blue-black line in marginal gingiva & sometimes confined to gingiva papilla.
- Nevi (moles) – most common on HARD PALATE; congenital nevi (>10com) have higher incidence of malignant transformation;
  - → **Tyndall Effect** – melanocytes deep in surface reflect shorter wavelength (blue).
  - → acquired nevi are more common than congenital intraorally; DYSPLASTIC; 5 subtypes:
  1. Intramucosal Nevus – most common intraorally; slightly raised & solid;
  2. Blue nevus – 2nd most common; painless.
  3. Compound nevus – raised & solid; rare orally; cells b/w epithelium & lamina dura;
  4. Junctional nevus – cells b/w epithelium & lamina dura; flat & premalignant.
  5. Intradermal nevus – common mole; most common skin lesion;



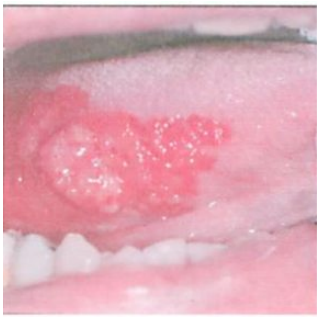


- Osler-Weber-Rendu Syndrome (**Hereditary Hemorrhagic Telagiectasia**) – telageictasia are dilated superficial blood vessels near skin/mucosa; autosomal dominant hereditary form of hemangioma;
  - → spider like telangiectases on face, neck, lips, gingiva, BM, & tongue;
  - → earliest sign – epistaxis (nose bleeds); onset often in childhood.
  - → Also known as **Polycythemia Vera**!
- Hemangioma – endothelial cells & CT origin; **proliferation of blood vessels**; biologically active; growth is independent of child's growth; F:M = 5:1; POSITIVE pressure test.
  - → Incisional biopsy contraindicated! Tx = laser therapy/surgery.
  - → Common on tongue, BM, lips, & palate; 3 types:
  1. Capillary
  2. Cavernous
  3. Hemangioendothelioma – stratified squamous epithelium covering loose, fibrous CT containing thin-walled engorged vascular spaces.
- Encephalotrigeminal Angiomatosis (**Sturge-Weber**) – uncommon w/ unknown etiology; sometimes classified as variant form of hemangioma; consists of facial lesion known as PORT-WINE STAIN, which is distributed over CN V; often unilateral; multiple supernumerary teeth!





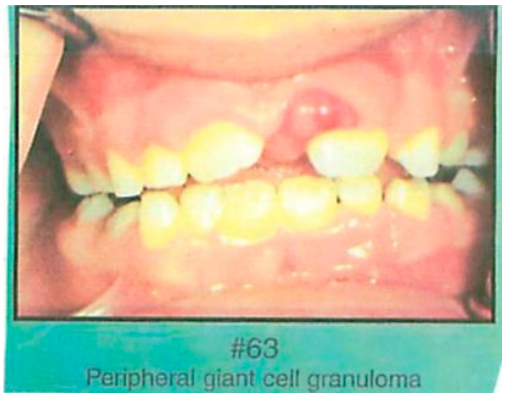
- Erythroplakia – microscopic pic of epithelia dysplasia;
  → common insitu & invasive SSC; mostly affects pts > 60yrs.
  → most likely in mucobuccal fold, oropharynx, & floor of the mouth;
  → PREMALIGNANT; less common than leukoplakia.



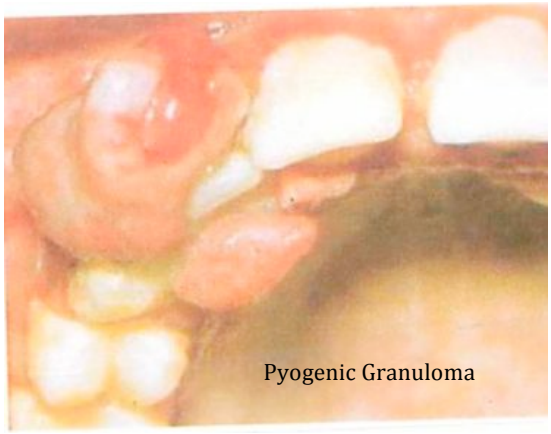
- B-K mole syndrome & Dyplastic Nevus Syndrome – both have large, pigmented atypical nevi which have high risk for malignant melanoma;
- **Focal Melanosis** – common circumstances where brown area of pigmentation occur in oral cavity; no tx is necessary; developmental; most common areas – gingiva, then lower lips; seen in Addison's disease, ACTH tumors, malignant tumors of pituitary gland & Metastatic malignant melanoma;

CONNECTIVE TISSUE LESIONS:

- Peripheral Giant Cell Granuloma – most common on alveolar mucosa or gingiva; believed to be reactive; local dental irritation contributes to lesion's development;
  → **70% of ant. segment of jaw & > in mand.**;
  → Pedunculated broad-based growth w/ smooth surface; redish blue in color & sometimes lobulated; >20yrs of age & F:M = 2:1;
  → Xray – cupping out/saucerization of alveolar bone; histologically identical to central GCG!

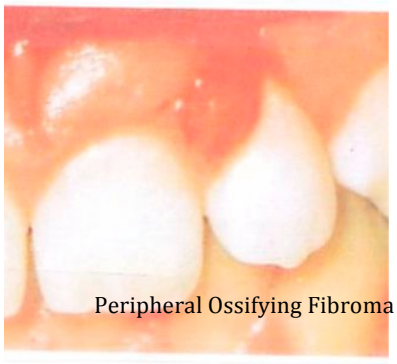


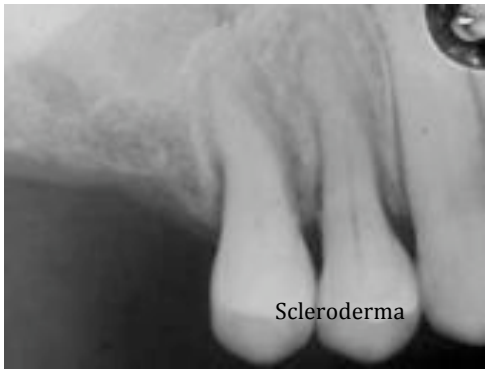
- Pyogenic Granuloma – soft, red lesion sometimes seen in pregnant women; increased vascularity; soft pedunculated broad-based growth w/ smooth red surface; common on gingival, lower lip, tongue & BM;
  → **arises from minor trauma** to tissues causing fast-growing reactive proliferation of endothelial cells; raspberry appearance; DON'T REMOVE til after PREGNANCY!
  → may be caused by hormone changes during 1st trimester!



- Peripheral Ossifying Fibroma – benign reactive lesion that may recur; hyperplastic tissue; tissue colored & maybe ulcerated;
  → painless, solid, firmly attached, gingival mass exclusively!
  → arises from PDL fibers & frequently causes separation of adjacent teeth; Tx = surgical excision;
  → ages 5-25 yrs; peaking @ 13 yrs; more females;
  → majority of lesion occur anterior to molar region and maxillary more than mandibular;
  → histologically - ↑ degree of cellularity; 2 other forms:
    i. Peripheral Odontogenic Fibroma – gingival mass composed of well-vascularized non-encapsulated fibrous connective tissue;
    ii. Giant cell Fibroma – fibrous hyperplasia composted of multi-nucleated CT.
- Scleroderma – autoimmune; inflammation & progressive tissue fibrosis & occlusion of microvasculature by excessive production & deposition of Type 1 & 3 collagen; women>men and ages 25-50 yrs; affects CT of joints, blood vessels, & interal organs.
  → x-rays show abnormal but uniform widening of PDL & bilateral resorption of angle of mandibule, codyle, or coronoid process; 2 types:
    1. *Localized Scleroderma* – isolated areas of skin/tissues; mild & no organs;

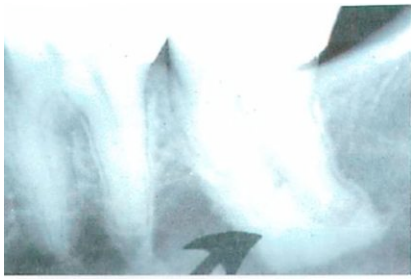
2. *Systemic Scleroderma* – skin & internal organs; may affect blood vessels, joints, digestive system, lungs, heart, kidney, & muscles;


Peripheral Ossifying Fibroma


Scleroderma

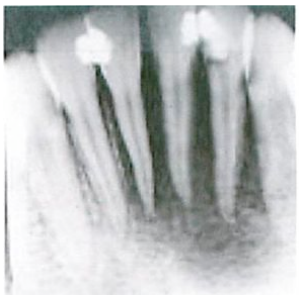

Scleroderma

- Crest Syndrome – mild form of scleroderma; tx – steroids; Clinical:
  - C – calcinosis cutis
  - R – raynaud's phenomenon
  - E – esophageal dysfunction
  - S – sclerodactyly
  - T – telangiectasias of face & vermillion border of lips & tongue.



INFLAMMATORY JAW LESIONS:

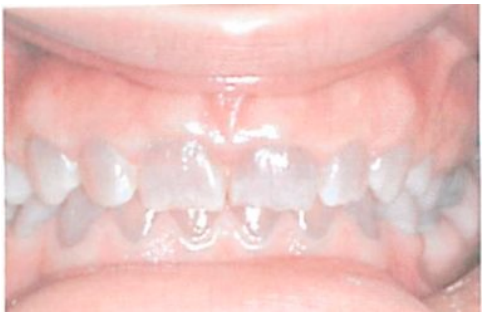
- Condesing Osteitis (AKA – Chronic Focal Sclerosing Osteomyelitis) – unusual rxn in high tissue resistance or low-grade infection; most common – 1st mand. molar;
  → Xray- well-circumscribed RO mass of sclerotic bone extending below apex of 1/both roots; differs from benign cementoblastoma in that w/ Osteitis, the root outline is clearly visible on the xray.



- Osteomyelitis – acute pyogenic (bacterial infections that make pus) infection of bone often from staph infection; often starts in another part of the body & spread to bone via blood;
  → bone may produce abscess causing pain, redness, swelling, fever, malaise.
  → In children – affects long bones; in adults – affects vertebrae & pelvis.
  → Risk factors – trauma, diabetes, hemodialysis, drug abuse, & no spleen pts.

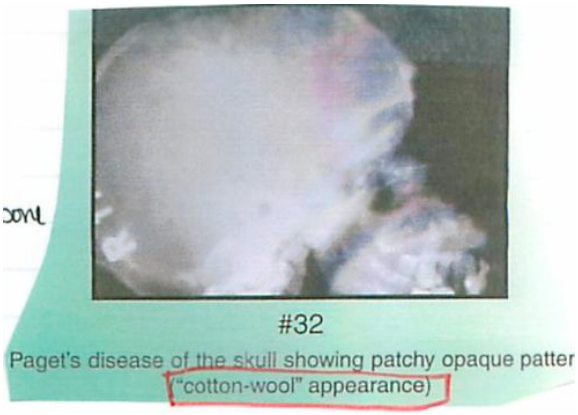
METABOLIC JAW DISEASES:

- Osteoporosis - most common bone disease; generalized hereditary condition of excessive bone mineralization; tx = supportive tx.
  → causing frequent fractures, lack of hematopoietic fuction & tendency for osteomyelitis of the jaw;
  → when body fails to form enough bone or when too much old bod is resorbed;
  → leading cause of osteoporosis is ↓ estrogen in women & ↓ testosterone in men.
  → In infantile osteoporosis, delayed eruption & ↑ in fractures (also w/ adults);
- Osteogenesis Imperfecta – genetic disorder of person w/ less collagen or poorer quality of collagen leading to bones that fracture easily; varries greatly form person to person; blue sclera.
  → Bones extremely fragile & porous, loose joints, low muscle tone, triangular face, tendency of spinal curvature; NO CURE!
  → Teeth – bulbous crowns, pulp obliterated, shorter/narrower roots; PRIMARY teeth affected more.
    1. **Type 1** – most common & mild and associated w/ DENTINOGENESIS IMPERFECTA;
    2. **Type 2** – most severe causing multiple fractures just from birth.



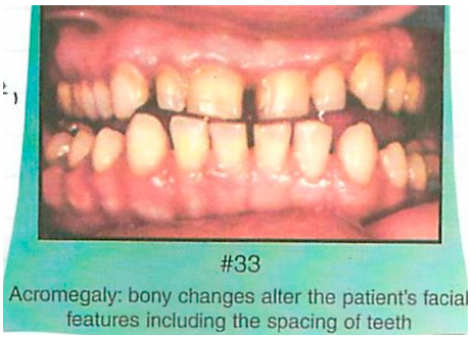
3. **Type 3** – most severe form beyond perinatal period.
4. **Type 4** – mild to moderate sever bone fragility;

- Hypophosphatasia – inherited metabolic bone disease from ↓ enzyme of alkaline phosphatase (calcifies bone); severity is variable from pt. to pt; resembles osteogenesis imperfecta; tx = calcitonin;
  → may have blue sclera, deformity of arms, legs, chest or frequent bouts of pneumonia & fractures;
  → Teeth – large pulp, deficient root development & alveolar bone loss; 4 types:
  1. **Neonatal** – severe, resp. failure, hypocalcification of skeleton.
  2. **Infantile** – hypercalcemia, premature loss of prim. teeth, failure to grow.
  3. **Childhood** – short stature, frontal bossing, normal Ca+ & PO_4 levels.
  4. **Odontohypophosphatasia** – children & adults only have dental problems.

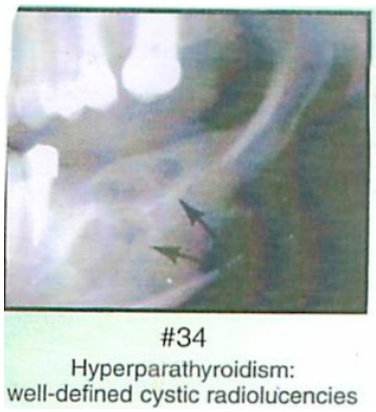

#32
Paget's disease of the skull showing patchy opaque patter
("cotton-wool" appearance)

ENDOCRINE DISORDERS:

- Paget's Disease of Bone (**Osteitis Deformans**) – chronic bone disorder causing enlarged & deformed bones;
  → slow development of symptoms – pain, bone deformity, fractures, headaches, hearing loss;
  → has ↑ alkaline phosphatase levels causing premature loss of teeth & hypocalcification; ↑urine Ca+ & hydroxyproline.
  → bones dense but fragile due to excessive breakdown & formation of bone; more in males; bones warm b/c ↑ vascularity;
  → xrays: skull & jaw display cotton-wool appearance; hypercementosis & loss of lamina dura;
  → pts prone to osteosarcomas; tx = CALCITRONINE which ↓ bone resorption.
- Giant Cell tumor – bone tumor of multinucleated giant cells resembling osteoclasts scattered in matrix of spindle cells; rarely in jaws; in long bones & associated w/ pre-existing Paget's disease in both jaws & long bones;
- Acromegaly – overproduction of growth hormone caused by benign tumor of pituitary gland called ADENOMA;
  → **Gigantism** – tumor prior to adolescense (non-fusion of epiphyses); enlarged tongue, prognathism, teeth tipped B/L; roots longer than normal;
  → **Acromegaly** – tumor after adolescence (fusion of epiphyses);
  → Oral – enlarged tongue, PROGNATHISM, space b/w teeth, longer roots; also HYPERCEMENTOSIS.


#33
Acromegaly: bony changes alter the patient's facial features including the spacing of teeth

- Dwarfism – arrested growth from undersecretion of growth hormone; Oral – delayed eruption & shedding of teeth, small crowns & roots & mandible underdevelopment.
- *Parathyroid hormone* - ↑Ca+ but ↓PO_4 levels in SERUM;
- HyPOparathyroidism - ↓Ca & ↑PO_4 in serum; rare disorder w/ ↓production of PTH & inability to make usuable form of PTH & inability of kidney & bone to respond to PTH; tx = Vit. D.
  → Cause – congenital disorders, iatrogenic causes, infiltration of parathyroid gland, suppression of parathyroid function, HIV, or idiopathic mechanisms;
  → Dental – delayed eruption, enamel hypoplasia (can be tx w/ Vit. D), & bluted roote apices;
  → *Hypocalcemia* – consequence of hypoparathyroidism; Ca+ ↓ to <2.5-3mg/100mL; causes TETANY – positive Chvostek's Sign: twitching of face when taping on facial nerve near parotid;


#34
Hyperparathyroidism:
well-defined cystic radiolucencies

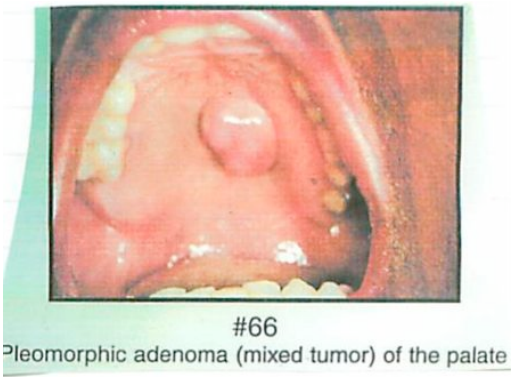
- HyPERparathyroidism – too much PTH so ↑Ca+ release from bone; more in middle-aged females;
  → ↓appetite, ↑thirst & urination, lethargy, muscle weakness, joint pain, & constipation;
  → 1st symptom – pathologic fracture from marked bone resorption; multinucleated giant cells;
  → malocclusion & teeth shifting; X-ray: well-defined cystic RLs, brown tumor of hyperparathyroidism; causes PREMATURE exfoliation of primary teeth;
    1. *Primary* – often >60 yrs; uncontrolled PTH production from parathyroid adenoma or hyperplasia; triad of symptoms – stones, bones, & abdominal groans.
    2. *Secondary* – when PTH continously produced in response to ↓Ca+ from renal failure.
    3. *Tertiary* – after secondary, when external factor corrected but PTH gland remains hyperplastic.
- Muscular Dystrophy – genetic disease marked by progressive weakness & degneration of skeletal or voluntary muscles which control mvmt;
  → dental - ↓OH, weakness in muscles of mastication, ↓biting force, mouth breathing, & open bite.
- HyPERthryoidism – too much thyroid hormone (thyroxine) due to overactivity of thyroid gland; thyroxine's role is to stimulate cellular metabolism, growth, & differentiation of all tissues;
  → ↑basal metabolism, fatigue, weight loss, excitability, ↑temp, & osteoporosis; 2 types:
    1. **Grave's Disease** – diffuse toxic goiter causing overactivity of entire thyroid gland; most common form and more in women >50 yrs; exopthalmos common & thickened skin over shin area;
    2. **Plummer's Disease** – 1/more nodules/lumps in thyroid that cause overactivity; toxic nodular goiter; affects both men & women >50 yrs.; often uni-system; may only present cardiac disease.
- HyPOthyroidism - ↓metabolism; more common than hyperthyroidism; most common cause is **Hashimoto's Thyroiditis** (autoimmune disease); 2nd most common cause is tx of hyperthyroidism;
  → in child = CRETINISM; in adults = MYXEDEMA.
  → Puffy face, eyelids, swelling of tongue & larynx, dry & rough skin, & sparse hair;
  → ↓ BMR & temp, poor muscle tone, low strength, & get tired easily;
  → **Cretinism** - ↓ thyroxine, retarded growht, & abnormal development of bones; improper development of CNS may cause mental retardation;
    - Dental – underdeveloped MAND, overdeveloped MAX, enlarged tongue, delayed eruption & shedding;
- Thyroiditis – inflammation of thyroid gland; 1st hyperthyroidism & then hypothyroism;
  → Hashimoto's thyroiditis is most common hypothyroidism;
  → then **Subacute Granulomatous Thyroiditis**; then **Silent Lymphocitic Thyroiditis**;
- Osteomalacia – bones failing to calcify; softening of bones b/c ↓Vitamin D; complication of steatorrhea secondary to chronic pancreatitis; blood test = ↓Vit. D, ↓Ca+, ↓phosphorous, & ↑alkaline phosphatase.
  → Osteopenia (↓bone density), mottled skull, pseudofractures, bone softening, hourglass thorax, bowing of longbones; ↑ fracture & biconcave vertebral bodies; more in women;
  → All bones involved but mostly epiphyseal growth plates;
- Rickets: osteomalacia of kids due to ↓Vit. D; irritability, muscle weakness, bowlegs, pigeon breast, & protruding stomach; delayed teeth eruption, malocclusion, ↑caries, abnormalities of dentin & enamel;
- Cystic Fibrosis – inherited, congenital metabolic disorder that causes exocrine glands to produce abnormal secretions; most importantly affecting digestive tract & lungs;
  → Inherited a defective copy of CF gene from each parent; affects pancreas, resp. & sweat glands
  → Viscous mucous secretions blocking airways & sweat glands due to faulty ↑Na & Cl transport.
  → **Staining from tetracycline**; mouth breathers, ↑calculus, ↓ caries, & salivary gland enlargement.
  → Symptoms – poor growth, ↑appetite, steatorrhea, BARREL-CHESTED, COPD, recurrent pneumonia, & clubbing of fingers & toes; most diagnostic – SWEAT TEST (shows elevated levels of Na & Cl).
  → Most common inherited disease in white people in the US; reduced rate of caries;

SALIVARY GLAND TUMORS/DISEASES:

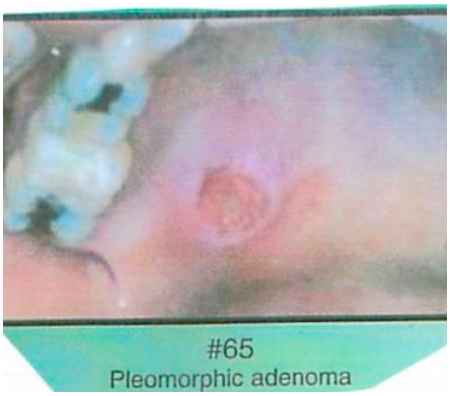
- Uncommon; only 2-4% of H&N tumors; Benign Salivary Gland Tumors (4):

1. Pleomorphic Adenoma – most common benign salivary gland tumor; slow growth w/ few symptoms;

→ Benign mixed tumor; ectodermal & mesenchymal origin.
→ F>M b/w 40-60 yrs; 93% in major salivary glands, 84% in Parotid;
→ Painless lumps below & anterior to ear; tx – excision.
→ 7% are palate w/ firm, swelling, not ulcerated but encapsulated & well-demarcated;
→ cells – round, polyhedral, elongated, or stellate cells which are small & stain uniformly;

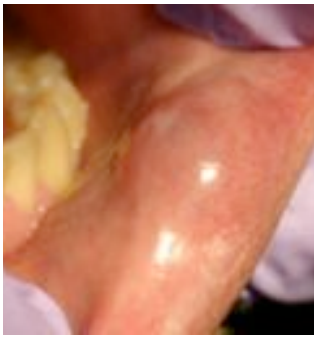

#66
Pleomorphic adenoma (mixed tumor) of the palate

2. Monomorphic Adenoma – group of benign lesions (often parotid gland) w/ variety of growth patterns;
3. Warthin's Tumor (**Papillary Cystadenoma Lymphomatosum**)– slow growing cystic tumor in older men; Parotid tumor; older men;
   → 5% bilateral; arises from heterotrophic ductal epithelium;
   → non-tender, slow growing, firm to fluctuant nodule over angle/ramus of mandible.


#65
Pleomorphic adenoma

4. Benign Lymphoepithelial Lesion (**Mikulicz's Disease**) – wide range of cystic changes but have atypical lymphoid hyperplasia; non-neoplastic; often HIV pts; middle aged women.
   → Progressive, asymptomatic enlargement of parotid & sumandibular glands; unilateral/bilateral;
   → Autoimmune as well as Sjodren's syndrome – both where salivary gland become antigenic.
   → Cells – lymphocytic infilatrate w/in which there are scattered epimyoepthelial islands, which may cause malignant transformation.

- Most common site of intraoral minor salivary gland neoplasms – PALATE.
- Most common site of intraoral major salivary gland neoplasms – PAROTID.
- Clinical features of BENIGN salivary gland tumors – mucosa NORMAL, painless, nodular, moveable, firm & slow-growing;
- Clinical features of MALIGNANT salivary gland tumors – ulcerated, firm, painful, nodular, fixed, & rapid growth; anaplastic (not well-differentiated); **Malignant salivary glands** – 1)Adenocarcinoma, 2) Adenoid, Cystic Carcinoma, 3) Acinic Cell Carcinoma, 4) Mucoepidermoid Carcinoma.
- Mucous Retention Cyst (**Mucocele**) – traumatic origin; 95% are labial mucosa of lower lip; involve minor salivary glands & their ducts; not true cyst;
  → Cells – spilled mucin surrounded by granulation tissue w/ many foamy histiocytes; may be deep or superficial;
  → Raised, circumscribed, bluish, translucent vesicle; several mm - cm in diameter; Deeper lesion appears as fluctuant swelling but tissue colored.

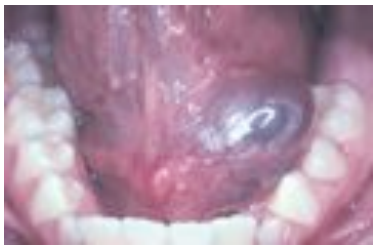


- Necrotizing Sialometaplasia – minor salivary gland lesion caused by necrosis of glandular parenchyma w/ associated squamous metaplasia & hyperplasia of ductal epithelium;
  → adult males; asymptomatic, necrotic, ulcerated area involving palatal mucosa;
  → hard palate – most common site; may stimulate malignancy;
  → tx postponed for 6-12 weeks after biopsy.

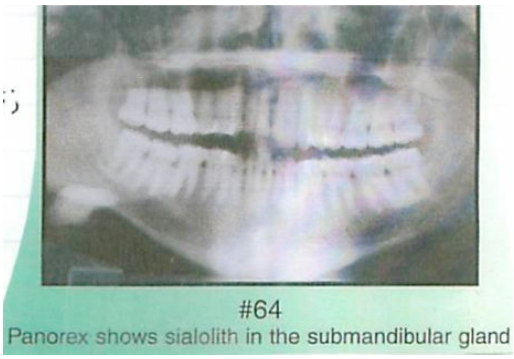

Fig 1 - Necrotizing Sialometaplasia

- Mumps – PARAMYXOVIRUS in same group as parainfluenza virus; aquired by respiratory droplets; replicates in nasopharynx & LNs; 90% < 14 yrs; major sign – sudden salivary gland swelling;
  → **Parotid in 90% of cases** & 2/3rd of cases are bilateral;

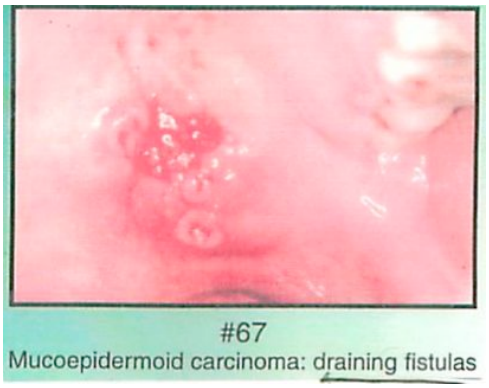
→ Fever, malaise, anorexia; self-limiting;
→ May cause orchitis (testical swelling), epididymitis, sterility, meningitis, enchephalitis, deafness, myocarditis, pancreatitis, oophoritis (inflammation of ovaries), & pyelonephritis (infection of kidneys & ureters);
→ ↑serum amylase; tx – live attenuated vaccine; may still cause non-suppurative salivary adenitis.



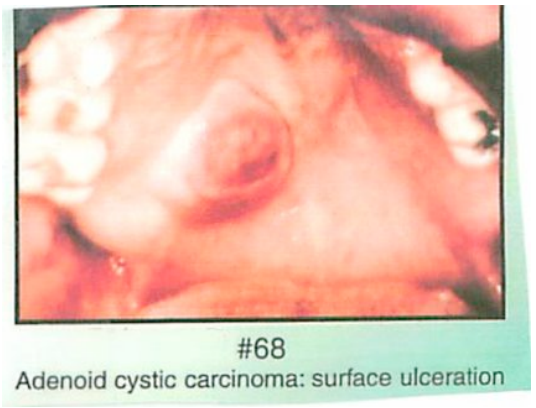
- Ranula (**Retention Cyst**) – floor of the mouth & unilaterally located; arises w/ secretory ducts of submand. & sublingual gland; usually **caused by obstruction**;
  → ↑in size just before/during a meal & ↓in between.
  → Translucent, bluish, well-rounded smooth-surface bulge;
- Sialolith – stone/salivary calculus w/in salivary gland/duct; sialolithiasis occurs from precipitation of Ca+ & PO_4 salts around nidus of mucous & bacterial debris;
  → Single/multiple & can cause swelling & pain.
  → ↑ pain during meals; **most often submand. duct & gland.**
  → Mand. occlusal for xray; tx = surgical extirpation.


#64
Panorex shows sialolith in the submandibular gland

- Salivary Gland Enlargement Diseases – Hypothyroidism, Sjogren's Syndrome, Sarcoidosis, Obsesity, Hypertension, Hyperlipidemia, Warthin's Tumor, Infections, Mikulicz's Disease, Malnutrition;
- Acinic Cell Carcinoma – derived from serous acinar & found exclusively in Parotid gland; less than 10% of salivary gland tumors; low grade malignancies;
- Adenocarcinoma NOS – MALIGNANT MINOR(50%) & MAJOR (50%) salivary glands of nose & paranasal sinuses; may originate from metastases elsewhere in the neck; 25% minor salivary glands, 15% of malignancies of parotid & 10% of malignancies of submand. gland.
- Sjogren's Syndrome – chronic inflammatory disorder of autoimmune nature w/ infiltration of exocrine glands (salivary/lacrimal); mostly post-menapausal women;
  → dry eyes & mouth & 50% have enlarged parotid & submandibular glands;
  → tx – symptoms; malignant lymphomas & pseudolymphomas may develop; most common disease causing XEROSTOMIA;
- Mucoepidermoid Carcinoma – most common malignant salivary gland neoplasm; chiefly parotid; slow growing & painless;
  → poorly encapsulated & often PALATE;
  → high-grade & low-grade forms exist; tx – excision.


#67
Mucoepidermoid carcinoma: draining fistulas

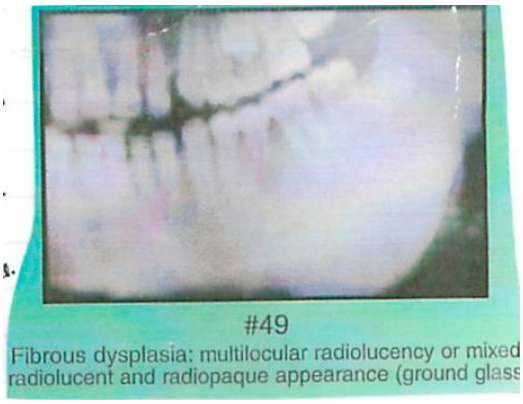
- Adenoid Cystic Carcinoma – most common malignant tumor of minor salivary glands & 23% of all salivary gland tumors; surface ulceration; slow growing but relentless w/ sever pains w/ occasional facial nerve paralysis;
  → Cells – abnormal "nests" or cords of epithelial cells to surround/infiltrate ducts/glandular structures (hyaline membranes);
  → May recur years later but metastasize to lung/liver;
- 8/10 salivary tumors are in PAROTID & mostly benign while sublingual tumors are mostly malignant;
- Malignant Mixed Tumors – 15-20% of parotid & submand. neoplasms; slow, protracted growth;
- Oncocytomas – benign salivary gland tumors (1%); 84% in


#68
Adenoid cystic carcinoma: surface ulceration

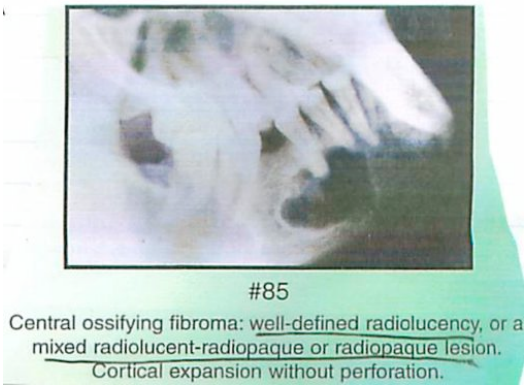
parotid; M=F but 6th decade; large cells w/ bright pink cytoplasm that is granular & eosinophilic due to abundent mitochondria; related to aging process; tx – excision.

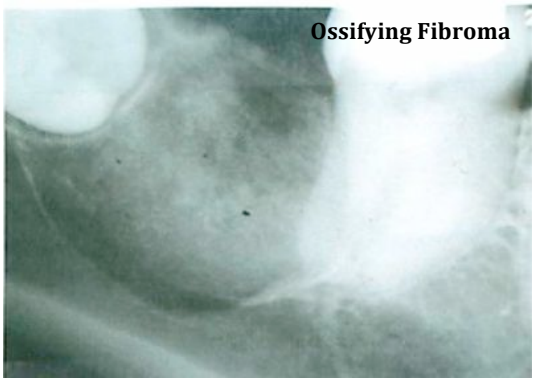
FIBROUS OSSEOUS DISEASES:

- Fibrous Dysplasia – possible malignant transformation; asymptomatic alteration of bone where normal bone replaced by fibrous tissue & non-functional trabeculae-like osseous structures;
  → Yg adults, may involve impacted/unerupted teeth; ↑ alkaline phosphatase;
  → Singular, slow growing, painless swelling; RO, not well-circumscribed & "GROUND GLASS" appearance;
  → 5 types – 1)Monostatic: most COMMON; mostly max.
  2)Polystatic
  3)Cranofacial form: (10-15% of FDs) - monostatic but 50% polystatic.
  4) McCane-Albright's Syndrome - polystatic
  5) Jaffe Syndrome - polystatic
  → both Albright & Jaffe Syndromes have café-au-lait spots & fibrous dysplasia; Albright's most severe form of FD!!


#49
Fibrous dysplasia: multilocular radiolucency or mixed radiolucent and radiopaque appearance (ground glass

- Mandibular Tori – bony exophytic growths lingual of mand. & superior to mylohyoid ridge; exotosis of hard palate = max. tori.
- Central Ossifying Fibroma - benign neoplasm of OSTEOBLASTS; presents as well-demarcated RL to mixed RL/RO mass w/ peripheral rim; post. mand in 90% of cases;
  → F:M = 5:1 at 35 yrs; tx = excision; early symptom – DISPLACEMENT OF TEETH;
  → Clinically similar to central cementifying fibroma (cementoblasts neoplasm)
  → Slow growing & expansive lesion w/ characteristic downward expansion.


#85
Central ossifying fibroma: well-defined radiolucency, or a mixed radiolucent-radiopaque or radiopaque lesion. Cortical expansion without perforation.


Ossifying Fibroma

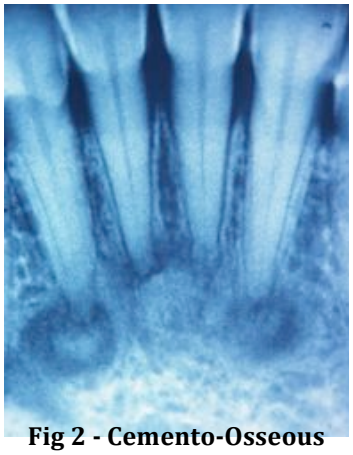
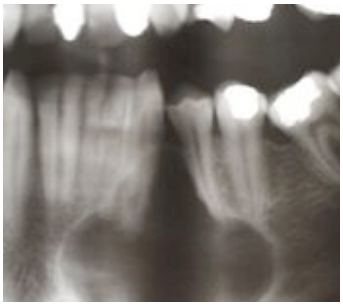


Fig 2 - Cemento-Osseous Dysplasia

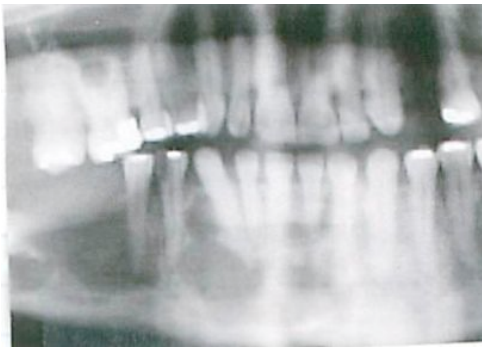
- Cemento-osseous Dysplasia – unknown etiology; disorder of production of bone & cementum-like tissue in tooth areas of jaw; RO/RL well-circumscribed lesions; PA are of mand. ant. teeth.

ODONTGENIC CYSTS:

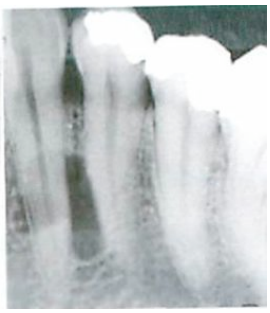
- Odontogenic Keratocyst (OKC) – dentigerous cyst; remnants of dental lamina; cant be distinguished radiographically form periodontal, primordal, or follicular cysts; often associated w/ impacted tooth.
  → more in males and mandible (70-80%); asymptomatic;
  → 10% of cases are nevoid BCC; 50% in mand. thirds area.
  → well-demarcated RL w/ SCALLOPED RO margin; uni/multilocular;
  → **great tendency toward recurrence (30%).**
- Nevoid Basal Cell Carcinoma – autosomal dominant disorder characterized by:



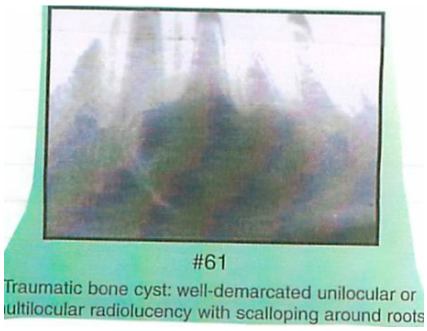
A. Cutaneous anomalies – multiple BCC & other dermal cysts & tumors.
B. Dental & Osseous anomalies – OKC, prognathism, nevus-bifid rib syndrome & verterbral anomalies.
C. Opthalmologic abnormalities – hypertelorism (↑distance between two organs/body parts) w/ wide nasal bridge & blindness.
D. Neurologic anomalies – retardation, dural calcification.
E. Hypogonadism in males & ovarian tumors in females.
F. X-ray – calcification of falx cerebri & multiple OKCs.



- Lateral Periodontal Cyst – inflammatory rxn; well-defined, round/tear-dropped RL; **95% MAND. CUSPID/BICUSPID AREA**; symptomless;
  → gingival cyst is soft tissue counterpart of this lesion.
- Only way to differentiate dental granuloma from radicular cysts (from pre-existing PA granuloma) is histologically;
- ↑ in osmotic pressure in cyst lumen is important in pathogenesis of radicular cyst.
- Dental granuloma is **most common sequelae of pulpitis**!
- Radicular cyst is most common odontogenic cyst; develops w/in pre-existing PA dental granuloma.

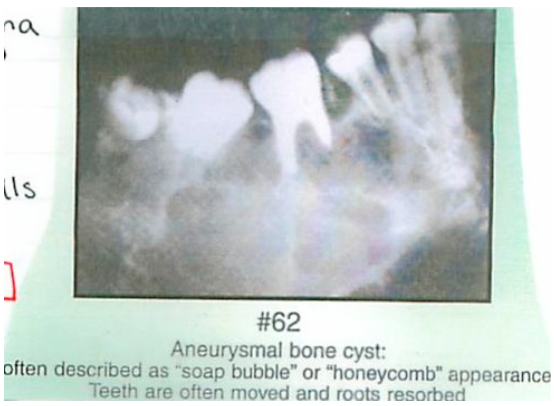


- Dentigerous Cyst – due to unerupted tooth; unilocular RL around unerupted tooth w/ well-defined sclerotic border; often thirds & max. canines! Attaches to tooth's CEJ; 2nd most common odontogenic cyst.
- Eruption Cyst – soft tissue analogue of dentigerous cyst.
- Primordal Cyst – follicular cyst w/ no calcified structures & lined w/ stratified squamous epithelium from enamel organ; often thirds!



PSEUDOCYSTS = (no epithelial lining):

- Traumatic Bone Cyst – intramedullary hemorrhage: blood clot liquefies & leaves empty space; asymptomatic intraosseous empty cavity;
  → due to trauma; often young pts;
  → located **primarily w/in posterior mand**. lined by thin loose CT membrane;
  → vital teeth; well-defined uni/multilocular RL w/ scalloping around roots;
- Aneurysmal Bone Cyst – often humerus, femur, tibia, or pelvis; M=F; uncommon lesion in post. max/mand (more often mand) & similar to central GCG;
  → no epithelial lining so pseudocyst;
  → fibrous CT stroma w/ fibroblasts, macrophages, & multi-nucleated giant cells;
  → X-ray – "HONEY-COMB"/ "SOAP-BUBBLE" appearance;
  → teeth often moved & roots resorbed.



NON-ODONTOGENIC CYSTS:

- Developmental Cysts (**Fissural Cysts**)– Nasopalatine, Nasolabial, Median Palatal, Median Alveolar, & Globulomaxillary.
- Nasopalatine Developmental Cyst – most common of max. developmental cysts; most frequent non-odontogenic cyst; asymptomatic but may produce elevation in palate; tx = enucleation;

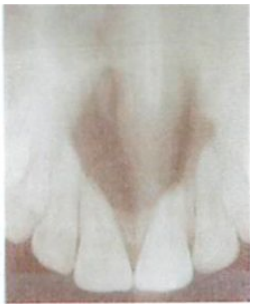


Fig - 2 - Nasopalatine Cyst

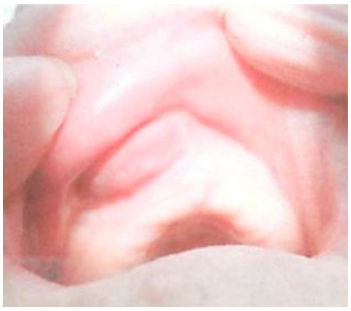
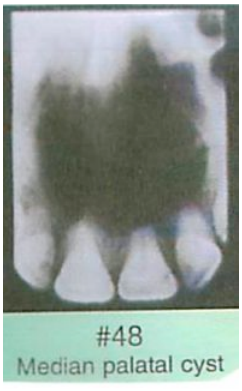
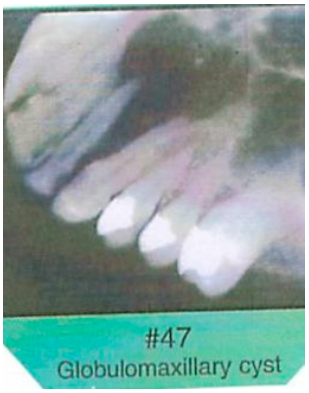


Fig - 1 - Nasolabial Cyst

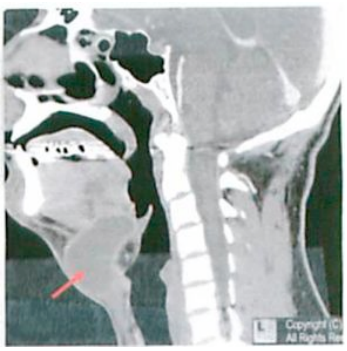
→ heart-shaped RL in midline b/w max. central incisors; teeth vital;

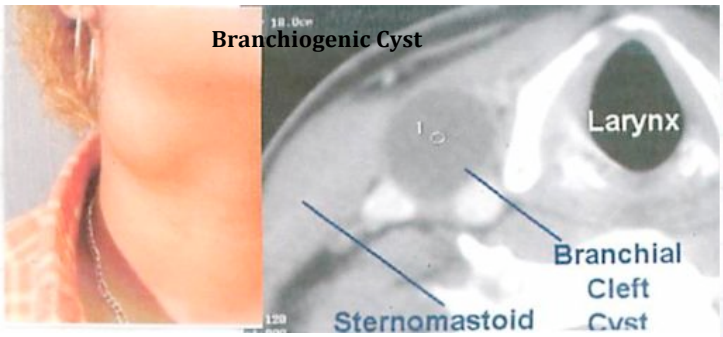
- Nasolabial Cyst – located in soft tissue of upper lip; CANT be seen on x-ray so extraosseous cyst;
  → Epithelial remnants from inferior & ant. portion of nasolacrimal duct;
  → Soft tissue cyst just beolow or inside nostril; not visible but may produce "cupping" of underlying bone;
- Median Palatal – rare; may produce swelling on palate; distinguish b/w other PA RLs by teeth adjacent are vital; most posterior presentation of nasopalatine cyst.
  → epithelial remnants in line of fusion b/w palatine processes; Soft fluctuant/crepitant swelling in midline of hard palate; May occur anywhere along median palate; posterior to premaxilla.

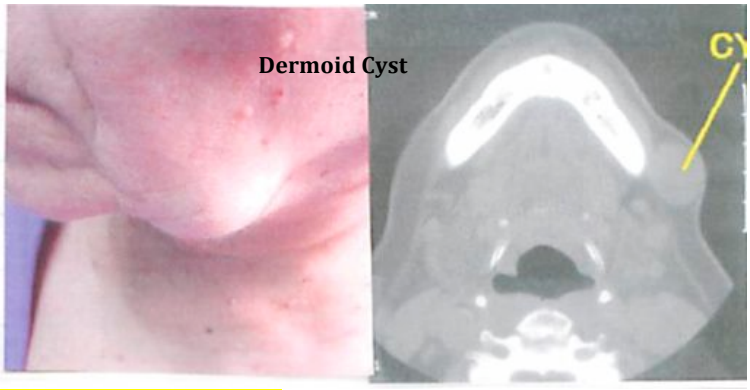

#48
Median palatal cyst


#47
Globulomaxillary cyst

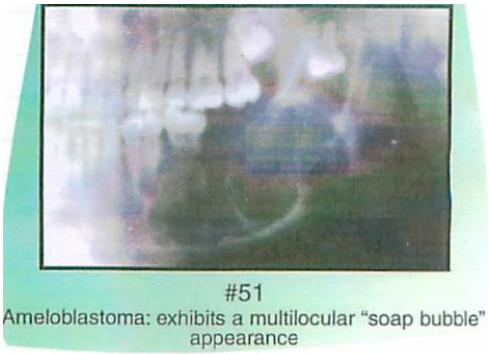
- Median Alveolar Cyst – rare & in bony alveolus **b/w central incisors**; adjacent teeth vital.
- Globulomaxillary Cyst – developmental/fissural cyst; **over 80% of lesion are PA origin**; Appears b/w roots of lateral incisor & canines;
  → "pear-shaped" causing roots to diverge; Asymptomatic & vital teeth but often associated w/ non-vital lateral incisors;
- Soft tissue variant of nasopalatine canal cyst is cyst of palatine papilla.
- **Congenital Cysts:**
  1. Thyroglossal Duct Cyst – may arise from any portion of thyroglossal duct (from foramen cecum to thyroid gland); midline position & vascular resembling hemangioma; hemmorhage into mouth;
     a. Midline of neck/to one side, floor of mouth, tongue, near thyroid cartilage.
  2. Branchiogenic Cyst – transformation of salivary gland tissue from 2nd branchial arch cleft; lined w/ ciliated & stratified squamous epithelium; milky & mucoid fluid;
     a. Freely moveable, well-circumscribed mass; **anterior border of SCM muscle.**
  3. Dermoid Cyst – epithelium trapped in CT during embryogenesis; uncommon in oral cavity;







contains hair, sebaceous & sweat glands as well as tooth structure;
  a. most common site – **floor of the mouth**; then submandibular & sublingual areas.
  b. well-circumscribed, compressible soft tissue enlargement;

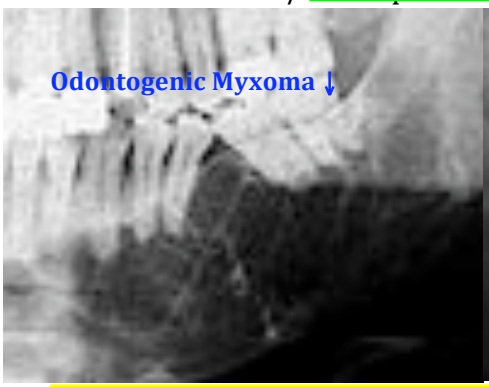

#51
Ameloblastoma: exhibits a multilocular "soap bubble" appearance

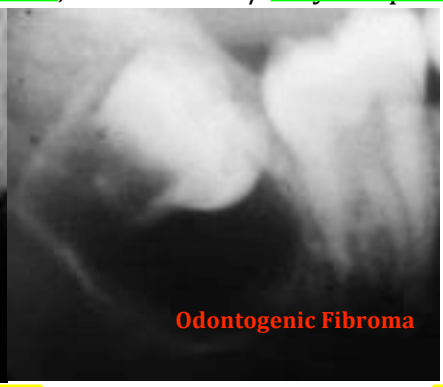
ODONTGENIC TUMORS:

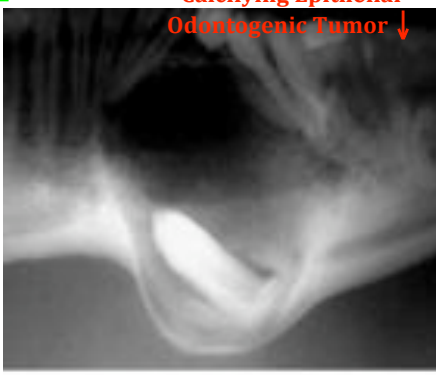
- Ameloblastoma – ENTIRELY odontogenic epithelium ; adolescents in mand. molar area; most aggressive & most common odontogenic epithelial tumor; many cases, mand. 3rds associated w/ RL.
  → Avg. age = 34; slow growing & painless swelling; capable of large facial deformities; RECURRENCE common;

→ Xray – uni/multi-locular; "SOAP-BUBBLE" appearance RL; vital teeth; similar to central giant cell granuloma; **most likely to develop on wall of dentigerous cyst**; 3 subtypes:
   a. Solid (polycystic) – more aggressive form; 86% of cases; surgical excision for tx.
   b. Extraosseous (peripheral) – least common form;
   c. Unicystic – most common in younger pts; enucleation for tx.

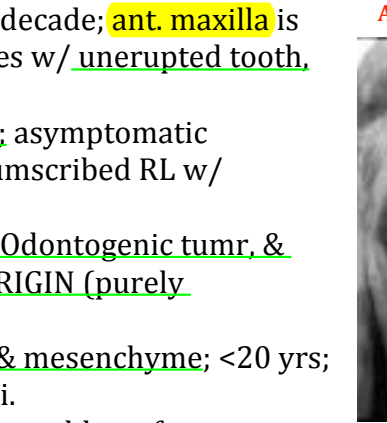
- Odontogenic Myxoma – rare jaw tumor that arised from connective tissue resembling pulp; from sac, papilla, & PDL so MESENCHYMAL ORIGIN; pts < 35 yrs; often POST. MAND.
  → slow growing & asymptomatic; "honey-comb" or multi-locular RL sometimes w/ unerupted teeth;
  → localized expansion of jaw; tx – curretage; recurrence is common;
- Odontogenic Fibroma – tumor of mesenchymal origin from either PDL, dental sac, or papilla; multilocular RL & sometimes w/ unerupted teeth; <20yrs and often in mand. molar region.
- Calcifying Epithelial Odontogenic Tumor (**Pindborg Tumor**)– polyhedral, neoplastic, & epithelial cells; during 4th decade; **mostly mand. molar area**;
  → RL-RO area w/ unerupted tooth; associated w/ amyloid production.

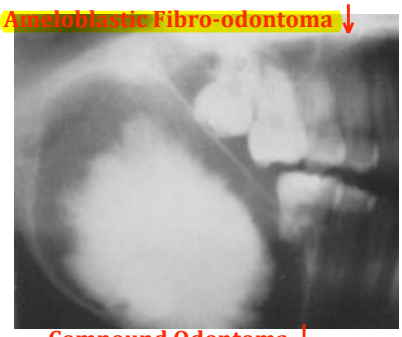


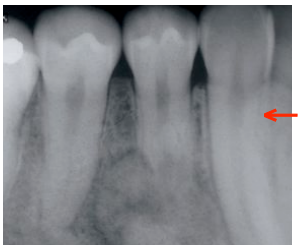


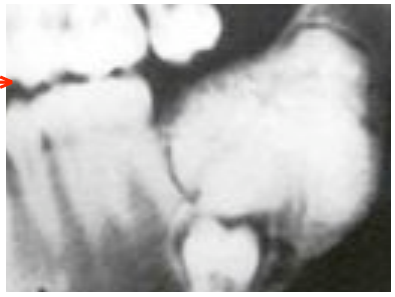


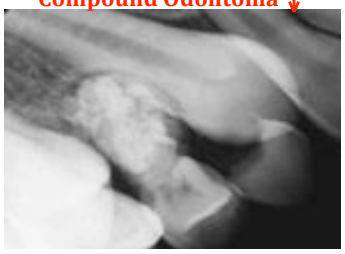
- Adenomatoid Odontogenic Tumor – enamel organ; 2nd decade; ant. maxilla is most common; unilocular RL w/ tiny RO foci; sometimes w/ unerupted tooth, often CANINES; tx = enucleation.
- Squamous Odontogenic Tumor – from rest of malassez; asymptomatic /painless swelling; often mobile teeth; triangular/circumscribed RL w/ unerupted/erupted teeth;
- Calcifying Epithelial odontogenic tumor, Adenomatoid Odontogenic tumr, & Squamous Odontogenic tumor are all ECTODERMAL ORIGIN (purely epithelial!).
- Ameloblastic Fibro-odontoma – neoplastic epithelium & mesenchyme; <20 yrs; max = mand; well-defined RL but may also have RO foci.
- Benign Cementoblastoma – benign neoplasm from cementoblasts from PDL; males<25 yrs;
  → mand. 1st PM region; vital tooth but usually solitary;
  → attached to tooth root; tx = TE tooth!
  → well-demarcated, mottled/densely RO mass w/ RL peripheray;
- Ameloblastic Odontoma – ectodermal/mesenchymal components of tooth germ; causes delayed eruption; <20yrs;
  → max & mand. bicuspid/molar regions;
  → well-defined RL w/ RO foci; may recur;









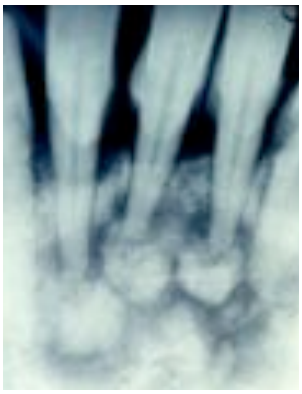


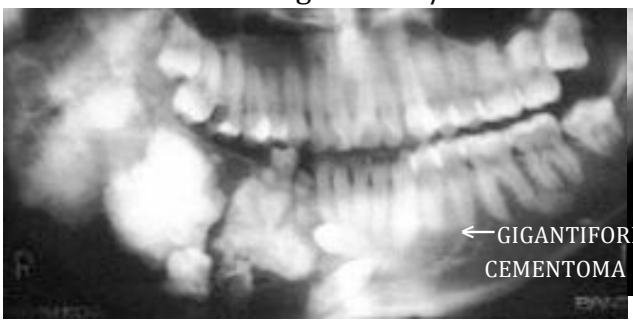
2 types:

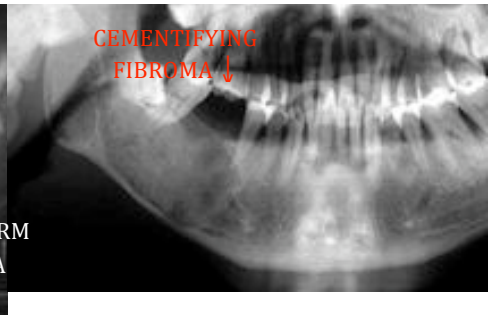
1. **Complex Odontoma** – well-defined RO mass of dental tissue surrounded by RL zone; mand. bicuspid/molar area.
2. **Compound Odontoma** – looks like a tooth; max. incisor/cuspid area.

- Gigantiform Cementoma – from PDL; common in middle-aged black women;
  → multiple & symmetrical; tx = excision
  → large, dense & often lobulated RO mass;
- Cementifying Fibroma – from PDL; adult, mand. swelling; well-defined RL w/ scattered RO foci;
- Cementoma – from PDL; common in middle aged black women; mand. incisor region; small, sharply circumscribed RO attached to or adjacent to apices of teeth; reactive & not neoplastic; actually arises from bone NOT teeth; vital teeth; 3 stages:
  1. Osteolytic – RL
  2. Cemtoblastic – begins calcification of RL area
  3. Mature Stage – RO w/ thin RL line around teeth.

Cementoma ↓

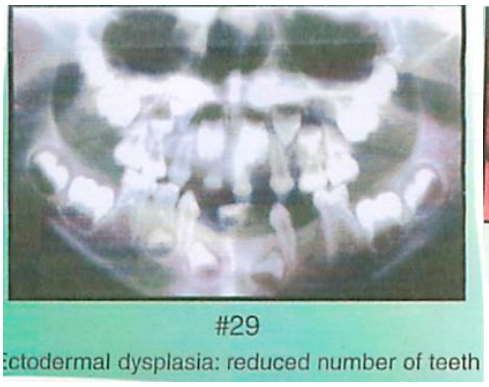


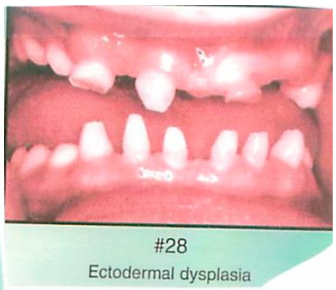


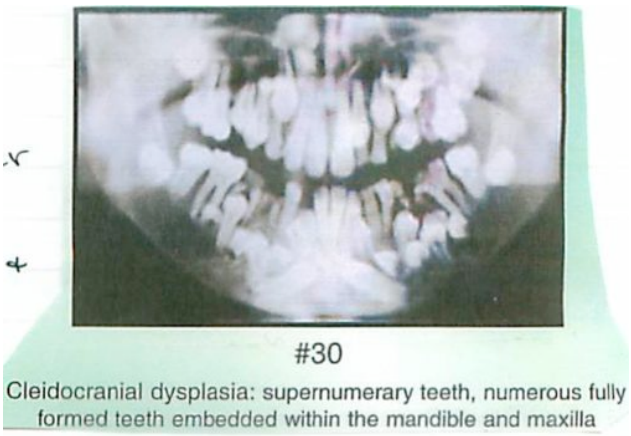


GENETIC JAW DISORDERS:

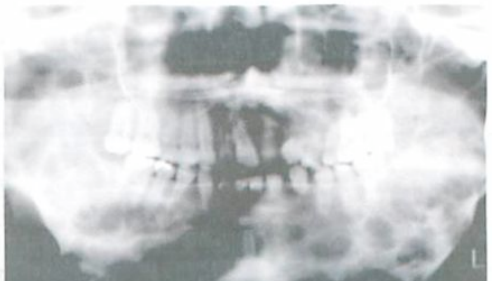
- Ectodermal Dysplasia – *X-linked recessive* (so ONLY in males) condition of abnormal growth of skin, hair, nails, teeth (cone-shaped) & sweat glands; all structures from ECTODERM; no tx;
  → *Hypotrichosis* (↓hair), *Anhidrosis* (↓sweat glands), anodontia/ oligodontia, depressed bridge of nose, lack of salivary glands, & child appears older; **conical-shaped anterior teeth**;
- Cleidocranial Dysplasia – *autosomal dominant* disorder of abscent or incompletely formed collar bones; protruding jaw, wide nasal bridge; **peg-shaped teeth**;
  → Dental - retained prim. teeth, malaligned teeth, multiple supernumerary teeth & unerupted teeth.


#29
ctodermal dysplasia: reduced number of teeth


#28
Ectodermal dysplasia

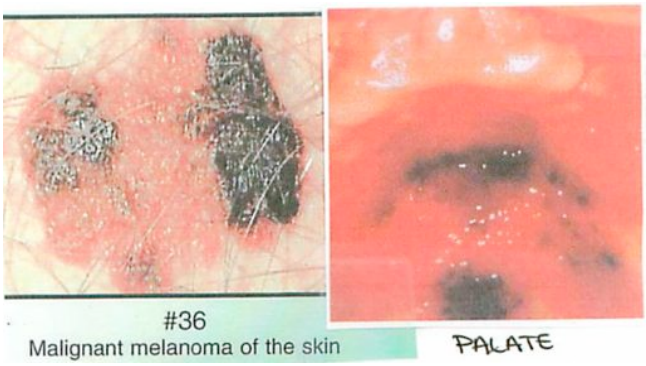

#30
Cleidocranial dysplasia: supernumerary teeth, numerous fully formed teeth embedded within the mandible and maxilla

- Pierre Robin Syndrome – neonatal inherited disorder; collagen gene 2A1 mutation; causes micro & retrognathic mandible, glossotosis, & cleft palate; also resp. problems;
- Cherubism – benign autosomal dominant disorder of max & mand (most often); often in children around 5 yrs; males 2:1; ↑ age = ↓ in bone deformity noticeability.

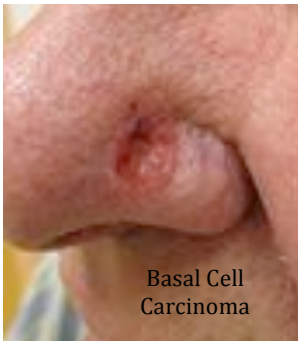


→ Jaw firm & hard to palpate & lymphadenopathy; no systemic problems; Premature shedding of prim. teeth but delated eruption of perm. teeth;
→ X-ray – multiple, well-defined multilocular RLs of the jaw;
→ Histologically – multinucleated giant cells w/ ovoid/spindle shaped cells; perivascular collagen cuffing; numerous small vascular spaces w/ large endothelial cells; similar to central giant cell granuloma, hyperparathyroidism & aneurysmal bone cyst.

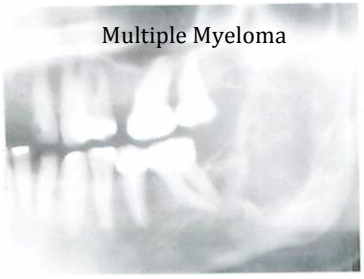
MALIGNANCIES OF THE JAW:



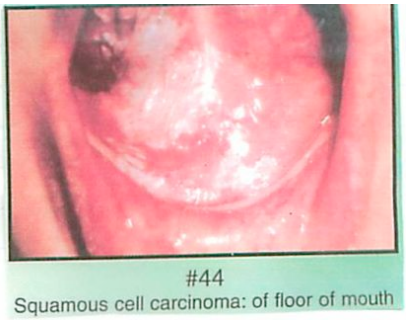
- Malignant Melanoma – 4% of all skin cancers but greatest skin cancer deaths; spreads very rapidly; melanocytes & melanin; more in males >50 yrs;
  → ABCD's – A = asymmetry, B = border irregularity, C = color variabilty, D = diameter > ¼ inch.
  → 5% survival rate is 7%
  → **usually palate or max. gingiva** but uncommon oral neoplasm;
  → Dysplastic (atypical) Nevi – larger than normal moles, flat & irregular, various shades, greater risk of malignant melanoma;
  → Tumor either RADIAL growth (initial growth in horizontal plane; NOT metastasized) or VERTICAL growth (metastasized); *4 types of MM:*
    1. *Superficial Spreading* – most common MM; most common in whites; radial growth occurs which is best prognosis(100% cure rate w/ surgery);
    2. *Nodular* – raised dark blackish/bluish red lesion; vertical growth which is poorest prognosis;
    3. *Lentigo Maligna* – elderly; develops from pre-existing lentigo naligna (Hutchinson freckles); **radial & vertical growth**.
    4. *Acral Lentiginous* – least common; palms, soles, under nails; common in african americans; **radial growth**.



- Basal Cell Carcinoma – malignant epithelial cell tumor characteristically begins as papule & enlargess peripherally as central crater erodes, crusts, & bleeds; metastasis is rare; tx = eradication of lesion; most common cancer.
  → Primary cause is excessive exposure to sun & xrays; more men > 40yrs; never in MOUTH.
  → Located primarily on H&N w/ nose being most common site.

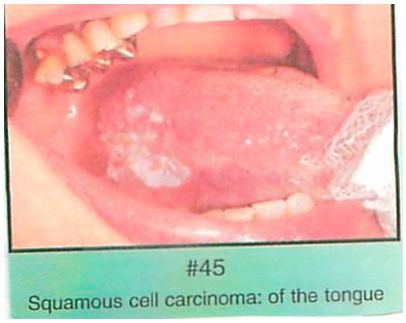


- Multiple Myeloma – PLASMA CELL MYELOMA; malignant neoplasm of bone characterized by progressive destruction of marrow w/ replacement of neoplastic plasma cells; men 2:1 & 40-70 yrs;
  → vertebra, ribs, & skull, pain in lumbar & thoracic region;
  → **molar-ramus area is most common intraoral site** w/ swelling pain, loosening of teeth & paresthesia; jaw is rarely PRIMARY site.
  → multiple, small, discreet "PUNCHED-OUT" RLs; use Lateral skull X-ray;
  → poor prognosis w/ 2-3 yrs; most likely FATAL.
  → hypergammaglobulinemia (↑IgG) & ↑Bence Jones Proteinuria in 60-85% of cases;
  → **Plasmacytoma** – localized collection of monoclonal plasma cells; split into primary or extramedullary; potential to progress to multiple myeloma.

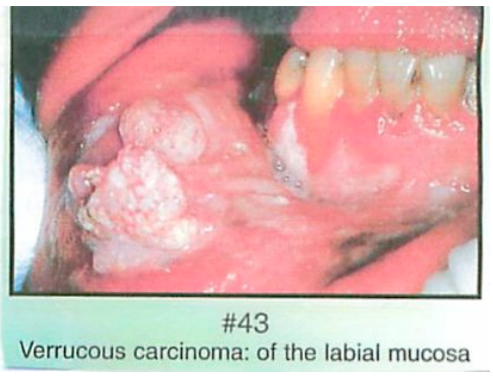


- SCC – tobacco is primary risk factor; >90% of oral cavity malignancies; 2nd most common cancer; most common sites:

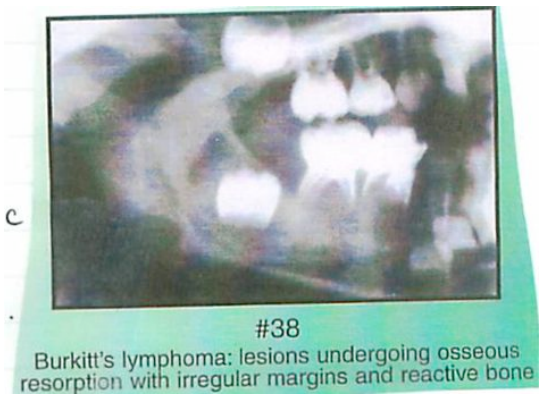
1. **Lip** = 25-30% (90% on lower lip)
2. **Lateral & ventral border of tongue**
3. **Floor of the Mouth** near orifices of salivary glands; poor prognosis b/c premalignant lesions often seen here.
   - Least common to most – Nasopharynx < Palate < Oropharynx < Max. Sinus; **most common malignancy in oral cavity!**

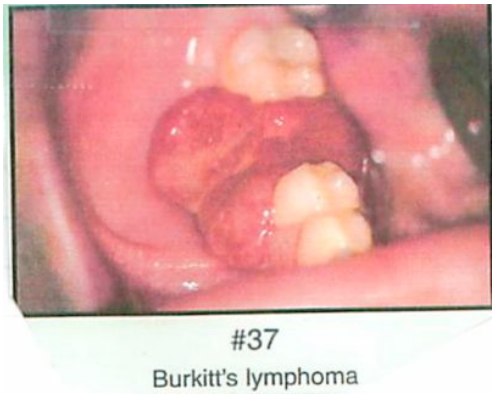

#45
Squamous cell carcinoma: of the tongue

- Verrucous Carcinoma (**Snuff Dipper's Cancer**) – well-differentiated squamous cell neoplasm of soft tissue of oral/laryngeal cavity; rarely metastasizes; often misdiagnosed as benign; males > females;
  → Distinct, diffuse, papillary, superficial non-metastasizing form of well-differentiated SSC;
  → Diffuse, white, cauliflower mass, mand. mucobuccal fold, alveolar mucosa & palate; Slow growing but painless.


#43
Verrucous carcinoma: of the labial mucosa

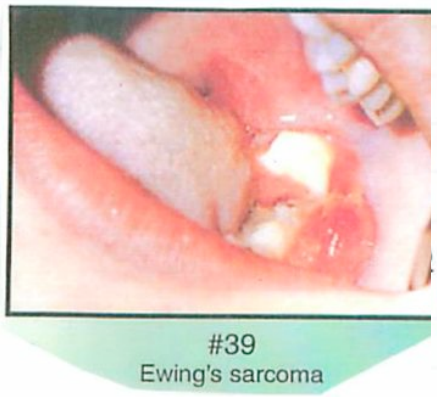
- Lymphoepithelioma – poorly differentiated SSC involving lymph of tonsils & nasopharynx; often young adults of east asia; very small & often hidden; 30% have 5 yr. survival rate;
  → Cells (squamous/undifferentiated) w/ slight to moderate amts of fibrous stroma w/ numerous lymphocytes; metastasis at early stage to cervical LNs;
  → Swelling of LN, sore throat, nasal obstruction, bloody nose, & headache;
- Burkitt's Lymphoma – high grade non-Hodgkin's Lymphoma; endemic in africa; large osteolytic lesion in jaw (african form – males, 3yrs) or abdominal mass (non-african form – M/F, 11 yrs);
  → Both forms histologically identical.
  → Jaw lesion – expanding masses on palate & gingiva, nodular, & hemorrhagic.
  → X-ray – moth-eaten, poorly destruction of bone.


#38
Burkitt's lymphoma: lesions undergoing osseous resorption with irregular margins and reactive bone

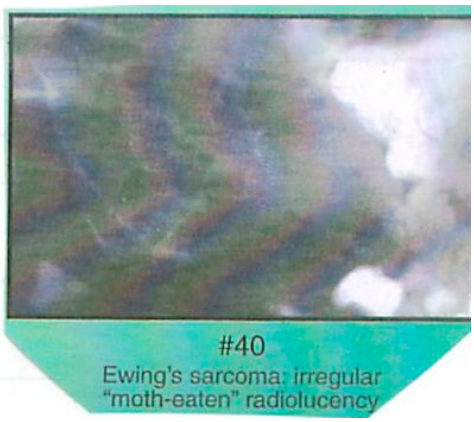

#37
Burkitt's lymphoma

  → **Epstein-Bar Virus** (*herpes-type virus*) – isolated from cultures of tumor cell & pts. w/ Burkitt's lymphoma have high titers of antibodies against EBV; involved in **Burkitt's Lymphoma** & **Nasopharyngeal carcinoma**; associated w/ infectious mononucleosis & oral hairly leukoplakia.
    - Infects B-lymphocytes & some epithelial cells; produces atypical lymphocytes & IgM heterophil antibodies in 80% of mono pts; causes **HSV-4**!!!!
  → **Hodgkin's Lymphoma** – malignant growth of cells in lymph; most common symptom is painless swelling of LN in neck, underarm, or groin.
- Ewing's Sarcoma – uncommon, highly lethal, malignant neoplasm of bone of uncertain origin; pelvis, thigh, & trunk involved; ages 10-20;
  → earliest S&S is pain & swelling; fever, leukocytosis, ↑ESR & anemia;
  → cells contain glycogen;

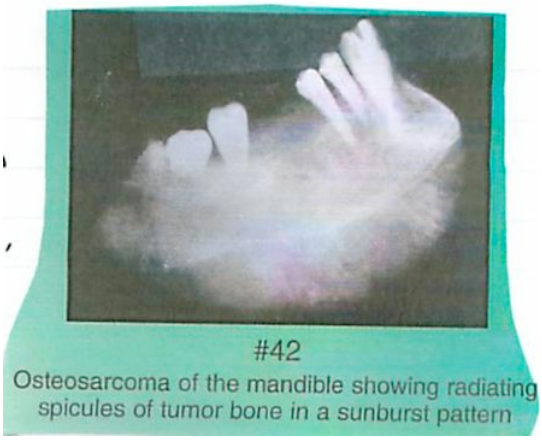
→ involves ramus of mand. w/ rapid swelling, loosening teeth, MOTH-EATEN RLs of medulla & cortex; variable periosteal "onion-skin" rxns;



#39
Ewing's sarcoma

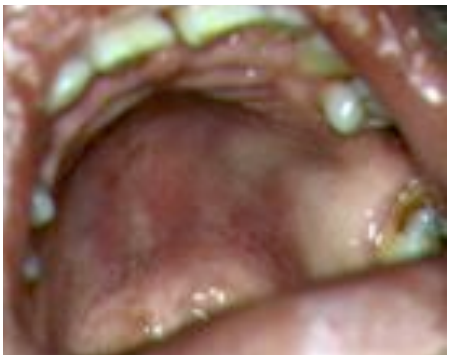


#40
Ewing's sarcoma: irregular "moth-eaten" radiolucency



#42
Osteosarcoma of the mandible showing radiating spicules of tumor bone in a sunburst pattern

- Osteosarcoma (Osteogenic sarcoma) – **most common malignant neoplasm from bone cells in jaw;** atypical osteoblasts or abnormal bone or osteoid formation;
  → Mostly men 30-40 yrs; WIDENING of PDL;
  → Firm, rapid growing swelling & pain w/ loosening of teeth & paresthesia;
  → Classified by dominant tissue (osteoblastic, chondroblastic, or fibroblastic) or site of origin.
  → X-ray – sclerotic (excessive bone production), SUN-RAY/SUNBURST appearance, lytic (irregular RL), mixed (both sclerotic & lytic) – most osteosarcomas;
- Kaposi's Sarcoma – form of angiosarcoma (blood vessel); most common cancer of AIDS pts; **malignant** neoplasm; possible herpes virus 8 related; NEGATIVE pressure test! (hemangioma is positive).
  → Abnormal vascular proliferation (angiosarcoma).
  → Initally small, red papules which enlarge & fuse to form purple to brown, spongy nodules; spreads to LNs & organs;
  → most common site – Palate, then gingiva, then BM;
  → 4 clinic presentations – Classic, Endemic (african), Immunosuppression, & AIDS related.

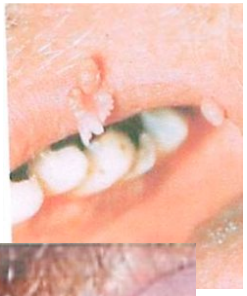


- Most to least common OSSEOUS malignances:
  → Osteosarcoma >Chondrosarcoma >Fibrosarcoma >Ewing's Sarcoma.
- TNM method of assessing prognosis & therapy of malignant neoplasms:
  1. T = size of primary tumor
  2. N = presence of lymph node involvment
  3. M = presence of distant metastasis

Tx	tumor can't be assessed.	Nx	LNs can't be assessed.	Mx	metastasis can't be assessed.
To	no tumor	No	no palpable LNs	Mo	no metastasis.
Tis	carcinoma in situ	N1	palpable LN, not fixed (hemolateral)	M1	evidence of metastasis.
T1	<2cm in diameter	N2	palpable central/bilateral LN, not fixed		
T2	2-4 cm in diameter	N3	palpable & fixed LNs		
T3	>4cm in diameter				

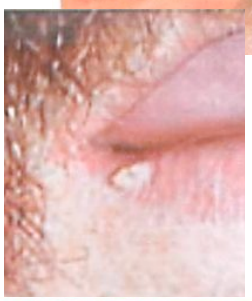
- **Vertical growth phase** – neoplastic cells populate under dermis; ↑ in size, change in color, nodularity, & ulceration; metastasis possible at this point; cure rate of 70%
- Cancers of the tongue cause more deaths than any other area of the H & N b/c its richly endowed w/ lymphatic & blood vessels; SSC of tongue metastasizes to cervical LN;
- Cancers of the buccal mucosa occurs along plane of occlusion.
- Cancer of the gingiva – mand > max & post > ant.

- Most common malignancy of skeletal bones – **METASTATIC CARCINOMA**(malignant cancer that arise from epithelial cells); metastases of jaw commonly originate from primary carcinomas of breast, kidney, lungs, colon, prostate, & thyroid; least common – brain.
- The lung is the most common source of metastases to the oral soft tissues, whereas the breast is the most common source for metastatic tumors to the jawbones. Primary carcinoma metastasising most frequently to the jaws are from breast (33%), thyroid (18%), kidney (16%), prostate in males (6%) and colon (6%).
- Features of metastatic jaw lesion:
  --asymptomatic
  --parasthesia/anesthesia of lip/chin
  --loose teeth; much more MAND. than max.
  --swelling/expansion of jaw
  --asymptomatic RLs

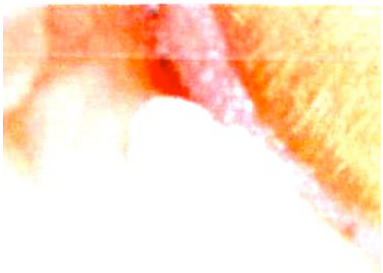


VERRUCAL PAPILLARY LESIONS:

- Papilloma – most common benign neoplasm of epithelial tissue origin; benign exophytic papillary growth of stratified squamous epithelium; MUST DO SURGERY.
  → similar to common wart (verruca vulgaris); anywhere on oral mucosa;
  → cauliflower-like; long duration; considerable keratin so white color; VIRAL.



- Verruca Vulgaris (common wart) – caused by HPV 2, 4, or 40; 79% in skin but also acral sites; **virus** remains latent in skin; tx – remove/excise;
  → can last 6 wks-1yr; skin, lips, palate; sessile, soft, cauliflower-like lesion;
  → autoinoculation is possible; NOT pedunculated (foot-shaped), like papillomas.
- Verruciform Xanthoma – verruciform means pointy projections/warty; papilloma like lesion; middle-ages & M:F = 1:2; gingiva or alveolar mucosa;
  → Well-demarcated, soft, painless, sessile, elevated w/ white, yellow, or red color & papillary/roughened surface; <2cm; **HISTIOCYTOSIS Y**;
  → Hyperplastic condition w/ lipid-laden histiocytes;



- Inflammatory Papillary Hyperplasia – hard palate; poor OH & ill-fitting dentures maybe cause; tx = excision;
  → AKA **Palatal Papillomatosis/Denture Papillomatosis**;
  → Reactive lesion from dentures, due to CANDIDA;
  → erythematous mucosa w/ numerous red papillary projections;
- Inflammatory Fibrous Hyperplasia (**Epulis Fissuratum**) – reactive from bad dentures; Common on denture borders; tx = excision;
  → folds of hyperplastic tissue in alveolar ridge; elongated, firm, and ulcerations;

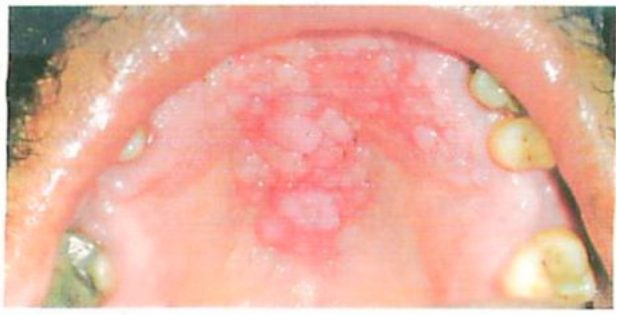


Fig - 4 - Inflammatory Papillary Hyperplasia

Fig - 3 - Inflammatory Fibrous Hyperplasia

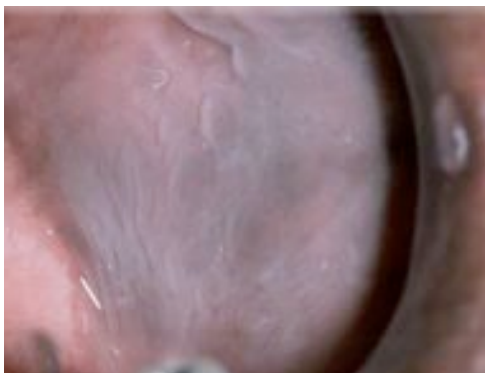
NERVE & MUSCLE DISORDERS:

- Bell's Palsy – disproportionately attacks pregnant women, diabetes, influenza, or upper resp. ailments; after sudden onset, usually subsides w/in 2-3 wks in over 85% of pts;
  → Can occur if hit Parotid gland during IA.

→ Triggers: otitis media (inflammation of middle ear), pressure change, exposure to cold, ischemia of CN VII (restricts blood supply to CN), **Melkersson-Rosenthal Syndrome**, & MS.
→ **Melkersson-Rosenthal Syndrome –** when cheilitis (inflammation of lip) occurs w/ facial palsy & plicated/fissured tongue.

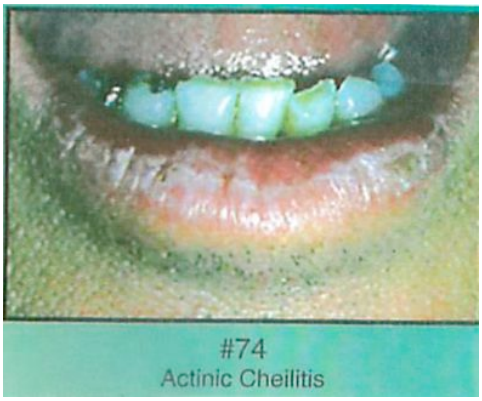
- Myofascial Pain-Dysfunction Syndrome(MPDS) – **initiated by muscle spasm**; women 20-40 yrs; mostly UNILATERAL; 4 signs - pain, muscle tenderness (often *lateral pterygoid*), TMJ clicking, limited jaw mvmt; arise in 3 ways:
    1. Muscular Overextension.
    2. Muscular Overcontraction
    3. Muscle Fatigue (most common)
- Glossopharyngeal Neuralgia – pain arises from CN IX; pain described as sharp, jabbing, electric, or shock-like pain located in deep throat; mostly UNILATERAL & triggered by swalling/chewing;
- Frey's Syndrome (**Auriculotemporal syndrome**) – not common; damage to auriculotemporal nerve & reinnervation of sweat glands by parasympathetic salivary fibers;
    → Chief symptom – GUSTATORY SWEATING – flush/sweating on side of face while eating;
    → May occur post-surgery of parotid tumor removal, mand. ramus or infection damaging auriculotemporal nerve (V3).
- Postherpetic Neuralgia – persistant burning, aching, itching, & hyperesthesia (↑sensitivity) w/ cutaneous nerve following attack of herpes zoster; may be few weeks to many months;
    → Involvement of CN VII & geniculate ganglion produces **Ramsey Hunt Sydrome** – facial paralysis + otalgia (earache).
- Myasthenia Gravis – AUTOIMMUNE disorder which antibodies form against Ach nicotinic postsynaptic receptors @ myoneural junction; so blocks Ach receptors; signs:
    → Flat smile & droopy eyes
    → Slow papillary light response & double vision
    → **Xerostomia & rampant caries**
    → Difficulty chewing & swallowing.
- Multiple Sclerosis – chronic, disabling AUTOIMMUNE disease attacking CNS; **demyelination**; more women 20-40 yrs; tingling, numbness to paralysis, blindness, facial & jaw weakness;
    → More frequently develop Bell's Palsy & Trigeminal Neuralgia;
- Trigeminal Neuralgia – sudden stab-like pains accompanied by brief facial spasm/tic; short but excruciating pains; provoked by touching "trigger zones";
    → degeneration of CN V or pressure applied to it; affects CN V unilaterally & sensory distribution;
    → spontaneous remission lasts 6 months during early phase of disease.
    → Tx = **Carbamazepine** (Tegretol) – analgesic & anticonvulsant;

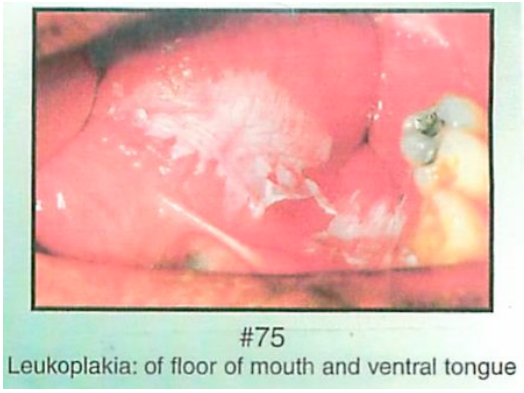
WHITE ORAL LESIONS:

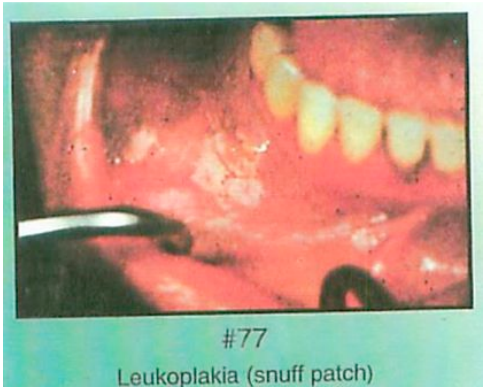


- Leukoedema – filmy opalescence of mucosa to definite gray-white cast w/ coarsely wrinkled surface; bilatera & common occlusal line of PMs & molars;
    → Disappears when applying clinical stretch test; no tx needed.
    → Epithelium parakeratotic & acanthotic w/ entracellular edema of spinus cells;
    → Water creates white appearance.
- Actinic Cheilitis – chronic & excessive exposure to UV radiation; causes irreversible damage to lower lip; variant of ORAL LEUKOPLAKIA; loss of elasticity & definition of vermillion border; more men b/w 30-40 yrs; malignant change can manifest & lead to SCC;

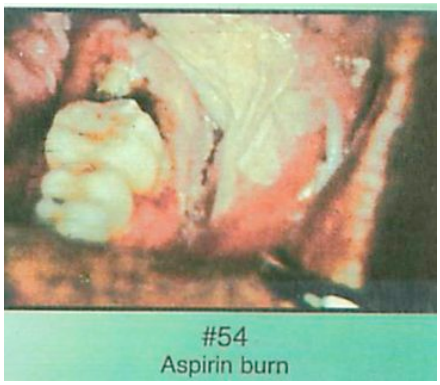
→ lips appear dry, mottled, & opalescent w/ elevated white/gray plaques that cant be rubbed off;

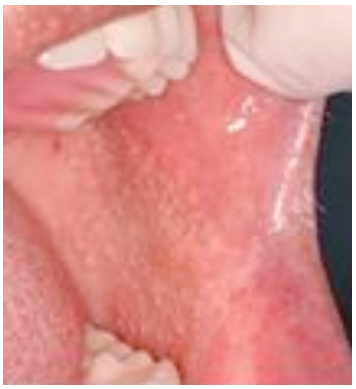

#74
Actinic Cheilitis

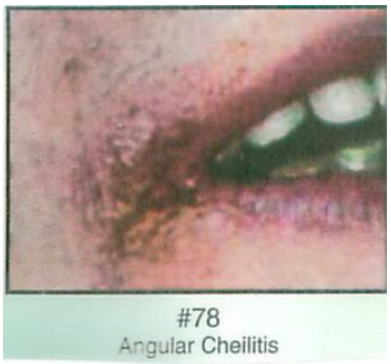

#75
Leukoplakia: of floor of mouth and ventral tongue


#77
Leukoplakia (snuff patch)

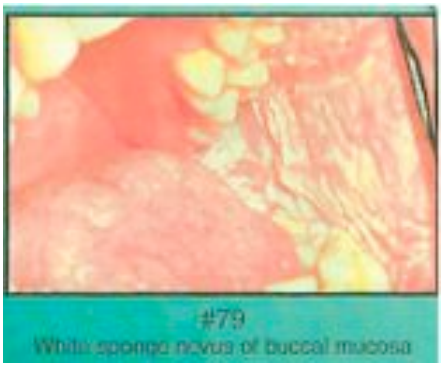
- Leukoplakia – premalignant lesion; **all leukoplakia's should be biopsied**; slow change in mucosa characterized by thickened, white, firmly attached patches that are slightly raised & sharply circumscribed; older men more b/c often caused by tobacco, esp. pipes;
  → floor of the mouth & base of tongue most aggressive; tx – complete excision; erythroplakia has greater potential for malignancy.
- Aspirin Burn – placing tablet against tooth ache; caused buring & whitened tissue; drug causes NECROSIS of oral mucosa;
- Fordyce's Granules – ectopic sebaceous glands in oral mucosa; 75% are adults; yellow & sometimes yellow-white submucosal clusters that are essentially normal;


#54
Aspirin burn

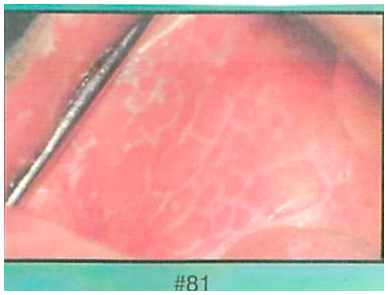



#78
Angular Cheilitis

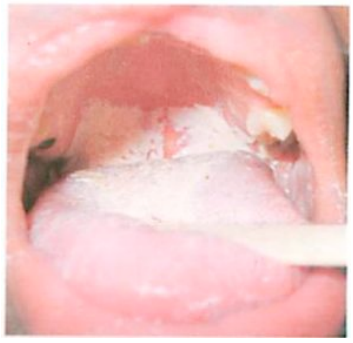
- Angular Cheilitis (Perleche) – small accumulations of saliva gathered in skin folds at commissural angles & are colonized by **CANDIDA** causing fissured erythematous alterations in skin;
  → Occurs w/ loss of VD; painful & irritating; often elderly pts.
- White Spongy Nevus – autosomal dominant disease; asymptomatic, pearly white, folded, & spongy & thickened;
  → most common site – BM bilaterally than labial mucosa, alveolar ridge, & floor of the mouth; no tx needed & benign;
  → unique: eosinophilic condensation in perinuclear region of cells in superficial layers of epithelium;


#79
White sponge nevus of buccal mucosa

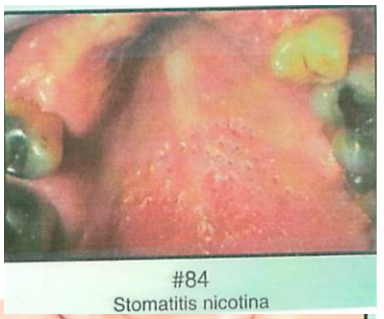
- Hyperkeratosis – abnormal ↑ in thickness of keratin layer of epithelium; commonly on cheek & presents as white, thick & scaly.
- Lichen Planus (**Wickham's Striae**) – lace-like white striae; bilateral BM; common inflammatory disease-cell mediated hypersensitivity rxn; tx – topical steroid therapy;
  → asymptomatic or sometimes burning;
  → F>M & middle ages; 2 forms:
  a. Bullous – fluid-filled vesicles projecting from surface;


#81

b. Erosive – lesions intensely red & raw-appearing; like desquamative gingivitis.
→ Histologically – hyperparakertosis w/ thickening of granular cell layer; "SAW-TOOTH" appearance of rete pegs; degeneration of basal layer of cells;

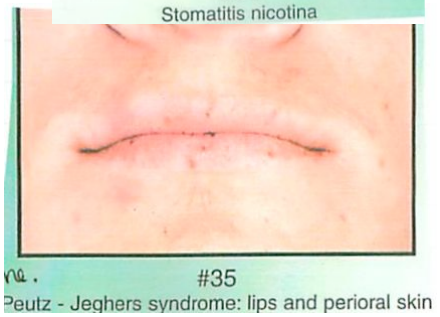


- Candidiasis (**Thrush**) - white curd-like patches in mouth/throat; tongue, cheeks, or palate; often in pts w/ ↓ immune systems; caused by yeast-like fungus C. Albicans; other signs – burning mouth, altered sense of taste.
  → Inflammatory, pruritic infection w/ thick, white discharge.
  → WIPEABLE, leaving red/raw/bleeding surface; tx – Nystatin troches
  → Common in newborns/young children after antibiotic therapy.
  → Acute Pseudomembranous Candidasis – **most common form**; BM, tongue, & palate; oral cytology smear reveals budding organism w/ branching pseudohyphae.
- Stomatitis Nicotina – palate; males; diffuse, gray-white, thickened, multinodular appearance w/ small red spot at center of each nodule; "Spot" = orifices of salivary glands; due to SMOKING;
  → In india, due to use of smokeless tobacco, people develop oral submucous fibrosis; ↑ risk of oral carcinoma.

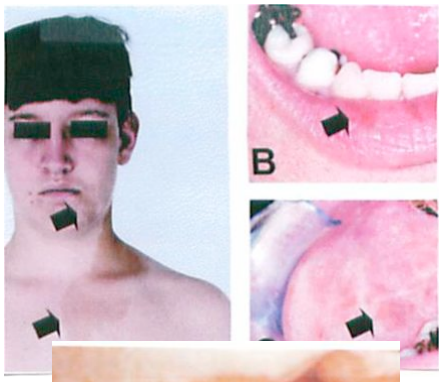


DISEASES with ORAL PIGMENTATION:

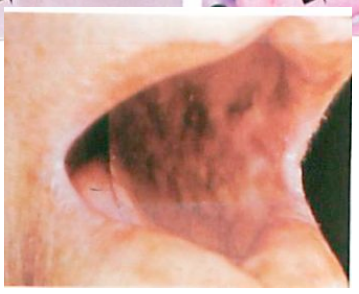
- Peutz-Jeghers Syndrome – autosomal dominant disorder causing hyperpigmentation of lips, BM, gingiva, & palate; followed by development of benign polps called hemartomas on small intestine.
  → Oral finding around age 10; macules may be blue/brown/black.
  → Oral pigments are harmless but polyps of colar undergo malignant changes.
  → Etiology – mutation of LKB1 of chromosome 19 for serine-threonine kinase.



- Albright's Syndrome – possible malignant transformation potential to *osteosarcomas*; **most severe form of polyostotic fibrous displasia**; young M&F; pathologic fractures are common.
  → CAFÉ-AU-LAIT spots on torso & sometime intraorally and endocrine abnormalities;
  → Hallmark sign is PREMATURE PUBERTY IN FEMALES;
  → Multiple, slow growing, painless expnsive bone lesions confined to craniofacial area or throughout skeleton.

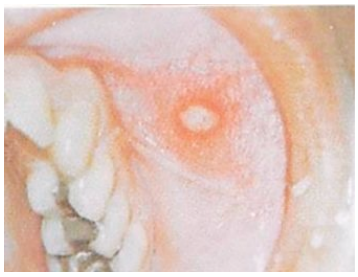


- Addison's Disease – **hypofunction (↓ cortisol) of adrenal cortex**; bronzing skin; diffuse persistant pigmentation of gingiva, tongue, hard palate, & BM; 90% of gland is destroyed when symptoms appear.

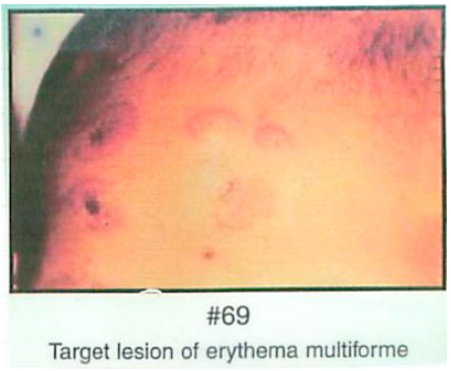


VESICULOBULLOUS DISEASES:

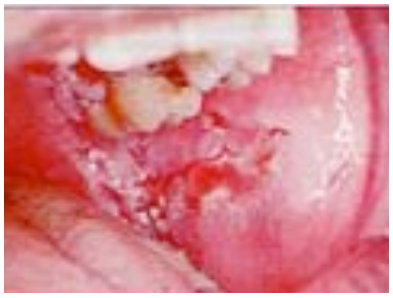
- Recurrent Apthous Ulcers – canker sores; caused by stress, or possible focal immune dysfuction where T-lymphocytes play a role; nonkeratinized mucosa; minor form is most COMMON; 3 TYPES:
  1. **Minor RAU** – very painful red macule, 2-3mm-10mm diameter; 7-10 days; 80% occur as non-keratinized movable mucoas; no scarring;
  2. **Major RAU** – lare painful ulcers; 5-20mm; upto 6 weeks; SCARRING; often HIV pts; soft palate, tonsil fauces, tongue, LM & BM;
  3. **Recurrent Herpetiform** – 100 at a time; any mucous surface; no



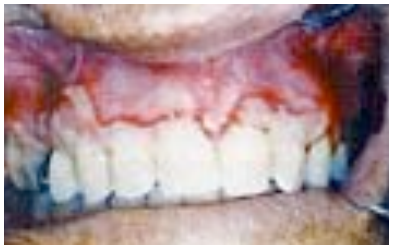
scarring; similar to HSV;
  - All 3 are painful & recurrent; Tx – topical/systemic steroids;
- Vesicles do NOT precede ulcers! Distinctive diagnostic feature;
- Erythema Multiform (EM) – acute, self-limiting eruption characterized by iris/TARGET lesion;
  → central lesion surrounded by concentric rings of pallor & redness over dorsal aspect of hands & forearmes;
  → hypersensitivity rxns w/ infections as precipitating factor; may be due to sulfa drugs, vaccinations, or HSV.
  → mostly children & young adults; 3 forms:
  1. **EM Minor** – mostly skin (25% oral); fever, malaise, headache, in first 4-7 days then target lesions which are covered by yellow-white membrane after rupture;
  2. **Chronic EM Minor** – MILDEST form; lesions small & short in duration; vary in oral lesions from focal to diffuse areas of erythema;
  3. **EM Major (SJS)** – acute from w/ skin & mucous membrane; large bullae form;
     - ⊕Nickolsky's Sign (peels off revealing pink ulcer) is common;
     - bullae collapse producing white pseudomembrane on mucosa & dark red crusty lesions;

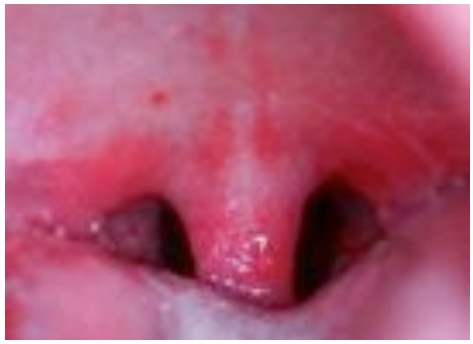

#69
Target lesion of erythema multiforme

- Steven-Johnson's Syndrome – immune-complex mediated hypersensitivity complex that is severe expression of erythema multiforme major; symptoms & lesions are severe and extensive; signs – sputum cough, headache, malaise, anthalgia; "BULLS-EYE SHAPED" target lesions present;
  → SJS TRIAD – eye lesions, genital lesions, & stomatitis;
  → Cause – drugs & malignancy in adults but infections in kids;
- Pemphigus – blistering of skin caused by binding of antibodies to surface cells of the epidermis;
  → Oral lesions are first sign of pemphigus; large areas of ulceration/erosions seen covered by white/blood-tingled exudates;
  → ⊕ Nickolsky's sign; tx = steroid/chemo (methotrexate)
  → histology – suprabasilar vesicles w/ intercellular edema loss of intercellular bridges w/ loss of cohesion – *acantholysis*; **tzanks cells** are clumps of cells often found floating free in vesicle space;



- Pemphigus Vulgaris – IgG antibodies & Tzanck cells bind to epidermis of skin & epithelial lining of mucous mucosa; 30-50 yrs & common in jewish people;
  → need 2 portions of tissue for biopsy, 1 in vile & another for **Michel's Solution** (tissue in this solution produces yellowish fluorescence where antibodies are against desmosomes;
  → severe oral ulcers, inflammation/erosion of lining of eye/eyelids, nasal mucosa, & genital mucosa;
- Benign Mucous Membrane Pemphigoid – oral lesions present as DESQUAMATIVE GINGIVITIS (sloughing of gingiva during eating/brushing); also may have ⊕ Nickolsky's sign
  → autoimmune/self-allergy disease; F>M & 40-60 yrs;
  → clear-fluid blister breaks rapidly to leave flat white tender ulcer w/ thin red line around it;
  → conjuctival involvement may lead to blindness; tx = systemic steroids; less severe than pemphigus b/c antibodies attack attachment fibrils (type 4 collagen);
  → Subepidermal & NO acantholysis while pemphigus vulgaris is suprabasilar & has acantholysis.

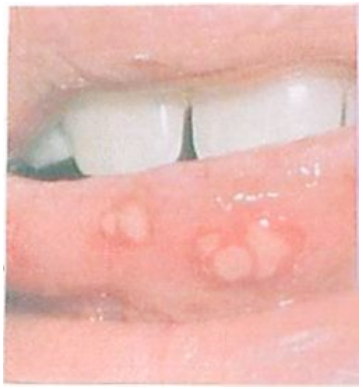




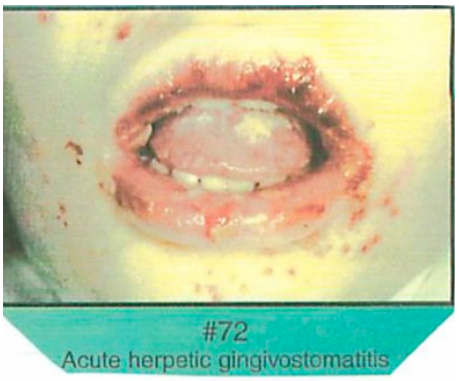
INFECTIOUS VESICULOBULLOUS DISEASES:

- ***Coxsackievirus*** – Group A has 2 types: Herpangina & Hand,Foot,& Mouth Disease;

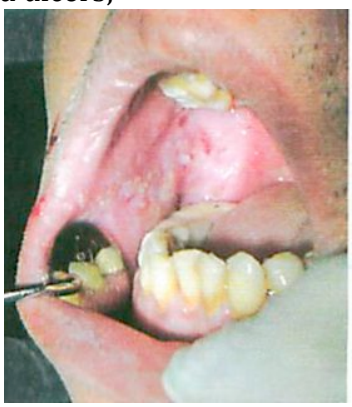
- Herpangina – non-treatable mild infection caused by Group A Coxsackie Virus; ulcers w/ white-gray base w/ red border; may be painful;
  → stomatitis (inflammation of mouth); soft palate & nasopharynx; common w/ infants/children; also common in summer;
  → spreads fecal-orally or resp. droplets; mild & short duration;
  → sore throat, difficulty swallowing, mild fever;
  → small vesicular & punctuate lesions w/ white base; course < 1 wk;



- Hand, Foot, & Mouth Disease – highly contageous infection from coxsackievirus A & w/ limited duration;
  → vesicular eruptions of palsm, soles, & anterior mucosa of mouth;
- HSV–1 - oral herpes; **HSV-2** = genital herpes; tx = symptomatic & supportive using *Acyclovir*, *Valacyclovir*, or *Fanciclovir*;
  → **corticoidsteroids are contraindicated;**
  → HSV-1 lies dormant in trigeminal ganglion; cells are **Lipschutz bodies** (giant cells w/ multiple nuclei) that can be seen on **Tzanck smear (**smear cells from fresh blister & stain w/ *Wright's stain*);
  → Other test: tissue culter, antibody titers, biopsy, or Fluorescent staining – HSV test + for fluorescence which is difference b/w HSV & herpes zoster;
  → 3 oral presentations of HSV-1:



  1. **Herpes Labialis** – fever blisters/cold sores; common due to sunlight; eruption of small & painful blisters on skin of lips, mouth, gingiva, or skin around mouth;
  2. **Acute Herpetic Gingivostomatitis** – heals on their own & lasts 7-14 days & common in children/young adults; may cause DEHYDRATION; PRIMARY INFECTION!
     i. prodromal symptoms (fever, malaise, irritability, headache) 1-2 days prior to local lesions;
     ii. small yellow vesicles that rupture quickly results in round, discrete ulcers w/ erythematous halo; free/attached ginigva; generalized marginal gingivitis may preced ulcers;
  3. **Recurrent Herpetic Stomatitis** –adult pts; triggers are trauma, fatigue, URI, stress, allergy, UV exposure causing release of latent HSV-1;
     i. Lips, hard palate, attached gingiva, & alveolar ridge; cold sores.
     ii. Day before vesicle may have tingling or itching of skin;
     iii. Cytopathic effect - BALLOONING degeneration of epithelial cells & loss of cohesion to adjacent cells.



- Varicella-Zoster Virus – herpes virus family(HSV-3); causes chickenpox (varicella) & shingles (herpes zoster); very contagious thru direct contact or droplets; 2 types:
  1. **Primary Infection** – macules, papules, vesicles & ulcers on skin/oral mucosa; malaise, fever, lymphadenopathy;
  2. **Secondary Infection** (recurrent)– painful vesicles, ulcers, & crusts in trigeminal & trunk area;
- Chickenpox – itchy/scabby pruritic vescular lesions; most contagious until one day before onset of rash; mostly children (benign) but in adults, may cause pneumonia/encephalitis; **ZIG** (**Zoster Immune Globulin**) – morbidity in high risk children;
- Shingles – reactivation of latent varicella-zoster virus; inflammatory response causing painful vesicles on skin/mucosal surfaces along distribution of sensory nerve in distinctive unilateral pattern;
- Rubella – GERMAN MEASLES; rash – flat, pink spots on face & spread to other body parts; benign viral disease; swollen LN, fever, achy; Petechiae-like spots of soft palate;

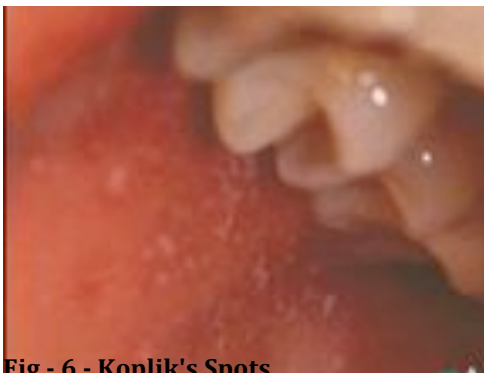
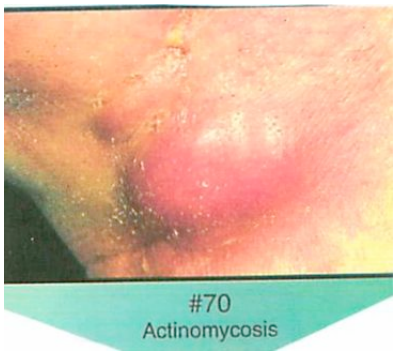


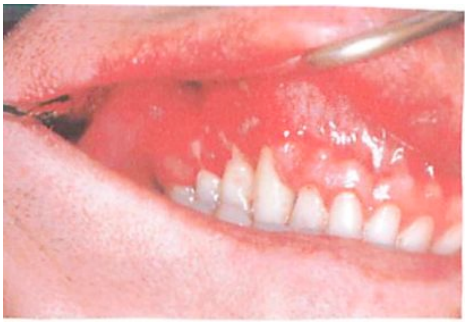
Fig - 6 - Koplik's Spots

- Paramyxoviruses – measles (rubeola) & mumps; KOPLIK's Spots (from rubeola/measles) in oral cavity – small, bluish white lesions surrounded by red ring; opposite molars;
- Measles – RUBEOLA; highly contageous viral disease characterized by fever, cough, & spreading rash; incubation pd = 1-2 weeks;
  → Koplik's Spots – on BM, 1-2mm, yellow-white necrotic ulcers surrouded w/ bright red margin;
- Small Pox – VARIOLA – acute viral disase; high fever, nausea, vomiting, chills, headache; skin lesions first as small macules & papules which first appear on face but rapidly spread to cover body;
  → Oral – ulcerations of oral mucosa & pharynx; sometimes tongue swollen & painful;

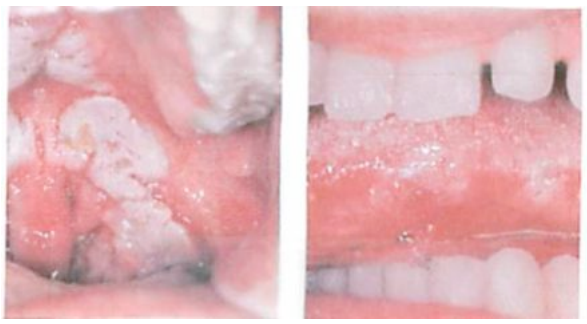
INFECTIOUS DISEASES:



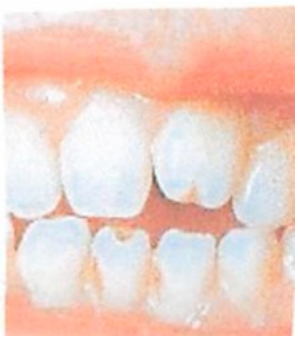
- Actinomycosis – LUMPY JAW; exudate of sulfur granules from trauma or infection caused by A. israilli (anaerobic gram +);
  → painful mand. swelling; forms abscess causing hard, red/purple lump in jaw; tx = PCN.
  → exudate creates wooden indurationw/ central soft spot.
  → not contagious but occur after trauma/surgery/previous infection;
  → most infections of thorax, abdomen, H&N (cervicofacial actinomycosis – most common form of actinomycosis);
- Histoplasmosis – chronic lung infection from **spores of Histoplasma Capsulatum**; chronic non-healing oral ulcer;
  → fever, malaise, cough, dyspnea, & cervical lymphadenopath;
  → tx – amphotericin B, itraconazole, or ketoconazole.



- Syphilis – infectious venereal disease caused by SPIROCHETE TREPONEMAPALLIDUM; less common STD; 10-90 days; 3 STAGES:
  1. **Primary** – non-painful chancre; 2-6 wks after exposure; disappear whether treated or not & if not treated, 1/3 of pts will progress to chronic stages; tongue, lips, penis, vagina;
  2. **Secondary** – highly infectious stage; 6 wks after no tx in primary stage; widely disseminated spirochetes causing mucous membranes to exhibit a reddish brown maculopapular cutaneous rash; also **condylomata lata** (elevated grey broad based plaque);
  3. **Tertiary** – many years after no tx of secondary stage; GUMMA typifies this stage – focal nodular mass on **palate or tongue**; bacterial damage to heart, eyes, brain, CNS, bones, joints;
  → Neurosyphilis = headache, stiff neck & fevers;



- Congenital Syphilis – infection of spirochete, treponema palladium during fetal period; nearly ½ die before or after birth; tx = PCN.
  → if tx at 4th/5th month than 95% have no manifestations;
  → newborn symptoms – irritability, bloody discharge from nose, no thriving, early/later rashes;
  → young child symptoms – Hutchinson's Incisors (screwdriver shaped centrals), bone pain, joint swelling, condyloma lata, saber shins, visual loss, CN VIII deafness & interstitial keratitis;
  → HUTCHINSON'S TRIAD – 1) screwdriver incisors & mulbery molars
  2) interstitial keratitis (inflammation of corneal stroma).
  3) CN VIII deafness.



- Mucormycosis – FUNGAL INFECTION for sinus, brain, or lungs; primarily in ppl w/ immune disorders; aggressive, opportunistic infection w/ high affinity for afflicting Diabetes pts;

→ genera – *mucor/rhizopus*;
→ **Orbitorhino-cerebral mucormycosis** – most common type; sinus & nasal involvement.
→ Diabetes pts predisposed to mucormycosis b/c ↓ability for their neutrophils to phagocytize & adhere to endothelial walls; also acidosis & hypoglycemic good for fungal growth;

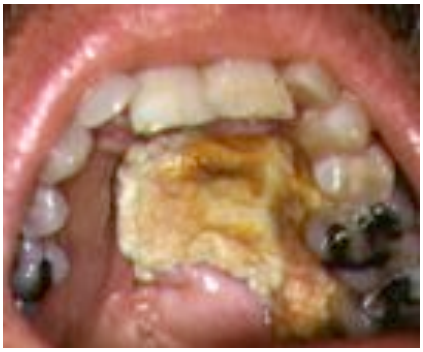


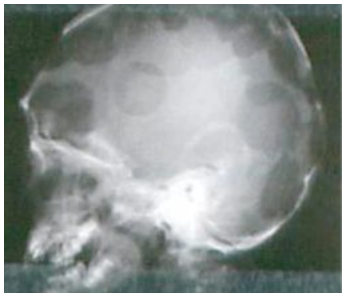
Fig - 7 - Mucormycosis

- Hepatitis A – highly contagious & affects liver but MILDEST FORM of hepatitis; fecal-oral route & parenterally;
  → **RNA enterovirus**; young adults mostly; incubation pd = 3-6 weeks;
  → symptoms – fever, malaise, abdominal pain, jaundice;
  → damages liver cells causing ↑serum enzymes like transaminases; recover in 4 months;
  → **oral complications – abnormal bleeding**;
  → presence of surface antigen A/B indicates Hep A carrier state; Hep A is very heat resistant;
- Hepatitis B – Double-stranded DNA virus thru parenteral/sex; incubation = 40-100 days; can be recovered from all body fluids; often longer duration but fully recovered;
- Hepatitis C – antigenically differ from A/B; from post-transfusion hepatitides; ↑incidence of chronic disease, cirrhosis, & hepatocellular carcinoma;
- Hepatitis D – only in pts w/ Hep B & makes Hep B more severe; often w/ drug addicts.
- Hepatitis E – transmitted enterically; occasionally epidemics in underdeveloped countries;

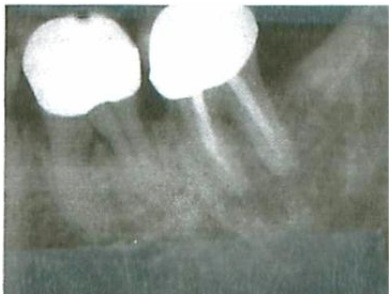
MISCELLANEOUS:

- Granulomatous Inflammation – chronic inflammation & contains epitheloids which are specialized macrophages & lymphocytes surrounding granulomas;
  → Granulomas produce multinucleated giant cells; caseous necrosis produced by infectious agents like Mycobacterium Tuberculosis.
  → Causes – infections, foreign material, sarcoidosis, Crohn's disease.
- Neck swelling are characteristic of MONO, Hodgkin's Disease, & TB;
- Ludwig's Angina – results from odontogenic infection; spreading infection involving submandibular & sublingual spaces bilaterally; rapid onset; board-like swelling of floor of the mouth;
  → Open-mouthed appearance, drooling, trismus, fever; involves oral flora (esp. anaerobes).
  → Elevation of tongue, difficulty eating, swallowing, or breathing.
  → Tachycardia & ↑respiratory rate; Tx = airway management, antibiotics, & IND.
  → Most serious is EDEMA OF GLOSSITIS (opening b/w vocal cords).
- **Sialoscintigraphy** – simple, non-invasive procedure that can separate benign entities from malignant tumors.
- ESR – erythrocyte sedimentation rate; RBC cells rate of settling out in tube of unclotted blood; measured as mm/hour; ESR measures progression of disease;
  → ↑ESR = inflammation, pregnancy, tissue degeneration, suppuration, necrosis;
- **Toxic dose** for Fluoride Ingestion = 5-10mg/kg; min for adults = 2g & child = 16mg/kg;
- Symptoms for Acute Fluoride Poisoning: nausea, abdominal pain, vomiting, diarrhea, convulsion, & hypotension; tx – call poison control, monitor vitals, initiate BLS, transport pt to hospital.
- Chronic Fluoride Poisoning causes – Osteosclerosis of bones (10-25ppms) & Fluorosis;
- **Sodium silicofluoride** fluoridates communal water supply;
- Best tx for XEROSTOMIA = Sodium Carboxymethyl Cellulose;
- Carcinoma In Situ – all histological signs of malignance but no invasiveness or extension into adjacent structures; when dysplastic changes are marked & involve entire thickness of epithelium;
  → 3 types of SCC: Verrucous Carcinoma, Carcinoma In Situ, & Carcinoma Invasive-Ex.

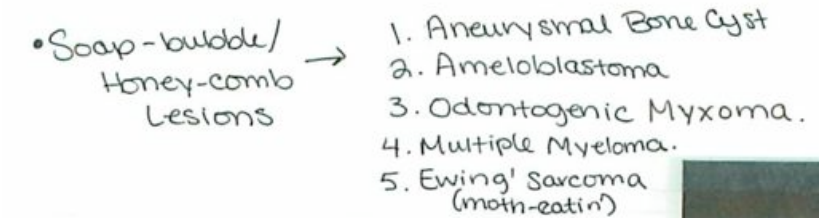
- Erythromelagia - rare disease of paroxysmal vasodilation w/ burning pain & ↑skin temp, redness of feet & hands.
- Cleft Lip – when medial nasal process fails to fuse w/ lateral of max. process of 1st brachial arch; usually during 6th & 7th weeks; more in males, max. lip, & left side;
- Cleft Palate – fissure of midline from failure of 2 sides to fuse; 8-10th weeks; more in females and most severe b/c can prevent normal speech/swallowing; often lateral incisor missing in cleft area;
  → Speech problems often from inability of soft palate to close airflow in nasal area;
- Achondroplasia - MOST common type of dwarfism; normal torso but short arms & legs; forehead prominent, nose is saddle-like, & mandible exhibits prognathism;
  → Overcrowding of teeth, speech problems, & ↑ ear infections.
  → Maxillary small causing overcrowding; class III malocclusion common;
  → Many children die in 1st year of life;
- Cerebral Palsy – disability affecting body mvmt & muscle coordination; insult/anomally of motor contrl centers interfering w/ messages from brain to body & body to brain; no identifiable cause.
  → spastic paralysis or impairment of control/coordination; mental retardation, seizures, & disorders of vision & communication; varies widely from person to person;
  → Difficulty masticating/swallowing, **↑periodontal disease & caries**, attrition, & trauma.
- Down's Syndrome – mand. prognathism, ↑ periodontal disease, delayed eruption, ↑ congenitally missing teeth, malocclusion, & enamel dysplasia; fissured tongue.
- Langerhan's Cell Histiocytosis X – name for group of disorders characterized by abnormal increase in histiocyte cells (monocytes, macrophages, & dentritic cells); langerhan's cells are dentritic cells; 3 types:



  1. Letterer-Siwe (acute disseminated disease) – aggressive; young kids, widespread disease; skin & mucosal lesions with visceral & bone involvement
  2. Hand-Schuller-Christian (Chronic Disseminated Disease) – exophthamus, diabetes, & lytic skull lesions; hepatosplenomegaly, skin & mucosal lesions;
     a. bad breath, sore mouth, loose teeth;
     b. punched out bone lesions in skull/jaw;
     c. RL associated w/ apices of teeth appearing as floating teeth.



  3. Eosinophilic Granuloma (focal chronic disease) – localized bone destruction, swelling, & pain; most benign form, more mandible; irregular RL areas involving superficial bone;
     a. cupped out RL resembling periodontal disease/PA lesion; single/multiple well-circumscribed radiolucencies.
- **Condylar Agenesis** – absence of all/portions of the coronoid process, condylar process, ramus, or mand. body; tx – costochondral graft w/ or w/out ortho.
- **Condylar Hypoplasia** – often trauma or infection; shortness of mandible & deviation toward affected side; child – costochondral graft; adults – shorten/lengthen one side; ORTHO NECESSARY!
- **Condylar Hyperplasia** – idiopathic; progressive, unilateral overgrowth of mandible; deviation toward unaffected side; x-ray **– normal condyle but elongated neck**; tx = condylectomy or orthognathic surgery.
- Von Recklinghausen, Albright, & Jaffe's Syndrome all have CAFÉ-AU-LAIT spots.



- Cushing's Syndrome - ↑cortisol; 20-50 yrs; obesity, roundface, buffalo hump, & thin arms & legs; no pigmentations.
- **Choristoma** – normal tissue present in abnormal location.
- **Hyperplasia** - ↑ in # of component cells; **Hypertrophy** - ↑ in size of component cells;
- **Cancer** – all malignant neoplasms; Carcinoma – malignant epithelial neoplasm;
- **Sarcoma** – malignant mesenchymal (CT) neoplasm;
- **Hamartoma** – developmental defect of overgrowth of tissue NORMAL to that organ;
- **Teratoma** – neoplasm of multiple tissues FOREIGN to the organ it arises from.
- **Malignant Neoplasms** – anaplasia (undifferentiated), pleomorphism; abundance of DNA & hyperchromatic; nuclear-cytoplasmic ratio = 1:1; degree of malignancy (mostly for SCC) based on degree of differentiation:
  Grade 1 = well differentiated
  Grade 2 = moderate well-differentiated.
  Grade 3 = poorly undifferentiated
  Grade 4 = undifferentiated.

Benign	Malignant
well-differentiated	not well-differentiated (anaplastic)
slow growth	rapid growth
encapsulated	invasion
localized	metastasis
movable	immovable

- Neuroblastoma – most common malignant tumor in NEONATES; originates in adrenal medula & other sympathetic nervous tissue; sites – abdomen, chest, neck, & pelvis.
- Pierre Robin Syndrome – hereditary disorder in neonates causing:
  1. Micrognathia (smaller than normal jaw)
  2. Glossoptosis
  3. High arched/cleft palate
- Lymphangioma – well-circumscribed nodule/mass of lymph vessels; often neck & axilla; red to blue translucent lesions that are spongy; tx – excisional biopsy; Most common site – TONGUE, macroglossia can occur.
- **Dysplasia** – principally in epithelia; loss in uniformity of individual cells as well as loss in architectural orientation; pleomorphism & hyperchromatism; often associated w/ CHRONIC irritation of tissue; atypical cells w/out invasion; exhibits acanthanosis (abnormal thickening of epithelium);
- **Metaplasia** – when one cell type changes to another cell type in response to stress; most common – replacement of columnar cells by stratified squamous epithelium.
- **Anaplasia** – absence of cell differentiation.
- **Nosocomial** – infection/disease originating in the hospital.
- **Neoplasm** – uncontrolled new growth of tissue; **Tumor** – localized swelling that may/may not be a true neoplasm.
- **Carcinoma** – malignant epithelial neoplasm; **Sarcoma** – malignant mesenchymal (CT) neoplasm;
- Conditions that present Giant cell lesions – Fibrous dysplasia, Central & Peripheral Giant Cell Granulomas, & Paget's Disease.
- Hairy Leukoplakia – NOT hairy tongue; appears 99% of time in HIV pts; always white furry lingual lesions on LATERAL tongue borders (mostly bilateral) caused by **EBV**!!!
- Recurrent apthous ulcers occur mainly on mobile mucosa, while intra-oral herpes lesions occur on tissue bound to periosteum; so diffentiate b/w the two based on LOCATION.

ORAL SURGERY

- Erythromycin not acceptable antibiotic for prophylactic.
- If allergy to PCN & clindamycin, use cephalexin, clarithromycin, or azithromycin.
- Normal Pulse = 72; BP = 120/80
- Temp: Oral = 98.6, Axillary (least accurate) = 97.6, Rectal (most accurate) = 99.6, Aural = 99.6.
- **ASA Classifications:**
  1. Normal healthy pt.
  2. Mild systemic disease/significant health risk factor (smoking, alcohol, obesity)
  3. Severe disease but not incapacitated
  4. Severe systemic disease that is constant threat to life.
  5. Moribund pt. not expected to survive unless operation.
  6. Brain-dead pt. whose organs removed for donation.
- Tests for admitting to hospital for surgery: CBC, WBC count, urinalysis, and if G.A. then chest x-ray, and over 40 yrs, then EKG.

PATHOLOGY:

- CBC: 1) Hematocrit: M=40-54%, W=37-47%; # of RBCs in your blood;
  - minimal for surgery is 30%

  2) Leukocytes: 5-10,000/mm^3; dental infection=15-20,000/mm^3.
  3) Hemoglobin: M=14-18g/dL, W=12-16g/dL
  4) Total Erythrocytes: M = $5x10^6/mm^3$, W = $4.5x10^6/mm^3$.
  5) Template Bleeding time = 1-9min;
  6) PT time = 11-16min – best test to determin if O.S. can be done w/ pt on COUMADIN; Pt must be w/in 5-7 secs of control sample.
  7) PTT Time = 25-36 sec; best test for hemophila; detects coagulation defects of intrinsic system;
  8) Platelets = 140,000-440,000/mL
     - Platelets at 50-100,000 is ok if platelet fct is healthy.
     - Thrombocytopenia =<50,000/mm^3 which is contraindicated for surgery.

  9) Urine pH = 6; Specific Gravity = 1.005-1.025
- Hydrocortisone (glucocorticoid) - 20 mg secreted by adrenal cortex/day; stimulated by ACTH (ant. pituitary); increases in concentrations under stress but decreases with excess steroids in system.
- Cushing's Syndrome – hormone disorder caused by prolonged exposure to high levels of cortisone (glucocorticoid) causing *hypercortisolism*; rare but more in females ages 20-50 yrs.
  - 10-15ppl out of 1 million affected each year; most common cause is pituitary adenomas.
  - Causes moon face, fat pads, buffalo hum, obesity, and purple striae.
  - Causes muscle weakness, bruising, weight gain, and growth retardation, excess hair.
  - Increase in BP, osteoporosis, fractures, impaired immune fct, glucose intolerance, and psychosis.
- Erythema Multiforme – hypersensitivity syndrome of polymorphous eruption of skin/mucous membranes; macule, papules, vesicles, "BULLS-EYE" shaped bulla.
  - Severe form = Steven's Johnson Syndrome.
  - Tx = corticosteroids (consult DR b/f treating these patients)
- Pts on Steroids:
  - Small doses (5mg/dy) will have suppression if been on regimen for month.
  - 100mg cortisol/day (20-30mg Prednisone/day) will have abnormal cortical fct for a week.
  - Short term therapy (ie high doses for 1-3days) will not alter cortical fct.
  - Adrenal crisis – IV/IM of hydrocortisone and supportive tx for decrease BP.
  - Person who has been on supressive steroids will take 1 year to regain full adrenal cortical fct.
- Pts taking chronic daily does of steroids (>10-20mg/day of prednisone) should be considered for steroid supplementation for oral surgery.

- If currently taking steroid, double daily dose of steroid for surgery day.
- If less than 2 weeks after steroids stopped, double dose of steroids on surgery day.

- As **dehydration** progress – turgor (fullness) of skin loss, then oliguria (decrease in urine), then severe cell dysfunction – water shifts from intracellular to extracellular space, particularly in brain.
    - BP falls w/ continuous dehydration.
- Diabetes Mellitus – mostly carbs/glucose & lipids owing to *lack of insulin secretion* by beta cells of pancreas; if well-controlled, not susceptible to infections but difficulty containing infections.
    - Hypoglycemic Symptoms: tachycardia, sweating, nausea, tremulousness, hunger.
    - Diabetes is most common pancreatic endocrine disorder/ metabolic disease.
    - Type 1 pt – absolute deficiency of insulin due to destruction of B cells.
    - Type 2 pt – resistance of insulin's action in peripheral tissues.
    - Causes Polydipsia(excessive thirst), Polyuria(excessive urination), & Polyphagia(excessive hunger).
    - Tx = for conscious pt – oral carbohydrate/sugar
    - Tx = for unconscious pt – 1mg glucagon IM or 50ml of 50% glucose IV.
    - #1 cause of Kidney Disease (40%); high bp is 2nd common cause.
    - Well-controlled diabetes are no more susceptible to infections than pts w/o diabetes but it is more difficult containing infections due to their altered leukocyte function.
- Dypspnea: difficulty breathing; Apnea: transient absence of breathing; Hyperapnea: deep & rapid breathing.
- Hyperventilation – ↓CO_2 from blood causing decrease BP & fainting; hypocapnea (loss of CO_2).
- Hypoventilation – ↑CO_2 in blood; hypercapnea (excess CO_2)
- CHF – 50% of ventricular ejection; usually left ventricle fails first;
    - most common sign of left CHF is pulmonary edema;
    - most common sign of right CHF is pedal edema or abdominal swelling.
    - Earliest & most common sign–*Paroxysmal Nocturnal Dyspnea*(pt wakes up gasping for air).
- Usually a post-infarction pt is not subjected to oral surgery w/in 6 months of his infarction.
- Pts taking diuretcis/vasodilators are prone to orthostatic hyptension and avoid excessive EPI.
- Normal blood pH = 7.33-7.44; normal blood bicarbonate to carbonic acid ratio = 20:1.
- Bicarbonate-carbonic acid ratio normal is 20:1.
- Acidosis – deceased blood pH; CNS depressed; 10:1 ratio indicating uncompensated acidosis – always occurs during CPR;
- Alkalosis – increased blood pH; over excitability of CNS causing tetany.
- Metabolic Acidosis = ↓bicarbonate; too much acid or too little base; causes CNS depression so disorientation, the comatosed; causes are Chronic renal failure, diabetic ketoacidosis, lactic acidosis, poisons, and diarrhea.
- Respiratory Acidosis = increase CO_2 b/c decreased resp. rate b/c poor lung function.
- Tx for Metabolic & Respiratory Acidosis = sodium bicarbonate.
- Metabolic Alkalosis – ↑bicarbonate; too much base/too little acid; causes overexcitability of the body.
    - Etiology – diuretics, cusshing's syndrome, vomiting;
- Respiratory Alkalosis – decreased C_{O2} b/c increased resp. rate.
- Tx for Metabolic & Respiratory Acidosis = aluminum chloride.
- Status Asthmaticus – severe form of asthma; if not tx, then chronic partial airway obstruction which may lead to respiratory acidosis.
- Rheumatic Fever – Sequela of previous Group A – β-hemolytic Staph infection of Upper Respiratory Tract; exudative & proliferative inflammatory lesion (NOT INFECTION) of connective tissue, esp. heart, joints, blood vessels, & subcutaneous tissue; Tx = PCN and rest.
    - Common in children 5-15 yrs; Carditis may cause permanent valve damage, like MVP;
    - Diagnosis made when 1 major & 1 minor criteria (JONES CRITERIA) are met:
        - Major – carditis, arthritis, chorea, erythema margnatum, and subcutaneous nodules.

- Minor – fever, arthralgias, history of RFD, EKG, and lab test.

- Hemophilia A & B – takes long time for blood to clot and abnormal bleeding occurs; sex-linked recessive (males affected & females carriers)
  A. Often <25 yrs; deficiency of **factor VIII** (anti-hemophilic factor).
  B. (Christmas disease) Deficiency of **factor IX** (plasma thromboplastin component).
  C. (Rosenthal's Syndrome) Deficiency of **factor XI** (plasma thromboplastin antecedent).
  - **true hemophiliac has increased PTT, normal PT & bleeding; however pts on anticoagulant therapy (wafarin, heparin, aspirin, or NSAID) will have prolonged PT and bleeding time.
- Von Willibrand's Disease – autosomal dominant bleeding disorder caused by deficiency in von Willebrand factor – binds to factor VIII and adhedes platelets to collagen.
- Thrombocytopenia – most common cause of hemorrhagic/bleeding disorders; abnormally low # of platelets (<150,000); Abnormal reductions of platelets caused by any of these 3 processes:
  1. Platelet production by bone marrow
  2. Trapping of platelets by the spleen
  3. Faster tha normal destruction of platelets.
  - S&S – petechiae, nosebleeds, GI bleeding, tendency to bruise, urinary tract bleeding.
  - 2 concerns w/ these pts – post-op hemorrhage and adrenal insufficiency (due to steroid tx).
  - Excessive bleeding causes formation of hematomas which increases chance of infection.
  - Drugs that potentiate bleeding after extraction: 1) aspirin
    2) anti-coagulants
    3) broad-spectrum antibiotics
    4) alcohol
    5) anticancer drugs.
- Pts on Anticoagulated Therapy – stop drugs for 5 days then perform surgery and restart the drug therapy THE DAY AFTER surgery if no bleeding is present.
- COPD – emphysema & chronic bronchitis & asthma or any combination of those 3 diseases; airway obstruction that is chronic & progressive; **causes secondary pulmonary hypertension.**
  1. Bronchial Asthma – disorder marked by dyspnea & wheezing expiration from narrowing airways.
  2. Emphysema – often w/ chronic bronchitis; labored breathing and increased chance of infections.
  3. Bronchiectasis – copious purulent sputum, hemoptysis, and recurrent pulmonary infection.
  4. Chronic Bronchitis – excessive bronchial mucous and productive cough (universal sign of chronic bronchitis) w/ sputum for 3 mo/more in at least 2 consecutive years w/out any other disease.
- COPD & aspirin may cause Hemoptysis – bursting of RBC.
- Chronic Bronchitis – causes hyperplasia of bronchial submucosal glands & bronchial smooth muscle hypertrophy quantified by **Reid Index;** predisposed w/ lung cancer;
  - associated w/ smoking; productive cough w/ wheezing; so need to be UPRIGHT during O.S.
  - COR PULMONALE (enlarged RV of heart);
  - airway narrowing & obstruction of bronchial tree.
- Emphysema – "BARREL-CHESTED" appearance; b/c distal air spaces become enlarged & lungs hyperinflated; **destruction of airsacs** in lungs where oxygen exchanged;
  - shortness of breath and difficulty exhaling.
- End-Stage Renal Disease – perm. & almost complete loss of kidney fct <10%; toxins slowly build-up;
  - On steroid therapy, increased post-op infections, increased bleeding tendency.
  - Oral surgery performed 1 day after dialysis; Consult dr. for prophylaxis.
  - Do not use NSAIDS; avoid drugs metabolized/excreted by kidneys.
- Atelectasis – mucous/foreign object obstructs airflow in mainstem bronchus causing collapse of affected lung tissue; often 36 hrs. post-op w/ mild dyspnea, low grade fever, hypoxia, & can lead to pneumonia;
  - most common ANESTHETIC COMPLICATION occuring in 1st 24 hrs.
  - Tx = incentive spirometer, pt. takes long deep breaths to expand the lung.

- Pneumothorax – air leaks into pleural space causing lung to recoil from chest wall; dyspnea, chest pain, need chest x-ray; can occur as post-op complications from aspiration of vomit into trachea.
  - Tx = remove air from pleural space w/ chest tube/small needle.
- **Pneumonitis (inflammation of lungs) & atelectasis – 2 most common causes of fever in pt. w/ G.A.
- **Calcium** regulated by parathyroid hormone causing increased bone resorption with increased Ca levels; calcium also regulated by kidney tubules and GI mucosa (↓pH =↑Ca);
  - ↓Ca causes hyperirritability of nerves and muscles.
  - ↑Ca = ↓PO_4
  - Ca increased in hyperparathyroidism, glomerulonephritis, hypervitaminosis D, & malignant diseases (ie multiple myeloma); Ca decreased in diabetes mellitus.
- **Phosphorus** concentration regulated by parathyroid hormone = ↑PTH =↑ Phosphorus in urine =↓ phosphorus in plasma.
- Good health = Ca:Phosphorus ratio is 10:4.
- Insulin = ↓ glucose; glucagon = ↑ glucose.
- Fasting glucose >140 and nonfasting glucose >200 = diabetes; Normal Glucose – 70-120mg/dl.
- Glucose regulated by liver w/ hormones from pancreas, adrenal medulla and cortex.
- Blood glucose increased w/ glucagon and decreased w/ insulin; glucose not in urine but filtured b/c reabsorbed in PROXIMAL CONVOLUTED TUBULE of kiney
- Osteomyelitis – inflammatory process w/in medullary bone that involves marrow spaces; caused by STAPH AUREUS; less in maxilla b/c rich blood supply; pus is produced in bone so may cause abscess.
  - Suppurative osteomyelitis – acute, chronic, or infantile osteomyelitis.
  - Nonsuppurative osteomyelitis – chronic sclerosing, Garre's Osteomyelitis and actinomycotic osteomyelitis.
  - Can affect adults(vertebrae & pelvis) /children(long bones) – affects adjacent ends of bones like femer & tibia or humerus & radius.
  - Garre's Osteomyelitis – in children/young adults; causes periosteal thickening and peripheral reactive bone formation resulting from mild irritation/infections; clinically- bony, hard non-tender swelling and associated w/ *painful carious tooth*.
  - Acute Osteomyelitis – **reduced blood supply predisposes bone to osteomyelitis**; like in mand.
- Dentigerous cyst – associated w/ crowns of unerupted teeth; AKA follicular or primordial cysts; result of degenerative changes in reduced enamel epithethelium.
    - Unerupted 2nd mand. molar on 14 y/o w/ dentigerous cyst around crown…tx – uncover crown and keep it exposed.
    - Eruption cyst form when tooth is erupting – tx = simple incision/deroofing.
- Characteristics of Malignancies: 1) erythroplasia – lesion red/speckled red & white
  2) ulceration
  3) duration > 2 wks; >40yrs. old pt
  4) rapid growth, bleeding, induration, fixation.
- Early carcinoma frequently appears as area of erythroplasia (red but not ulcerated area of mucous membrane).
- Squamous Cell Carcinoma = 90% of oral cavity and oropharyngeal malignancies;
    - most common site is LIP (25-30%, also GOOD PROGNOSIS); often ulcerated.
    - 2nd most common site is tongue, often anterior tongue (lateral border).
    - 3rd most common site is floor of the mouth; often older men who smoke/drink.

EMERGENCIES:

- Reducing cardiac output is MAIN FACTOR in all types of shock;
- S&S of shock: tiredness,cofusion, cold skin, sweaty, bluish, pale, rapid but weak pulse, and BP drops.
- Characterizations of Shock: 1) increased HR & vascular resistance
  2) decreased cardiac output

3) tachycardia
4) adrenergic response
5) ischemia/mental change

- Stages of Shock: 1) Compensatory stage – increase HR and peripheral resistance.
  2) Progressive stage – metabolic acidosis
  3) Irreversible/Refractory stage – organ damage, survival not possible.
- Categories of Shock: 1) Hypovolemic Shock – produced by decreased blood volume.
  2) Cardiogenic Shock – caused by massive MI; circulatory collapse from **pump failure of L.V**.;
  3) Septic Shock – severe infection from endotoxin of gram – bacteria.
  4) Neurogenic Shock – severe injury/trauma to CNS.
  5) Anaphylactic shock – severe allergic rxn.
- Epinephrine is given during shock b/c prevents release of substances from mast cells & antagonizes the action of histamine & leukotrienes of smooth muscle.
- LA is sedative/depressant on CNS; toxicity causes drowsiness, slurred speech, coma, culvusions, resp. depression, decreased cardiac output; initial effect may be stimulation, agitation, talking, ↑BP, ↑HR, ↑Resp; Tx = oxygen and diazepam IV.
- First CLINICAL SIGN of mild lido toxicity is NERVOUSNESS!
- First CNS manifestation of LA toxicity is short CNS excitation then **drowsiness** then unconsciousness and resp. depression/ arrest; CV effects are depressant causing dec. BP.
- *Allergic rxns to LA* is from either the LA or methylparaben (*preservative*); if there is allergic rxn to LA, use dipheynylhydramine/benadryl; allergic rxns to LA caused by antigen-antibody rxn.
  - Presents w/ swelling, itching, and oral mucosa swelling.
- Syncope = transient cerebral hypoxia; tx = oxygen 3-4L/min;
  - MOA = increases amounts of catecholamines causing decrease peripheral resistant, tachycardia, sweating; so syncope caused by overcompensating for increase BP so creates bradycardia.
  - Most common early sign of syncope = pallor/paleness
  - Oxygen indicated for all syncope unless caused by hyperventilation and contra for COPD.
  - Inhaled ammonium irritates the trigeminal nerve sensory endings causing reflex stimmulation of medullary respiratory & vasomotor centers;
  - Types: Vasovagal, Neurogenic, Orthostatic, Hyperventilation.
- Hyperventilation in anxious dental pt. leads to carpopedal spasm = spasm of the hands, thumbs, foot, or toes.
- Asthma – dyspnea, cough, & wheezing caused by bronchospasm which results from hyperirritability of tracheobronchial tree; If bronchodilator doesn't work during asthma attack use EPI (.3ml of 1:1000 dilution), then oxygen; Sit pt in erect or semi-erect position during asthma attack.
- Epinephrine is drug of choice in acute allergic rxn w/ bronchospasm and hypotension.
- If asthmatic, avoid: aspirin, NSAID, barbs, narcotics, erythromycin; use B_2-agonist (albuterol) for asthma tx.
- If CPR is effective, then pupils constrict; **if too much pressure on xyphoid process – **liver** injured!
- If interrupt chest compression – fall of BP to 0 and reduced blood flow.
- For BP cuff – bladder length and width of cuff should be 80% and 40% of arm circumference.
- BP – 5mg Hg higher when sitting; difference in arms bp is 20%; inflate cuff 30mg HG until pt. radial pulse disappears; the sphygmomanometer should be reduced at 2-3mm/sec.
- Most common error in recording BP is applying cuff too LOOSELY and gives false elevated reading.
- Activate EMS immediately for adults and after 1 min for infant and child.
- **Rescue breathing** (has pulse but not breathing) - 1 breath every 5-6 sec (every 3 sec for child) or 10-12 breaths/min (15/20 for child/infant).
- For compressions, depress sternum 1.5-2mm(1-1.5 child, .5-1 for infant); 30 compression q 2 breaths for adult & kids but 15 compressions q 2 breaths for 2 rescuers; (80 -100/min) and 5:1 for infant.

- In anaphylaxis, pt should be in **Trendelenburg position** – body laid down and inclined at 45° w/feet & legs above head.
- Meperidine/Demoral – narcotic used to relieve moderate/severe pain and a cough suppressant.
    - Most widely used narcotic in hospitals;
    - Most abused drug by health professionals.
- Concomitant administration of Meperidine & MAO inhibitors (like Phenelzine) can cause life-threatening **hyperpyrexic** rxns that can end up in seizures/coma.

ANESTHESIA:

- Nitrous (**blue** tank!) = blood/gas partial coefficient of 0.47 so poorly soluble in blood and lack of potency; *excreted unchanged* by lung; only *inorganic* anesthetic; primary disadv: lack of potency.
    - Mainly effects reticular activating system and limbic system and CNS; room air = 21% oxygen so need pt. to receive this much oxygen when getting NO.
    - First symptom of nitrous is tingling of hands; good for timid/scared kids.
    - Keep reservoir bat 1/3 to 2/3 full; only inoganic substance.
    - Inhalation anesthetic w/ fastest onset of action! Oldest gaseous anesthetic;
    - Nausea is most common side effect; diffusion hypoxia if not give 100% O_2 at end of procedure; 100% oxygen CONTRAINDICATED in COPD pt.
    - Most common complication of nitrous is behavioral problem.
    - In conscious sedation, pt retains all reflexes but doesn't under G.A.
- Full cylinder of oxygen = 600L at 2000psi **(green** tank!)
- Spirometer measures respiratory air volumes:
    1. Functional Residual Capacity – amt of air remaining in lungs at end of expiration; **nitrous takes longer if more FRC**; FRC = ERV + RV.
    2. Tital Volume – amt of air remaining in lungs at end of expiration.
    3. Expiratory Reserve Volume – amt of air forced out of lungs in max. expiration.
    4. Inspiratory Reserve Volume – amt of air inhaled at max. inspiration.
    5. Vital Capacity = TV + ERV + IRV.
    6. Residual Volume – volume of air remains in lungs at all times (can't be measured)
    7. Total Lung Capacity = VC + RV
- Pulmonary volumes 20-25% less in females than males and larger in athletic ppl so nitrous adjustments needed.
- Stages of Anesthesia:
    I. **Amnesia & Analgesia** – administration of anesthesia; verbal responses (best monitor).
    II. **Delirium & Excitement** – loss of consciousness & onset of total anesthesia; may become violent with irregular BP & Respirations.
    III. **Surgical Anesthesia** – regular pattern of breathing and total loss of consciousness; eye mvmt stops!; when signs of resp or CV failure first appear; this stage has 4 PLANES!
        a. Pt has no pain reflexes.
    IV. **Premortem**– signals danger; decrease BP; cardiac arrest imminent; **medullary paralysis!**
        a. Eyes are greatly enlarged/maximally dilated pupils.
- Induction Phase: Stage I & II of G.A.; Maintenance Phase: keeps pt in surgical anesthesia; Recovery Phase: begins when surgery complete and anesthetic terminated and end when anesthetic eliminated from body.
- Most resistant part of G.A. is *medulla oblongata* (CV, vasomotor, resp. center)
- Most controllable root of GA is inhalation; sedation can be reversed rapidly when using inhalation.
- Emergency most often experienced during outpatient G.A. is respiratory obstruction.
- Minimum Alveolar Concentration (MAC) – alveolar concentration of anesthetic where 50% of pt unresponsive to surgical stimulus.
- Meyer Overton Theory – anesthetic begins when reaches certain molar conc. in hydrophobic phase.

- Second Gas Effect – potent agents administered with nitrous so agents delivered in increased amts to alveoli as gas rushes to replace nitrous absorbed by pulmonary blood.
- Eyes greatly enlarge and nonreactive to light – circulation to brain has stopped!
- Eyes taped shut during GA to prevent corneal abrasion.
- Cyanosis/↑Pulse – indicates oxygen is needed during GA.
- During G.A., pt loses laryngeal reflex so if blood & saliva collect near the vocal cords, they close (laryngospasm); this is an adverse effect of *ketamine*;
    - Laryngospasm – acute spasm of vocal cords and epiglottis that can result in airway occlusion and death.
    - Tx = oxygen & succinylcholine (cholinergic) – a skeletal muscle relaxant.
- Stridor (CROWING SOUNDS) – universal sign for laryngeal obstruction; cerebral blood permits up to 2 min of consciousness and lack of oxygen but neurologic damage at 3-5 min.
    - Invasive tx = 1) Tracheotomy – for long-term airway, not ER airways.
      2) Cricothyrotomy – for ER airway (last resort); for anaphylaxis; 1st epi, then oxygen, then cricothyrotomy if loses consciousness.
- Common *barbituates* for induction of anesthesia:
    - Thiopental = 2.5% solution; 3-5mg/kg produces loss of consciousness w/in 30 secs & recovery in 5-10 min; ½ life = 6-12 hrs; IV is irritating.
    - Methohexital (Brevital)= 1-2 mg/kg produces loss of consciousness in less than 20 sec & recovery time 4-5 min; ½ life = 3 hrs; less lipid soluble & less ionized at physiological pH;
        - metabolized in liver & excreted by kidney; causes hiccoughs – most common side effect;
        - MOST COMMON DRUG for G.A. anesthesia.
- Primary advantage of IV sedation is ability to titrate individualized dosages.
- Main target of INHALATION ANESTHETIC is brain; Lipophilic molecules; administration of anesthetic preceded by IV/IM barbituate w/ endotracheal intubation; 5 volatile liquids that require vaporization & may irritate respiratory tract & cause *malignant hyperthermiaI;* they cause ↓in arterial pressure.
    1. **Enflurane** – less potent but rapid onset with risks of seizures; CNS irritant effect.
    2. **Halothane** – powerful but toxin in adult liver; sensitizes heart to catecholamines.
    3. **Isoflurane** – combo with IV anesthetics; can cause heart irregularities.
    4. **Sevoflurane** – good for kids, less irritating with rapid awakening.
    5. **Desflurane** – heating component; irritating so used w/ IV agents but awaken faster than any other inhalant; has low blood:gas partition coefficient, but not used to induce anesthesia.
- Drugs to avoid in pts taking barbituates: phenothiazines, alcohol, antihistamines, & antihypertensives b/c these drugs enhance CNS depression of barbituates.
- At IV of barbituate, last tissue to become saturated as a result of redistribution is FAT (not vascular).
- Barbituates overdose may occur b/c its effective dose is close to the lethal dose; barbituates can cause hyperanagesia (sensitivity to pain).
- Most effective tx for resp. depression from overdose of barb is oxygen under positive pressue.
- Best anesthetic technique used in O.S. to avoid aspiration during G.A. is endotracheal intubation w/ pharyngeal packs.
- Effects **Speed of Induction** of inhalation anesthetics:
    1) Solubility
    2) Gas Partial Pressure
    3) Ventilation Rate
    4) Pulmonary Blood Flow
    5) Arteriovenous conc. gradient
- Malignant Hyperthermia: autosomal dominant, pharmacogenetic disease of skeletal muscle; no signs til given anesthesia; triggers are inhalation agents and depolarizing muscle relaxants.
    - sudden rapid rise of temp, tachycardia, sweating, cyanosis, increased CO_2, and muscle rigidity;
    - Tx = Dantrolene – impairs calcium dependent muscle contraction.

- IV Sedation - optimum site is *median cephalic vein* (lateral aspect of ant. of elbow); avoid brachial artery b/c will cause burning, blotch skin & weak pulse.
    - w/ 21 gauge needle, use valium = 1ml/min = 5mg of valium (contraindicated w/ glaucoma);
    - injection discontinue when eyelids droop; **Verrill's Sign = 50% ptosis**;
    - signs sedation working = blurry vision, slurred speech, and verrill's sign.
- Neurolept Anesthesia – and unconsciousness produced by combining Neuroleptic & Narcotic & NO; The neuroleptic and narcotic provide neurolept analgesia while the neuroleptic and NO provide anesthesia & unconscious state;
    - Pt. sedated but conscious and can answer questions; induction of anesthesia is slow but consciousness returns quickly.
    - Nitrous & Ethylene are useful ONLY for sedation & analgesia.
- *Post-op Hypotension* causes: 1) anesthesia/analgesic on myocardium
  2) intravascular hypovolemia
  3) rewarming vasodilation
  4) hypothyroidism
    - Tx = narcan (narcotic antagonist) or atropine (anticholinergic) if bradycardia.
- *Post-op Hypertension* causes: 1) post-op pain/anxiety
  2) hypercapnia (too much carbon) or hypoxia (lack of oxygen)
  3) overdistention of bladder
- For psychogenic rxn use following Rx 1 hr b/f appt:
  1. Diazepam (Valium): 5-10mg orally
  2. Pentobarbital (Nembutal): 50-100 mg orally
  3. Secobarbital (Seconal): 50-100 mg orally
  4. Promethazine (Phenergan): 25 mg orally
- Dissociative Anesthesia – method of pain control to decrease anxiety and produce trancelike state which feels like they are separated from their body but not asleep; useful in children.
    - Produces amnesia during procedure.
    - Tx = ketamine – trancelike state for 10-30 min but pain control 30-45min; sedative often given b/f ketamin to reduce anxiety; ketamine increases saliva, BP, & HR & causes delerium.
- Enteral sedation: use of pharmacoligical method to produce a minimally depressed level of consciousness.
- Somatogen Rxn: rxn from organic pathophysiologic causes.
- Phlebitis - irritation/inflammation of vein; maybe caused by **propylene glycol** in valium; common in smokers and women taking BCP; Tx = elevate limb, moist heat, IV antibiotics (Cefazolin – 1g) or anti-coagulants; S & S:
    1. Vessels feel hard, thready, or cord-like
    2. Extremely sensitive to pressure
    3. Surrounding area may be erythematous and warm to touch
    4. Entire limb may be pale, cold, and swollen.

LOCAL ANESTHESIA:

- Nerve loss of fct from LA affects in order from first to last:
    - PAIN>TEMP(cold then warm)>TOUCH>DEEP PRESSURE>PROPRIOCEPTION>SKELETAL MUSCLE.
- Loss of sympathetic fibers occur first; smaller and myelinated fibers are the first to fail to conduct.
- Sensory Fibers (pain) – high firing rate and long action potential duration.
    - ie: A delta and C fibers – small diameters so blocked sooner.
- Motor fibers fire at slower rate and shorter action potential.
    - ie: A alpha motor fibers – to skeletal muscle so blocked last.
- Vasoconstrictors like epi act on alpha receptors to constrict arterioles;
    - ie: cocaine – increases pressor activity of epi & norepi.

- **Vasoconstrictor** in LA:
  1. limits uptake of anesthetic into vasculatures so increase duration of LA & decrease systemic effects.
  2. Reduces toxicity b/c less LA is needed. (DOESN'T REDUCE CHANCE OF ALLERGIC RXN!)
  3. Reduce rate of vascular absorption thru vasoconstriction
  4. Help make LA more profound by increasing concentrations of LA at nerve membrane.
- The presense of vasoconstrictor doesn't prevent an intravascular injection/systemic absorption.
- Lidocaine, prilocaine, & etidocaine are for pregnant/lactating women.
- Novocaine = procaine = ester LA; procaine was prototype ester LA used.
- Volatile anesthetics not concern for COPD but nitrous is; nitrous is not contraindicated for asthma.
- 1cc of 2%lido = 20mg lido, .01mg epi, 6mg NaCl, .5mg Na–metabisulfate (preservative to stabilizes epi), 1mg methylparaben(preservative), NaOH to stabilize pH.
- 1.8cc of 2% lido = 36mg lido, .018 mg epi, 10.8mg NaCL, .9mg Na-metabisulfate, 1.8 methyparaben, & NaOH.
- Amide LA – biotransformation in **liver** but 20% excreted unchanged.
  - Longest DOA = bupivacaine (marcaine).
- Ester LA – biotransformation in **blood plasma** by pseudocholinesterase.
  - Tetracaine is commonly used;
- LA – produces anesthesia by reversibly binding to & **inactivating Na** channels; stops depolarization; site of action of LA is lipoprotein sheath of nerves.
  - So LA decreases membrane's permeability to Na and decreases membrane's excitability and prolongs refractory period.
  - More effective when pH>7; effectiveness depends on lipid solubility b/c 90% of nerve cell membrane is lipid; potency of LA increases w/ increased lipid solubility.
  - LA in ionized (cation) & non-ionized (base) with non-ionized for blocking Na channels; more non-ionized form has faster onset of action while ionized/acidic, like w/ inflammation, causes delay in onset; pH of LA = 7.8
  - When injection of LA in solution of increased pH due to buffers in body causes increase percent of noncharged LA so can readily penetrate lipid barriers; ↓pKa =↑ pH = ↑ onset of action.
  - Max dose of 2% lido w/ 1:100k epi = 3.2 mg lido/lb.; 1kg = 2.2 lbs.
  - For carbocaine w/o epi, max dose = 3.0 mg/lb.
  - Max dose of epi in cardiac pt. is 0.04mg or .2mg of levonordefrin. (equals 1 carp of 1:50,000 or 2 carps of 1:100,000 epi).
- Trismus is caused by IA injection *below mandibular foramen* into *medial pterygoid muscle*; arises 1-6 days after injection; IA injection into the Parotid gland may cause Bell's Palsy.
- *Buccinator* pierced when giving IA.
- If IA causes tingling or complete numbess of lower lip, may be due to trauma/piercing of nerve trunk by needle; more often occurs w/ mental block; may last 2wks-6mos but usually complete recovery.
- PSA (AKA – tuberosity block/zygomatic block) - blocks 1st, 2nd, & 3rd molars but need greater palatine inj for palate and infiltration for MB canal of 1st molar.
- MSA - block max. PMs & MB of 1st molar.
- ASA – blocks max. centrals, laterals, & canines.
- Long buccal inj. must be given to extract all molars and 2nd PM, don't need to for canine and 1st PM if giving IA & lingual block.
- Greater Palatine nerve – branch of V2 that profides soft tissue innervation to the posterior $^2/_3$ of the hard palate; inject b/w 2nd & 3rd max. molars, 1cm from palatal gingival margin toward midline.

ANALGESICS:

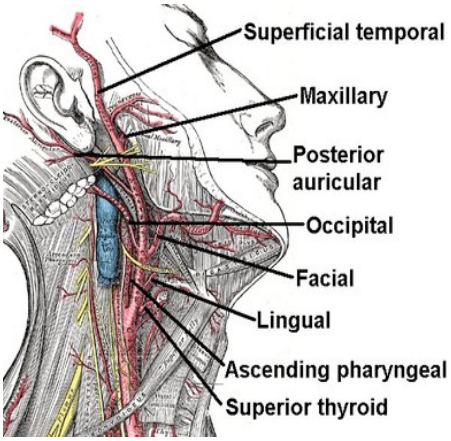
- Analgesics are under 2 categories = NSAIDS & Narcotics.
- Phenothiazines (anti-psychotic) are dangerous when mixed with sedative drugs (benzodiazepines/ tranquilizers) b/c phenothiazines can potentiate their action.

- Chloral Hydrate – sedative/hypnotic for pedo sedation.
- Proproxyphen (Darvon) – oral synthetic **opoid** analgesic structure similar to methadone.
    - Darvon Compound-65 = aspirin + caffeine + propoxyphene; AKA *Talwin Compound* (no euphoria).
- Acetominophen + Propoxyphen = Darvocent + Wygesic; used for tx of sever pain in dental procedures.
- Analgesics to avoid w/ RENAL disease = aspirin, acetaminophen, NSAIDS, meperidine, morphine.
- Barbituate Therapeutic Functions – anesthesia, anticonvulsant, anxiety; Rx interactions with barbs, CNS depressors, alcohol, and opoids.
- Anticholinergics: Tertiary's more readily penetrate CNS than quaternary.
    - Tertiary = atropine (penetrates CNS poorly & most common for DENTISTRY), scopolamine, benzotropine, dicyclomine, and trihexyphenidyl.
    - Quaternary = glycopyrrolate, ipratropium, probanthine.
    - In dentistry (often atropine), they DECREASE saliva, secretions from resp. glands (for G.A.), bradycardia.
    - they interfere w/ binding of Ach at its receptor;
- Pts premedicated w/ ATROPINE will exhibit mydriasis (dilated pupils); atropine is contraindicated for glaucoma and nursing mothers.
- Scopolamine (anticholinergic)– effective for preventing motion sickness; prolongs amnesia, psychic sedation, and **decreases** salivation; structurally similar to acetycholine; mydriasis (dilation of pupils).
    - depresses CNS so used as sedative before anesthesia and ad anti-spasmodic.

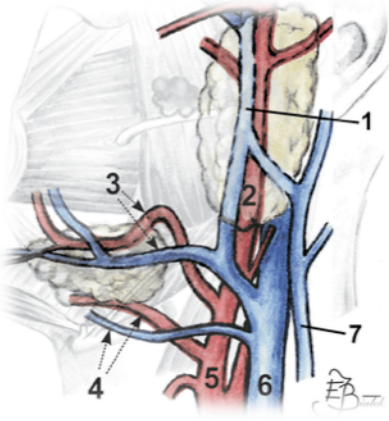
ANATOMY:

- Lymph Nodes: all pass to deep cervical LN.
    - Parotid LN: lymph from scalp above parotid, ant. wall of external auditory meatus, and lateral parts of eyelids and middle ear.
    - Submandibular LN: lymph from front of scalp, nose, cheek, upper & lower lip, ant. 2/3 of tongue, paranasal sinuses, floor of the mouth, max. & mand. teeth & gingiva.
        - Paranasal sinuses – series of mucous membrane lined air spaced that lighten skull & enhance voice resonance; within frontal, ethmoid, sphenoid, maxillary bones.
    - Submental LN: lymph from tip of tongue, floor of mouth below tip of tongue, mand. incisors & gingiva, center part of lower lip & skin & chin.
    - Lymph drainage:
        - Superficial cervical LN → Deep Cervical LN → Rt/Lt Jugular Lymph trunks → thoracic duct or ↓→ right lymphatic duct.
- Lymphadenopathy is most common swelling of submandibular triangle.
- Hard palate perforated by following foramina:
    - Incisive foramen: posterior to max. incisors; nasopalatine nerves & sphenopalatine artery.
    - Greater Palatine: medial to 3rd molar; greater palatine nerves and vessels.
    - Lesser Palatine: posterior to greater palatine foramen; lesser palatine nerves and vessels.
- Palatal Nerves:
    - Sensory – supplied by CN V-2 - Ant. Hard Palate = nasopalatine nerve.
      Post. Hard Palate = greater palatine nerve
      Soft Palate = lesser palatine nerve.
    - Motor – supplied by motor root of CN V – tensor veli palatine muscle.
      -other muscles innervated by CN X via pharyngeal plexus.
- Facial Nerves: from pons transverses facial canal of temporal bone and exits cranium thru **stylomastoid foramen;** 4 COMPONENTS:
    - Branchial Motor – muscles of facial expression, post. digastric, stylohyoid, and stapedius.
    - Visceral Motor – parasympathetic to lacrimal, submandibular, and sublingual glands.
    - Special Sensory – taste on anter. 2/3 of tongue, palate.
    - General Sensory – general sensation from skin of concha of auricle and small area behind ear.

- Pterygomandibular Raphe – where superior pharyngeal constrictor and buccinator insert; passes b/w tip of hamulus and internal surface of mandible at point posterior/superior limit of mylohyoid ridge.
    - Length of raphe increases as mandible moves.
- **Deep tendon of temporalis** and **superior pharyngeal constrictor** form V-shaped landmark for IA.
- Glossopharyngeal Nerve supplies parasympathetic secretomotor innervation for **PAROTID GLAND**;
    - start from lesser superficial petrosal nerve and leaves thru FORAMEN OVALE w/ V-3;
    - these preganglionic fibers synapse at **otic ganglion** and join auriculotemporal nerve (V-3) to distribute to gland.
- Parotid Gland – largest gland and purely SEROUS (like von Ebner's); divided by stylomandibular tunnel into deep "to ramus" and superficial "to ramus" lobes;
    - drained by STENSON's DUCT = which drains opposite max. 2nd molar and pierces **buccinator** but crosses **masseter**.
    - Arteries of Parotid are external carotid, superficial temporal, and maxillary arteries.
    - Lymph drainage to superior deep jugular LNs.
    - Mumps = viral disease of parotid gland



- External Carotid Artery supplies most of the head & neck, except brain (internal carotid & vertebral arteries); Splits into…
    1. *Maxillary Artery* – to muscles of mastication, all teeth, and palatal & nasal cavity.
    2. *Superior Temporal Artery* – supplies scalp.
- IA artery & Palatine arteries are branches of maxillary arteries;
- Mandibular teeth supplied by IA artery; Maxillary teeth – post = PSA artery, ant = ASA & MSA arteries.
- Lingual Artery Branches:
    a. *Suprahyoid* – supplies suprahyoid region.
    b. *Dorsal Lingual* – supplies dorsum of tongue.
    c. *Sublingual* – supplies floor of the mouth & sublingual gland.
    d. *Deep Lingual* – supplies anterior 2/3rds of tongue ; *terminal artery.
- Vertebral arteries arise form subclavian arteries and join basilar artery which is blood supply to brain stem & circle of willis.
- Venous return on both arches is Pterygoid Plexus of Veins.
- Submandibular Glands: located in submandibular/digastric triangle; innervated by CN VII which runs in chorda tympani & lingual nerve (V3) & synapses in submand. ganglion (*same for sublingual gland*).
    - Submandibular/WHARTON's Duct – emerges from anterior end of deep part of gland and passes forward along side of tongue and beneath mucous membrane of floor of mouth.
    - Blood supply from external carotid artery and facial artery.
    - MIXED gland with mucous & serous cells.
- Sublingual Gland – numerous small ducts (RIVIAN DUCTS) that open into the floor of the mouth secreting mostly MUCOUS acini w/ serous demilunes;
    - smallest salivary gland that contains mostly mucous.
    - Blood supply from sublingual artery.
    - consists mostly of Mucous acini capped with serous demilunes and is therefore categorized as a MIXED gland.
    - S-times sublingual ducts join to form Bartholin's Duct which drains into submandibular ducts.



- Von Ebner's Glands – around circumvallate papilla of tongue to wash food after tasted; PURELY SEROUS – only gland w/ parotid.

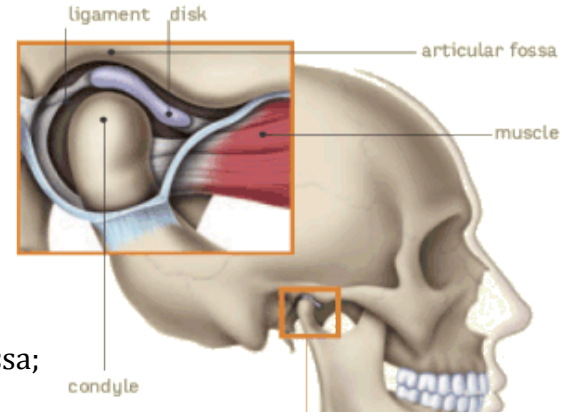
- Genial tubercles (4 of them) – lingual surface of mandible midway b/w superior & inferior borders; area of muscle attachment for suprahyoid muscles, if removed, tongue will end up flaccid, so genial tubercles are never removed!
- Carotid Sheath – deep to SCM, extends from base of skull to 1st rib and sternum; it contains:
  1. Carotid artery
  2. Internal jugular vein
  3. CN X
  4. Deep cervical LNs
- Facial (3) & Retromandibular (1) Vein → Internal Jugular (6)→ +Subclavian → brachiocephalic → Superior Vena Cava → Right Atrium of the heart.
- Mylohyoid Muscle – V-3; inferior to sublingual gland but superior to submand. gland; elevates: hyoid bone, base of tongue, and floor of mouth;
  - Gets in way of doing PA of mand. molars.
  - Mylohyoid and genioglossus detached when floor of mouth lowered surgically.
- Olfactory Nerve – sense of smell
- Optic Nerve – sense of sight
- Occulomotor Nerve – motor supply to all muscles, controlling lense shape & pupil size EXCEPT superior oblique muscle.
- Trochlear Nerve – motor supply to superior oblique muscle of the eye.
- Trigeminal Nerve – largest of 12 CNs; principal general sensory nerve to head & face.
  I. **Opthamic Div** (Superior Oblique Fissure) – sensory to cornea, scalp, eyelids, mucous membrane of paranasal cavity.
  II. **Maxillary Div** (Foramen Rotundum) – sensory for skin over maxilla, upper teeth & gums, mucous membrane of nose, max. sinus, & palate.
  III. **Mandibular Div** (Foramen Ovale) – innervates EIGHT muscles; motor of muscles of mastication, sensory from skin of face over mandible, lower teeth & gums, TMJ, mucous membrane of floor of mouth and anterior of tongue.
  - *V3 innervation:*
    - Cheek & Mand. buccal gingiva – long buccal nerve (sensory)
    - TMJ, Auricle, & external auditory meatus – auriculotemporal nerve (sensory)
    - Floor of mouth, mand lingual gingiva, ant. $^{2}/_{3rd}$ of tongue – lingual nerve (sensory)
    - Mand. teeth, skin of chin & lower lip – IA (sensory & motor)
  - 3 Nuclei of Trigeminal Sensory Nuclear Complex:
    1. **Mesencephalic Nucleus** – mediates proprioception (ie. Muscle spindle)
    2. **Main Sensory Nucleus** – mediates general sensation. (ie. Touch)
    3. **Spinal Nucleus** – mediate pain & temp from head & neck.
  - Proprioceptive first order neurons of the TMJ are in the *mesenphalic nucleus* of trigeminal nerve.
  - *Branchiometric motor fibers* innervate muscles of mastication, ant. digastric, mylohyoid, tensor tympani, tenso veli palatini.
- Buccinator fuction is to compress cheeks agains the molar teeth for sucking & blowing.
- Innervation of the Tongue - Motor – Hypoglossus (XII)
  Sensory – Ant 2/3rd - Taste = chorda tympani (VII)
  - Sensation = lingual branch of V3
  - Posterior - Taste & Sensation = Glossopharyngeal Nerve (IX)
- Abducens Nerve – motor supply to lateral rectus muscle.
- Facial Nerve – facial expression, submand., sublingual, & lacrimal glands; **taste for ant. part of tongue** (via chorda tympani), palate, & floor of the mouth and sensory input for outer ear;
  - exits cranium thru stylomastoid foramen.
  - Passes THROUGH parotid gland.
  - Facial nerve trauma destroys ability to contract facial musles on affected side of face and taste.
- Vestibulocochlear Nerve - 1) vestibular division = balance and head position.

2) cochlear division = sense of hearing

- Glossopharyngeal Nerve – motor to stylopharyngeus muscle and **PAROTID** salivary gland; **taste of post. 3rd of tongue**; sensory – bp receptors of carotid artery; sensory to tonsile, nasopharynx, & pharynx.
- Vagus Nerve – motor to pharynx, larynx, trachea, bronchi, lung, heart, esophagus, stomach, intestines, liver, pancreas, kidneys.
- Accessory Nerve – motor to SCM & trapezius, muscles of soft palate, pharynx, & larynx.
- Hypoglossal – motor supply to muscles controlling tongue EXCEPT *palatoglossus muscle*!!
  - Injury to hypoglossal nerve produced paralysis and atrophy on **affected** side which will **deviate to that side**; Dysarthria (inability to articulate) may also be found;
  - This injury is due to unopposed action of genioglossus muscle (pulls tongue forward); genioglossus muscle arises lateral and inserts at mandible midline.
  - If genioglossus paralyzed, tongue may cause suffocation.
- CN 3, 7, 9, 10 all have parasympathetic activity.
- Lateral Pterygoid Injury – mand. will deviate toward side of injury; when ankylosis of condyle or unilateral condyle fracture.
    - Will deviate AWAY from affected side w/ condylar hyperplasia from malocclusion.
    - Tx = closed procedure involving intermaxillary fixation.
    - Lateral Pterygoids – open, protrude, and move mandible side-to-side!
    - For RIGHT lateral excursive mvmts – LEFT lateral pterygoid is primary mover.
- Bone of maxilla MORE POROUS than mandible so can be infiltrated anywhere.
- Maxillary Sinus opens into **Hiatus Semilunaris** – groove in middle meatus of nasal cavity & contains frontal nasal duct & ant. ethmoid air cells.
    - Develops after perm. teeth erupted and continues growth thru adulthood.
    - Innervated by V2 – ASA, MSA, PSA & infraorbital nerve.
- Max. Sinusitis – pain in midface, cheek, & pain on percussion of max. posterior teeth.
- Ethmoid Sinusitis – pain b/w eyes & near bridge of nose.
- Frontal Sinusitis – forehead pain.
- Sphenoid Sinusitis – pain behind eyes or back of head.
- Tx for sinusitis: Ampicillin if cause is URI; PCN & amoxicillin if caused by odontogenic foci.
- Pterygomandibular Space – b/w med. Pterygoid muscle & mand. ramus w/ roof of lateral pterygoid muscle; contains IA nerve & artery & lingual nerve.
    - When draining abscess of pterygomandibular space intraorally, buccinator often incised.
- Infratemporal Fossa – behind maxilla; roof – greater wing of sphenoid; medial – lateral pterygoid plate; limited by coronoid process & ramus of mandible;
  - Communicates w/ pterygopalatine fossa thru pterygomaxillary fissure (cleft b/w lateral pterygoid plate & maxilla).
  - Communicates w/ orbit thru inferior orbital fissure (b/w maxilla & greater sphenoid wing). contains some muscles of mastication, max. artery, pterygoid venus plexus, mand. nerve, otic ganglion, & chorda tympani.
- Pterygopalatine fossa – small space behind & below orbital cavity; maxillary nerve & artery pass thru it.
- Submandibular Space – drains infection from mand. PMs and molar b/c below mylohyoid muscle.
    - Bound ant. & medial by tongue.
    - Bound laterally by deep cervical fascia
    - Bound inferiorly by hyoid bone
    - Split into *sublingual* (superior) & *submaxillary* (inferior) space by mylohyoid muscle; medial part of submaxillary space = submental space.
    - *Submental space* drains median of lower lip, tip of tongue, and mouth of floor; drains infections of mand. incisors & caninies b/c apices lie ABOVE the mylohyoid muscle.

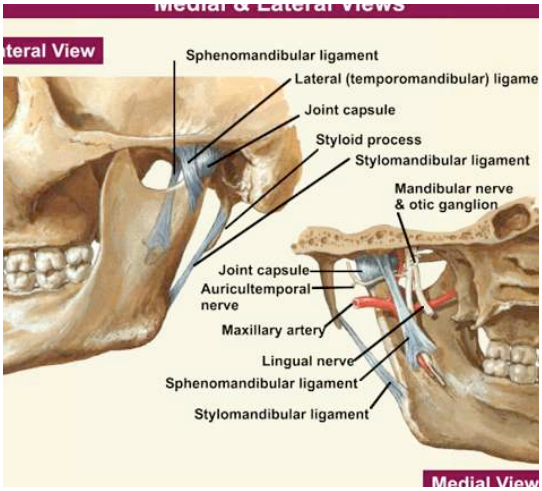
- Masticatory Space = masseteric space, pterygomandibular space, and temporal space; infections of this space usually dental origin (esp. mandibular molar region); needle tract infection from IA enter pterygomandibular space.
  - S&S of masticator space infection – TRISMUS, pain, and swelling; signs peak 3-7 days w/ spontaneous intraoral drainage on 4th & 8th day.
- Ludwig's angina – most common neck space infection (sublingual, submental, & submandibular).
- Lymphadenopathy is most common cause of swelling of the submandibular triangle tissues.
- Cavernus Sinus Thrombosis – blood clot w/in cavernus sinus which is a large channel of venus blood and contains CN III, IV, V1, V2, & VI; caused by Staph Aureus infection.
  - Infections of the face can cause septic thrombosis (often in opthalmic vein b/c no valves) of cavernous sinus; furunculosis & infected hair follicles are frequent causes.
  - TE of max. anterior teeth w/ infection can cause this; life-threatening!
  - Pts. presents w/ proptosis, orbital swelling, neurologic signs, and fever.

TMJ:



- TMJ not hyaline cartilage, just dense FIBROUS CONNECTIVE TISSUE;
- TMJ: has **ginglymoarthrodial** joint meaning it has hinge-like rotation and gliding mvmts; 4 components:
  1. *Mandibular Condyle* – functional part is superior & anterior head of condyle and covered with fibrous connective tissue; surface covered w/ vascular layer of fibrous C.T.; long axis oriented mediolaterally.
  2. *Articular Fossa* – anterior 3/4th of larger mandibular fossa; **non-functional** part of joint; bounded in front by articular eminence & behind by temporal bone; concave!
  3. *Articular Eminence* – ridge extends mediolaterally in front of mand. fossa; functional part of joint; lined w/ thick fibrous C.T.; convex!
  4. *Articular Disc/Meniscus* – **biconcave, fibrocartilaginous disc** b/w condyle & mand. fossa; gliding surface of condyle and central point is avascular and NO nerves (only periphery).
- Articular disc varies in thickness; has 2 thicker band than central bands:
  - Posterior band – thickest band and attached to retrodiscal tissue; Retrodiscal Tissue (bilaminar zone) – posterior loose connective tissue; highly vascularized & innervated.
  - Anterior band – contiguous w/ capsular ligament, condyle, & superior belly of lateral pteryogoid muscle.
- Posterior aspect of condyle is round and convex while anterior inferior aspect is concave.
- Condyles are NOT symmetrical/identical;
- Palpate external posterior surface w/ mouth open when examining.
- Condyle held in place by *collateral/discal ligaments* (restricts mvmts of disc away from condyle during function) at medial and lateral poles of condyle.
  - Held in position anteriorly by lateral pterygoid muscle.
  - When collateral ligaments become torn, condyle displaced **anteriomedially** causing clicking sound & disc displacement.
- Arteries to TMJ: 1) superior temporal artery
  2) max. artery & external carotid artery
  3) smaller masseteric
  4) posterior deep temporal
  5) lateral pterygoid arteries anteriorly
- Venous drainage thru diffuse plexus around capsule.
- Fibrous capsule of TMJ – innervation Auricular Temporal Nerve (V3)
- Anterior region of TMJ – masseteric nerve (V3) and posterior deep temporal nerve (V3)

- SENSORY innervation of TMJ – trigeminal nerve; Anterior TMJ supplied by Masseteric nerve, Posterior TMJ supplied by Auriculotemporal nerve; NO motor innervation.
- **TMJ Ligaments:**
  1. Temporomandibular Ligament (lateral ligament) – provides lateral reinforcement & **prevents inferior & posterior displacement** of condyle; *main stabilizing ligament and only ligament provides DIRECT support.
     a. From articular eminence to condyle.
     b. Keeps condyle head in place if fractured.
  2. Sphenomandibular Ligament – attaches to lingula of mandible; most often damaged in IA block; limits mvmt.
     a. IA nerve passes lateral to this ligament.
  3. Stylomandibular Ligament – attaches to angle of mand and styloid bone.



- 3 groups responsible for Displacing Condyle:
  1) Masseter, med. Pterygoid & temporalis - ELEVATE MANDIBLE so upward & medial displacement.
  2) Digastric, mylohyoid, geniohyoid, and lateral pterygoid – DEPRESS MANDIBLE so inferior & posterior displacement.
  3) Lateral Pterygoid – forward & medial displacement; however, right lateral pterygoid cant contract during protrusion.
- **Crepidation** from degeneration of condyle (maybe osteoporosis).
- *Dull thud* – self-reducing subluxation of condyle.
- Preauricular – best surgical approach to exposing TMJ.
- Submandibular Approach (Risdon Approach) – surgical approach for ramus of mandible and neck of condyle.
- Trauma is common cause of TMJ ankylosis but ankylosis is most common complication of Rheumatoid Arthritis.
- **Disorders of TMJ:**
  1. Myofascial Pain Dysfunction: main cause of TMJ pain; unilateral dull pain that increases with muscular spasm; masticatory muscle spasm and limited jaw opening;
     a. Complaints: referred pain, headache, otalgia(ear pain), tinnitus, burning tongue.
     b. Often due to stress; Tx = nightguard.
  2. Internal Derangement: when disc pulled **anteriorly** by superior head of lateral pterygoid muscle;
     a. With reduction – disc anterior at rest but returns when opening and closing; pain and clicking may occur; $^1/_3$ of population; normal opening or "S" shaped.
     b. w/o reduction – disc always anterior, no sound but max opening < 30mm.
     c. *Subluxation*/dislocation/open lock – pt cant close after keep open for a long time due to posterior band stretching and joint traveling in front of eminence;
     d. Tx = conservative for 4-6 wks and the consider surgery; 95% improve w/o surgery.
     e. There isnt a reproducible reciprocal click; most disc displacements are ANTERIOR & MEDIAL.
  3. Degenerative Joint Disease (osteoarthritis): $1^o/2^o$ trauma; old ppl b/c of wear and tear; asymptomatic unless it is in young ppl where it is more severe;

BIOPSY:

- After tissue remove for biopsy, place in 10% formalin (4% formaldehyde) that's 20x the volume of the tissue.
- Biopsies: Incisional – take only part of lesion.
  Excisional – entire lesion removed.
  Needle – aspirational biopsy
  Exfoliative Cytology – pap smear

- All oral ulcers caused by trauma will heal in 2 wks so biopsy needed if longer than 2 weeks;
- also biopsy: pigmented lesions, tissue associated w/ paresthesia, & when a lesion enlarges, hyperkeratotic changes in lesion, if doesn't responsd to antibiotics for 14 days, or persistent swelling.
- Always aspirate a central bone lesion to rule out vascular lesion.
- Stethoscope is used to listen for bruit (unusual sound that blood makes when it rushes past an obstruction (called turbulent flow) in an artery).
- All leukoplakias should be biopsied because they are premalignant.
- Block preferred for anesthesia rather an infiltration for biopsy; anesthesia > 1cm away from lesion.
- Get some normal tissue as well as diseased for biopsy.

IMPLANTS:

- **Bone-Implant Integration:**
  1. Fibrous-Osseous Integration: connective tissue encapsulated implant w/in bone; success rate 50% over 10 yrs; not see often w/ newer materials.
  2. Osseousintegration: direct connection b/w living bone & implant (w/o soft tissue); ONLY **endosseous & transosseous implant;** *most predictable longterm stability*; uses radiographic & light microscopic analysis;
  3. Biointegration: implant interface w/ bioactive materials (hydroxyapetite) or bioglass that bonds directly to bone; develop bone faster than non-coated but cant tell after 1 year.
- Best time to augment soft tissue to develop keratinized tissue around implant is stage II surgery.
- Guided Tissue Regeneration: surgically eliminated bony defect around implant to decrease C.T. growth while increasing bone; don't heat bone >116^{o}F/47^{o}C.
- For successful implant:
  1. need adequate transfer of force and biocompatibility.
  2. Histologically 35-90% bone contact, C.T. adhesion above bone, and non-inflamed JE.
- For implant, use low speed and high torque handpieces; use superfloss/yarn.
- Need 10mm bone height to place endosseous/root form implant; need 2mm b/w apex of mand. post. implant & IA canal; implants placed 3mm apart and 1mm apart away from adjacent tooth;
- Titanium/Titanium alloy are most common for 2-stage endosseous implants;
- Smoking affects healing of bone & tissue so NO IMPLANTS!
- Pt. w/ uncontrolled systemic disease – use extreme caution w/ implant placement.
- Max. ant. implants – highest failure rate;
- Mobility is most common sign of implant failure.
- Max. amount of taper for draw of overdenture = 15^{o}.
- 2 types of Implant Placement:
  1. Submerged – 2 stage surgical procedure to uncover fixture.
  2. Nonsubmerged – only 1 stage.
- 3 Categories of Implants:
  1. Endosseous Implants – surgically inserted into jawbone; most used implant; 2 forms:
     a. Root-formed implants – cylindrical shape, titanium; 3 phases – surgical, healing, & prosthetic.
        i. Most popular! 80% of all implants are ENDOSSEOUS (into bone).
     b. Blade Implants – flatter in appearance for insufficient bone width but adequate depth; titanium; either single/2 stage;
  2. Subperiosteal Implants – rides on bone; fits on top supporting structures under mucoperiosteum.
  3. Transosseous Implants – inserted into jaw bone but penetrates entire jaw and emerges at opposite entry site(usually chin); indications: very atrophic mandible.

EXTRACTIONS:

- Maxillary 3rd occasionally displaced to: 1)Max. Sinus – use caldwell-luc approach to remove.
  2) Infratemporal space – may need oral surgeon.
- If root tip 2-3mm or less gets into max. sinus then NO tx needed.

- Palatal root of max. 1st molar most often dislodged into max. sinus.
- Caldwell-Luc Approach – opening made into max. sinus by incision into canine fossa above PM roots; figure 8 sutures, antibiotics, nasal spray & decongestant.
- Most frequently impacted teeth are mandibular 3rds, the max. 3rds, then MAX canines.
- Root tip of mand. 3rd molar disappears into submandibular space.
- IA nerve often lies buccal to roots of mand. thirds;
- bone rarely removed from lingual aspect of mandible b/c likeihood of damaging lingual nerve.
- When removing mylohyoid ridge, be careful to protect lingual nerve.
- Most common causes of paresthesia to lower lip is removal of mand. 3rd molars.
- Extract max b/f mand and post b/f ant.
- After removing max. teeth, upper jaw should be at same height as dentist's shoulders.
- Mandibular arch parallel to floor when doing mand. extractions.
- Contraindications for Extractions: -acute infection w/ uncontrolled cellulitis
  -acute pericoronitis or stomatitis or ANUG
  -malignant disease or irradiated jaws.
- Direction of luxating primary Max. molars – palatal; perm. max molars – buccal.
- If perm PM wedged b/w bell-shaped roots of primary tooth – section & remove.
- Do NOT use cowhorns on mand. primary molars.
- Dead Space: wound in area that remains devoid of tissue after wound closure; usually fill w/ blood causing hematoma & high potential for infection;
  - Tx = resolves on its own or open and drain.
  - Eliminate by: close wound in layers, apply pressure, use drains to remove bleeding, place packing into void til bleeding stops.
- Fracture of maxillary tuberosity most common result from extraction of erupted max. 3rd molar; if tuberosity fracture but intact, reposition and suture;
  - ***beware of lone molar – often ankylosed & emits atypical, sharp sound on percussion.
- When removing mand. tori – use envelope flap design w/ no vertical component.
- **Maxillary Tori** often seen b/f age 30 & more in females; removal of max. palatal tori:
    - Stent fabricated; Use double Y-incision
    - Use osteotome to remove small portions
    - Use bur/bone file to smooth area
    - Irrigate & loosely place sutures & use stent to prevent hematoma & support flap.
    - Most often located at midline of hard palate.
- **Classifications of Impactions:** w/ difficult of removal from *easy to hard* w/ MAND 3rds:
    - Mesioangular (43%)
    - Horizontal (3%)
    - Vertical (38%)
    - Distoangular (6%)
  - OPPOSITE for max. molars!!! Distoangular is easiest!
  - Most mand. 3rds angled in lingual direction.
- If sinus communication after TE – no add'l surgical tx…
  - Post-op: avoid nose blowing for 7 days.
    Open mouth when sneezing
    Avoid vigorous rinsing
    Soft diet for 3 dys.
  - Meds: 1) Afrin (local decongestant), 2) antibiotics (amoxicillin), 3) actifed (systemic decongestant)
- If sinus opening moderate (2-6mm), place figure 8 suture over socket.
- If sinus opening larger (>7mm), close socket w/ flap procedure.
- Class II lever used for tooth extractions.
- Luxation – loosening of tooth by progressive severing of PDL; luxation forces perpendicular to long axis of tooth; can use rotational forces on single rotted teeth.

    - Mvmts firm and primarily to the facial w/ secondary mvmts to the lingual.
- Teeth resistant to crush but not resistant to shear so beaks applied to line parallel w/ long access of tooth.
- Ideal time to remove impacted 3rds – when roots 2/3rds formed b/c bone more flexible and no root curves & rarely fracture; around age 17-21 yrs. old.
- Older individual have most postoperative difficulties.
- Bite on tea bag if bleeding persists after TE; the *tannic acid* promotes hemostasis.
- Autotransplanting teeth – often 3rd molar replacing carious mand. 1st molar;
    - Most important criteria is adequate bone support in recipient sign.
    - Best result if donor tooth's roots are $^1/_3$ to $^2/_3$ completed root development.
    - Most likely cause of failure is chronic, progressive external root resorption.
    - Universal sequelae of **allogenic** tooth transplant is ankylosis & root resorption.
- Periocoronitis – causes food debris & bacterial waste products and tissue often traumatized during mastication; max 3rds most frequent contributing factor to pericoronitis of mand. 3rds.
    - S&S – pain, bad taste, inflammation, pus; can be a recurrent condition and an abscess can form unless cause is removed.
    - Definite criteria for removing 3rd molars; Tx = irrigate area, place on antibiotics and rinse with warm saline solutions and once symptoms relieved, then extract.
- Post-op Ecchymosis – trauma to underlying blood vessels > 1cm; common after TE's in elderly pts b/c fragile vessel walls; pt. complains of diffuse, non-painful, yellowing discoloration of skin; may first present as bluish lesion; more predisposed in pts w/ clotting & bleeding disorders; tx = heat.
- An abscess should NOT be contraindicated to a TE b/c infections resolve quickly after tooth is removed.
- Conditions that require prophylaxis prior to oral surgery: (NOT pacemakers)
    1. Prosthetic heart valve
    2. Rheumatic Valve disease
    3. Most congenital heart malformations.

INCISIONS & SUTURES:

- *Advantages of interrupted suture*: most common, independent, strength, & flexibility; if one sutures is loose, the other ones stay put; disadv: time.
- *Advantages of continuous suture:* ease and speed of placement, distribution of tension over whole suture; more watertight closure.
- Sutures should NOT be closed under tension and should be 2-3mm apart; suture placed from mobile tissue into fixed tissue and from thin into thick tissue.
- Suture size based on strength & diameter; as diameter decreased, the 0s added or numbers followed by 0s = 000 = 3-0 ----same size; 9-0 has least strength and smallest diameter.
- B/c sutures are foreign body, smallest diameter suture sufficient; most OS use 3-0 or 4-0 sutures.
- Resorbable sutures evoke intense inflammatory rxn; not for skin wounds; recommend non-resorbable for TE sites and remove in 5-7 dys.
- Monofilament sutures consist of material from single strand and resist infections; RESORBABLE:
    - *Plain gut* – sheep intestine, susceptable to rapid digestion but retained for **5-7 days**; most severe tissue rxns w/ this suture material.
    - *Chromic gut* – chromatized to be more resistant to digestion and retained for **9-14 days**; moderate tissue rxn.
    - *Polyglycolic Acid* – doesn't enzyme break down, undergoes slow hydrolysis, less stiff but more expensive.; minimal tissue rxn.
- Polyfilament sutures – multiple fibers either braided or twisted; NON-RESORBABLE:
    - *Silk* – braided, black, inexpensive, good handling but severe tissue rxn.
    - *Nylon* – strong, not used orally but is suture material of choice for facial lacerations.
    - *Polypropylene* – least tendency for inflammation but fair handling.

- Non-resorbable sutures should be removed in **5-7 days.**
- Vertical releasing incision made at tooth line angle.
- 3 types of incisions: 1) linear – straight line incision for apicoectomies.
  2) releasing – adding vertical leg to horizontal incision; for TE & augmentations; incision along tooth line angle.
  3) semi-lunar – curved incision for apicoectomies.
- #15 scalpal universally used for OS procedures.
- Sutures over single extraction socket are NOT usually placed unless papillae have been excised, bleeding from gingiva, or gingival cuff torn/lose;
- Most common cause of post extraction bleeding is failure of patient to follow post-extraction instructions.
- Osteoradionecrosis - Most serious complication after extractions from areas previously irradaited; condition of non-vital bone that can result in tissue injury;

HEALING:

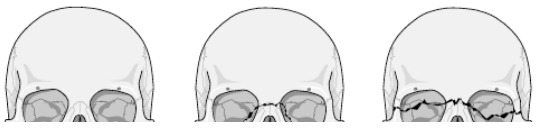
- 5 stages of healing – (same as soft tissue – inflammation → fibroplasia → remodeling)
  1. Clot formation
  2. Granulation tissue (can be retarded by Glucocorticoids)
  3. Connective tissue
  4. Fibrillar bone
  5. Bone recontouring.
- Stages of Wound Healing:
  1. *Inflammatory Stage* (vascular & cellular phase) – neutrophils & lymphocytes predominate w/ macrophages(most important inflammatory cell for wound healing).
  2. *Proliferative Stage* (fibroblastic stage) – collagen & new blood vessels produced; mediated by fibroblasts.
  3. *Maturation Stage* (remodeling stage) – collagen fibers continue to increase tensile strength.
- Bone heals by primary and secondary intention like soft tissue:
  1. **Primary Intention** – endosteal (in bone) & periosteal (w/in connective tissue covering bone) proliferation; occurs when incomplete fracture or reapproximating fracture ends of bone; little fibrous tissue w/ *minimal* callous formation. (Ie – well repaired reduced bone fractures)
     a) Minimal re-epithelization and collagen formation; allows wound to be sealed w/in 24 hrs.
  2. **Secondary Intention** – endosteal proliferation; used when fracture bones >1mm apart; lots of fibrous tissue & callus is formed (which ossifies). (Ie – TE sockets, poorly reduced fractures)
     b) Re-epithelization via migration from wound edges; site fill w/ granulation tissue; slower healing;
- Bone healing in 3 overlapping phases:
  1. Hemorrhage – first 10 days.
  2. Callus formation – 10-20 days primary callous; 20-60 days secondary callous.
  3. Functional Reconstruction – 2-3 years to completely reform a fracture.
- 3 Phases of Hemostasis:
  1. *Vascular* – vasoconstriction, begins immediately after injury.
  2. *Platelet* – platelets and vessels become sticky; mechanical plug of platelets seal off cut vessels; seconds after injury.
  3. *Coagulation* – blood loss in surrounding areas coagulate thru extrinsic and common pathways while vessels in area of injury use intrinsic and common pathways; slower than other pathways.
- 5 ways to obtain hemostasis – hemostat on vessel, heat cut vessels, suture ligation of vessel, pressure dressing, vasocontrictive substances like epi.
- Dry Socket – increased fibrinolytic activity causing increased lysis of blood clot; most commonly following TE of mand. molars; causes: smoking, mouthrinses, hot liquids, trauma, oral contraceptives.

- Symptoms – pts develops severe, dull throbbing pain 2-4 days after TE; foul odor and taste and extraction site filled w/ necrotic tissue which delays wound healing.
- Tx = -flush w/ warm saline but no currettage.
  -Place eugenol sedative dressing & replace every 48 hrs til asymptomatic.
  -Analgesics but NO antibiotics needed.

- 3% hydrogen peroxide agent for debridement of intraoral wounds.
- Order of tx for acute infection = localize infection, IND, then culture; if infection produces cellulitis of region involved, called **induration** (appears hard, dense, and brawny).
- Incision & Drainage – only performed for acute infection if localization of infection has occurred.
  - Culture after InD if antibiotics is not sufficient to resolve abscess.

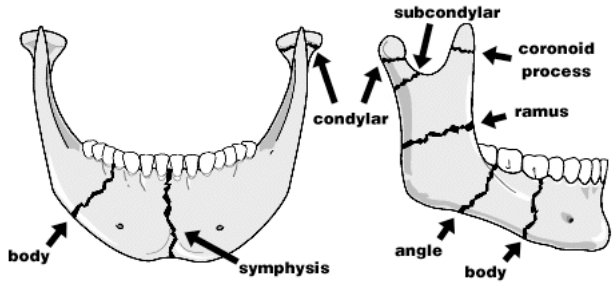
FRACTURES:

- most common cause for facial fractures = auto accidents (80%); highest incidence of fractures in young males ages 15-24.
- Fracture type prevalence: -Zygomaticomaxillary complex (40%) – tripod fracture
  -Lefort Fracture I (15%), II (10%), III (10%).
  -Zygomatic arch (10%)
  -Maxilla alveolar process (5%), Smash Fractures (5%)
- Control of airway is vital to any treat of pt w/ facial fractures.
- 4 reason fracture doesn't heal:
  1. Ischemia – poorly vascularized so ischemic necrosis after fracture.
  2. Excessive mobility – healing prevented & pseudoarthritis or pseudojoint occurs.
  3. Interposition – of soft tissue and occur b/w fractured ends
  4. Infection – **compound** fractures have tendency to become infected.
- Fat embolism often sequela of fractures.
- Inappropriate healing: delayed healing (>6wks), non-union, mal-union.
- Mandible must be immobilized for 3-6 wks for fractures: (4 forms)
  1. Barton Bandage – simplest form; used 1st aid measure til definitive therapy.
  2. Intermaxillary Fixation – use prefabricated arch bars and wire teeth together; class way to mobilize fracture after closed reduction; most common technique for IMF is to use prefabricated arch bars.
  3. External Skeletal Fixation – if IMF not satisfactory, use screws, pins, and use cold cure acrylic bar to hold screws in place.
  4. Direct Intraosseous Wiring – combing w/ IMF and traditionally used after open reduction.
- Closed reductions often for condylar neck fractures; often used when both fragments have teeth;
- Zygomatic arch fractures – best seen by **submental vertex view**; may cause damage to superior orbital fissure; complications: parasthesia, hematoma of sinus, & impaired occular muscle balance.
- Zygomatic complex fractures are most common midface fracture but 2nd most common facial fracture behind nasal bone fractures.
- Infraorbital Rim Fractures: presents w/ numbness of upper lip, cheek, and nose.
  - **Water's view** best to evaluate orbital rim areas.
- S&S of Mand. Fracture: *malocclusion*, lower lip numbness, mobility, pain, bleeding at fracture.
- Open reduction – direct exposure and reduction of fracture thru surgical incision;
  - Procedure: open reduction then direct intraosseous wiring with IMF for 3-6wks.
  - Most common site is angle of mandible; Performed for displaced angle or body fractures.
  - Best used to reduce a fracture when teeth are missing in one or more of the fractured segments.
- Fracture of angle of edentulous mandible often displaced anteriorly and superiorly.
- Le Fort Fractures: from severe frontal blows; associated w/ intracranial damage, CSF leak; types:

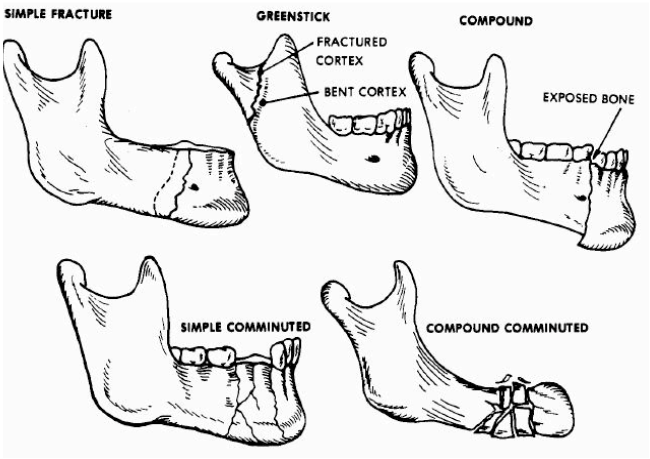


I. Horizontal fracture thru maxilla just above max. teeth; causing **open bite**!
II. Fracture which maxilla separated from facial skeleton w/ separated bone being **pyramidal** in shape and includes palate and max. teeth; S&S - edema, ecchymosis, hemorrhage, and nose bleeding.
III. Horizontal fracture where entire maxilla and 1/more facial bone separated from upper face; pts. have restricted mand. mvmt.

- Blows to maxilla cause maxilla to be driven backward and downwards; may cause open bite or impingement of airway.
- Location & extent of fracture based on direction & intensity of blow & points of weakness in mand.
- Common sites for fractures: Body - 30-40%
  Angle - 25-31%
  Condyle – 15-17%
  Symphysis – 7-15%
  Ramus – 3-9%
  Coronoid – 1-2%



- Bilateral dislocated fractures of condylar necks cause anterior open bite and cant protrude mandible.
- Unilateral fracture thru neck of condyle caused forward displacement of head of condyle.
- <u>Mandibular Fractures:</u>
  1. *Simple* – divides bone in 2 parts w/ no external communication; it's a closed fracture w/no laceration of mucosa/facial tissue.
  2. *Compound* – open fracture that communicates w/ outside env't; may have lacerations of oral tissue; infections are common.
  3. *Communited* – multiple fracture of single bone; may be single/compound.
  4. *Greenstick* – fracture only thru corticol portion of bone w/o complete fracture of bone; closed fracture; often in children w/ orbital and frontal fractures common.

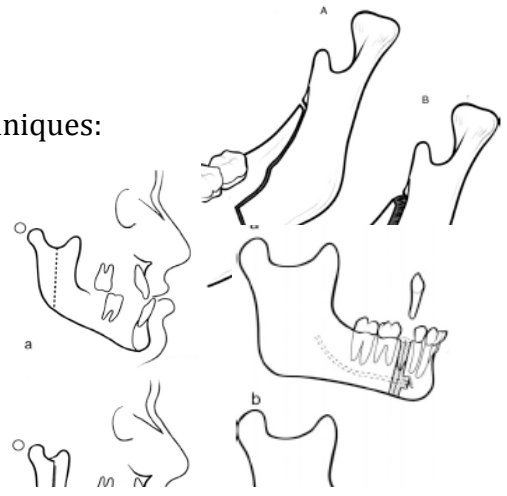


- Most common complication of fracture is infection.
- Most common sign of mand. fracture= malocclusion.
- First step to treat mid-facial fractures is to re-establish a proper occlusal relationship.
- Line of fracture determines whether muscle will be able to displace the fracture segments from original position:
  - favorable fracture – if fracture line prevents fracture displacement by muscle pull.
  - unfavorable fracture – if fracture line results in muscle pull displacing fracture.
- Maxillary fractures have a greater tendency to produce facial deformities than mandibular fractures.

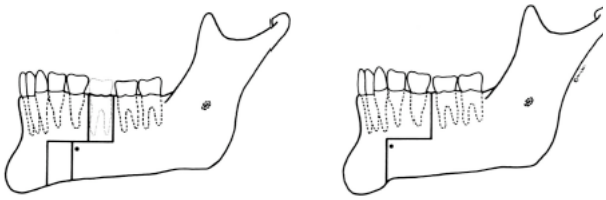
<u>GRAFTS:</u>

- Ideal graft is replaced by host bone and assists osteogenic processes of the host.
- 3 forms of grafts:
  1. <u>Cortical Grafts</u> – withstand early mechanical forces but require more revascularizing.
  2. <u>Cancellous Grafts</u> – increase healing rate; most abundant supply from iliac crest; disadv – inability to provide mechanical stability.
  3. <u>Corticocancellous Grafts</u> – provides mechanical stability and increase osteogenesis but not as well as cancellous grafts b/c layer of nonporous corticol bone.
- Iliac crest provides bone marrow for grafting mandible and maxilla and ridge augmentation.
- Costochondral rib graft for cartilaginous part simulating TMJ & condyle.

- For fixating bone grafts – bone plates, biphasic pins, titanium mesh, and intraosseus wire.
- Greatest osteogenic potential occur w/ autogenous cancellous graft and hemopoietic marrow.
- Classes of Grafts:
  1. Autogenous Graft – tissue from same individual; common in OS but frequently present surgical/technical problems;
     i. Mandible is most commonly resected for oncological surgery of all facial bones.
  2. Allogenic Grafts – tissue from individual of same species but not genetically related; often human cadaver bone; 3 forms:
     i. *Fresh frozen* – rarely used b/c transmission of disease.
     ii. *Freeze-dried* – osteoconductive but no osteogenic or osteoinductive capabilities; used in conjuction w/ autogenous grafts.
     iii. *Demineralized Freeze-dried* – lack strength but has osteoconductive and osteoinductive capabilities; exposed bone morphogenic proteins.
  3. Xenogenic Grafts – tissues from donor of another species. (both xenogenic and allogenic grafts are most common grafts for rejection).
  4. Isogenic Grafts – tissue from same species and genetically related to recipient.
  5. Alloplastic Graft – synthetic, inert, man-made synthetic materials;
     i. often hydroxyapatite is used to augment the mandible; granular/particle is used; it is biocompatible & non-resorbable; hydroxyapatite bonds physically and chemically to bone;
     ii. May cause chin prominence erosion and unpleasant cold sensation in implant region.
- 3 processed bone repairs/regenerates:
  1. osteogenesis – ability to form new bone in graft by transplanting viable osteoblasts.
  2. osteoconduction – ability of graft to allow vascular and cellular invasion by host.
  3. osteoinduction – ability of graft to stimulate differentiation of mesenchymal cells into osteoblasts at recipient site.
- Sliding Genioplasty – surgically improving a person's chin; horizontal sliding osteotomy; removing horseshoe shaped piece of chin bone and sliding either forward/backwards and fixing it with screws.
  a. Problems with alloplastic materials for genioplasty: migration, erosion, & cold sensation.
- High-speed hand pieces can cause tissue emphysema or air embolus when removing bone during O.S.; the tissue emphysema can be caused by air pressure syringes or atomizing spray bottles.
- Main reason to use water irrigation when cutting bone is b/c heat generated by drill affects bone vitality and don't want to burn bone. Duh.
- **Marsupialization**, *decompression*, and *Partsch operation* refer to creating a surgical window in wall of cyst which is uncovered or deroofed and *emptied*.
  - Marsupialization is tx for ranula when cyst is large and close to vital structures; if recurrent ranula also excise sublingual gland; **cyst lining made continuous with oral cavity**.
- Enucleation – total removal of cyst and preferred tx of cysts; tx for congenital & odontogenic cysts & mucoceles.
- Operculectomy – removal of operculum – flap of tissue over unerupted/partially erupted tooth.
- Frenum provides support or restricts mvmt; 3 Frenectomy Techniques:
  1. Diamond excision & 2. Z-Plasty – both effective when mucosal & fibrous tissue is narrow; these techniques relax the pull of the frenum.
  3. V-Y advancement – preferred when frenal attachment has wide base; less scarring and good for lengthening tissue.
- Mandibular Ramus Sagittal Split Osteotomy – common performed mand. orthognathic procedure; used to either advance or set back the mand.;

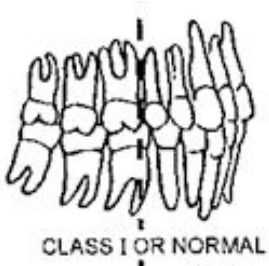


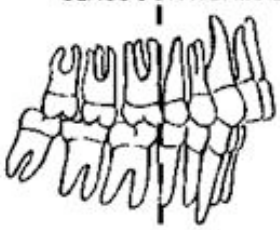
    - position of condyle UNCHANGED;
    - for correcting Class 2 malocclusion.
- Vertical Ramus Osteotomy – to set mand. posteriorly for prognathism.
- Vertical Body Osteotomy – TE mand. teeth (PMs) bilaterally and set mand. back.; corrects class 3 malocclusion.
- Le Fort I Osteotomy – most common to fix max. retrognathia.
- Step Osteotomy – for mand. prognathism, retrognathism, asymmetry, and apertognathia; 3 independent pieces.

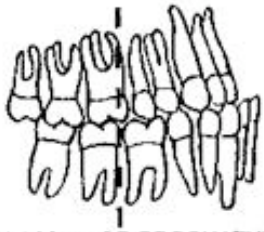


ORTHODONTICS

OCCLUSION:


CLASS I OR NORMAL


CLASS II OR RETROGNATHIC


CLASS III OR PROGNATHIC

- Class I – MB cusp of max. 1st molar lines up w/ BUCCAL GROOVE of mand. 1st molar; Orthognathic profile; 70% of population;
  - → Most prevalent characteristic of Class I malocclusion is CROWDING;
  - → If crowding <4mm – strip some enamel off interproximals of mand. teeth.
  - → If crowding >4mm – extraction;
- Class II – MB cusp of max 1st molar b/w Mand. 2nd PM & 1st Molar; max. canine mesial to mand. canine; retrognathic profile (**overbite**); 25% of population; convex profile;
  - → Div 1 – ALL max. incisors protruded in extreme labioverision & mand. incisors tipped forward;
  - → Div 2 – Max. centrals tiped palatally & in retruded position (linguoversion) but Lateral incisors tipped labially & mesially (labioversion); if this only occurs unilaterally = SUBDIVISION;
- Class III – MB cusp b/w mand 1st molar & 2nd molar; max. canine distal to mand. canine; prognathic profile (underbite); max. incisors tipped lingually.
  - → "f" or "v" sounds affected by Class III malocclusion;
- Pseudo-Class III Malocclusion – mandibular incisors forward in relation to maxillary incisors when in C.O. but can move mandible back w/out strain.
  - → Most instances edge to edge; tx = elimination of CO-CR discrepancy.
- Sunday Bite – forward postural position of mandible which is adopted by people w/ people w/ Class II profiles in order to improve esthetics;
- Physiological Occlusion – may not be ideal occlusion but its an occlusion that adapts to stress of function & can be maintained.
- Pathological Occlusion – cant function w/out contributing to own destruction; may cause:
  1. Excessive tooth wear
  2. TMJ problems
  3. Pulpal changes
  4. Periodontal changes
- Bimaxillary Dentoalveolar Protrusion – in both jaws the teeth protrude; Signs are
  1. Separation of lips at rest
  2. Severe lip strain
  3. Prominence of lips in profile view
- Common dental condition that can benefit from ortho tx prior to prosthetic tx is long-term loss of mand. 1st molar; better to tip 2nd molar distal than move mesial.
- On a child, if permanent 1st molar extracted, best approach is to allow 2nd molar to mesial drift into that area;

PRIMARY & MIXED DENTITION:

- Mixed Dentition Analysis (Transitional Analysis) – determines space available vs space required; based on tooth size; Procedure:
  1. Measure MD of mand. incisors & add together
  2. Measure space available
  3. Subtract #1 from #2; a negative number indicates crowding;
  4. Measure the space available for the canine & premolars on each side of the arch
  5. Calculate from the prediction table the size of the canine & premolars.
  6. Subtract #6 from #5 on each side; negative number indicates crowding.
  7. Then add these 3 numbers together (# from incisor crowding/space, # of right canine & PM crowding/space, # of left canine & PM crowding/space); (-) = crowding, (+) = space!

- Moyer's Mixed Dentition Analysis – predicts size of unerupted canines & PMs by looking at MAND. INCISORS that have already erupted; the incisors determine both mand & max posterior teeth.
  - → Predicts the amount of crowding AFTER the permanent teeth erupt.
  - → Both MAX & MAND space determined from MAND. incisors.
- Mandibular anterior crowding usually results from tooth size-arch length deficiency;
- Supervision of child's occlusion most critical at ages 7-10 because malocclusion most identifiable in children 7-9 yo.
- Leeway Space – serves to accommodate PERMANENT CANINES (which are larger than primary);
  - → the difference in sum of MD width of primary canine, 1st molars, 2nd molars & permanent canine, 1st PM, & 2nd PM.
  - → Mand. leeway space = 3-4mm; Max. leeway space = 2-2.5mm.
- Permanent successors often smaller than primary successors;
- *Late Mesial Shift of 1st molar* – loss of arch length when primary 2nd molar are lost & 1st permanent molar shifts into leeway space.
- Permanent MAND. canines erupt FACIALLY/RIGHT IN LINE to primary canines;
- In max. & mand. arches, perm. tooth buds for incisors **lie LINGUALLY** & **APICALLY** to prim. incisors causing mandibular incisors to erupt LINGUALLY;
- Permanent teeth normally move **OCCLUSALLY** & **BUCCALLY** while erupting;
- Max arch = 128mm; Mand. arch = 126mm.
- Primary molar relationship = STEP relationship;
- Mesial Step (primary teeth)= distal surface of mand. 2nd molar is mesial to distal surface of max. 2nd molar; normally results in Class I occlusion of perm. teeth;
- Flush-Terminal Plane – the NORMAL relationship of primary molars in primary teeth; **most common** initial relationship; when distal surfaces of mand. & max. 2nd molars are end to end relationship;
  - → permanent teeth don't erupt immediately in normal occlusion, first Class II, but around 10/11 yo (during late mesial shift), the move into Class I occlusion;
  - → if late mesial shift doesn't occur, then stays in Class II occlusion.
  - → Terminal plane relationship determines future anteroposterior positions of permanent 1st molars!
- Distal Step – creates permanent Class II occlusion;
- Mesial Step, Flush-Terminal Plane, Distal Step are all determined by observing 2nd Primary Molars!
- Child w/ class III malocclusion, they will have edge to edge contact w/ primary incisors;
- **Primate Space** – Max. arch = b/w Lateral incisors & canines.
  Mand. arch = b/w canines & 1st molars.
  - → Spacing is normal thru out the primary dentition, but these areas are the most NOTICEABLE.
  - → Caused by growth of dental arches.
- If no spacing & primary teeth were in contact b/f loss, a collapse in arch after loss of primary incisors is almost certain;
  - → not true for loss of perm. incisors – space closure occurs rapidly whether spacing/not.
- Most common cause of malocclusion – inadequate space management following early loss of prim. teeth;
- Premature exfoliation of primary canine may indicated arch length deficiency & may cause lingual & lateral collapse/migration of mandibular anterior teeth;
- Premature loss of primary max. 2nd molar produces Class II malocclusion;
- As child matures, face becomes less convex.
- The most reliable indicator of readiness of eruption of succedaneous tooth is extent of root development;

OPEN BITE & CROSSBITE:

- Thumbsucking may cause **Class II malocclusion**, **unilateral/bilateral crossbite**, **constricts MAX. arch**, **anterior crossbite, proclination of max. incisors, & retroclination of mand. incisors.**
  - → As the hand rests on the chin, it retards mandibular growth, causing Class II.

→ Constriction of the maxilla due to pressue from buccinator, NOT negative pressure;

- ANTERIOR OPEN BITE (**APERTOGNATHISM)**is most common sequelae of *digital sucking habit*; assymmetrical w/ normal posterior occlusion; it is a malocclusion;
- Skeletal open bite (long face syndrome) is most often associated w/ mouth breathing.
- Ant. crossbite rare b/c mandibular growth lags behind maxillary growth, unless Class III relationship; most often associated w/ retention of primary teeth;
- Cross bite is associated w/ **jaw-size discrepancy, hereditary, reverse over-jet,** & **scissor bite**;
- Neither crossbite or open bite are caused by tongue thrusting.
- **Anterior Crossbite** in primary teeth is indicative of 1)Skeletal Growth Problem & 2) Class III malocclusion; Results from: 1) Labial situated supernumerary tooth
  2) Trauma
  3) Arch Length Discrepancy
  - → should always be treated in mixed dentition stage;
  - → most often associated with prolonged retention of a primary tooth;
  - → most essential factor in correction is amount of MD space available.
  - → More common in african-americans, while open bite is more common in caucasians.
- Delayed treatment of anterior crossbite can cause loss of arch length and the most important factor is space availability mesial distally.
- Anterior crossbite best retained by normal incisor relationship achieved by treatment (the overbite) not appliances; Anterior crossbite – easily retained after ortho tx by overbite achieve during tx.
- **Overbite** (deep bite) – vertical overlapping; **Overjet** – horizontal overlapping.
- **Reverse overjet** – Class III malocclusion w/ > 2 max. anterior teeth in linguoversion;
- **Scissorbite** (bilateral lingual crossbite) – from narrow mandible or wide maxilla; when posterior mand. teeth lingual to maxillary teeth.
- Open bite may cause tongue thrust swallowing but tongue thrust swallowing doesn't cause anterior open bite;
- Posterior Crossbite: -- Transverse plane problem
  -- corrected ASAP
  -- Thoroughly diagnosed as dental, functional, or skeletal orgin.
  -- maybe corrected w/ palatal expansion – causes diastema & expansion of nasal floor;
  -- Maybe associated w/ mandibular shift
  -- correct in 1st stage of tx along with MILD ant. crossbite (2nd stage is severe).
  -- skeletal crossbite demonstrates smooth closure to C.O.
  --due to prolonged thumb sucking & anterior crossbite!
- The MOST COMMON active tooth movement in primary dentition is to correct a posterior crossbite – a TRANSVERSE plane of space problem.
- 1st step of treatment for crossbite is maxillary expander – 1-2 months of turn key then another 3 months; then braces are used b/ of spacing produced by expansion;
- An anterior open bite may make it difficult to make sounds – **th, sh, ch; also s, & z** (due to lisp).
- Large diastema can also cause a lisp so difficult to produece **s & z** sounds;
- Irregular incisors can make it difficult to produce sounds **t & d**.
- Class III can cause difficulty with **F & V** sounds.

BONE GROWTH:

- Don't confuse bone growth and bone formation; Once bone is formed, it then grows by appositional growth = growth by addition of new layers on top of previous formed layers;
- Bone formation begins in embryo where mesenchymal cells differentiate into either fibrous membrane or cartilage; 2 paths of bone development:
  1. **Intramembranous Ossification** – in membrane of CT; osteoprogenitor cells in membrane differentiate into osteoblasts & a collagen matrix is formed undergoing ossification.

    a. How mandible & maxilla are formed; also flat bones of skull & clavicle.
2. **Endochondral Ossification** – take place in HYALINE CARTILAGE; cartilage cells replaced by bone cells (osteocytes replace chondrocytes), matrix is laid down & Ca & PO_4 are deposited;
    a. Forms long & short bones – ethmoid, sphenoid, temporal bones;

- Mandible & Maxilla grow DOWN & FORWARD;
- **Mandible Growth -** 1) growth in condyle increases anteroposterior dimension of mandible.
  2) increase resorption of anterior border of ramus
  3) increase apposition of bone on posterior border of ramus
  4) apposition of alveolar bone increases superior/inferior dimension of mandible.
  → Space b/w jaws is provided by growth of condyle – major site of VERTICAL GROWTH due to cartilage proliferation;
  → Resorption occurs along anterior surface of ramus while bone apposition occurs along posterior surface of ramus;
  → Mand. main growth site – CONDYLAR CARTILAGE; The "V Principal" of growth is illustrated with growht of mandibular ramus;
  → Growth at mand. condyle during puberty usually results in increase in posterior facial height.
  → The main growth thrust is UPWARD & BACKWARD directiong causing the body of the mandible to move DOWNWARD & FORWARD, same as Maxilla;
- **Maxilla Growth -** 1) growth at spheno-occipital & sphenoethmoidal junctions.
  2) growth at nasal cartilaginous septum
  → Sutures for secondary growth: 1) Frontomaxillary suture
  2) Zygomaticotemporal suture
  3) Pyramidal process of palatal bone
  4) Alveolar process.
  → Maxillary arch elongates, moves posterior, and increases height.
  → Posterior movement is due to resorption of labio-alveolar sufrace & apposition of the lingual surface;
- Posterior bone remodeling at ramus ceases before 3rd molar eruption often causing impaction;
- <u>Cartilage Growth:</u>
  1. **Appositional Growth** – recruit fresh cells (chondroblasts) from perichondral stem cells & add new matrix to surface.
     a. Appositional growth occurs below covering layer of bone (periosteal); periosteum has other fibrous layer & cellular inner layer of osteoblasts which lay down bone;
  2. **Interstitial Growth** – mitotic division & deposition of more matrix; chondrocytes already established in cartilage;
     a. ie – Condyle (hyaline cartilage), nasal septum, sphenooccipital synchondrosis;
     b. Hyaline cartilage differs from bone in that hyaline cartialge may grow by interstitial growth.
- At age 6, greatest increase in mandible size occurs distal to 1st molars;
- Bone deposition in tuberosity region responsible for lengthening arch & posterior mvmt;
- Alveolar growth responsible for increase in height of maxillary bones;
- Incisor crowding due to LATE mandibular growth.
- Alveolar process bone exists only to support teeth so if tooth fails to erupt, alveolar bone will never form in that area; if tooth extracted, alveolus resorbs.
- **Late mandibular growth** is theory that best explains why there is a strong tendency for mandibular anterior crowding in later teens & early 20s;
  → The concept is that incisor crowding develops as the mand. incisors & possibly the entire mand. dentition move DISTALLY relative to the body of the mand. late in mand. growth;
  → Mandible undergoes more growth in late teens than in the maxilla;
  → Late incisor crowding occurs in ppl w/o 3rds so not a factor in crowding but late mandibular growth is a critical variable.

- Most rapid losses in arch perimeter are usually due to mesial tipping & rotation of permanent first molar after removal of primary second molar.

ORTHO PROCEDURES/TREATMENT:

- Most important aspect of ortho = RETENTION; accomplished w/ fixed/removable appliances;
- Gradual withdrawal of ortho appliance is of NO value!
- Indirect Method of Bonding Brackets is more technique sensitive and reduces chairside time; controls FLASH (excess of resin); used when visibility is a problem;
- 35-50% unbuffered phosphoric acid is used as bonding agent before direct bonding of orthodontic brackets (for 1 min).
- topical flouride should NOT be used before etching b/c it decreases solubility of enamel;
- **Indications for using Bands instead of Bonding Brackets**:
    1. Better anchorage for greater tooth movement
    2. Teeth that need both lingual & labial attachment
    3. Short clinical crowns
    4. Tooth surfaces that are incompatible w/ successful bonding.
- GI cements are replacing Zinc Phosphate because 1)Fluoride releasing & 2) Retentive Strengths.
- Frozen Slab Technique – allows more powder into liquid increasing strength.
- Cross-Elastics – from maxillary lingual to mandibular labial can be used to correct single-tooth crossbite;
- Serial Extraction – orderly removal of selected primary/permanent teeth;
    → For severe Class I malocclusion in mixed dentition w/ insufficient arch length; if >10mm.
    → 1st extract Primary Canines, 2nd – Primary 1st Molars, 3rd – Permanent 1st PMs;
    → key to success is to extract the 1st PMs before the permanent canines erupt.
    → must leave 6-15 months b/w extractions; for support & retention, use for… Mandible – lingual arch, Maxilla – Hawley Appliance.

ANGLES:

- Facial Profile Analysis (Poor man's Ceph Analysis) – same info as lateral ceph but less detailed; give the following info:
    1) Anterior/Posterior Position/Protrusion of Jaws
    2) Lip Posture & Incisor Prominence
    3) Vertical Facial Proportions
    4) Inclination of Mandibular Plane Angle
- Within lower 1/3 of anterior face height, the mouth should be about 1/3 of the way b/w nose & chin.
- Steep Mandibular Plane Angle – correlates w/ long anterior facial vertical dimension & anterior open bite malocclusion;
- Flat Mandibular Plane Angle – correlates w/ short anterior facial vertical dimension & anterior deep bite malocclusion.
- Max-Mand Plane Angle – angle b/w mand. plane & max. plane = **27° (+/-4)**; greater the value, the longer the face height.
- High mandibular plane angle is most significant complication of molar uprighting – can cause increased open bite & loss of anterior guidance;
- Long face predisposes to Class II, while short face predisposes to Class III.
- SNA Angle – angle formed by line from SELLA TURNICA to NASION to Pt. A;
    → SNA > 82° = Max. Prognathism.
    → SNA < 82° = Max. Retrognathism.
- SNB Angle – angle formed by line from SELLA TURNICA to NASION to Pt. B; defines sagittal location of mand. denture base;
    → SNB > 80° = Mand. Prognathism
    → SNB < 80° = Mand. Retrognathism
- **ANB Angle:** - ANB angle = 2° = Class I

- ANB angle < 0^o = Class III
- ANB angle > 4^o = Class II

- Physiological/developmental age judged by wrist/hand x-ray; Landmarks – 1) Ulnar Sesamoid
  2) Hamate Bones
- Frankfort-Horizontal Plane – connects **Porion** (mid point of upper contour of metal ear rod of ceph) & **Orbitale** (lowest point on inferior margin of orbit); best representation of natural orientation of the skull;
- Some important Ceph. Landmarks:
  - Sphenocciptal Synchondrosis – junction b/w occipital & basisphenoid bones.
  - Sella – midpoint of cavity of sella turnica;
  - Pt. A = subspinale = innermost point of premaxilla
  - Pt. B = supramentale = innermost point on contour of mandible;
  - Pogonion – most anterior point of contour of chin
  - Menton – most inferior point on mandibular symphysis (bottom of chin)
  - Gonion – lowest posterior point of mandible w/ teeth in occlusion
  - Nasion – anterior pt of intersection b/w nasal & frontal bones;
- Ceph includes measurements from hard & soft tissue;
- Most stable area to evaluate craniofacial growth is ANTERIOR CRANIAL BASE because of its early cessation of growth.
- Cephs often show 7-8% magnification; good for tooth-tooth, bone-bone, & tooth-bone relationships.

APPLIANCES:

- Band & Loop – has limited strength so only replaces 1 tooth; most often used when PRIMARY FIRST MOLAR prematurely extracted.
- Distal Shoe – used when 2nd PRIMARY MOLARS lost very prematurily & prior to eruption of 1st perm. molars; prevents mesial tipping of permanent molar;
- Lingual arch space maintainer – used if loss of bilateral molars but incisors erupted; 2 bands around either prim. 2 molars or perm. 1st molars & wire rests on cingula of incisors;
  → DOESN'T restore function & should be completely passive.
- Nance Appliance – for premature bilateral loss of max. primary teeth; small acrylic button that rest on palatal tissues that are attached to bands that are bilaterally cemented on permant max. molars;
  → Prevents MESIAL rotation & drifting of perm. max. molars it is attached too.
- Removable appliances arent used often b/c appliance not being work or easily broken/lost.
- Quad Helix Appliance – fixed appliance, not functional but contains 4 helices (2 ant, 2 post); for POSTERIOR CROSSBITE w/ digit sucking habit;
- Functional Appliances are either tooth-borne or tissue-bourne;
- **Tooth Borne Appliances**:
  A. Activator – advances mand. into edge to edge postion to induce mand. growth & inhibit max. growth; **improves deep bite in Class II cases**;
  B. Bionator – trimmed down version of activator appliance for comfort;
  C. Herbst – fixed/partially removable; metal rod & tube telescopic apparatus attached bilaterally to max. 1st molar & mand. 1st PM; **used to posture mandible forward & induce growth**;
  D. Twin Block – 2 piece acrylic appliance to posture mandible forward w/ help of occlusal incline & guiding planes & bite blocks(determines vertical separation);
- **Tissue Borne Appliances**:
  A. Frankel Functional Appliance – serves to EXPAND ARCH by padding against pressure of lips & cheeks; protrudes mand. forward & downward; REMOVABLE functional appliance used for abnormal soft tissue patterns;
- Best method for tipping max &mand. anterior teeth is with **FINGER SPRINGS** which are attached to removable appliance; most common problems: 1) lack of pt cooperation
  2) Poor design/lack of retention

3) Improper activation
4) Root apex movement

- Force of Spring = **F α dr4/13**; d = distance of spring, r = radius of spring; force of spring is inversely proportional to length of spring.
- **Z Springs** – can also be used for tipping but excessive heavy force & lack of range of motion;
- **Buccal Springs –** used to try & regain space by pushing a tooth mesial/distally, but may cause rotation of that tooth;
- Maxillary incisor rotation not fixed til after all permanent teeth have erupted except for crossbite which should be corrected ASAP.
- Whip-Spring Appliances – used to de-rotate 1 or 2 teeth;
- Fixed Ortho appliances offer controlled tooth movement in all 3 planes of space;
- 3 planes of space in malocclusion – Antero-posterior, Transverse, & Vertical;
- Removable Appliances – generally restricted to tipping teeth;
    a. **Attached Removable Appliances**:
        i. Active Appliance – contains extraoral traction devices (headgear), lip bumpers, active plates, vacuum formed appliances;
        ii. Passive Appliance – contains bite planes, splints, & retainers.
    b. **Loose Removable Appliances**.
- Indications for Removable Appliance: 1) Retention after comprehensive tx
    2) Limited tipping movements
    3) Growth modifications during mixed dentition
- Components of Removable Appliance:
    1) Retentive Component – retains appliance's function w/ clasps.
    2) Framework/Base – acrylic, provides anchorage
    3) Tooth-moving elements – spring/screws
    4) Anchorage Component – resists active components
    5) Active components – springs, screws, elastics;
- For appliances to be effective, must be capable of exerting torque.
- 4 basic components of **Fixed Appliances**: 1) Bands
    2) Brackets
    3) Archwires
    4) Auxilliaries (elastics/ligatures)
- Alloys for ortho – **Stainless steel** (can be supplied soft & w/ good formability), **Chromiun-Cobalt** (increased strength & spring), & **Titanium**.
- Ideal wire material should possess: -- Increased strength
    -- Decreased stiffness
    -- Increased range
    -- Increased Formability
- Loops & helices incorporated in archwires to increase activation range;
- Edgewise Appliance – bands on all teeth, tubes on last molar & brackets on all teeth; 1 labial used as a time - .0125x.028 in diameter, which fits in bracket slott of .022" wide from top to bottom;
    → Best appliance for tx of comprehensive malocclusions of permanent dentitions;
    → Variations include double/tandem brackets & narrow (.018) slottle brackets.
    → Components - 1) Siamese twin bracket – maxillar anter. Teeth
        2) Broussard buccal tube – segmented arch technique to intrude teeth.
        3) Straight wire bracket
        4) Bracket w/ .022x.028 rectangular slot;
    → Straight-wire Appliance – version of edgewise w/ features that allow placement of ideal rectangular archwire w/o bends;
- 1st order bend in ortho wire is HORIZONTAL PLANE;
- Begg Appliance – uses round wires which fit loosely in vertical slot of bracket;

- Hawley Retainer – incorporates clasps on molar teeth & a characteristic bow w/ adjustment loops, spanning from canine to canine; palatal coverage w/ acrylic – major source of anchorage;
    - → Tx for pt w/ **excessive overbite**; can be max or mand.
    - → MOST COMMON REMOVABLE RETAINER.

HEADGEAR:

- Advantage of extraoral anchorage (headgear) is it permits posterior movement in an arch and doesn't touch opposing arch;
- Req'd force for anchorage= 250g for 10hrs/day; Req'd force for traction= 500g for 14-16 hrs/day.
- Headgear extraoral components – neck strap, chin cup, & head cap.
- Headgear intraoral components – facebow.
- **Facebow –** intraoral headgear component; has outer & inner bow; inner bow relates to resistance of tooth & effects anchorage/traction;
- High-Pull Headgear – produced distal & upward force on maxillary teeth & maxilla; headcap & facebow; **Helps w/ Class II, Div. I Malocclusion w/ open bite**.
- Cervical-Pull Headgear – neck strap & facebow; produces distal & downward force on maxillary teeth & maxilla; possible extrusion of max. molars;
    - → causes opening of bite & 1st molar moves distally & forward growth of maxilla decreases;
    - → for **Class II, Div. I malocclusion**.
- Straight Pull Headgear – places force in straight distal direction from maxillary molar; for Class II, Div 1 malocclusion;
- Reverse Pull Headgear – extraoral component supported by chin, cheek, forehead; for Class III malocclusion, for protruding maxilla.

PATHOLOGY:

- Hyperparathyroidism – causes premature exfoliation of primary teeth;
- Primary Failure of eruption is caused by eruption mechanism itselft but can be caused by:
    1. **Hereditary Gingival Fibromatosis**
    2. **Down's Syndrome**
    3. **Rickets**
- Localized caused of failed/delayed eruption are:
    1) Congenital Absence
    2) Abnormal Position of Crypt
    3) Lack of space
    4) Supernumerary tooth
    5) Dilacerated roots.
- Prolonged ortho tx has long been associated w/ caussation of inflammatory periodontal disease;
- Mouth Breathing causes:
    1. Skeletal Open Bite (longface syndrome) – worsens over time;
        a. anterior open bite = **APERTOGNATHISM**.
    2. Narrow face
    3. Narrow oropharyngeal space
    4. Chronic rhinitis, deviated nasal septum.
    5. Tonsilitis, allergies
- Conditions w/ multiple supernumerary teeth:
    1) Gardner's Syndrome
    2) Down's Syndrome
    3) Sturge-weber syndrome
    4) Cleidocranial Dysplasia
- Supernumerary teeth have predilection 2:1 for males; most common site is b/w CENTRALS;
- An impacted mesiodens can cause diastema but an INVERTED mesiodens can cause delayed eruption of centrals;
- **Oligodontia** – absence of 1/more teeth; more females than males; smaller than avg tooth size ratio.

MISCELLANEOUS:

- Dental arch form determined by interaction of environmental influences on genetic pattern.
- Malocclusion is MOST OFTEN hereditary.
- 98% of 6 year olds have diastema while 49% of 11 year olds do too;
- Diastema closes after canines erupt if <2mm but if >2mm willl not close so need tx:
  - → If abnormal frenum – do ortho tx THEN do a frenectomy.
  - → Use lingual arch w/ finger springs
  - → Use Hawley appliance w/ finger springs
  - → Cemented ortho band w/ inter-tooth traction.
- Maxillary canine is most commonly impacted tooth after thirds; in older pts, there is an increased risk that impacted tooth is ankylosed.
- Tx of impacted tooth – during surgical exposure, flaps reflected so tooth is ultimately pulled into arch thru KERATINIZED TISSUE not alveolar mucosa;
- Ectopic Eruption – tooth erupts in wrong place; common in MAX. 1st MOLARS & MAND. INCISORS;
  - → Common in **Class II** in 2-6% of population & correctin in 60% of population;
  - → If max. 1st molar – tx is place brass wire b/w primary 2nd molar & permanent 1st molar;
- Uprighting a molar can take 6-12 months:
  - → Tx – fixed edgewise ortho appliance w/ .022" or .018" wire sizes
  - → Tipped 2nd molar should be banded b/c masticatory forces;
  - → Severly **lingually** tipped mand. molar MORE DIFFICULT to control & upright.
  - → High mandibular plane angle also make it very difficult to upright a molar (may cause open bite).
  - → Stabilization should last til lamina dura & PDL reorganize (2-6 months);
  - → Retention w/ well-fitted provisional.
  - → Slow progress in molar uprighting – due to occlusal interference;
- **6 Types of Tooth movement:**
  1. *Tipping* – crown moves in 1 direction & root tip in opposite direction (often w/ appliance); common w/ anterior incisor teeth;
  2. *Translation* (bodily movement) – root movement in same direction as tooth movement; difficult!
  3. *Extrusion* – displacement of tooth from socket in direction of eruption.
  4. *Intrusion* – movement into socket along long axis of tooth; difficult!
  5. Torque – root movement while crown is stable; Mesial distal root mvmt = AKA- UPRIGHTING.
  6. Rotation – revolving tooth along long axis; need adequate retention to prevent relapse.
- Side toward tooth movement = osteoclasts – break down bone;
- Side away from tooth movement = osteoblasts – bone forming cells.
- Collagen fibers (like rubber bands) in supra-alveolar tissue are responsible for relapse of orthodontically rotated teeth as well as redevelopment of spaces b/w orthodontically moved teeth
  - → Primary component of gingiva & get stretched during ortho tx.
- Circumferential Supracrestal Fibrotomy – simple incision in sulcus to bone; incises collagen fibers inserted into root of tooth; eliminates potential relapse & allows new fibers to form in new position.
  - → Good candidate for procedure is a rotated maxillary lateral incisor.
- Collagen fibers in SUPRA-ALVEOLAR tissue are primarily responsible for relapse of orthodontically rotated teeth & for redevelopment of spaces b/w orthodontically moved teeth.
  - → Collagen fibers are main component of attached gingiva.

OSHA & PATIENT MANAGEMENT

BEHAVIORAL SCIENCE:

- Behavior is determined, purposeful unit of activity;
- 4 major fields of behavior: Personal Social, Motor, Language, & Adaptive;
- Most researchers believe changes in behavior are a prerequisite to changes in attitude;
- The **most effective** way to teach oral hygieve skills is by having pt participate in repeated supervised trainining sessions;
- Maintaining a 4 year old child's healthy dentition starts w/ educating the parent;
- Behavior Modification – type of psychotherapy that attempts to modify observable, maladjusted behavior patterns by substituting a new response or set of responses to a given stimulus; *5 Types*:
  1. **Classical Conditioning** (pavlovian/respondent conditioning) – a form of learning in which a previously neutral stimulus comes to elicit a given response through associative training;
     a. Operates by associating one stimulus w/ another;
  2. **Operant Conditioning** – consequence of a behavior is in itself a stimulus that can affect future behavior; a form of learning where the person undergoing therapy is rewarded for correct response & punished for incorrec response;
     a. 4 types: Positive & Negative reinforcement, omission, & punishment;
     b. **Behavior Shaping** (successive approximation) – an operant conditioning technique in which a new behavior is produced by providing reinforcement for progressively closer approximations of the final desired behavior; sometimes called *Stimulus Response Therapy*;
  3. **Aversion Conditioning** – technique in which punishment or painful stimuli are used in suppression of undesirable behavior; ie – Hand over mouth technique;
  4. **Observational Learning** (modeling/behavior shaping) – behavior acquired through initiation of a behavior observed in a social context;
     a. 2 stages – observational learning acquisition and actual performance of behavior;
  5. **Systemic Desensitization** – a technique used to eliminate maladaptive anxiety associated w/ phobias; construction by the person of a hierarchy of anxiety producing stimuli & general presentation of these stimuli until they no longer elicit an initial response of fear;
- **Flooding** – intense & prolonged exposure to a feared stimulus while using coping skills;
- **Biofeedback** – teaching 1 to have control over his or her physiological arousal thru the use of auditory/visual monitoring of arousal level;
- **Cognitive Coping** (reframing) – assisting pts in changing their thinking about something to a more adaptive or realistic thinking style;
- The Premack Principle – making a behavior that has a higher probability of being performed contigent upon (used a reinforcement) the performance of a less frequent behavior may increase performance of the less frequent behavior;
- **Extinction** – identifying the positive consequences or reinforcements that maintain a behavior & ceasing or withholding these reinforcments or consequences;
- **Incompatible behavior/stimulus control** - use of an incompatible behavior to decrease the frequency of an undesirable behavior;
- **Eye contact** is the primary non-verbal cue that 2/more people use to regulate verbal communication;
- The best way to show a pt you care about what they are telling you is to use **eye contact;**
- When presenting treatment plans always use **open-ended questions**; they are the MOST EFFECTIVE way to help pts understand the proposed tx plan;
- **Constructive Aggression –** an act of self-assertivenes in response to a threatened action for purpose of self-protection & preservation;
- **Destructive Aggression** – act of hostility unnecessary for self-protection/preservation directed toward an external object or person;
- *Anxious* pts are usually considered the most difficult pts; most pts who are anxious have a traumatic experience in dental/medical setting;

- **Fear** – anticipation of a threat elicited by an external object; it is distinguished from anxiety on the basis of the person's ability to locate the threatening agent & recognize the presence of a behavior that will reduce perceived danger;
- **Stress** – general disturbance in psycho-physiological adaptation; mostly associated w/ response aspects;
- Overprotective parents usually have children who are **shy, docile,** & **manageable;**
- Health Belief Model - conceptual framework that describes a person's health behavior as an expression of his/her health beliefts; suggests that individuals will act to prevent disease only when they belive they are susceptible to disease; Components of the model:
    1. Person's own perception of susceptibility to a disease/condition.
    2. Likelihood of contracting that disease/condition.
    3. Person's perception of severity of consequences of contracting the condition/disease.
    4. Perceived benefits of care & barriers to preventive behavior.
    5. Internal/external stimuli that result in appropriate health behavior by the person.

OSHA:

- Standard/Universal Infection Control Precautions – method of infection control which all human blood & certain body fluids (saliva in dentistry) are treated as if show to be infectious for HIV, HBV, HCV, & other bloodborne pathogens; first recommended by CDC in 1987;
- Occupational Safety & Health Administration (OSHA) – federal agency created by congress in 1970 to protect workers from hazards in the work place; they are concerned w/ REGULATED WASTE in dental office;
- **Hazardous Waste** – waste causing harm/injury to environment; doesn't have to be toxic/poisonous;
- **Infectious Waste** – waste that contains strong enough pathogens in sufficient quantity to cause disease;
- AIDS prompted OSHA to adopt *Bloodborne Pathogens Standard for Dentistry* – a comprehensive rule that sets forth the specific requirements OSHA believes will prevent the transmission of bloodborne diseases to **EMPLOYEES** not patients or employers;
- OSHA directs that uniform clothing worn in dental office is laundered at dental office or by an outside service, NOT employee's home;
- Only in dental procedures is SALIVA considered a potentially infectious material;
- Fluid-resistant gowns are not required unless it is anticipated that large amounts of blood, saliva, or other body fluids will soak thru gown to the employee's clothing;
- When handling chemical agents or cleaning a dental office, always wear protective eyewear, mask, & heavy duty utility or nitrile gloves;
- CDC suggests new mask for each patient; masks should have at least 95-99% filtering efficiency for small particle aerosols 1-3m;
- HIV is MOST INFECTIOUS TARGET of standard/universal blood precautions but HBV is MOST INFECTIOUS BLOODBORNE PATHOGEN, not most infectious agent;
- HBV – poses the **greatest occupational healthcare worker risk for bloodborne infection**;
    - → HBV concentrations in blood of a chronic carrier can range b/w 1-100 million virions/ml, in contrast to significantly lower viral loads shown for both HIV & ADS infected pts;
    - → Exposed employees who have declined the HBV vaccine can change their mind at any time & receive FREE vacination;
    - → Exposed employees who have begun their HBV vaccine series can work at their job even though the series is not complete;
    - → Dentist must provide "at-risk" employees w/ protection from HBV; federal standard for occupational exposure to bloodborne pathogens REQUIRE employers to proved the HBV vaccine;
    - → Employe may refuse vaccination but OSHA will require proof that employee has refused;
    - → Employers must offer the vaccination to a new employee w/in 10 working days of initial assignment to a position involving exposure; Training must be provided prior to offer of vaccine;

→ **HBV infection commonly occurs by sex, prenatal transfer, & percutaneous inoculation;**

- HCV – transmitted primarily in infected blood via accidnetal needle-sticks, blood transfusions, or drug addicts sharing contaminated syringes;
  → Historically, drug users, ppl receiving tansfusions, organ recipients, & hemophiliacs receiving **Factor VII or IX** are at high risk for the virus, but now ppl getting tattoos & piercings are at risk;
  → Viral conc detected in HCV infected pts range b/w numbers for HBV & HIV;
- Occupational Exposure – any reasonably anticipated skin, mucosal, eye, or parental contact w/ blood or other potentially infectious fluids during the course of one's duties while at work;
  → Infection control training records & medical records if employee involved in occupational exposure must be maintained;
  → Medical records must be maintained for duration of employement plus 30 years & strictly confidential;
  → if you go out of business or new owner, must notify *Director of National Institute of Occupational Safety & Healthy* at LEAST 3 months b/f you intend to dispose records & offer to transmit the records to NIOSH;
- Exposure Incident – specific occupational incident involving eyes, mouth, other mucous membranes, non-intact skin, or parenteral contact w/ blood or potentially infectious materials;
  → **Any injury from a contaminated sharp is the most common exposure incident.**
  → EmployER must provide EmployEE with any meds needed after or before exposure, CONSELING, and evaluation weeks after incident;
- Exposure Control Plan – requires that every employer have a written exposure control plan to elimate/minimize employee exposure to bloodborne diseaseas;
  → Must be updated at least ANNUALLY & whenever necessary to reflect office changes;
  → The plan must be provided to OSHA upon request;
- Employers must ensure that ALL employees w/ occupational expsosure participate in training program **at NO cost, during working hours, w/ material for education, literacy, & language of the employee!**
- Contaminated sharps are any object that can penetrate skin, like needles, scalpels, broken glass, broken capillary tubes, & exposed ends of dental wire;
- **Anti-Retraction Valves** – used on handpiece & air-water syringe hoses to prevent retraction of fluid back into the tubing; prevents pts fluid from getting into water lines;
  → **CDC** recommends minimum of 20-30 secs of flushing wanter lines b/w patients and several minutes if the system has been idle for awhile, like over the weekend;
- FDA – branch of *Health & Human services* that determines which drugs & medical services can be marketed in US; also responsible for regulating handpieces & recommending sterilization procedures to CDC;
- DEA – branch of *Department of Justice* that determines degree of control for substances w/ abuse potential;
- The most commonly used dental materials deemd hazardous by OSHA are **mercury, nitrous, & chemicals used to develop film;**
- Amalgam scrap is stored in tighly sealed containers covered w/ **sulfide solution**;
- Acceptable max exposure level allowed by OSHA for nitrous is 1000ppm;
- Material Safety Data Sheet – document that contains info concerning hazardous chemicals; chemica manufacturers & importers are required to obtain a MSDS for each hazardous chemical;
  → Must be readily accessible to employees
- EPA – regulates waste TRANSPORATION from dental office;
- OSHA considers part-time, temporary, & probational workers as employees;

PUBLIC HEALTH:

- Quality Assessment – measure of the quality of care provided in a particular setting; limited to appraisal of whether or not standards of quality have been met;

- Quality Assurance – measurement of quality of care & IMPLEMENTATION of all necessary changes to maintain/improves the quality of care rendered; contains 3 Concepts:
    a. Structure – layout & equipment of facility;
    b. Process – the actual service the dentist provides for pts;
    c. Outcome – change in health status that occurs b/c of care delivered;
- Sensitivity & Specificity are INVERSELY proportional; as the specificity of a test increases, the sensitivity decreases;
- **Sensitivity** – ability of test to diagnose correctly a condition/disease that actually exists; measures the proportion of people w/ a disease who are correctly identified by a positive test;
    → Defined as # of true positive (TP) divided by total # of potential positive findings (true positives & false negatives) in sample; Sensitivity = TP/(TP+FN)
- **Specificity** – ability of test to classify health; defined by # of true negative results dived by total # of false positive & true negative results in sample; Specificity = TN/(FP + TN)
- **Prevalence** – # of OLD cases of disease present in population at risk at a specific period of time; the proportion of persons in population suffering from particular disease at given point in time;
    → Expressed as percentage of population;
- **Incidence** – # of NEW cases of specific disease occurring w/in a population at certain amount of time; expressed as a rate (cases)/(population)/(time); incidence is a rate that requires a unit of time;
    → Incidence is a RATE & prevalence is a PROPORTION;
- Frequency = a count;
- Abuse – dentist are morally, ethically, & legally obligated to report a suspected case of child abuse; dentist's first & immediate responsibility is to protect the child;
    → Dentist also ethically obligated to identify & refer cases of domestic violence;
    → 68% of battered women injuries involve face, 45% the eyes, & 12% the neck;
- Managed Care – arrangement where 3rd party payer mediates b/w doctors & patients negotiating fees for services & overseeing types of tx provided; types = HMO, PPO, & IPA;
    → PPO (preferred provider orgnaization) – typically involves contracts b/w insurers & dentist and patients can choose their dentinst depending on if the dentist participates in PPO;
    → Participants of HMO are much more limited in their dentist selection b/c they have to stay w/in network;
- **Capitation -** fixed monthly payment paid by carrier to a dentist based on # of pts assigned to dentist for treatment; fee is same regardless of how much or how often care is delivered;
    → Most popular managed care payment method;
- HMO = capitation; PPO = reduced fee for service;
- **Dental Index** – data collection instrument used to numerically express oral health status of population; 8 Indices:
    1. **DMFT Index** (Decayed-Missing-Filled Teeth) → irreversible index (measures that cant be reversed like caries) applied only to PERMANENT teeth;
        i. It yields a groups caries susceptibility; received universal acceptance & is probably the best known of all dental indices;
    2. **DEFT Index** (Decayed-Extracted-Filled Teeth) → used for PRIMARY TEETH;
    3. **DMFS Index** (Decayed-Missing-Filled Surfaces) → same as DMFT but records involve tooth surfaces;
    4. **Gingival Index** (GI) – reversible index used to asses severity of gingivitis based on color, consistency, & BOP;
        i. Gingivitis most commonly scored w/ **Gingival Index of Loe & Silness** which grades gingiva on 4 surfaces of each tooth based on inflammation & bleeding;
        ii. **GI, Papillary, Marginal & Attached Gingival Index** (PMA Index) – measurement w/in gingiva; records the prevalence & severity of gingivitis in school children;

5. **Periodontal Index** – reversible index that measures conditions that can be changed, like plaque & bleeding; condition of gingival (less weight) AND BONE (more weight) estimated for each tooth;
6. **Simplified Oral Hygiene Index** – reversible index used to measure oral hygiene status by estimating tooth surface covered w/ material alba &/or calculus;
7. **Plaque Index (PI) of Silness & Loe** – reversible index to assess THICKNESS of plaque at the gingival margin; scores from 0 to 3;
   0 = tooth surface is plaque free
   1 = plaque not observed on tooth but is on probe
   2 = thin plaque observed on tooth
   3 = heavy accumulation of plaque on tooth;
   → Extensively used but not universally accepted;
   → 80-90% of children have perio disease by age 15; most common form is localized acute gingivitis;
8. **Sulcus Bleeding Index** – used to determine bleeding & gingival health;

- Vital Statistics – quantitative methods to monitor & evaluate the life history of a specific population;
  → identifies community health needs, estimates healthcare costs, & evaluates health program effectiveness;
  → data monitored is mortality, morbidity, natality, birth-death ratio, & crude death ratio;
- 3 Principles of Public Heath – problem exists, solutions exists, & solutionsto problem is applied;
- most important concept of Winslow's definition of public health is **promotion through organized community heath;**
- Dental public health is a form of dental practice that serves the community as a patient rather than serving the individual;
- **Fundamental principles of public health are prevention, cost-efficiency, & teamwork;**
- **Prevention** is major objective of public health programs; more ethical to prevent disease than cure it;
- Randomized Study – study where ALL subjects have equal chance of being assigned to either the study or control group;
- Blind Study – study where subjects are unaware if they are in a test or control group; this is achieved by using **placebos;**
- Cross-Sectional Study – study in which the health conditions in a group of people who are, or are assumed to be, a sample of a particular population (a cross-section) is assessed at one time;
- Case Control Study – people w/ a condition (case) are compared w/ people w/o it (control) but who are similar in other characteristics;
- Cohort Study – 2 types: prospective cohort study & retrospective cohort study;
  → *Prospective Cohort Study* – a general population is followed thru time to see who develops the disease, & then the various exposure factors that affected the group are evaluated;
    - Ie – studying a sample of subjects who don't yet have cancer but measuring the risk factors of each subject that may predict the subsequent outcome.
  → *Retrospective Cohort Study* – used to evaluate the effect that a specific exposure has had on a population; measuring the risk factors of subjects who had the outcome of interest;
- The ethical principles found in the ADA's *Principles of Ethics & Code of Professional Conduct* are:
  1. **Justice** – the quality of being impartial & fair;
  2. **Autonomy** – to inform patient about treatment, be truthful, & protect their confidentiality;
  3. **Beneficence** – to be kind & give highest quality of care one is capable of providing;
- Good Samaritan Law – law enacted in all states that provides IMMUNITY from suit for specified health practitioners who render emergency aid to victims of accidens, provided there is no evidence of gross negligence; **Not all states include dentists in Good Samaritan Law;**
- **Mean** = average; **Median** = middle measurement in set of data; **Mode** = most frequent measurement;
- **Range** = the simplest measure of variability; **Variance =** method of ascertaining the way individual values are located around the mean; **Standard Deviation** = typical/avg deviation from the mean;
- Chi-square test – measure association between 2 categorical variables;
- T-test – used to analyze the statistical difference b/w 2 means;

INFECTION CONTROL:

- Opportunistic Infection – infection caused by normally non-pathogenic microorganisms in a host whose resistance has been decreased/compromised;
  → Percentage of ppl living w/ wide variety of immun compromised conditions continues to increase;
- Exposure is not synonymous w/ infection; Do not disinfect when you can sterilize;
- **It is not possible/necessary to sterilize all environmental surfaces that become contaminated during patient care;**
- Sterilization of all clincal instruments & inanimate surfaces NOT manditory**;**
- Bactericidal agents preferred over bacteriostatic chemicals;
- Sanitization – type of antimicrobial treatment (used for drinking water) to lower total microbial load to safe public health levels;
- Sterilization – process of killing/removing all microorganisms, including spores, on an object/in a material; limiting requirement is destruction of heat-resistant spores; **abscess of all living forms;**
- Heat is most efficient, reliable, & biologically monitorable sterilization method;
- Pre-Cleaning – MOST IMPORTANT STEP in instrument sterilization b/c debris acts as a barrier to the sterilant & sterilization prcces;
  → **Ultrasonic instrument cleaning** is safest & most efficacious method of precleaning;
- Immersion of dental instruments in cold disinfectants will not destroy spores/hepatitis viruses;
- Liquids are generally sterilized by filtration; most common filter is composed of *nitrocellulose* & has **pore size of 0.22m**;
- **Rapid Heat Tranfer Sterilization** – very fast cycle time, no dulling of instruments & drys instruments after cycle; forced air, dry heat convection ovens are are appropriate for sterilizing heat-stable instruments & other reusuable items used in patient care;
  → Higher temp than other dry heat units; can sterilize much faster than traditional dry heat sterilizers;
  → Requires 375°F (191°C) for 12 min for wrapped instruments & 6 min for unwrapped instruments;
- **Dry Heat Sterilization** – Dry heat destroys microorganisms causing **coagulation of proteins;**
  → requires 320°F (160°C) for 2 hours or 340°F (170°C) for 1 hour;
  → instruments must be dry before using this sterilization & *ethylene oxide sterilization*;
  → doesn't dull or corrode instruments but long cycle & poor penetration;
- **Autoclave** – destroys bacterial by denaturation of high protein-containing bacteria;
  → Requires 250°F (121°C) for 15-20 min under 15psi or 270°F (134°C) at pressue of 30psi for 3 min (**flash cycle);** flash cycle best indicated for unwrapped instruments;
  → the pressure greatly speeds up the protein denaturation process; only 10 min required to destroy all bacterial but increased time allows penetration when instruments wrapped in thick towels;
  → Spore testing for autoclave units recommended WEEKLY; the spores *Bacillus Stearothermophillus* are used;
  → Spores are resistant to boiling (100°C) so temp increased & pressure needed;
  → This kills even highly heat resistant spores like *Clostridium Botulinum*;
- **Unsaturated Chemical Vapor Sterilization** – requires 270oF (132oC) for 20-40 min; yields 20lbs of sterilizing vapor pressure; Doesn't rust or corrode instruments;
  → doesn't use distilled water, uses solution of alcohol, formaldehyde, ketone, acetone, & water to produce the sterilizing vapor;
- **Glutaraldehyde** (2%) – an alkalizing agent highly lethal to essentially all microorganisms; takes 10 HOURS to kill SPORES when instrument placed in 2% glutaraldehyde solution;
  → long time, allergenic, & extremely toxic to tissues;
  → used in hospital to sterilize respiratory therapy equipment;
  → Faceshields disinfected w/ Iodophors or Glutaraldehydes;
  → This disinfectiant often 28-30 day life span;

- **Ethelene Oxide Gas Sterilization** – kills by **alkylating proteins & nucleic acids & proteins**; used extensively in hospitals to sterilize heat-sensitive materials like surgical instruments & plastics;
  - → Slow process taking 10-16 hours; toxic to humans & flammable, so limited use;
  - → Highly penetrative, doesn't damage heat-sensitive material, evaporates w/o leaving residue;
- Antiseptics – chemical safe to be administered to external body surfaces or mucous membrane to ↓ microbial numbers; cant take internally; similar to disinfectants but can be applied to living tissue;
  - → Best relates to handwash agent like **chlorhexidine gluconate, parachlorametaxylenol, idophors, & triclosan;**
  - → Alcohol is MOST WIDELY USED ANTISEPTIC & reduces the number of microorganisms on skin surface in wounded area; it acts by: 1) denaturing proteins 2) extracts membrane lipids 3) dehydrating agent
  - → Even some viruses (lipophilic) are inactivated by alcohol;
  - → Alcohols are bactericidal, tuberculocidal, & economical; NOT sporicidal; it evaporates too quickly and diminished activity against viruses in dried blood, saliva, & other secretions;
  - → **Isopropyl alcohol** is major form used in hospitals;
  - → **Ethanol** – widely used to clean skin prior to immunization or venupuncture;
  - → **Iodine –** MOSTE EFFECTIVE skin antiseptic used in medical practice that acts as an oxidizing agent, & irreversibly combines w/ proteins;
  - → Phenol was original disinfectant but rarely used today b/c too caustic;
- Disinfection – process of reducing the # or inhibiting growth of microorganisms, especially pathogens to the point where they don't pose a threat of disease; not all pathogens or spores!
- Disinfectants – antimicrobial chemical agents used to destroy/kill microorganisms when applied to inanimate objects/surfaces; not safe on living tissues;
  - → Ie – **Alcohol, Chlorhexidine, & Quaternary Ammonium Compounds;**
  - → Water-based disinfectants are better than alcohol-based disinfectants;
  - → Pump spray disinfectants are better than aerosol spray disinfectants;
  - → **Quaternary Ammonium Compounds** – cationic detergents used as disinfectant & antiseptic agains gram (+) bacteria which are most susceptible to destruction; inactivated by anionic detergents (soaps & iron found in hard water); ie – *Benzalkonium Chloride;*
  - → Cleaning surfaces prior to disinfection is required to REDUCE concentration of pathogens;
  - → *Mycobacterium Tuberculosis* is the marker microorganism for intermediate surface disinfection;
  - → **Chlorhexidine Gluconate & Triclosan -** handwash agents w/ broad antimicrobial effect; have substantivity or residual action on washed tissues for extended periods of time;
- **Chlorine** – powerful OXIDIZING agent that inactivates bacteria & most viruses by oxidizing free sulfhdryl groups; active component of hypochlorite & used as disinfectant;
- Pasteurization – tx of dairy foods for short intervals using HEAT to kill certain disease-causing microorganisms; target of pasteurization is to **destroy *Mycobacterium Tuberculosis;***
- **Concentration & Time** are critical factors that determine effectiveness of antimicrobial agent;
- Individuals predisposed to readily developing hypersensitivity rxns can become SENSITIZED to latex allergnes more readily than people w/ few or no allergies;
- *Hevea Brasiliensis* – water-soluble macromolecules that can leach out of latex gloves when a person perspires or may be detected on surfaces of other product containing natural rubber latex;
  - → These proteins cause Type IV, IgE mediated reactions to natural rubber latex;
  - → Products designated HYPOALLERGENIC are no longer labelled latex alternatives since they contain latex w/ a chemical coating over the latex;
- **Irritation Dermatitis** is MOST COMMON form of an adverse epithelial rxn noted for healthcare professionals; 20-30% of healthcare workers suffer occasional or chronic dermatitis on their hands;

- Americans w/ Disabilities Act – both state & federal statues define disability as having "a physical or mental impairment that substantially limits one/more major life activities of the individual, a record of such impairment exist, & the patient is regarded as having such impairment."
    - → Dentists CANNOT deny anyone care due to disability & cannot dismiss employees due to disability.
    - → Dental offices must undergo structural changes to allow access for the disabled.
    - → HIV pts are protected under this act;

PEDIATRIC DENTISTRY

TOOTH ANATOMY:

- **Primary mand. 1st molar** – like no other tooth; difficult to do a Class II, no central fossa;
- **Primary mand. 2nd molar** – greatest FL diameter of all primary teeth.
- **Primary max. central incisor –** NO MAMELONS; incisocervical height < MD width.
- **Primary mand. central incisor -** similar to permanent LATERAL incisor.
- **Primary mand. lateral incisor –** similar to permanent CENTRAL incisor.
- **Primary max. 1st molar –** FL diameter > MD diameter (different than other primary molars); 5th cusp; often resembles permanent max. PM; oblique ridge; MB – largest pulp horm; MB cusp > ML cusp; grooves form H pattern w/ 3 fossa; has 3 Roots, resembling perm. 1st max molar.
- **Primary max. canine –** mesial cusp ridge > distal cusp ridge & mesial cusp longer & sharper; both facts differ than permanent canines.
- **Permanent Max. Canines** – most likely to be crowded out of maxillary arch.
- **Permanent Mand. 2nd PMs** – most likely to be crowded out of mandibular arch!
- Facial part of remaining primary root is longest.
- Labial & Lingual cervical ridges prominent on all primary incisors!
- Largest primary tooth – mand. 2nd molar; Smallest primary tooth – mand. lateral incisor.
- Largest permanent tooth – Max. 2nd molar; Smallest – mand. central incisor.
- Primary molars – 1) B & L surfaces are flatter
  2) Shorter & narrower MD at cervical 1/3
  3) Longer & slender roots.
- Primary Anteriors - 1) Wider MD & shorter IC
  2) root tapers more rapidly
- Enamel ends abruptly at cervical line on all primary teeth;
- Lateral incisor is most common PRIMARY congenital missing tooth.
- Primary teeth less opaque on xray than permanent teeth b/c > inorganic (Ca+, Phosphorus, hydroxyapatite) content; Organic content is collagen type 1.
- Enamel on primary molars = 1mm while permanent molars = 2.5 mm of enamel.
- Sum of MD widths of primary molars in any 1 quadrant is 2-5mm greater than perm. teeth that succeed them (premolars);
- Last primary tooth to be replaced by permanent tooth is maxillary canine.
- Occlusal table on primary molars are narrower facial lingually.
- **Cementum** (thicker apically than cervically) & **PDL fibers** increase as you age;
- Child Gingiva - 1) more red, 2) less stippling, 3) flabbier tissue, 4) rounded/rolled gingiva, 5) PDL runs parallel to teeth, 6) alveolar bone thinner;

ERUPTION & CALCIFICATION:

- Primary teeth begin to form at 6 weeks in utero; Permanent teeth begin to develop 4 months in utero.
- When tooth erupts, ½ - 2/3 of root formed; apex fully formed in 2-3 yrs (perm teeth); root completely forms in 18 months for primary teeth.
- all 20 primary teeth begin calcification at 4-6 months in utero; 10 months for complete calcification;
- primary teeth begin to form at 6 weeks; All Primary teeth calcification in utero!
- After permanent teeth have reached full occlusion, small tooth mvmts occur to compensate for wear on contacts (mesial drift) & occlusal surfaces (deposition of cementum at root apex).
- Hard tissue formation of primary teeth at 18 weeks;
- **Succedaneous tooth** – permanent tooth that moves into position formerly occupied by primary tooth; NEVER MOLARS!
- Tooth buds generally initiated after birth – PMs, 2nd molars, & 3rd molars.
- Best tx for permanent tooth trying to erupt but primary tooth is still in places is EXTRACTION;
- Primary tooth takes 1.5 to 2 months from eruption to occlusion; CANINES take the longest;

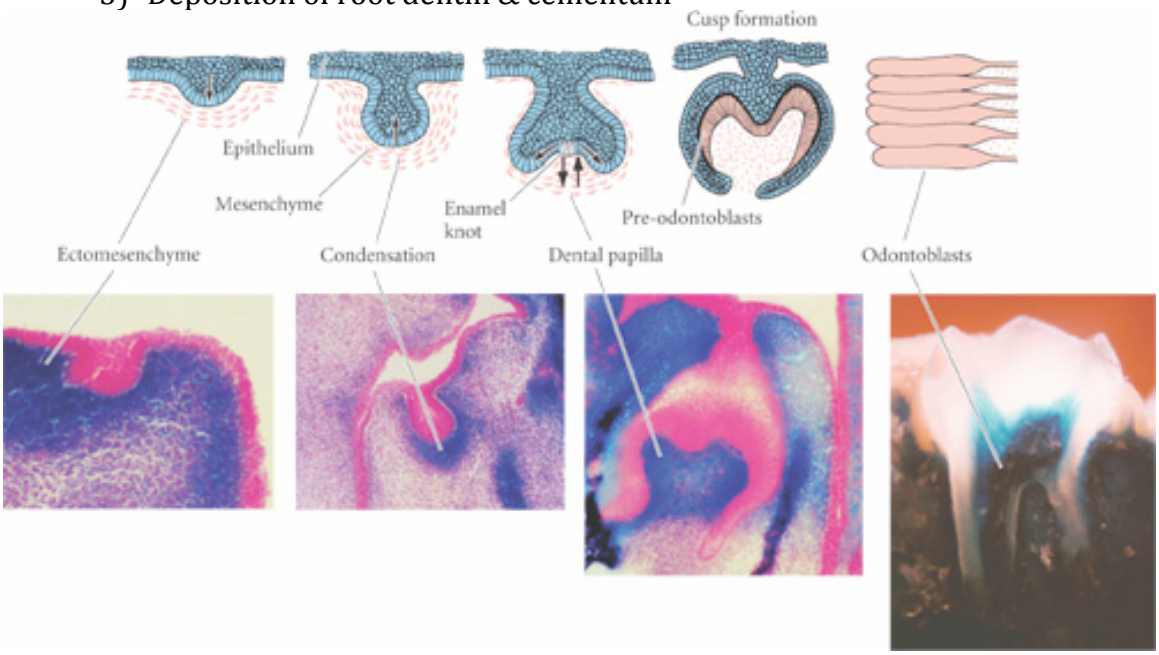
- Calcification of roots by age 3 or 4; Calcification of primary teeth during 2nd TRIMESTER.
- After primary teeth fall out, extra space on Mand = 3.1mm/quad (6.2) & Max = 1.3mm/quad (2.6).
- Mand. 3rd molars are last to begin calcification at 8-10 years.
- Girls teeth erupt before boys; girls reach puberty 2 years before boys.
-

PRIMARY TEETH ERUPTION SEQUENCE: Rule of 4, every 4 months, 4 new teeth!		
7 months	4 teeth	Mand. incisors
11 months	8 teeth	Mand. & Max incisors
15 months	12 teeth	All incisors & 1st Molars
19 months	16 teeth	All teeth but 2nd molars
23 months	20 teeth	All teeth!

Permanent Teeth Calcification	
Birth	1st Molars
6 months	Anter. teeth except Max. L. I.
12 months	Max. L. I.
18 months	1st PMs
24 months	2nd PMs
30 months	2nd Molars

TOOTH DEVELOPMENT:

- Tooth development initiated by mesenchyme's inductive influence on overlying ectoderm;
- Enamel of tooth from ectoderm while other tissues of tooth from mesenchyme.
- Ectodermal cells responsible for crown root & shape;
- Histogenesis of Tooth: once ectomesenchyme influences oral epithelium to grow into **ectomesenchyme** & become tooth germ:
    1) Elongation of inner enamel epithelium cells to enamel organ
    2) Differenciate into odontoblasts
    3) Deposition of first layer of dentin
    4) Deposition of first layer of enamel
    5) Deposition of root dentin & cementum



- **Korff's Fibers** – rope-like fibers at periphery of pulp dealing w/ formation of dentin matrix.
- **Lobes** – primary centers of calcification; separated by developmental grooves in posterior teeth & developmental depressions in anterior teeth.
    → Anterior teeth – 3 labial & 1 lingual lobe
    → PMs – 3 labial & 1 lingual lobe (Mand. 2nd PM – 3 labial & 2 Lingual)
    → 1st Molars – 5 lobes – 1 for each cusp.
    → 2nd & 3rd Molars – 4 lobes – 1 for each cusp.
    → No mamelons in permanent teeth unless malocclusion like anterior open bite!
- **Hertwig's Epithelial Root Sheath** – determines #, size, & shape of roots; inductor of dentin formation in developing root;

→ Uniform growth = single root tooth; Medial growth = evaginations/multi-rooted teeth;
→ formed when outer enamel epitheilium & inner enamel epithelium combine at cervical loop region to form this bilayered structure.

- 6 stages of Tooth Development:
  1) *Induction* – **induction**, **5th week**, formation of dental lamina from epithelium & mesenchyme.
  2) *Bud Stage* – **proliferation**, **8th week**, dental lamina into 10 buds per arch; shape of tooth evident & enamel organ forms;
  3) *Cap Stage* – **proliferation & differentiation** (either morphodifferentiation or histodifferentiation), **9th & 10th week**;
     a. tooth germ complete w/ enamel organ, dental papilla (pulp & dentin) & sac.
  4) *Bell Stage* – **11th & 12th week**; dental papilla (either outer cells or central cells); dental sac has increase in collagen; 4 cell types in enamel organ:
     i. OEE – cuboidal
     ii. IEE – columnar
     iii. Stelate Reticulum – star-shaped
     iv. Stratum Intermedium – flat to cuboidal
  5) *Appositional Stage* – deposite specific dental tissues (enamel, dentin, cementum, & pulp).
  6) *Maturation Stage* – mineralization at DEJ & continues til tooth development 2 years later.

FRACTURES:

- **Ellis Fractures -** 1) Class I – little/no dentin; tx – enamelplasty/bonding.
  2) Class II – fracture crown w/ lot of dentin but no pulp; tx – restore w/ CaOH & GI.
  3) Class III – fracture w/ pulp exposure; tx – Pulp therapy & restore.
  4) Class IV – fracture entire crown; tx – pulpectomy & SSC.
  5) Class V – tooth avulsed.
  6) Class VI – fracture root but not crown.
  7) Class VII – displacement of tooth.
  8) Class VIII - fracture crown en masse (as a whole).
  9) Class IX – injury to primary teeth.
- Prognosis less favorable in horizontally fracture primary teeth versus permanent teeth;
- Fractured maxillary anterior teeth most often in kids w/ Class II, Division 1 malocclusion.
- Chief cause of failure of replantation of permanent teeth is EXTERNAL RESORPTION.
- Thickness of dentin in primary teeth = ½ of dentin in permanent teeth.

VITAL PULP THERAPY:

- Pulpotomy:
  → No pulpotomy if tooth painful/swelling.
  → **Formocresol Pulpotomy** – tx for primary teeth w/ carious exposure; success of formocresol pulpotomy for primary tooth depends primarily on **vital root tip**;
    - ZOE is placed over chamber & restored;
    - allows resorption & exfoliation of primary tooth but preserves space maintainer;
    - formocresol causes surface fixation of pulp tissue accompanied by degeneration of odontoblasts.
  → **CaOH Pulpotomy** – not often used on primary teeth b/c alkaline pH can irritate pulp causing internal resorption; must be symptom free; forms NECROTIC dentin layer under CaOH.
    - for permanent teeth w/ carious exposure but immature root development & healthy pulp in root canals.
- Pulpectomy – canals debrided, enlarged, & disinfected; filled w/ ZOE so it will resorb when roots resorb; tx of choice when there is periapical pathology.
- Apexogenesis – vital pulp to encourage physiological development & formation of root end; MTA used;

- Contraindications for IPC - 1) Spontaneous Pain
  2) Furcation involvement
  3) Pulp Involvement;
  4) **Primary teeth**
- Chronic pulp infection in primary molars is noted in x-rays as a change in bony furcation.

OPERATIVE:

- Primary molars have exaggerated cervical constriction & enamel rods in gingival 1/3 extend **OCCLUSALLY** from DEJ so no gingival bevel!! But Axio-pulpal line angle BEVELED!
- Class 2 Amalgam on primary teeth - 1) Box broader cervical than occlusal
  2) B/L/G walls break contact & can fit explorer thru it.
  3) B & L walls create 90^{o} angle w/ enamel.
  4) Flat pulpal floor
  5) isthmus = 1/3 of intercuspal width.
- If amalgam fracture occurs, it is most likely to occur here; prefer rounded angles in prep!
- "Extension for Prevention" – only for amalgam, because you can use sealant for composite;
- For SCC, reduce cusp 1-1.5mm, while proximal surfaces are reduced & caried gingivally to extent that contact w/ adjacent teeth is broken; 2 types – Pretrimmed SSC or Precontoured SSC;
  - Remove sharp line angles and distinct buccal bulge especially in primary 1st molar.
- Larger pulpal space in primary teeth limits depth of amalgam prep. Duh.
- Cervical constriction in primary molars make gingival floor not ideal & difficult to adapt matrix band to the tooth.
- Facial & lingual walls of proximal box should be paralles to external surfaces & converge slightly.

LA/DRUGS/MEDS:

- Mandibular foramen in child is slightly below plane of occlusion and more anterior than adults;
- Max dose of lido in kids = **4.5**mg/kg per appointment.
- Bupivacaine/Marcaine should NOT be used on kids.
- Most frequent inhalation agent for sedating pts = NITROUS; earliest symptom of conscous sedation is Light Headedness;

Chloral Hydrate Dosages:

Weight (lbs)	Age	Dosage
25-50	2-4	500-760 mg
50-75	4-7	750-900 mg
75-100	7 & up	1000-1500mg

ANTIBIOTIC PROPHYLACTIC REGIMENS FOR CERTAIN DENTAL PROCEDURES.*

SITUATION	ANTIBIOTIC†	REGIMEN‡
Standard Prophylaxis	Amoxicillin	Adults, 2.0 grams; children 50 milligrams/kilogram orally one hour before procedure
Cannot Use Oral Medications	Ampicillin	Adults, 2.0 g IM§ or IV§; children, 50 mg/kg IM or IV within 30 minutes before procedure
Allergic to Penicillin	Clindamycin	Adults, 600 mg; children, 20 mg/kg orally one hour before procedure
	Cephalexin or cefadroxil	Adults, 2.0 g; children, 50 mg/kg orally one hour before procedure
	Azithromycin or clarithromycin	Adults, 500 mg; children, 15 mg/kg orally one hour before procedure
Allergic to Penicillin and Unable to Take Oral Medications	Clindamycin	Adults, 600 mg; children, 15 mg/kg IV one hour before procedure
	Cefazolin	Adults, 1.0 g; children, 25 mg/kg IM or IV within 30 minutes before procedur

EMERGENCY TREATMENT:

- Emergency Treatment for Fractures of Permanent teeth w/ immature apices:
  1. **Class I** – smooth enamel edges and restore.

2. **Class II** – apply CaOH & restore.
3. **Class III** – apply CaOH & place temporary; if large, perform CaOH pulpotomy; after apex closes, do pulpectomy;
4. **Class IV** – CaOH pulpotomy and after apex closes, due pulpectomy;

- Intruded primary anterior tooth – NO TX; repositioning of primary teeth not recommended; However, if the intruded incisor is contacting the perm. tooth bud(take xray), then prim. tooth should be TE'ed.
- Darker primary teeth from trauma is due to pulp bleeding & diffusion of BILIVERDIN in dentin tubules; if discolored primary teeth is asymptomatic & no radiographic changes, the NO TX.
- Underdeveloped motor coordination is most common cause of dental trauma in kids 1.5-2.5 yrs old.
- Root fractures of primary teeth are UNCOMMON b/c more pliable alveolar bone; However, if root fracture, same tx as perm teeth but LESS favorable prognosis; Splinting is NOT recommended for primary teeth;
- THERMAL test is most reliable in primary teeth but pulp vitality isnt commonly tested in these teeth.

FLUORIDE:

- CDC recommends at least 0.7ppm of fluoride be present in drinking water; max amt = 1.2ppm.
- Water fluoridation & supplements may affect tooth morphology;
- Types of fluoride added to water: 1) Sodium fluoride
  2) Hydrofluosilicic acid
  3) Sodium silicofluoride
- As fluoride concentration increases beyond 1ppm, then increase in fluorosis prevalence but no increase in reduction of dental decay;
- 43 states have water fluoridation, 62% of population; Fluoridation cost 72 cents/person/year.
- School water fluoridation concentration is 4x the city water due to less water consumption at school.
- The most cost-effective method of delivering fluoride to 6-12 year old children (in non-fluoridated community) is through school water fluoridation.
- Over the counter fluoride rinces: ACT, Fluoriguard, Prevident; all contain 0.2-0.5% **NaF**.
- Fluoride in toothpastes: 1) Stannous Fluoride
  2) Sodium Monofluorophosphate
  3) Sodium Fluoride
  4) Sodium Fluoride & Calcium Phosphate
- Fluoride concentration in USA is 0.1% (1,000ppm) = .22% NaF = .76% NaMFP = .4% SnF_2.
- Most desirable form of Fl^- is fluorohydroxyapatite (less acid soluble, more resistant to caries) & most efficient means of forming this rxn is prolonged exposure of enamel to ↓concentration of fluoride.
- Major mechanism of fluoride is caries inhibition which **increases remineralization of enamel**;
- Fluoride also inhibits glycolysis (where sugar is converted to acid by bacteria);
- Fluoride is BACTERICIDAL; decreases enamel solubility; least effective on root surfaces;
- Fluoride works by stopping or even REVERSING tooth decay; greatest effect on newly erupted teeth.
- Enamel demineralization starts at pH = 5.5.
- Greatest concentration of fluoride ions exist on outermost layer of enamel;
- Acute fluoride toxicity tx = syrup of IPECAC to induce vomiting & call 911; calcium binding products like milk decrease absorption.
- Death by acute fluoride toxicity is cardiac failure & respiratory paralysis; fluoride toxicity shows up in the bones as OSTEOSCLEROSIS;
- Child lethal dose = 15mg/kg; Adult lethal dose = 4-5gm; completely weight dependent;
- Fluoride absorbed thru stomach & small intestine & excreted by kidney;
- Fluoride's main effect occurs AFTER the tooth has erupted above the gingiva!
- 3 types of TOPICAL FLUORIDE:
  - **Sodium Fluoride** (NaF) – **2%**; neutral/basic pH of 9.2; acceptable taste; 29% efficacy;
  - **Stannous Fluoride** (SnF2) – **8%**; doesn't etch porcelain; BAD TASTE & stains silicate restorations; pH = 2.1-2.3; main advantage – SINGLE APPT but not used in U.S.

- **Acidulated Phosphate Fluoride – 1.23%;** acceptable taste (bitter w/o flavoring) but damages porcelain & contraindicated in implant restorations;
    a. MOST COMMONLY used in practice;

Recommended Dosages of Supplemental Fluoride:			
AGE	**<0.3ppm**	**0.3-0.7ppm**	**>0.7ppm**
6mo-3yrs	.25mg	none	none
3-6yrs	.5mg	.25mg	none
6-16 yrs	1mg	.5mg	none

- Fluorosis – irreversible diffuse symmetric HYPOMINERALIZATION disorder of ameloblasts during CALCIFICATION period of tooth development.

SEALANTS:
- Fissue sealants succeed by altering host susceptibility.
- Low viscosity sealants wet acid-etched tooth surfaces the best;
- Sealants need MICRO-MECHANICAL RETENTION;
- Acid etched w/ 30-50% phosphoric acid;
- Properties of sealants are closer to unfilled direct resins than filled resins like composite;
- Sealants are best retained on max & mand PREMOLARS!
- The principal feature of a sealant required for success is adequate retention.
- Components of Pit & Fissure Sealants:
    a. Bis-GMA – monomer diluted w/ TEGDMA to reduce viscosity.
    b. Initiator – Benzyol Peroxide in self-cured sealants & Diketone in visible-like cured.
    c. Accelerator – amine is self-cured.
    d. Opaque Filler – small amounts of titanium oxide to make different color than enamel.

PEDS PATHOLOGY:
- Cleft Palate & Lip are MOST COMMON craniofacial malformation, accouting for 50% of all defects!
- Cleft Palate – failure of fusion of palatal shelves of Max. process w/ primary palate; more FEMALES; impairs speech & swallowing; occurs during 1st trimester of preganancy (6-9 wks) 4 classes:
    1) Class I – only soft palate
    2) Class II – Soft & hard palate
    3) Class III – Class 2 & alveolar process
    4) Class IV – Class 3 & through alveolus on both sides of premaxilla.
- Cleft Lip – failure of medial nasal swellings & maxillary swelling to fuse; Left > Right; more males; lip & primary palate develop @ 4-5 weeks gestation period; during 4-6 wks of pregnancy; 4 classes:
    1) Class I – unilateral notching of vermillion
    2) Class II – Class 1 & extends to lip.
    3) Class III – Class 2 & extends to floor of nose.
    4) Class IV – bilateral clefting of lip.
- Atrophic Gingivitis – recession w/out alveolar bone loss; minor gingival inflammation;
- Cretinism – HYPOTHYROIDISM due to absence of thyroxine from thyroid gland; defective mental & physical development; curved spine & pendulous abdomne; features are coarse; thickened lips.
    → Underdeveloped mandible & overdeveloped maxilla w/ enlarged tongue;
    → Anterior open bite & flaring; delayed eruption; unerupted but fully developed perm. teeth.
- ADHD – M:F = 10:1; 3-5% of children; child doesn't usually need special dental treatment;
    → Tx = Methylphenidate (**Ritalin**) – CNS stimulant; Amphetamines (**Dextropamphetamine**).
- Scarlet Fever – EXOTOXIN-mediated disease arising from *group A β-hemolytic strep infections*; mostly in 4-8 yrs old; strep throat, fever, headache, nausea, vomiting, pain, & fatigue;
    → **Strawberry tongue** – enlargement of FUNGIFORM papillae above the level of desquamating filiform papillae; appearance of unriped strawberry; tx = PCN.

- Diptheria – acute contagious disease caused by *Bacterium Corynebacterium Diptheria*, characterized by production of systemic toxin; damaging to heart & CNS; immunization available.
- **Nursing Bottle Caries / Baby Bottle Tooth Decay** – most affect MAX. INCISORS; rampant decay from sleep-time bottle feeding & activity of strep mutans;
- Congenital Porphyria – autosomal recessive; skin become light brown & sensitive to sunlight & photosensitivity expressed as large bullous lesions;
  → teeth are pink/brown but scarlet under UV light due to excessive porphyrins in blood during mineralization; 3 complaints: 1)Photodermatitis
  2)Neuropsychiatric complaints
  3)Visceral complains (abdominal pain/cramping)
- Down's Syndrome – underdeveloped midfacial regions**; Class III**; open bite; chronic mouth breathing, delayed tooth eruption, ↑rate of missing teeth; roots short & conical; heart defects are common;
  → Need comprehensive preventive plan; difficulty accepting dental care but cooperation improved by using gradual exposure to dental office;
- Type 1 Diabetes – body cant properly use/store glucose; body completely stops producing insulin; **Xerostomia**, infections, poor healing, ↑periodontal disease, burning mouth syndrome, blindness;
- Apert Syndrome – cranial/limb anomalies; skull, midface, hands, & feet malformations; Shovel-shaped incisors; Lefort 3 surgery for retruding midface; supernumerary teeth, Class III malocclusion.
- Autism presents in the first 3 years of life; neurological disorder that affects brain function; 4x more prevalent in males than females;
- Crouzon Syndrome – **autosomal dominant** craniofacial disorder; maxillary hypoplasia, crossbite; dysmorphic facial features;
- Rieger's Syndrome – delayed sexual development & hypothyroidism; hypodontia, underdeveloped premaxilla, cleft palate, & protruding lower lip;
- Treacher Collins Syndrome – mandibular facial dysostosis (disorder of developing bone); autosomal dominant; sunken cheekbones, receding chin, malformed ears, mandibular hypoplasia, narrow face.
- **Seizures** – grand mal (2-5 min) is most common (90%); 3 phases of seizures:
  1) *Aura* – smell, taste, vision, hearing, emotions
  2) *Ictus* – larger event; tx = supine position, BLS, oxygen (if cyanotic)
  3) *Postictal* – drowsiness & confusion; brain recovery; tx = IV of 25-50ml of 50% dextrose, then 10 mg IV of Diazepam;
- Hemangioma is most common benign tumor of infants; vascular birthmarks that are biologically active so independent of child's growth; 5x more common in girls;
- 3 stages of Odontogenic Infection:
  1) *PA osteitis* – inflammation w/in alveolar bone; NO soft tissue swelling but sensitive to percussion.
  2) *Cellulitis* – infection spreads from bone to soft tissue; inflammation & edema occurs; sensitive to palpation; may be caused by necrotic primary/permanent tooth; discolored tissue; bacteria – *Group A Strep & Staph Aureus*.
     a. Often Ludwig's Angina in kids which causes DEHYDRATION!
  3) *Supparation* – inflammation localized to discrete, fluctuant abscess;
- Conditions causing Delayed Exfoliation & Delayed Eruption: Cleidocranial Dysostosis, Ectodermal Dysplasia, Down's Syndrome, Gardner's Syndrome, Osteogenesis Imperfecta, Rickets, severe congenital heart disease, & mental retardation; Hypothyroidism, Hypopituitarism, Hypoparathyroidism, & genetics (most common reason for missing teeth);

MISCELLANEOUS:

- Child should have PANO by age 6; frequency of xrays depends on child's risk of decay;
- 1st BWs should be taken when the spaces b/w the posterior teeth have closed.
- Within 6 months of 1st tooth eruption – dental visit (b/f 1st birthday!);
- 30-60% loss in mineralization b/f caries is radiographically evident.

- At age 6, childs head is 90% of adults.
- At birth - 1) jaw can accommodate all primary teeth
  2) **width** of face at greatest % of adults
  3) palate is flat
  4) can't differentiate sour, salt, or bitter taste
  5) cranial vault very near size of adult
  6) brain & cranial base fully developed.
- Tonsils in early life function to filter bacteria & program production of antibodies;
- Age 6-12, lymph tissue 200% of adult tissue; lymph tissue decreases at puberty while genital tissue is developing;
- If permanent tooth bud is accidently extracted while removing primary molar, immediately orient the tooth bud, replant the bud using digital pressure, & suture.
- **Hydrodynamic Theory** – pain results from indirect innervation caused by dentinal fluid movement in tubules which stimulates mechanoreceptors near the predentin.
- The most personal behavior by the dentist is touching the patient gently in the arm.
- The main advantage of using rubber dam is it AIDS in child management; it works for very nervous/anxious pts;
- A very young child is best managed under GA; premedication w/ barbituate may cause paradoxical excitement in a young child.
- Post-anesthetic lip biting is common post-treatment complication in children;

PERIODONTICS

- Tx of Perio-Endo Abscess: 1) RCT – re-evaluate in 2-3 mo.
  2) Antibiotic
  3) Sc/Rp
  4) Perio surgury if needed 2-3 mo. after RCT
- Periodontal Cyst – cant be differentiated radiographically from periodontal abscess; common in mand. Canine/PM area; teeth vital; no periodontal pockets; presents as a local tender swelling; tx = excision.
- Periodontal Abscess – vital teeth with deep pockets; acute pain that is constant, severe, and throbbing; increase in mobility; tx = PCN.
- Periodontal Tx Planning:
  I) OHI, extraction of hopeless teeth, SRP, Occlusal adjustments/Nightguard, Splinting; RE-Eval.
  II) Perio Surgery
  III) Restorative Phase
  IV) Maintenance Phase

POCKETS:

- Gingival Pocket – no apical migration of junctional epithelium; coronal expansion of marginal tissue;
- Periodontal Pocket – Junctional epithelium to migrate apically along cementum; attachement loss!
  - Suprabony pocket – base of pocket coronal to crest of bone; *horizontal* destruction of bone; not intraosseous.
  - Infrabony pocket – base of pocke apical to crest of bone; periodontal osseous defect; angular/vertical destruction of bone; **contraindication of Mucogingival Surgery!
- Infrabony/Intrabony pockets – *vertical* bone loss; classified as:
  1. 1-walled = hemiseptum (only prox. walls present) or ramp (only F/L wall present).
  2. 2-walled = interdental crater
  3. 3-walled = intrabony defect; contraindication for mucogingival surgery.
  4. 4-walled = circumferencial/moat defects.
     - 3 and 4 walled defects have best prognosis for treatment!
     - 0 (zero) walled defect = *dehiscences* and fenestrations; NO TX with osseous surgery!
- Dehiscence – loss of buccal/lingual bone overlaying root portion of tooth leaving area covered by soft tissue only.
- Osseous craters – concavities in crest of bone confied within facial or lingual walls; 1/3 of all defects and 2/3 of mandibular defects; TX = osseous surgery and recontouring.
- Horizontal bone loss parallels CEJ's of adjacent teeth and is usually generalized while vertical bone loss is often localized.
- Only way to determine # of walls surrounding tooth is exploratory surgery.
- 2 most critical parameters in prognosis of tooth – mobility and attachment loss.
- Pseudopocketing – pocketing w/o attachment loss and marginal tissue moves coronally; pseudopicks are suprabony.
- First detectable sign of inflammation is increase in sulcus fluid; bleeding is the most reliable indicator of gingival/periodontal inflammation.
- Best criterion to evaluate success of SRP is NO BLEEDING on probing!
- If after SPR pt returns in 1 wk, w/ hard & black deposits of calculus around gingival margin, indicates reduction in inflammation and old calculus is now exposed.
- When the gingival margin coincides with the CEJ, the loss of attachment = the pocket depth.

FURCATIONS & MOBILITY:

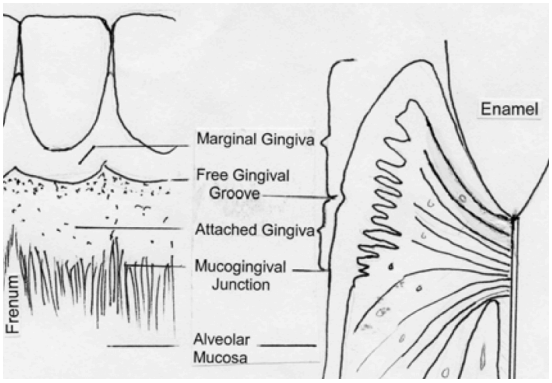
- *Classes of Furcations: (GLICKMAN FURCATION CLASSIFICATIONS)*
  I. Incipient bone loss; probe feels **depression** of furcation opening.

II. Partial bone loss; probe tip under roof of furcation; lesion is **Cul-de-sac**, not tunnel!
III. Total bone loss; thru and thru furcation (**TUNNEL**); furcation entrance isnt visible.
IV. Grade III fucation but entrance **visible**.

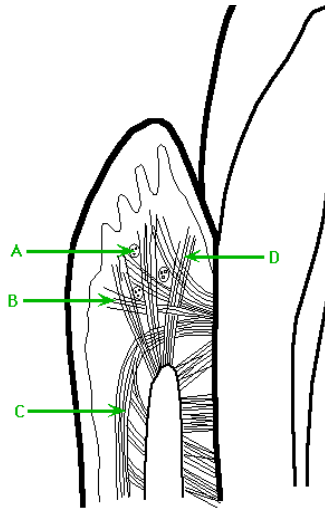
- Tx = guided tissue regeneration; Grade II furcations have good prognosis.
- Max 2nd Molars have poorest prognosis.
- *Mobility Classes:*
  0. No Mobility
  1. Barely distinguishable mvmt. (.5-1mm)
  2. Mvmt 1-2mm
  3. Mvmt > 2mm OR teeth depressed or rotate in socket.

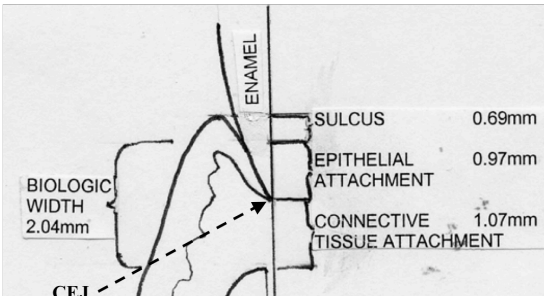
PERIODONTIUM ANATOMY:

- Gingival unit = free gingiva + attached gingiva + alveolar mucosa
- Attachment Apparatus = PDL + cementum + alveolar bone
- Free gingival groove demarcates jct b/w free gingiva and attached gingiva; only present in 33% of adults.
- Attached gingiva and free gingiva is KERATINIZED! Gingiva coronal to the mucogingival junction is keratinized and gingiva apical is non-keratinized.
- Width of facial attached gingiva greatest on facial surface of MAX. L. Incisors and narrowest b/w MAND. Canines and 1st PMs.
- Attached gingiva is coral pink color but it depends on degree of keratinization, thickness of epithelium, presence of melanin, and # of blood vessels.
- Attached gingiva is measured by subtracting pocket depth from width of gingiva from free gingival margin to mucogingival margin.



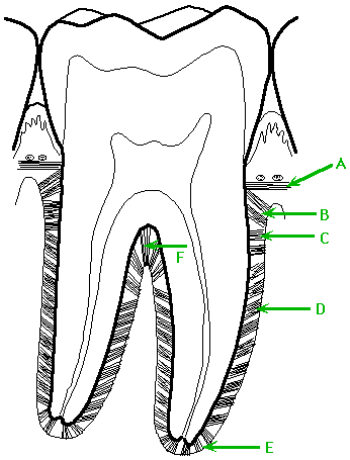
- Stippling – irregular surface texture of attached gingiva; intersection of epithelial ridges that cause depression and interspersing connective tissue papilla.
  - In healthy attached gingiva, it shows signs of stippling = orange-peel appearance.
- Gingival apparatus = gingival fibers + epithelia attachement.
- Gingival Ligament = dentogingival + alveologingival + circular fibers.
- Indifferent Fiber Plexus = in PDL; small collagen fibers that run indifferent directions.
- Gingival fibers are type 1 collagen fibers that extend from cervical cementum into gingiva; just free gingiva but part of PDL; supports gingiva and keeps it closely adapted to tooth.
  - A- Circular Fibers – resist rotational forces; encircle tooth around most cervical part of root; insert into cementum, lamina propria, and alveolar crest
  - B-Dentogingival Fibers – extend from cementum apical to epithelia attachement and course laterally.
  - C-Dentoperiosteal Fibers – from cervical cementum over alveolar crest to periosteum of bone;
  - D-Alveologingival Fibers – insert in crest of alveolar process and spread into free gingiva.
- Gingival collagen different than rest of body with the collagen turnover not as rapid as PDL; collagen is 60% of





gingival protein; but gingival collagen has significantly greater turnover rate than tendons and palate!

- Epithelial Attachement – mediates attachment of reduced enamel epithelium (1° attachment) or junctional epithelium (2° attachment), namely internal basal lamina and hemidesmosomes; joins free gingiva to tooth surface.
  - The attachment apparatus that joins JE to tooth surface.
- Junctional epithelium (.25-1.35mm) - stratified squamous, non-keratinized epithelium that surrounds tooth like collar; 2 basal laminas; in healthy gingiva, JE is entirely on enamel above CEJ.
  - Firmly attached to tooth by ***hemidesmosomes***; DOESN'T contain rete pegs while free gingiva does.
  - 10-12 cells thick near sulcus and 2-3 cells thick near apex.
  - Has a proliferative cell layer responsible for most cell divisions and in contact with C.T.
  - JE had desquamative/shedding surface located at coronal end and forms bottom of gingival sulcus.
  - Long JE refers to JE in disease.
- The PDL is highly vascular & cellular connective tissue that surrounds the roots of teeth and bridges root cementum with alveolar bone; PDL is specialized rom of C.T. derived from dental sac.
- PDL Principal Fibers (type 1 collagen) : connect root cementum to alveolar bone.
  A. *Transeptal* – tooth to tooth; keeps teeth aligned; not in facial aspect.
  B. *Alveolar crest* – cementum – alveolar crest; slants apically and resists LATERAL mvmt and counterbalance occlusal forces.
  C. *Horizontal* – runs perpendicular form bone to cementum; resists LATERAL mvmt.
  D. *Oblique* – slants occlusally from cementum to bone; resistant to MASTICATORY forces; 1/3 of fibers so most numerous.
  E. *Apical* – radiate apcial from cementum to bone; INITIAL resistant to OCCLUSAL forces.
  F. *Irradicular* – cementum-furcation; only multi-rooted teeth.



- Sharpey's Fibers – terminal portions of the collagen fibers that insert into cementum and alveolar bone; diameter > on bone side than cementum side.
- The PDL is hour-glass shaped w/ narrowest part in the middle of the root.
- PDL fuctions: -formative (connective tissue)
  -remodeling (resorb cementum)
  -sensory (proprioceptive and tactile sensitivity
  -physical and nutritive
  - .2mm wide and decrease width as you increase in age; immature elastin = oxytalan +eluanin;
  - *Oxytalan* fibers run parallel to root surface in vertical direction and bend to attach to cementum in the cervical third of the root; regulates vascular flow.
  - Major cells of PDL: 1) FIBROBLASTS, macrophages, and ectomesenchymal cells.
    2) cementoblasts and clasts
    3) osteoblasts and clasts
    4) cell rests of malassez
    5) vascular and neural elements.
  - Nerve endings in PDL = 1) free unmyelinated nerves – convey pain
    2) encapsulated myelinated nerves – convey pressure.
- 4 traits that affect PDL health: 1) ant. teeth have slight/no contact in MIC.
  2) occlusal table <60% of overall F/L width of teeth
  3) occlusal table 90° to tooth's long axis.
  4) mandibular crowns inclined 15-20% toward the lingual.
- Epithelial Rests of Malassez – groups of epithelial cells located in PDL; remnants of epithelial root sheath that remain following disintegration during root formation.
- PDL is thicker in functioning teeth than non-functioning teeth.
- CEJ curves toward the apex F/L and away from apex M/D; curvature gets smaller as approach molars.

- greatest contour of cervical lines and gingival attachements occur on the MESIAL surface of anterior teeth with the greatest cervical line curvature on the mesial of the max. central incisor.
- Attached gingiva can withstand frictional forces but alveolar mucosa cant.
- *Functional Adequate Zone of Gingiva* is keratinized and firmly bond to bone; 2mm or > in width and resistant to probing.
- Keratinized Tissue (all stratified squamous epithelium) – hard palate, vermillion border of lips, dorsum of tongue, and gingiva.
- Masticatory Mucosa – free & attached gingiva and hard palate; keratinized;
- Lining/Reflective Mucosa – mucosa that lines most of the oral cavity; non-keratinized epithelium.
- Specialized Mucosa – covers dorsum of tongue and taste buds; keratinized.
- Junction of lining mucosa with masticatory mucosa is Mucogingival Junction.
- Alveolar Process (2 Parts).
  1) Alveolar Bone Proper – part of alveolar process that immediately surrounds the root of tooth and PDL fibers are attached;
     a. Perforates Cribiform Plate (2 layers) – 1)Compact Lameller Bone(spongy and compact)
        2)Layer of bundle bone (PDL fibers insert into it)
  2) Supporting Alveolar Bone – surrounds alveolar bone proper and supports the socket; 2 layers:
     a. Corticol Plate (thicker in mand.)
     b. Spongy bone (fills in b/w corticol plate of bone); it is not in ant. region or radicular buccal bone of max. post. teeth where cortical plate fused to cribiform plate.
  - Compact bone - 1) cribiform plate (socket) – bundle bone (PDL attaches)
    2) cortical plate (under gingiva)
    -both are separated by spongy bone.
- Epithelial attachement has no rete pegs.
- Hydrodynamic Theory – root sensitivity caused from indirect innervation from dentinal fluid mvmt in tubules, which stimulates mechanoreceptors in pulp.
- *Vitamin C* is needed for collagen formation for hydroxylation of proline to lysine.
- CEMENTUM – thickness from 0.05-0.6mm; radicular cementum (thicker than coronal) is cementum on root and coronal cementum is cementum on enamel; deposition of new cementum continues periodically throughout life so root fractures can be repaired.
  - Cellular cementum contains cementocytes and mostly in apical 1/3 of root and furcations; formed after tooth reaches occlusal plane.
  - Acellular cementum is cementum w/o cells and mostly in coronal 2/3 of root and thinnest at CEJ; major role is tooth anchorage; first formed cementum.
  - Main function of cementum:1) can resorb but cant remodel!
    2) **the attachements of principal fibers of PDL**
    3) protects root surface from resorption
    4) compensates for loss of tooth structure from occlusal wear by apical deposition of cementum.
    5) reparative fct that allows reattachment of C.T. after perio tx.
  - 2 collagen fibers in cementum are sharpey's fibers (perpendicular to cementum) and type 1 collagen (parallel to cementum).

PLAQUE & CALCULUS:

- Layer of **biofilm** covers calculus which causes plaque to attach.
- Plaque bacterial development: gram ⊕ facultative to gram – anaerobic bacteria.
- Plaque - accumulation of mixed bacterial community (>1010 bacteria/mg) in a DEXTRAN MATRIX;
- PLAQUE = 80% water & 20% solids (95% bacteria); also contains calcium & phosphorus (from saliva)
- Plaque is most likely to accumulate on INTERPROXIMAL tooth surfaces first.
- Plaque – small # of epithelial cells, leukocytes, and macrophages; cells contain extracellular matrix with proteins, polysaccharides, and lipids;

- Extracellular/dextran matrix is insoluble and sticky;
- Gram ⊕ Facultative= S. Mutans and Sanguis and Actinomyces viscosus.
- Gram – Anaerobic = Aa, Capnocytophypa species, Eikenella Corrodens, P. Gingivalis.
- Pellicle – glycoprotein deposite (plaque).
- Formation: 1) Formation of pellicle – albumin, lysozyme, amylase, IgA, proteins, & mucins.
  2) Bacterial Colonization – 1) primary colonizers = gram ⊕ - S. Sanguis & Mutans & Actinomyces viscosus.
  2) secondary colonizers = gram – at 1-3 days of plaque --Fusobactium, Prevotella, Capnocytophaga
  3) tertiary colonizers --P. Gingivalis, Campylobactar, Eikinella, Aa, & Treponema.
  3) Maturation Stage – bacterial intercellular adhesion results.
  4) Day 1-2 = cocci
  5) Day 2-4 = cocci dominant with filaments and rods.
  6) Day 4-7 = increase in filaments and mixed flora begins.
  7) Day 7-14 = vibrios and spirochets w/ WBC's, more gram – anaerobes; -signs of inflammation.
  8) Day 14-21 = vibrios and spirochets in older plaque with filamentous forms; -gingivits evident clinically.

- Calculus = inorganic content of 70-90% with Calcium, Phosphate, Magnesium and Carbonate.
  -2/3 of the inorganic matter is hydroxyapatite;
  -organic components are microorganisms, epithelial cells, leukocytes, and mucin.
  -Calculus Formation takes about 12 days; it is formed by bathing the plaque in highly concentration solution of calcium and phosphorus from saliva.
- Supra-G Calculus – white/yellow; *lingual* of mand. Inc and *buccal* of max. molars the most b/c salivary gland; attaches by salivary pellicle; attached or tooth associated; Saliva & Diet alter its bacterial composition.
  - Gram positive facultative cocci – S. Sanguis and Mutans, Actinomyces Viscosus.
- Sub-G Calculus – dark color b/c blood breakdown products and more dense than Supra-G calculus; formed from gingival fluid secretions; attaches by irregularities in Cementum; unattached or loosely adherent; Saliva & Diet DON'T alter its bacterial compostion.
  - Gram negative anaerobic rods/spirochets – P. Gingivalis, Fusobacterium Nuclatum, Prevotella Intermedia, Bacteroides.
  - Sub-G root surface roughness doesn't interfere with healing after SRP.
- Microbiologic etiologic factor in periodontal diseases is PLAQUE while calculus is the most significant LOCAL contributing factor.
- Primary reason to remove calculus is b/c it harbors plaque organisms.
- S. Viridins is an alpha-hemolytic streptococci that are common oral flora!

INSTRUMENTS:

- Most effective instrument of sub-G Sc/Rp is sharp curet; working angle $<90^{o}$ or $>45^{o}$.
- RP promotes soft-tissue attachement/**re-epitheliazation which occurs in 7-10 days.**
- Most important factor to determine amount of shrinkage is degree of edema.
- Healing begins with blood clot formation and neutrophils predominate immediately after curettage (1^{st} 12 hrs).
- Chisel is best for removing supra-G calculus interproximal for ant. teeth; single straight cutting edge with flat blade beveled at 45^{o}.
- Currettage – removal of sulcular epithelium and inflammed connective tissue; NEUTROPHILS predominate immediately after currettage; incidental currettage occurs during Sc/Rp.
  - Objective: Maximum shrinkage after gingival currettage of tissue that is edematous.
  - Contraindications for Currettage: 1) acute perio inflammation

2) fibrotic tissue
3) infrabony pockets
4) mucogingival involvements
5) when later wall is too thin.

- In order for new attachment, need enough undifferentiated mesenchymal cells present, complete removal of calculus, and complete removal of junctional/pocket epithelium.

- Gracey Curets: (60^o to cutting surface)
  I. #1/2 & 3/4 – short shank distance and for ant. proximals and B/L posteriors.
  II. #5/6 – 2 different shank lengths but same as #1/2.
  III. #7/8 – universal (cutting surface is 90^o)
  IV. #9/10 – B/L of PM & molars; long contra-angle design.
  V. #11/12 – mesial of post. teeth.
  VI. #13/14 & #15/16 – distal of posterior teeth.
- Graceys: 1) offset blade beveled 60-70^o.
  2) curved in 2 planes.
  3) 1 cutting edge.
  -lower shank is parallel to tooth surface.
- Universal: 1) not offset with 90^o to shank.
  2) 2 cutting edges.
  3) curved in 1 plane.
  -lower shank **slightly tilted toward the tooth**.
- Curettes are smaller than scalers and have greater tactile sensitivity than scalers so best instrument for Sub-G calculus detection & removal.
- when sharpening, avoid producing "wire-edge" by finishing with down stroke.
- Properly sharped instrument with NO ROUND SURFACES will not reflect light.
- when sharpening, lubricant allows metallic particles to be suspended in lubricant so prevents scratching/glazing of stone; use oil with national stones and water w/ artifical stones.
- manual sharpening is preferred;
- instruments whose cutting edge is >90^o will slip over the calculus.
- Sharpening gracey and universal curettes are essentially the same.
- <u>Curet</u> – greater tactile sensitivity than scaler; cutting edge parallel and curved; smaller than gracey; first do short strokes and then long strokes.
- Root planing strokes are longer and lighter than scaling strokes.
- <u>Periodontal Files:</u> (cutting edge 90^o) crush/fracture accessible Supra G calculus; best on B/L surface; good for distal of last molar; use Vertical Pull-type strokes and can reduce amalgam overhands.
- <u>Hoes:</u> (single and straight cutting edge 90^o) only vertical pull-type strokes; B/L surfaces are best;
- Hoes and Files are used exclusively for HEAVY Supra-G calculus removal but may be used sub-G if gross calculus only and tissue is flexible and easily displaced; both have thick blades and lack of tactile sensitivity and adaptability; curettes used after hoe and files are used!
- Most important plaque retentive factor is calculus!
- Probe angle 10^o to detect crater but mostly parallel to long axis of tooth; probe has 0.5 tapered shaft.
- Periodontal probe is adapted in proximal areas so touches contact area with tip angled SLIGHTLY BELOW & BEYOND the contact area.
- Clinical probing > histologic/pocket depth; accuracy +/= 1mm.
- Most important reason for using periodontal probe is to determine ATTACHMENT LOSS!
- Naber's 2N or Hamp Probe are used to detect furcations.
- Correct probe force is 10-20 g so depresses thumb pad 1-2 mm.
- Record pocket depths > 3 mm and when gingival crest < 2mm at/below CEJ.
- In healthy gums, crest of alveolar bone is 1-2mm below CEJ.
- Most common error during probing in EXCESSIVELY ANGLING the probe interproximally.
- Probe should always be in contact with tooth and FLAT against the tooth.

- Recession (gingival atrophy) is measured as positive value so if gingival margin coronal to CEJ then recession is negative.
- Bacteremia can occur even with mastication or brushing, so must premedicate if probing.
- Toothbrush trauma (abrasion) – usually occurs on canine and PMs; most common is left canine of right handed people; MOST COMMON etiology factor for gingival recession.
  - Dentin abraded 25x more than enamel and cementum 35x more.
  - Gingival Clefts – narrow groves that extend from crest of gingival to attached gingiva.
- Most difficult arears to Sc/Rp are trifucations of Max. Molars.
- Cementum, dentin and calculus are all removed during Sc/Rp.
- In RP, working stroke begins at apical edge of junctional epithelium (base of sulcus/pocket).
- Probing/Working Stroke is upward & downward movement w/in pocket.
- Scaling storke is short and powerful PULL stroke; the motion to initiate a scaling stroek is from the FOREARM.
- Common clinical changes 1 wk after SRP include reduced pockets and gingival inflammation.
- 3 Basic Strokes: 1) Exploratory/Assessment Stroke
  2) Scaling Stroke – short and powerful pull stroke.
  3) Root Planing Stroke – long overlapping pull strokes. (less pressure)
- Order of strokes for Sc/Rp = vertical, oblique and then horizontal.
- Correct angulcation of currette facial surface to tooth is **70-80º.**
- straight shanks for anterior areas and contra-angle shanks for posterior areas.
- After perio tx, the 1st recal should be in 3 mo. and then can be lengthened to 4-6 months.
- Most difficult areas to scale are: 1)mesial of max. PMs
  2)proximals of mand. Incisors.
  3) **trifurcations of max. molars**. (MOST DIFFICULT!)
- Best clinical aid to determin if Sub-G calculus has been removed is explorer & BWs.
- If Curette tip breaks off: 1) use another currette ina spoon-like stroke to pull the fragment out of sulcus;
  2) take PA and place pt. UPRIGHT.
  3) check floor of the mouth and mucobuccal fold.
  4) best way to prevent curette breakage is proper sharpening technique.
- Power-Driven Scalers: use either magnetostrictive (ELLIPTICAL VIBRATION PATTERN) or piezoelectric technology (LINEAR VIBRATION PATTERN) to convert electrical energy to physical energy at tip; based on use/principal of HIGH-FREQENCY SOUND WAVES;
  - vibrates from 25,000-40,000 cycles/sec and amplitude = 10-13 μm.
  - Use side of tip with water for cooling which causes water "cavitation" which releases dissolved gases.
- Sonic intruments do not release heat the way untrasonics do, they are air-turbine intruments that use air pressure to produce tip vibrations form 2,000-6,000.

OHI:

- The primary cause of disease recurrence is dentist team failure to motivate pt to practice effective plaque control.
- Dentinal hypersensitivity (cold sensitivity) is common after perio surgery due to clinical exposure of root surfaces; best tx = diligent OH!
- Orange, green, and brown stains on anterior teeth are caused by poor OH!
- Extrinsic Dental Stains: 1)brown stain – due to pellicle; color from TANNIN.
  2) black stain – chromogenic bacteria (actinomyces)
  3) green/green-yellow stain – common in kids due to fluorescent bacteria.
  4) metallic stain – vary from green to black depending on metal.
- Tooth brush must have soft, nylon bristles and a small head.
- Methods for tooth brushing:

1. Bass Method/Sulcular Technique – brush bristles place 45° to tooth and brush moved in back and forth motion for 20 strokes; PREFERRED METHOD FOR BRUSHING!
2. Modified Stillman Method/Rolled Technique – brush resting partial on teeth and partially on gingiva; gingiva is blanched by tooth brush and moved back and forth strokes with brush moving coronally simultaneously.
3. Charter's Method – brush pointed away from gingival margin at 45°.

- 3 components of SUPERFLOSS: 1) stiff-end threader – for under appliances
  2) spongy floss – b/w wide spaces
  3) regular floss – for interproximal plaque.
- Tooth Paste Ingredients: 1) Fluoride
  2) abrasives – calcium phosphate or calcium carbonate
  -removes stain & plaque
  3) surfactants/detergents – sodium lauryl sulfate (for foam)
  4) humectants – glycerin/water (for texture/moisture)
  5) binder/thickener – cellulose gum
  6) flavoring agents and sweeteners
  7) coloring agent – titanium dioxide
- Chlorohexidine Gluconate 12% (peridex/perioguard) – 30 sec for 2x/day; helps control gingivits and greatest risidual concentration in mouth after its use; NOT teratogenic.
  - Causes reversible, yellow-brown to brown stains in teeth, tongue, and resin restorations; impairs taste perception; the stain is due to presense of aldehydes & ketones.
  - Retention properties that are concentration and time dependent.
  - Its effectiveness due to greatest residual concentration in mouth after its use.
- Gingivitis decreases with *Phenol-based* mouth rinses – LISTERINE and *Quaternary Ammonium* compounds – SCOPE & CEPACOL;
  - Phenol based rinses contain 20-27% alcohol; essential oils are flavoring agents.
- Perio Aid – tapered round tooth pick for tracing motion along gingival margins; cleans class II furcations.
- Stim-U-Dent – balsa wood wedges for gingival massage, interdental recession, and dislodging interproximal debris.
- Proxabrush – for interproximal brushing.
- Interdental stimulator – rubber tip of smooth/ribbed conical shape; massages and stimulates circulation of interdental gingiva; don't use if normal and filled gingiva.
- Water Irrigation Devices – around bridges and ortho appliances; doesn't remove all plaque.
  - Oral irrigation devices are contraindicated in pts with periodontal inflammation and pts requiring antibiotic premedication.
- Polishing teeth is contraindicated in: 1) communicable disease
  2) respiratory problems
  3) green stains
  4) newly erupted teeth
  5) pt at risk for dental caries
- Disinfectants/Antibiotics:
  1. Actisite – ethylene vinyl acetate flexible fiber impregnated with 12.7 mg of tetracycline HCl; for 7-10 days Sub G then removed.
  2. Atridox – biodegradable controlled release gel (7 dy)containing doxycycline; delivered via syringe.
  3. Perio Chip – gelatin chip contains 2.5 mg of chlorohexidine gluconate; bio-absorbable over 8 days.
  4. Periostate – 2x/day tablet of 20 mg doxycline.

PERIODONTAL DISEASE:

- periodontal disease may be autoimmune disorder; periodontitis always begins w/ gingivitis!
- BWs are most accurate to assess alveolar bone resorption

- Smoking/nicotine – *increase* inflammation by **reducing oxygen** in gingival tissue and trigger overproduction of cytokines; smoking can cause bone loss and recession even in absence of periodontal disease; risk of periodontitis is directly affected by # of cigarettes smoked.
    - Smoking cigars and pipes carries equal risk as cigarettes.
- Patients with diabetes have 15x's increase risk of periodontal disease than nondiabetics; they have higher levels of specific inflammatory chemicals like interleukins.
- Periodontal diseases is associated with:
    1) Down's syndrome
    2) HIV/AIDS
    3) Hormone imbalances
    4) uncontrolled Type 1 & 2 diabetes mellitus
    5) WBC disorders & Autoimmune diseases
    6) Medications
    7) Smoking
    8) Osteoporosis
- Osteoporosis (loss of bone density) - associated with periodontal disease in post-menapausal women.
- Criteria for diagnosis Gingivitis -
    -color (most common color change is cyanosis - bluish)
    -contour (gingiva should be scalloped)
    -tone (normal consistency)
    -size (knife edge thickness)
    -plaque/calculus
    - Gingivitis is the PREDOMINANT periodontal disease.
    - Best way to evaluate amt and distribution of plaque is with disclosing solution.
    - IgG is most abundant immunoglobin in gingival exudates and common in gingivitis.
- 3 stages of Gingivitis:
    1. Transient Stage – 2-4 days after cessation of OH; margination of leukocytes on junctional epithelium.
    2. Developing Stage – collagen destruction increases and fluid fills in destruction with IgG; *lymphocytes predominate* and macrophages.
    3. Chronic Stage – *Plasma cells predominant* in lamina dura; IgG (from plasma cells) and IgA (from salive) and IgM (rarely).
- Agranulocytosis & neutropenia associated with periodontal disease.
- Localized Acute Gingivitis is most common form of gingival periodontal disease in school-aged kids.
- Pregnancy Gingivitis – common sign is gingival hemorrhage to gentle pressue;
    - increase levels of Prevotella Intermedia – this bacteria craves progesterone of its metabolism.
    - Gingival changes common in pregnancy because increase progesterone and increase in masts cells.
    - Sc/Rp, polishing and OHI ok during 1st and 2nd trimester.
- Radiographic changes in Peridontitis:
    1) loss of lamina dura
    2) horizontal/vertical bone loss
    3) widening of PDL
- Inflammatory Gingival Enlargement – significant increase in pockets causing pseudopockets.
- Dilantin Hyperplasia = progressive proliferation response to gingiva associated with use of sodium dilantin/Phenytoin; caused by plaque accumulation and increased accumulation of inflammatory cells; 50-60% of people on dilantin will get hyperplasia; if OH is good, prolly wont obtain hyperplasia.
- 20% of people on calcium channel blockers will get gingival hyperplasia.
- 20-30% of people on cyclosporin A (immunosuppressant) will get gingival hyperplasia.
- Hereditary Gingivofibromatosis – rare genetic diseases causing generalized diffuse gingival enlargement, enough to cover the teeth; *lack of inflammatory cells and proliferating capillaries*.
    - Erythmatous changes are result of secondary bacterial involvement.
- Tx for Inflammatory Gingival Enlargement and Hereditary Gingivofibromatosis is GINGIVECTOMY.
- Aggressive Periodontitis (formerly Juvenile Periodontitis) – 2 forms:

1. <u>*Generalized*</u> – 12-25 yrs old; rapid severe periodontal destruction around most teeth and severe attachment loss; *Prevotella Intermedia* and *Eikenella Corrodens*.
2. <u>*Localized*</u> – 12-19 yrs old; rapid and severe attachement confined to incisors or 1st molars with absence of plaque; etiology – genetics or neutrophil dysfunction; *Aa* and *Capnocytophaga* (both are also associated with periodontitis in juvenile diabetes.
   - Good tx for Periodontitis with Aa bacteria is TETRACYCLINE!

- Periodontitis progresses slowly and painlessly but is ARRESTED with proper therapy.
- At least 30% of bone mass at the alveolar crest must be lost for a change in bone height to be recognized in xray; reduction in .5-1mm thickness of cortical plate is sufficient to see bone destruction in radiograph.
- Periodontitis cant be diagnosed w/o xrays but xrays are not definitive diagnostic tool with furcation involvement or interdental craters.
- <u>Desquamative Gingivitis</u> – fiery red marginal and attached gingiva which demonstrates ulcerated and necrotic epithelium that sloughs off with air blasts.
  - Maybe manifestation of lichen planus or vesiculobullous disorder like pemphigoid.
  - **Atrophic**/eroded gingiva; loss of stippling; middle-aged to elderly females.
  - Affects B/L attached tissue; rete pegs short/abscent.
  - Other Etiologies – allergy, crohn's disease, psoriasis, or chronic ulcerative stomatitis.
  - Tx = steroids/corticosteroids depending on etiology; if dermatologic etiology then usually resolves when skin disease resolves.
- <u>ANUG</u> – 18-30 yrs; AKA – vincent's infection or trench mouth; acute recurring gingival infection of complex etiology with necrosis of papilla; no attachment loss;
  - History of soreness/pain and bleeding gums form eating/brusing; fetor oris (odor), low-grade fever, lymphadenopathy and malaise.
  - interproximal necrosis and pseudomembrane formation on marginal tissue;
  - Prevotella intermedia and Treponema spirochetes and Fusiform spirochetes.
  - Dominant WBC = neutrophils; predisposed if smoke or neglect.
  - Tx = debridgement, hydrogen peroxide rinses and antibiotics (**PCN V**, if not PCN, then tetracycline); pts with HIV and ANUG require gentle debridement and antimicrobial rinses.

<u>BACTERIA:</u>

- The most likely source of bacteria found in diseased periodontal tissue is Sub – G plaque!
- In healthy mouth, more than 350 species of bacteria, w/ periodontal infections linked to < 5%.
- Periodontal HEALTH = gram positive NONMOTILE FACULTATIVE ANAEROBES.
  - S. Gordininii & Actinomyces
- Periodontal DISEASE = gram negative MOTILE STRICT ANAEROBES.
- <u>Aggressive & Localized Aggressive Peridontitis</u> - Actinobacillus Actinomycetemcomitans (Aa)
- <u>Chronic Periodontitis</u> – Porphyromonas Gingivalis
- <u>Deep Pockets and ANUG</u> – Prevotella Intermedia, Treponema, Denticola, Sokranskii
- Also associated with Periodontitis – Bacteroids Forsythus
- <u>Endotoxin</u> – Lipopolysaccharide base in cell wall of gram negative bacteria; exists in plaque and gingiva; promotes bone resorption by decreasing osteogenesis and chemotaxis of neutrophils;
- Plaque Bacteria produces enzymes that initiate peridontal disease:
  1) Collagenase – catalyzes degradation of collagen (produced by *Bacteroides*)
  2) Hyaluronidase (produced by *S. Mitans & Salivarius*) & 3) Chondroitin Sulfatase (produced by *Diptheroids*) – leads to destruction of amorphous ground substance.
- Acute gingivitis = gram ⊕ bacteria like Actinomyces and Strep.
- Chronic gingivitis = gram – bacteria like Fusobacterium, Prevotella, and Capnocytophaga.
- Oxygen is major determining factor in different bacteria.
- Oral cavity is sterile at birth but bacteria present at 10-12 hrs after birth;
  - After 1 yr – S. Salivarious (most abundent), Staph, Neisseria, Actinomyces, Fusobacterium.

- At age 4-5, oral flora like adults.

INFLAMMATION:

- PMNs (neutrophilic leukocytes) are the first line of defense and first cells to migrate to gingival sulcus when inflammation is caused by plaque formation; while Polymorphonuclear Leukocytes are main cell components in CHRONIC inflammation.
- bacteria that forms plaque/calculus relase toxins that stimulate immune system to overproduce powerful infection fighting factors called CYTOKINES:
  - cytokines are related to all periodontal disease: ie – TNF α, IL – 1B, IL – 4, and prostaglandin E-2.
  - Cytokines are for healing but can cause inflammation from overproducing *collagenase* which breaks down proteins including connective tissue around teeth;
  - often have hyperinflammatory monocyte/macrophage phenotype.
- Lymphocytes:
  1) B-Cells – wbc that mature in bone marrow and migrate to lymphoid organs; antibody-producing plasma cells involved in antibody-mediated immunity; travels to spleen/lymph to differenciate.
  2) T-Cells – wbc that mature in thymus and become thymocytes; important in cell-mediated immunity and type 4 hypersensitivity rxns and modulation of antibody-mediated immunity;
     a. Classes: T-helper cells, Suppressor T-cells, and cytotoxic (killer) cells.
     b. Pts with periodontal disease have T-lymphocytes sensitized to plaque bacterial antigens.
- Inflammation of Gingivitis: 1) Initial (2-4 dys) – neutrophils.
  2) Gingivitis (4-7 dys) – lymphocytes, macrophages, IgG, and mast cells.
  3) Chronic (wks – yrs) – increase in plasma cells (IgG) and B lymphocytes.
- When gingivitis turns to periodontitis – gaine lymphocytes, plasma cells, and **macrophages** (represent transition b/w acute and chronic inflammation).
- *3 phases of Acute Inflammation* –
  1) Vascular – vasocontriction, vasodilation, and increased vascular permeability; basophils, mast cells, and platelets.
  2) Cellular – first defense cells are leukocytes/**neutrophils** (via chemotaxis – chemotatic factors C5a and Leukotriene B_4 {LTB_4}); PMNs engulf matter by phagocytosis - phagosome & phagolysosome.
  3) Repair – either by regeneration or replacement.
- *4 signs of Acute Inflammation* - 1) redness – dilation of capillaries (from histamine)
  2) heat – increased blood flow
  3) swelling – increased capillary permeability (from histamine)
  4) pain – lysis of blood cells that trigger bradykinin and prostaglandins.
- Mast cells increase in number with increased inflammation; releases heparin/**histamine** in response to injury/inflammation; mast cells participate in early phase of inflammation.
  - Major storage sights for histamine are mast cells, platelets and basophils.
  - Anaphylactic response is characterized by degranulation of mast cells.
- Eosinophils are not in vascular phase but are predominant in allergic rxns and parasitic infections.

TRAUMA & INFECTIONS:

- Radiographic signs of reversible occlusal trauma: 1) widening of PDL
  2) thickening lamina dura
  3) angular bone loss
  4) root resorption
  5) hypercementosis
- Other signs of occlusal trauma: 1) alternating repair and resorption of bone
  2) fibrosis of alveolar bone marrow spaces
  3) cemental resorption leading to dentinal resorption
  4) cemental tears

5) ankylosis
6) pulpal necrosis/calcification

- Primary occlusal trauma – when occlusal trauma is principal etiology in changes in periodontium.
  - Early effect is hemorrhage and thrombosis of blood vessels in PDL.
- Secondary occlusal trauma – when peridontium is already compromised by inflammation and bone loss so cant withstand occlusal forces well;
  - Early effect is mobility
- Rosin in periodontal swelling used as filler for strength;
- Types of periodontal dressings:
  a. Eugenol dressing (hard pack) = powder + liquid (eugenol); ie – PPC, Wards.
  b. Non-Eugenol (soft pack) = base + accelerator; ie – Coe-Pak & PerioCare; today periodontal dressings don't contain eugenol b/c it causes its own tissue injury and necrosis.
  c. Light-Cure = syringe; ie – Barricaid
- Periodontal dressings have no well-defined effect on process of wound healing or surgical outcomes; Must be removed in 7-10 days.
- After acute periodontal abscesses exude, they become chronic.
- Bruxism: primary causes – occlusal prematurities, muscle tension, and emotional factors.
  - S & S: PDL widening and thickening of lamina dura, sore muscles, and jaw pain, difficulty opening mouth, increased mobility, and occlusal wear facets.
- If periodontal abscess is localized then perform IND; if not then Rx antibiotics; the most prevalent symptom is acute pain and can cause rapid alveolar bone loss.
- Splinting Teeth: primary reason for splinting is to IMMOBILIZE excessively mobile teeth for patient comfort; provides even distribution for occlusal forces; often on teeth with reduced periodontal support;
  - teeth tend to loosen B/L not M/D. *Types of Splints*:
  1. External – ligatures, tooth bonding, etc.; unesthestic and unhygienic; lack durability and fit but no tooth structure is removed.
     a. Night Guards – primary purpose is to modify/control bruxism or to REDIRECT FORCES inot a non-traumatic pattern; use CR occlusal splints.
  2. Intracoronal – amalgam/acrylic w/ embedded wire and acrylic for provisional splints; tooth structure removed; more serviceable than external splints but tend to break and plaque build-up.
- Steps in adjusting occlusion: eliminate prematurities in CR, in protrusive mvmt, and lateral excursive mvmt.

PERIO SURGERY:

- Autogenous free gingival graft – totally dependent on the bed of **recipient** blood vessels! This tx is good for increased width of attached gingiva for widening recession of gingiva and prophylactically to prevent recession in thin gingiva;
- Allograft – graft taken from 1 human and placed in another human; a freeze-dried decalcified bone graft aken from a human donor & placed in a periodontal defect in another human is also an allograft.
- Hemopoietic marrow is the bone donor graft with the greatest osteogenic potential.
- Free Gingival Graft – autogenous graft placed on viable connective tissue bed on B/L mucosa; donor site is often **edentulous area** or **palatal area**; success depends on graft being immobilized at recipient site.
  - Graft epithelium first *degenerates*, then sloughs, and reconstructed in a week; at 2 wks, the tissue reformed but maturation takes 10-16 wks.
  - Top layer of graft is revascularized last; re-epithelization occurs by proliferation of epithelial cells from adjacent tissue and surviving basal cells of the graft tissue.
  - Healing time is proportional to graft thickness and the greatest amt of thickness occurs in 1st 6 mo.

- Free gingival graft is not as successful w/ deep wide recession so use laterally reposition flap/pedicle graft which has a greater predictability.
- Often used in conjunction with frenectomy.
- Rarely used on F/L of mand. 3rd molars.

- Hemisection – vertical sectioning thru both crown and root; ofter Mand. Molars; ½ of tooth extracted and tx like premolar.
- Root Amputation - separating root from crown; mostly max. 1st & 2nd molars;
- Both hemisection and root amputation result in irreversible pulpal damage requiring RCT after resection.
- Osseous Recontouring – used to eliminate pockets! Also other treatment for eliminating pockets:
    a. Maintenance
    b. Bone grafts
    c. Reattachment – filled procedures
    d. Hemisection/root amputation
- Palatal flaps cant be displaced!!
- Flaps are most common perio surgeries and full thinkness flaps are most common!
- Full-thickness flaps are used where attached gingiva is thin (<2mm wide).
- Partial thickness flap includes only mucosa and bone not exposed; used when a dehiscence/fenestration is present; used when attached gingiva is thick (base of flap is 2mm/more).
- Internal Bevel Incision – the incision from which the flap is reflected to expose the bone/root; the incision… 1) removes pocket lining
    2) conserves relative uninvolved outer gingiva
    3) sharp thin flap margin for adapting tooth-bone junction
- Distal Wedge – simplest distal flap for retromolar reduction; performed after TE of 3rd s b/c bone fill is poor leaving periodontal defect; base of wedge is periosteum and apex is gingival surface; performed if:
    - Sufficient space distal to last molar
    - Max tuberosity
    - Mand retromolar triangle
    - Distal to last tooth in arch.
- Gingivectomy – pocket depth eliminated by resecting the tissue coronal to pocket base; also bevel/contour the coronal margin; must have adequate attached gingiva and no infrabony defects.
    - Factors affecting surgery – pocket depth, access to bone, amt of attached gingiva.
    - When determining gingivectomy vs. periodontal flap – if base of pocket is located at the mucogingival junction or apical to the alveolar crest DO NOT perform a gingivectomy.
- Gingivoplasty – reshapes gingiva and papilla for correcting deformities; objective is more physiological tissue contour *not reduced pockets*; common tx for ANUG.
- Primary objective to surgical flap procedures is to provide access to root surfaces for debridement.
- Modified Widman Flap – modification of replaced flap; full-thickness flap; for open flap debridement and regenerative periodontal procedures; *objectives*:
    - Gain access
    - Reduce pocket depths
    - Preserve adequate attached gingiva
    - Provide env't for healing by primary closure
    - Indications: 1) pockets with bases located coronal to mucogingival junction
        2) little/no thickening of marginal bone
        3) shallow to moderate pocket depths can be reduced.
        4) where esthetics are important.
- Reposition Flaps: 1) Replaced flaps, 2) MWF, 3) Excisional new attachement procedures.

- Heal by repair & they are pocket reduction procedures that gain clinical attachment mediated by repair.

- Positioned Flaps: when coronal margins of flap are lifted from an area adjacent to recipient site but flap isnt free'd up.
  - 1) laterally repositioned flaps, 2) coronally positioned flaps, & 3) apically positioned flaps;
  - vascular supply maintained so no necrotic sloughing; heal by repair.
- Apically Positioned Flap: full thickness, mucoperiosteal flap; high degree of predictability and "work-horse" of perio therapy; *indications*:
    - Moderate to deep pockets
    - Furcation involved teeth
    - Crown lengthening
  - Flap is sutured more apically, so exposing alveolar margin to form broader zone of gingiva
  - Objective is to surgically eliminate deep pockets by positioning the flap apically while retaining the attached gingiva.
  - Max molars palatal surface – trim flap margin to proper length;
  - *Contraindications*: pt risk for root caries and unesthetic if tooth exposed.
- Coronally Positioned Flap – full-thickness flap exclusively used to restore gingival height and zone of attached gingiva over isolated areas of recession.
- Pedicle Graft (lateral positioned flap) – first perio surgery for root coverage; defect covered by stretching flap laterally until free end comes over it; superior esthetics but less versatile;
  - base of graft remains attached to donor site for uninterrupted blood supply so position and repositioned flaps can be pedicle grafts; often full-thickness flaps.
  - Indications: 1) widen zone of attached gingiva
    2) repair isolated recession
  - Advantages: 1) predictable correction/prevention of recession
    2) minor post-op discomfort
    3) good esthetics.
  - Contraindications: 1) lacks attached gingiva
    2) donor site has fenestration/dehiscence of supporting bone.
- Guided Tissue Regeneration – blocks repopulation of root surface to allow cells from PDL and bone to repopulate bone defect; use either:
  - Non-resorbable barriers – expanded polytetrafluoroethylene (teflon)
  - Resorbable barriers – type 1 collagen, calcium sulfate (plaster of paris) , or polyactic acid.
- Most common reason for free gingival graft failures = disruption of blood supply b/f engraftment and 2nd reason is infection.
- Double Papilla Flap = variation of laterally positioned flap; gingiva b/w teeth on either side are moved over exposed root; indications:
    - Trauma from brushing
    - Covering exposed root surfaces
- Dental alveolar process less susceptible to permanent damage after surgical exposure than B/L plates of bone;
- Four rules of flap design: 1) base of flap wider than free margin
  2) lines of incision not placed over any defect
  3) incisions that traverse bony eminence (canine) should be avoided.
  4) all corners of flap should be rounded.
- Free Mucosal Autografts – when transplant of connective tissue w/o epithelial covering (differ from free gingival grafts); formation of keratinized tissue even if not keratinized recipient; often canines where little keratinized gingiva.
- Osteoplasty – reshaping/recontouring bone that is non-supportive bone (not attached to PDL);
  indications: 1)deep proximal pockets of buccal bone.
  2) pockets on B/L/P surfaces where resorpion causes ledges

3) tilted 2nd molar adjacent to no 1st molar

- Ostectomy – removal of osseous defects or infrabony pockets (below the crest of bone) by eliminating bony pocket walls; bone is supportive in nature; *indications*: 1) interproximal craters
  2) deep interproximal pockets where neighbor areas are intact.
  3) shallow infrabony defect (proximal) where reattachment failed.
  - *Contraindications*: 1) if weakens support for adjacent tooth.
- In some surgial procedures, it is necessary to leave interradicular bone exposed which may result in bone loss.
- Without direct visualization provided by a flap, it is rare that a clinician can effectively root plane beyond 5mm of probing depth or into root furcations of lesser value.
- Most critical factor in determining tooth prognosis is amt. of attachment loss!
- Defects that "will hold water" offer excellent opportunities for bone graft containment and periodontal regeneration procedures.
- Bone graft success depends on # of bony walls of defect; 3-walled defect is best and worst is thru-thru furcation of max. molar.
- Best indicator of success of periodontal flap procedure is postoperative maintenance and plaque control by the patient.
- Root resorption most likely side effect of autogenous bone graft.

PHARMACOLOGY

SYMPATHOMIMETICS:

- Autonomic Nervous System:
    1. SYMPATHETIC ("fight or flight")
        a. Preganglionic→CHOLINERGIC→Acetylcholine.
        b. Postganglionic→ADRENERGIC→NOREPI, EPI, & Dopamine (exception – innervation to sweat glands is cholinergic and secrete Ach)
    2. PARASYMPATHETIC ("rest & digest")
        a. Preganglionic→CHOLINERGIC→Acetylcholine
        b. Postganglionic→CHOLINERGIC→Acetylcholine (Muscarinic Response)
- Drugs that produce tissue responses resembling those produced by the sympathestic nervous system; adrenergic agonists; ie – dopamine, epi, norepi, isoproterenol, and phenylephrine.
    - α_1 – causes *contraction* & *vasoconstriction* of blood vessels so decreases hypotension;
        → Controls hemorrhage(**EPI**/adrenalin), allergic shock(**EPI**/adrenalin), nasal congestion(phenylephrine – **Neo-synephrine**);
        → Contracts sphincter muscles in intestines, urinary bladder & uterus; while β relaxes those muscles; also in fat cells & platelets.
    - α_2 – nerve endings; found in presynaptic nerve endings to inhibits NE release and postsynaptic nerve endings to decrease sympathetic tone.
    - β_1 receptor – increases cardiac output & conraction via cardiac muscle; least common receptor.
        - Cardiac stimulation(**isoproterenol –** for asthma);
    - β_2 receptor – ↑dilation of bronchi and relaxation of arterioles; ONLY EPI!; also ↑blood glucose; bronchodilation (albuterol); Beta receptors mostly *vasodilation* & *relaxation*.
    - alphas predominantly excitatory while betas are excitatory in hear but inhibitory elsewhere.
- Post Junction α1 – smooth muscle of iris, arterioles, veins, and GI tract (relaxes it!).
- Pre Junction α2 – inhibits norepi release; found on post-synaptic endings in CNS to↓sympathetic tone.
- Post Junction β1 – in heart(mainly b1 receptors), intestine smooth muscle, and adipose tissue.
- Post Junction β2 – bronchodilator and vascular smooth muscle.
- Cranial nerves w/ parasympathetic activity – 3, 7, 9, & 10.
- Catecholamines – sympathomimetic compounds composed of catechol molecule & aliphatic portion of amine; ie – epi, norepi, & isoproterenol: all direct acting catecholamines; also, Ach, Dopa, dobutamine, seratonin, GABA, opoids, & glutamate & aspartate; they pass blood brain barrier very poorly.
- Epinephrine – catecholamine; physical properties unknown; rapid onset and *prolongs duration of LA*; stimulates α & β adrenergic receptors w/in sympathetic division of ANS.
    - Epi is the prototypical **adrenergic agonist;**
    - During anaphylaxis, extreme reduction in BP & bronchospasms, EPI stimulates α1 (vasocontriction), stimulates β2 (dilates bronchioles), stimulates β1 (increase cardiac output).
    - it produces physiologic actions that are opposite the effects of HISTAMINE.
    - It also decrease blood volume in nasal tissues and relieves nasal, sinus, & throat congestion.
    - Restores cardiac activity in cardiac arrest; tx for glaucoma by reducing internal eye pressure.
    - Can be administered thru IV, sublingually, subcutaneously, or intramuscularly;
    - Contraindication – pts w/ ANGINA; side effects – headache, anxiety, tachycardia; caution in pts w/ high BP and hyperthyroidism.
- Norepinephrine – catecholamine that works on alpha 1 & 2, and beta 1 receptors.
    - For vasoconstriciton in hypotension.
- Isoproterenol – is b1 & b2 agonist and the MOST POTENT bronchodilator; cause cardiac stimulation.
- Dopamine – immediate precursor to NE; catecholamine w/ 2 subtypes: D1 – activates adenylcyclase & D2 – inhibits adenylcyclase.
    - Dopamine & Dobutamine both used for shock & heart failure.

- Seratonin – 5-Hydroxytryptamine work thru 14 subreceptor "trytominergic" type neurons.
- Glutamate & Aspartate – amino acids that have powerful EXCITATORY effect on every region in CNS;
- Sympathetic activation of eye – *mydriasis* (dilation), heart – *tachycardia* (↑HR), salivary gland – thick, ropey saliva (↓*saliva*); activation of parasympathetic division of ANS causes opposite of these rxns!
- Ephedrine – non-catecholamine for urinary inconstinence & vasoconstriction in hypotension.
- Phenylephrine – non-catecholamine for mydriasis, vasoconstriction, & decongestion.
- Oxymetazoline & Xylometazoline – causes nasal decongestion.
- Adrenergic agonists are direct acting or indirecting acting (store and release NOREPI).
- Amphetamines – *sympathomimetic amines* stimulate both CNS & PNS; pass readily thru CNS and release NE; potent CNS stimulants; increase systolic & diastolic BPs and weak bronchodilators; Used for treatments in…
    1. ADHD – **dexedrine**, **adderall** (dextroamphentamine) instead of ritalin(**methyphenidate**).
    2. Narcolepsy – **dexedrine** (prevents daytime sleep)
    3. Weight Loss – **lonamine** (phentermine)
- ADHD Treatment:
    1) **Methylphenidate** (**Ritalin**) – mild CNS stimulant.
    2) **Focalin** – ner form of ritalin called Dexmethylphenidate.
    3) **Concerta** – long-acting form of methylphenidate.
    4) **Adderall** – mixed amphetamine salts (mix of dextroamphetamine & amphetamine).
    5) **Strattera** – name for atemoxetine (1st non-stimulant)
    6) **Metadate CR** – controlled delivery of methylphenidate.
    7) **Dexedrine** – Dextroamphetamine.
- Selective Direct-Acting Adrenergic Agonists:
    1. **Phenylephrine** (Neo-synephrine) – a1 selective agonist; nasal decongestant and tx orthostatic hypotension and prevents LA diffusion away from injection site; 100x less potent than epi.
    2. **Clonidine** (Catapres) – a2 selective agonist; anti-hypertensive agent.
    3. **Dobutamine** – b1 selective agonists.
    4. **Terbutaline** – b2 selective agonist; administered orally, subcutaneously, or inhalation to treat longterm obstructive disease and ER tx of bronchospasm.
    5. **Albuterol** – b2 selective agonist;
- α_1 Adrenergic Blockers (-ZOSIN)– cause tachycardia, vasodilation, ↓BP, and orthostatic hypotension.
    - Ie – **Doxazosin**(long DOA) & **Prazosin** - ↑BP; **Terazosin** – tx for benign prostate hyperplasia.
- Anti-Hypertensives – 4 forms:
    1. β - adrenergic blockers (-OLOL): common side effect is drowsiness & weakness;
        a. **Propranolol, Timolol, Nadolol**– Block both β1 & β2 receptors; ↓BP by ↓CO; contraindicated in pts w/ asthma or COPD b/c cause fatal bronchospasm; also contraindicated in insulin-dependent diabetes pts b/ block hypoglycemia recovery.
            i. *Propranolol* – major anti-anginal effect by blocking β-adrenergic heart receptors; drug of choice for adrenergically induced arrythmias.
        b. **Metaprolol** (Lopressor) & **Atenolol** (Tenormin)– cardioselectively block β1 receptors.
            i. *Metaprolol* – B1 blocker for tx for angina & ↑BP; causes drowsiness.
            ii. *Atenolol* – B1 blocker w/ long DOA; tx for chronic angina & ↑BP; low lipid solubility and renally eliminated; long duration of action.
            iii. Both Metaprolol & Atenolo are longer-acting & more predictable than Propranolol and safer to use in pts w/ asthma or bronchitis.
        c. **Acebutolol** (Sectral) – cardioselective B1 blocker & partial B2 blocker; tx for ↑BP & ventricular arrythmias; ↓solubility & mild intrinsic sympathomimetic (similar to Pindolol);
    2. α - adrenergic blockers: cause tachycardia, lower BP, vasodilation, & orthostatic hypotension.
        a. *Non-Selective blockers:* don't treat cardiac conditions b/c can cause tachycardia & palpitations.

i. **Phentolamine Hydrochloride** & **Phenoxybenzamine Hydrochloride**– block both α1 & α2 for tx of presurgical management of pheochromocytoma (tumor of adrenal glands that releases excessive EPI & NE).

b. *Selective Blockers:* blocks a1 to treat hypertension & benign prostatic hyperplasia(BPH).
   i. **Doxazosin** – blocks α1 to tx hypertension w/ long DOA.
   ii. **Prazosin** – blocks α1 but rarely used to tx hypertension.
   iii. **Terazosin** – blocks α1 to manage mild to moderate hypertension and BPH.
   iv. **Tolazoline** – blocks α2 for tx of pulmonary hypertension in newborn; causes direct peripheral vasodilation.

c. Major adverse affect is hypotension;

d. α - adrenergic blockers can cause EPI REVERSAL; the anti-adrenergics reverse pressor action of adrenalin/EPI; they block both EPI & NE but then EPI causes low BP b/c stimulates β2 receptors too and they are not blocked by alpha blockers.

3. Central Acting Agents: a2 selective AGONISTS that inhibit adrenergic nerve transmission thru actions w/in CNS;
   a. **Clonidine**, **Guanfacine**, **Gaunabenz**, **Methyldopa**.
      i. *Clonidine* – a2 selective agonist
      ii. *Methyldopa* – hypertensive tx for renal damage (good w/ diuretic); produces false transmitter that replaces NE; side effects – CV, CNS, GI, hepatitis, and cirrhosis.
      iii. *Guanfacine* & *Guanabenz* – stimulated centrally α2 and ↓SNS flow & reduce vascular resistance; Tx – antihypertensive; used either alone or w/ diuretic.
4. Neuronal Depleting Agents: deplete catecholamine (NE) & seratonin from adrenergic terminals and in the brain;
   a. **Reserpine** (blocks NE, EPI & seratonin) & **Guanethidine** (blocks NE).

- α blockers block epi(adrenaline) and the depressor response mediated by β2 receptors (↓BP).
- α & β blocking agents act as COMPETITIVE INHIBITION on post-junctional receptors.
- Drugs for Asthma – β2 agonists (bronchodilate) – Epi, **Albuterol**, **Salmeterol**, and **Metaproterenol**.
  - **Aminophylline** – theophylline compound – bronchodilator & relaxes smooth muscle of bronchi.

CHOLINERGICS:

- Cholinergic drugs stimulate acetylcholine cholinergic receptors; they cause **↑salivation, sweating, GI motility, miosis(constriction), ↑flushing & bradycardia; ↑secretions & muscle weakness!**
  - Direct-Acting (Esters & Alkaloids): **Methocholine**, **Carbochol**, **Bethanecol**, **Pilocarpine**.
  - Indirect-Acting (Cholinesterase Inhibitors) : **Neostigmine**, **Physostigmine**, **Edrophonium**, & **Pyridostigmine**;
  - 2 Cholinergic AGONISTS drugs in Dentistry:
    1) **Pilocarpine** (Salagen)– tx for xerostomia from salivary gland hypofunction in cancer pts.
    2) **Cevimeline** (Evoxac)– specific for M3 receptor in salivary glands; tx of xerostomia in Sjrogen's Syndrome.
- 3 classes of *Cholinergic Agonists:* stimulate muscarinic site & *mimic Ach*; if any of these cholinergic agents are administered b/f ACh, the action of Ach is enhanced & prolonged.
  1. Choline Esters:↓BP w/ generalized vasodilation; ↓HR, ↑GI tone, miosis thru ↓intraocular pressure;
     a. **Acetylcholine Chloride** – tx to produce miosis; methacholine (not used as much).
     b. **Bethanecol** – post-op abdominal distension & urinary retention.
     c. **Carbachol** – tx to produce miosis.
     d. **Methacholine** – not used much anymore.
  2. Cholinergic Alkaloids: Muscarine, Pilocarpine, Nicotine, Lobeline;
     a. **Pilocarpine** - most useful alkaloid for miotic & tx of glaucoma & xerostomia.
     b. Both Choline esters & Cholinergic alkaloids stimulate smooth muscle activity and both are direct-acting cholinomimetic agents.

3. Cholinesterase Inhibitors: inhibit acetylcholinesterase at both muscarinic & nicotinic sites (indirect acting cholinomimetic agents); cholinesterase inhiibitors also ↑secretions b/c they ↓ACh metabolism; they increase effects of Ach w/in autonomic nervous system & at NMJ.
    a. **Physostigmine**, **Neostigmine**, **Endrophonium**, **Pyridostigmine**, **Malathion**, **Parathion**.
    b. **Endrophium** – drug of choice in diagnosing myasthenia gravis b/c rapid onset and reversibility; distinguishes myasthenia gravis from cholinergic crisis b/c improves MG but worsens cholinergic crisis.
    c. **Neostigmine** & **Pyridostigmine** – tx for myasthenia gravis.
    d. **Malathion** & **Parathion** – insecticides.

- Organophosphates (CHOLINERGIC)– esters of phosphoric acid & alcohol that inhibit cholinesterase;
  - **Isofluorophate**(glaucoma), **Malathion**(insecticide), **Parathion**(insectiside), **Echothiophate**(glauoma), **Tabun**(toxic nerve gas), **Metrifonate**(destroys intestinal worms).
- Pralidoxime (Protopam) – anti-cholinergic → cholinesterase reactivator which reverses muscle paralysis from organophosphate anti-cholinesterase pesticide poisoning;
    - Reversed effects of overdose of anti-chol agents used in tx of myasthenia gravis.
    - S&S of poisoning - ↑salivation, bronchoconstriction, diarrhea, & twitching.
- Stimulation of skeletal muscle by excess Ach eventually results in muscle paralysis.
- Anti-Cholinergics – block post-ganglionic cholinergic fibers; cause **XEROSTOMIA**, **MYDRIASIS**, **TACHYCARDIA & ↑body temp**, **↓SPASMS of smooth muscle of bladder**, **bronchi**, & **intestines**;
  - Anti-chols - no intrinsic activity, but cause xerostomia by blocking *postganglionic* cholinergic fibers and prevent Ach from occupying same receptor!
  - Contraindications - glaucoma, CV problems, asthma, GI obstruction;
  - Ie- **Beladona derivatives**, **Propantheline Bromide**.
  - **Glycopyrrolate** (Robinul) – treats traveler's diarrhea & anti-secretory.
  - **Benztropine Mesylate & Trihexyphenidyl HCl** – treat Parkinson's (anti-parkinsonism).
  - **Atropine sulphate** – produces mydriasis & cycloplegia (paralysis of the ciliary muscle of the eye).
  - **Scopolamine** (pre-op med) – prevents/reduces motion sickness.
  - **Mecampylamine** (Inversine)– nicotinic ganglion-blocking agent.
- Anti-Sialogogues – drugs that control salivary secretions; anti-cholinergics; also reduce spasms of smooth muscle and accelerate impulse conduction thru the myocardium by blocking vagal impulses.
- Acetylcholine – chemical mediator of all AUTONOMIC ganglia & parasympathetic post-ganglionic synapses; ACh alters cell membrane permeability & is secreted by cholinergic fibers; affects CNS by acting on these 2 receptors:
  1. *Muscarinic Receptors:* primarily in autonomic effector cells(heart, vascular endothelium, smooth muscle, presynaptic nerves terminals & exocrine glands) in CNS (also responds to Muscarine).
  2. *Nicotinic Receptors:* located in ganglia, skeletal muscle end plates & in CNS(also responds to nicotine); drugs like Ach mirror effects of para-post-ganglionic activity; 2 receptors:
     i. Receptors @**Neuromuscular Jcts** of somatic nervous system; Neuromuscular blockers act here.
     ii. Receptors @Autonomic Ganglia of both PSNS & SNS; Ganglionic blockers act here.
- LA prevents/reduces liberation of Ach at neuro-muscular jct of skeletal muscle;
- 2 types of Nicotinic Receptors:
  1. *Neuromuscular Blockers* – at neuromuscular jct of somatic system.
  2. *Ganglionic Blockers* – at autonomic ganglia (both symp & parasymp); rarely used because cause pronounced xerostomia, constipation, blurred vision, and postural hypotension.
     - **Mecamylamine** & **Trimethaphan** are used for ↑BP, ER ↑ in BP, & bloodless field surgery.
- Neuromuscular Blocking Agents: produce complete skeletal muscle relaxation & facilitate endotracheal intubation; interact w/ nicotinic receptors at NMJ; two types:
  1. *Nondepolarizing* – competitively compete w/ Ach at nicotinic receptors & prevent Ach from stimulating motor nerves & *can result in paralysis*;

a. prototype of Non-depolarizing NMJ blocker = **Tubocurare**
b. **Mivacurium**, **Vecurium**, **Doxacurium**, **Pancuronium**, **Atracurium**, **Cisatracurium**, & **Rocuronium**; Neostigmine & Pyridostigmine can reverse these!

2. *Depolarizing* – noncompetitive;
   c. **Succinylcholine** (Anectine) – nicotinic agonist & depolarizes the neuromuscular end plate; prototype for Depolarizing NMJ blocking agent.
      - Used w/ caution in pts w/ ↓levels of pseudocholinesterase, which breaks down succinycholine – resp. failure may result; may cause muscarinic response like bradycardia & increased glandular secretions; used if laryngospasm occurs during GA.

- Spasmolytic Drugs (skeletal muscle relaxants) – relieve muscle spasms w/o paralysis; act on CNS & skelatal muscle cells; used in MS, cerebral palsy, cerbrovascular accidents/strokes).
- Treatment for Chronic Muscle Spasms:
  1. **Baclofen** – derivative of GABA (site of action in reducing muscle spasms) that tx chronic muscle spasms; tx of MS & other spinal cord diseases;
  2. **Carisoprodal** (Soma) – tx of muscle spasms & acute TMJ pain.
- Treatment of Acute Muscle Spasms:
  1. **Cyclobenzaprine** – relieves muscle spasm thru central action.
  2. **Methocarbamol** – centrally acting muscle relaxant to relieve acute pain & tetanus.
- **Quinidine** – tx for nocturnal leg crapms;

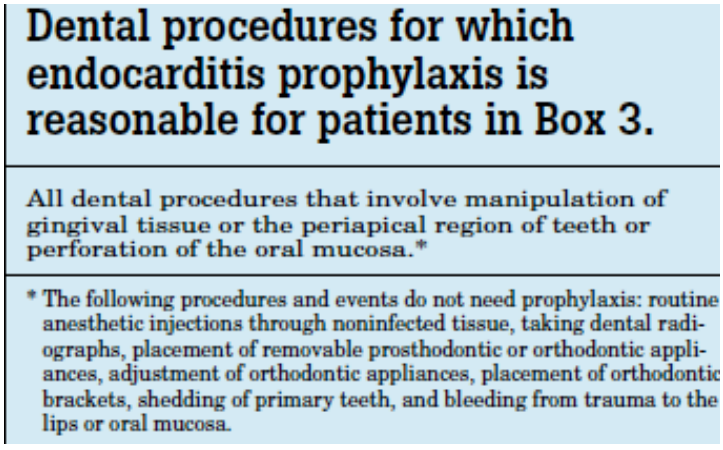
ANESTHESIA:

- IV agents for GA:
  1. Barbituates – Thiopental, Methohexital, Ketamine, Etomidate, Propofol.
  2. Benzodiazepines – Diazepam, Midazolam, Lorazepam.
  3. Neuroleptic Opoids – neurolept analgesics & fentanyl, and droperidol.
- Nitrous (BLUE)– rapid onset w/ recovery in 5 min; less soluble in blood than alveolar air; considered sedative but not GA unless >80% which can cause hypoxia; gas at room temp & pressure.
  - Sweet smelling, colorless & inert gas; coupled w/ no less than 20% O_2.(fail safe method).
  - Used to produce SEDATION & MILD ANALGESIA but must be coupled w/ LA.
  - Excreted unchanged by lungs; stored as liquid under pressure; onset of sedation = 5min.
  - Pt given oxygen for 5-10 min after taken off Nitrous to prevent diffusion hypoxia.
  - Dose response for NO: 10-20% - extremity tingling
    20-40%(usually 30-50%) – sleepiness & relaxation
    >50% - too much, nausea & sweating.
  - *Contraindications* – pts w/ URI, pregnancy(1st trimester), bronchitis, emphysema, and speech problems and pts w/ contagious diseases.
  - Most common complaint from pts on NO is mild NAUSEA.
- Chloral Hydrate – only non-barbituate sedative hypnotic agent & induces sleep;
  - DOESN'T RELIEVE PAIN.
  - Orally for preop management of anxious kids; kids excited and then sedated;
  - Rapid onset (15-30 min) & DOA = 4 hrs; kids – 50mg/kg w/ max 1gm in 500mg/5mL solution.
  - unpleasant ordor & taste; prodrug & metabolized to trichloroethanol(displaces warfarin).
- Toxicity of LA – causes bradycardia and decrease cardiac output; affects CNS & CV system; may cause restlessness, stimulation, tremors, seizures, CNS depression, slowed respiration, & coma.
- Allergy to LA – may present as nasolabial swelling, itching, and oral mucosal swelling;
- **LA** reversibly blocks sodium from going from outside to inside of axon; so LA decreases sodium UPTAKE thru the axon's sodium channels; no effect on potassium; decreases pain by blocking propogation of nerve impulses;
  - Small, unmyelinated nerves (pain) affected 1st because greater surface volume.;
  - Nonionized free-base form penetrate tissue; fat soluble/lipophilic drugs; converted to hydrophilic salts(water soluble) to prepare as injectable solution; pH=7.8.

- ↓pKa = ↑pH = more free-base available for injection.
- At physiological pH of 7.4, 5-20% of LA in free-base form so enough to anesthetized.
- Action of all LA's depends on anesthetic salt ability to liberate free-base
- Max dose = 300mg; 4.4mg/kg for kids; Max carps →Lido – 8.3 carps, Mepivacaine (3%) – 5.6, Prilocaine(4%) – 5.6, Bupivacaine(.05%) – 10 carps.
- 1kg = 2.3lbs; MAX DOSE of LIDO = 300mg or 4.4mg/kg for kids.
- Amide LAs are metabolized in liver, so toxicity is more likely if amides given to pts w/ liver dysfunction.

- ***POINT – potential action of all LA depends on ability of anesthetic SALT to LIBERATE FREE-BASE.
- **Articaine** (4% HCl) – amide LA; has ester group so could be inactivated by plasma cholinesterase; only amide metabolized in bloodstream; onset = 1-6min & DOA =1hr;
  → volume = 1.7mL & Max dose = 7mg/kg or 490kg.
  → contraindicated in pts w/ bisulfite or LA amide allergy.
- **Prilocaine(Citanest) –**intermediate DOA, **longer acting than Lido** but less potent & less vasodilation than Lido; metabolized as orthotoluidine -causes *methemoglobinemia*– not for hypoxic pts;
  - MAX DOSE = 400mg
- **Bupivacaine (Marcaine)** – has longest DOA of any LA; Radiotoxic in some pts & used w/ causing in CV disease, elderly, & peds; MAX DOSE = 90mg
- **Lidocaine** – anti-arrythmic agent of the ventricle; acts on fibrillating ventricles to decrease cardiac excitability & spares the atria;
- ***Lidocaine & Mepivacaine most likely to show cross-allergy.
- **Mepivacaine** (Carbocaine) – equal to lido in efficacy but ineffective as topical agent; short DOA and toxic to NEONATES; MAX DOSE = 300mg.
- Ester LA – mainly used as topical (BENZOCAINE) due to allergies; procaine/novocaine metabolized & forms paraminobenzoic acid (PABA) which pts can be allergic to; no longer used in dentistry; rapid onset & short DOA except tetracaine which has longer DOA.
- **Cocaine** – 1st LA ever; ester of benzoic acid; definite **vasoconstriction**; ONLY LA that increases pressor activity of EPI & NE by inhibiting catecholamine uptake by adrenergic nerve terminals.
- Bisulfites (preservative for epi) can cause allergy in LA; only in LA w/ epi so 3% **mepivacaine** (carbocaine) doesn't have epi so no bisulfites; most pts w/ allergy to LA have history of asthma and airway hyperactivity to sulfites.

ANTIBIOTICS:



Dental procedures for which endocarditis prophylaxis is reasonable for patients in Box 3.

All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa.*

* The following procedures and events do not need prophylaxis: routine anesthetic injections through noninfected tissue, taking dental radiographs, placement of removable prosthodontic or orthodontic appliances, adjustment of orthodontic appliances, placement of orthodontic brackets, shedding of primary teeth, and bleeding from trauma to the lips or oral mucosa.

DON'T NEED PROPHYLAXIS
1. Restorative Dentistry w/o retraction cord.
2. LA injections but nonintraligament inj.
3. Intracanal RCT
4. Rubber Dam placement
5. Post-op suture removal
6. Impressions & Fl^- tx

- The following procedures were identified as having a higher incidence of bacteremia: dental extractions; periodontal procedures, including surgery, subgingival placement of antiobiotic fibers/strips, scaling and root planing, probing, recall maintenance; dental implant placement and replantation of avulsed teeth; endodontic (root canal) instrumentation or surgery only beyond the apex; initial placement of orthodontic bands but not brackets; intraligamentary & intraosseous local anesthetic injections; prophylactic cleaning of teeth or implants where bleeding is anticipated.

Cardiac conditions associated with the highest risk of adverse outcome from endocarditis for which prophylaxis with dental procedures is reasonable.

- Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
- Previous infective endocarditis
- Congenital heart disease (CHD)*
  - Unrepaired cyanotic CHD, including palliative shunts and conduits
  - Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure†
  - Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

- Other Conditions for YES for Prophylaxis:
  1. Tetrology of Fallot
  2. Total Joint Replacement ONLY if surgery w/in the past 2 years!
- Other Conditions for NO for Prophylaxis:
  1. Rheumatic Heart Disease
  2. MVP w/ or w/o Regurgitation
  3. Septal Defects or Patent Ductus Arteriosus
  4. Hypertrophic Cardiomyopathy
  5. Bypass Graft Surgery
  6. Heart Murmurs & Kawasaki Disease
  7. Cardiac Pacemakers & Implanted Defibrillators
- If a patient is already receiving antibiotic therapy with a medication that is also recommended for infective endocarditis (IE) prophylaxis, the guidelines state that it is prudent to select an antibiotic from another class rather than to increase the dose of the currently administered antibiotic. For example, if a patient is already taking amoxicillin, the dentist should select clindamycin, azithromycin, or clarithromycin for IE prophylaxis.If you don't want to take antibiotic from different class then delay procedure 9-14 days after pt completes antibiotic.
- If unanticipated bleeding occurs, administer prophylaxis w/in 2 hours after procedure.

ANTIBIOTIC PROPHYLACTIC REGIMENS FOR CERTAIN DENTAL PROCEDURES.*

SITUATION	ANTIBIOTIC†	REGIMEN‡
Standard Prophylaxis	Amoxicillin	Adults, 2.0 grams; children 50 milligrams/kilogram orally one hour before procedure
Cannot Use Oral Medications	Ampicillin	Adults, 2.0 g IM§ or IV§; children, 50 mg/kg IM or IV within 30 minutes before procedure
Allergic to Penicillin	Clindamycin	Adults, 600 mg; children, 20 mg/kg orally one hour before procedure
	Cephalexin or cefadroxil	Adults, 2.0 g; children, 50 mg/kg orally one hour before procedure
	Azithromycin or clarithromycin	Adults, 500 mg; children, 15 mg/kg orally one hour before procedure
Allergic to Penicillin and Unable to Take Oral Medications	Clindamycin	Adults, 600 mg; children, 15 mg/kg IV one hour before procedure
	Cefazolin	Adults, 1.0 g; children, 25 mg/kg IM or IV within 30 minutes before procedur

- **# of Capulses for Antibiotics of Prophylaxis:**
  - → **Amoxicillin** – 4 capsules (500mg/capsule)
  - → **Clindamycin** – 2 capsules (300mg/capsule)
  - → **Cephalexin** – 4 capsules (500mg/capsule)
  - → **Cefadroxil** – 4 capsules (500mg/ capsule)
- **Probenecid –** used w/ antibiotic to delay renal clearance of antibiotic; interferes w/ organic acids at nephron & diminishes the PCN tubular secretion;
  - → affects **PCNs** & **cephalosporins** other β-lactam antibiotics like **Aztreonam** & **Imipenem**.
  - → Drug of choice for tx of GOUT.
- Antibiotics **AFFECTING CELL WALL:**
  - → **PCN, Cephalosporins, Vancomycen, Imipenem, Cycloserine, Bacitracin, Aztreonam;**
- **Penicillin** – derivative of 6-aminopenicilllanic acid & contains β-lactam ring joined by thiazolidine ring; β-lactam (3C & 1N) ring is responsible for antibiotic activity;
  - → synthesized from L-cysteine & L-valine.
  - → PCN is good for ANUG pts; 10% of population allergic to PCN.
  - → β-lactam antibiotics – **PCN**, **Cephalosporins**, **Carbepenems**, & **Monobactams**.
  - → Excreted DIRECTLY into urine via renal tubular cell secretion.
- **Pen VK** – antibiotic w/ narrow spectrum & bacteriocidal; good for minimizing resistance; used to treat ORAL infections b/c more acid stable; highest incidence of drug allergy;
  - → drug of choice for gram+ staphylococcal infection;
- **PCN G** – PCN prototype due to basice 6-aminopenicillanic acid molecule; add side chains to make it semi-synthetic PCN – more stable and broader spectrum & more penicillinase resistant.
  - → PCN G Procaine (Crysticillin) – IM route
  - → PCN G Benzathine – IM route; tx for syphilis & prevent rheumatic fever; longer DOA.
- **Ampicillin** (IV/oral) & **Amoxicillin** (oral) – both AMINOPENICILLINS (also **Becampicillin**) b/c characterized by amino substitution of PCN G; neither penicillinase resistant; extended spectrum PCN.
  - → AminoPCNs work against many gram (-) more readilly than natural PCNs like *Haemophilus influenzae*, *Escherichia coli*, *Proteus mirabilis*.
  - → Both are preferred tx for UTI caused by enterococci; also tx for URI, otitis media, bronchitis, sinusitis, & bacterial cystitis.
  - → **Ampicillin** is good for pts who can take oral drugs and are NOT allergic to PCN;
  - → **Amoxicillin** significantly interacts w/ **Methotrexate**; Amox inhibits renal tubular secretion of methotrexate; methotrexate can cause ulceration of oral tissues.
  - → **Amoxicillin** - ↑oral absorption, ↑serum levels, ↑half-life, ↓GI effects than ampicillin; for gram+ cocci & gram (-) bacilli.
- **Methicillin** – part of the PCN family; not often used due to nephritis but give IV in sever PCN-producing STAPH infections;
  - **MRSA** (methicillin-resistant Staph Aureus) – resistant to all antibiotics including vacomycin.
  - Methicillin, PCN G, & Carbenicillin are degraded by stomach acid.
- **Carbenicillin**, **Piperacillin**, & **Ticaillin** – WIDEST broad spectrum of PCNs(Carbenicillin); all against gram (+) rods & cocci, like *Pseudomonas*, *Proteus*, *Klebsiella*, & *Bacteroides*;
  - → tx for UTI caused by Pseudomonas & Proteus; given parenterally (IV).
- **Bacampicillin** – tx for URI & LRI, UTI, & skin infections; hydrolyzed to amoxicillin when absorbed by GI; better absorption than ampicillin and less GI effects.
- **Bacitracin –** gram (+) bacteria; for topical use b/c nephrotoxic.
- **Polymyxin B –** cationic detergents that scrub bacteria cell membranes; topical use b/c nephrotoxity potential; against gram (-) rods = Pseudomonas; triple antibiotic ointment for superficial lacerations;
- Beta-Lactamase – enzyme of gram (+) & (-) bacteria that works agains PCNs & cephalosporins; adding *clavulanic acid* w/ PCN can inhibit the bacterial enzyme; MOA of enzyme is splitting open the β-lactam ring structure to render the antibiotic ineffective.

→ **Augmentin** – Amoxicillin & Clavulanic Acid.
→ **Unasyn** – Amoxicillin & Sulbactum; IV or IM.

- Penicillinase is a specific type of β-lactamase, showing specificity for penicillins, by hydrolysing the beta-lactam ring.
- Penicillinase-Resistant PCNs – **Methicillin(IV)**, **Nafcillin(IV)**, **Oxacillin(IV)**, **Cloxacillin(Oral)**, **Dicloxacillin(Oral)**; they have protected β-lactam ring which prevent penicillinase effects;
  - these PCNs are effective agains penicillinase-producing Staph Aureus.
  - **Ampicillin** (**unasyn**) & **Amoxicillin** (**augmentin**) – block penicillinase from reaching beta-lactam ring b/c contain clavulanate potassium & sulbactum.
  - **Dicloxacillin** – similar spectrum as Pen VK but active against penicillinase producing Staph.
- IV PCNs **– Methicillin, Carbenicillin, PCN G.**
- Acid Stable PCNs (Oral) **– PCN VK, Amox., Amp., Nafcillin, Oxacillin, Cloxacillin, & Dicloxacillin.**
- Extended Spectrum PCNs – Aminopenicillins (**Amp** & **Amox**).
- Broad Spectrum PCNs – **Carbenicillin**, **Piperacillin**, **Ticarcillin** – WIDEST spectrum of PCNs.
- **Cephalosporins**: PCN-like b/c affect cell wall; bacteriocidal; broad spectrum antibiotics(both gram(-) & ⊕; Increase in gram (-) but decrease in gram (+) as you increase generations; 4 generations:
  1. 1st Gen. – **Cephalexin**, **Cephradin**, **Cefadroxil**, **Cefazolin** – used to as antibiotic prophylactic in pts w/ non-immediate allergic rxn to PCN; **Cephalexin** & **Cephradine** are 1st choice for prophylactic in pts not allergic to PCN w/ Total Joint Replacement w/in 2 yrs.
  2. 2nd Gen. – **Cefaclar**, **Cefuroxime**, **Cefoxitin –** tx for oro-dental infections caused by gram (+) & (-) bacteria and against anaerobic bacteria causing periapical abcesses.
  3. 3rd Gen. – **Cefixime**, **Cefoperzone**
  4. 4th Gen. – **Cefepime**.

  → Used in PCN-allergic pts w/ Staph infections.
- Imipenem – β-lactam antibiotic from thienamycin & 1st drug classified as carbapenem antibiotic;
  → tx for Enterobacter infections; combined w/ **Cilastin** for tx of severe/resistant infections, esp nosocomial infections. .
- Aztreonam – synthestic β-lactam antibiotic; against gram (-) rods, like Klebsiella, Pseudomonas, & Serratia; synergistic w/ aminoglycosides.
- 10% of pts allergic to PCN are allergic to cephalosporins.
- 3 types of PCN allergic rxns:
  1. Anaphylactic Shock – 30 min; IgE mediated; characterized by urticaria, angioedema, bronchoconstriction, GI disturbances, & shock (hypotension); Tx immediately w/ EPI.
  2. Accelerated – 30-48 hours after; uticaria(hives), pruritis, wheezing, edema.
  3. Delayed – 2-3 days after; skin rashes; **80-90% of PCN allergies.**
- Rash is most common sign of allergy to PCN.
- Antibiotics **INTERFERING W/ PROTEIN SYNTHESIS:**
  → **Clindamycin**(50S), **Tetracycline** (30S), **Erythromycin**(50S), **Azithromycin, Aminoglycosides**(30S), **Linomycin, Clarithromycin, Chloramphenicol.**
- **Clindamycin** – bacteriostatic agains gram (+) like *Staph* & *Strep* & anaerobic gram (-) like *Bacteroid fagilis*; causes diarrhea and **pseudomembranous colitis** caused by overgrowth of *clostridium difficile.*
  → No cross allergenicity b/w PCNs & Clindamycin.
- **Tetracycline**: limited oral treatment; can cause candidiasis and photosensitivity; absorption into GI tract inhibited by cations (Ca, Mg, Fe, & Al) so don't take w/ milk, vitamins, or minerals; 3 types:
  1. **Tetracycline** – used for Local Aggressive(Juvenile) periodontitis, because good w/ AA bacteria, ANUG (if PCN is not used),
     a. acne, gonorrhea, syphilis, mycoplasma pneumonia, chlamydia, rickettsia, bronchitis.
  2. **Minocycline** – acne, anthrax, and meningococcal prophy; Ie – **Arestin**: used to tx periodontal pockets causing pocket to shrink.
  3. **Doxycline** – Syphilis, Rickettisa, Chlamydia, and mycoplasma infection.

→ Contraindicated w/ child<8yrs & pregnant women while Doxycline & Minocycline – both contraindicated in pregnant women.
→ BROAD-SPECTRUM antibiotic, for Gram (+) and Gram(–) bacteria; Tetracyclines arrest rapid bone loss via tissue regeneration & enhanced repair due to their collagenase inhibiting effect.
→ Absorption of tetracycline from GI tract inhibited by these cations – Ca, Mg, Fe, & Al; these cations form CHELATION PRODUCTS w/ tetracycline to prevent their absorption; so not given w/ milk, mineral supplements, or antacids.
→ Adverse Effects – photosensitivity, nausea, diarrhea, fungal superinfections (Candidiasis), teeth discoloration, & enamel hypoplasia in kids.

- MACROLIDE FAMILY OF ANTIBIOTICS – erythromycin-type antibiotitics that are effective against Gram (+) but NOT gram (-); GI upset; includes **azithromycin**, **clarithromycin**, & **dirithromycin**
- **Erythromycin** – causes 21% GI problems & tinnitus (deafness); metabolized in liver & excreted by bile; *enteric coated* – prevents release and absorption til reach intestines; poor oral bioavailability.
    a. 2 types: Erythromycin Stearate & Erythromycin Estolate; can cause liver toxicity.
    b. 2nd choice of antibiotic to PCN to tx Oro-dental infections caused by gram (+) bacteria.
    c. Previously used as alternate to PCN-allergic pts but no longer used due to GI upset, the most common side effect, so take with food;
- **Azithromycin** (Zithromax - 1x/day) & **Clarithromycin** (Z-Pak - 2x/day)– Azithromycin – 5% GI effects, Clarithromycin – 10% GI effects; prolonged elimination half-life.
    - both have similar bacterial spectrums as erythromycin but better against *H. influenza*;
    - concentration on macrophages so good against *Mycobacterium avium intracellulare*.
- Aminoglycosides (IV/IM)– may cause muscle weakness so may aggravate pts w/ myasthenia gravis, infant botulism, or Parkinsons; rapidly excreted by kidneys;
    → causes ototoxicity & nephrotoxicity so must be used for serious infections.
    → bacteriocidal & broad spectrum – aerobic gram (-) infections.
    → **Streptomycin** – 1st aminoglycoside for TB tx; rarely used.
    → **Gentamicin**, **Amikacin**, **Tobramycin**, **Netilmicin**, **Spectinomycin** (tx for Gonorrhea).
    → **Neomycin** (topically used b/c high toxicity potential) & **Kanamycin** (rarely used b/c of ototoxicity);
- **Chloramphenicol** – broad spectrum gram(+) & (-) & bacteriostatic; used as 2nd or 3rd line of drugs for serious infections b/c causes 3 toxicities: 1) aplastic anemia 2) bone marrow suppression 3) Gray's syndrome (circulatory collapse)
- Antibiotics **INTERFERING W/ BIOSYNTHETIC PATHWAYS:**
    → **Sulfonamides, Fluoroquinolones, Trimethoprim.**
- Sulfonamides (sulfa drugs) – similar structure to Para-aminobenzoic acid (PABA), which is used to synthesize folic acid in bacteria, which is used to help bacterial cell growth; BACTERIOSTATIC.
    → competes w/ PABA & *inhibits folic acid synthesis*, so inhibiting cell growth.
    → Tx for UTI; **Bactrim** = Trimethoprim(antimicrobial) + Sulfamethoxazole (sulfonamide); Bactrim is drug of choice for UTI.
    → NOT for dental infections;
- Tuberculosis – caused by Mycobacterium Tuberculosis (needs combination of drugs since mycobacterium tends to develop resistants to any single anti-tubular drug)).
    1. **Isoniazid** – 4 drug regimin w/ **rifampin**, **pyrazinamide**, & **ethambutol**; also used for prophylactic; may cause peripheral neuritis(paresthesia) caused by pyridoxine (vit B6) deficiency.
    2. **Streptomycin** – combo w/ isoniazid; aminoglycoside.
    3. **Rifampin** – prevents transcription; most potent anti-leprosy agent.
    4. **Ethambutol** – in combo; may cause optic neuritis, hyperuricemia, & color vision disturbances.
    5. **Pyrazinamid** – in combo; enters CSF to treat tuberculosis meningitis.
    6. Rifabutin – active against MAI complex.

- **ANTIPROTOZOALS**:
  1. **Nitrazoxanide** – tx of Giardia(diarrhea) which is common protozoan infection;
     a. tx of infections from Giardia Lamblia & Cryptosporidium Parvum.
     b. MOA – interfers w/ electron transfer rxn w/in protozoa that is essential to its metabolism.
  2. **Atovaquone** – tx of Pneumocystitis Carinii Penumonia (PCP), in pts intolerant to Co-trimazole(combination of Trimethoprime+Sulfamethoxazole – which is drug of choice for PCP by inhibiting folic acid synthesis.
  3. **Eflornithine** – orphan drug status for meningoencephalitic stage of Trypanosoma Brucei Gambiense Infection (Sleeping Sickness).
  4. **Furazolidone** – tx of diarrhea from Giardia Lamblia or Vibrio Cholerae.
  5. **Metronidazole** – antibacteria & antiprotozoal for Trichomonas Vaginalis; affects cell walls!
     a. not true antibiotic b/c SYNTHETIC & lab fabricated;
     b. most effective Rx against anaerobic bacterial infections;
     c. causes dizziness, headaches, and nausea.

- **ANTI-MALARIA AGENTS:**
  1. **Mefloquine** - against Plasmodium falciparum, P. vivas maraliae, P. ovale; active alone agains multi-drug resistant Plasmodium flaciparum.
  2. **Cloroquine** – eradicates RBC forms by inhibiting plasmodial heme polymerase; tx for erythrocytic forms of Plasmodium falciparum & vivax; systemic amebic liver abscess & extraintestinal amebias.
  3. **Quinine –** back up agent for chloroquine used in combination w/ Fansidar chloroquine-resistant malarial strains; adverse effects – Cinchonism -nausea, vomiting, vertigo, tinnitus.
  4. **Atovaquone** + **Proguanil** (Malarone)
  5. **Sulfadoxine** + **Pyrimethamine** (Fansidar)
  6. Halofantrine
  7. **Pyrimethamine-folate antagonist**: active against P.falciparum, P.malariae, & Toxoplasma gondii.

ANTIVIRALS:
- Viruses lack cell membrane, wall, & metabolic machinery, thus are Obligate Intracellular Parasites.
- Oseltamivir(tamiflu) & Zanamivir(relenza): antiviral neuraminidase inhibitors; tx for influenza A & B.
- Acyclovir (zovirax)– antiviral that inhibits DNA synthesis.
- *Herpes Simplex Type 1 Treatment:*
  1. **Penciclovir** (Denavir) – CREAM; tx of recurrent herpes labialis (cold sores) for adults; inhibits herpes viral DNA synthesis which inhibits viral replication.
  2. **Acyclovir –** inhibits viral DNA polyermase/viral DNA synthesis; TABLET/CREAM to tx HSV-1, HSV-2, & varicella zoster(chicken pox/shingles);
     a. Drug of choice for HSV Encephalitis, genital herpes, herpes labialis, & varicella-zoster virus;
     b. Enters CSF & accumulates during renal failure.
  3. **Docuosanol**(Abreva) & **Lysine** – anti-virals that tx Herpes Labialis.
  4. **Valacyclovir** (valtrax) – PRODRUG of acyclovir given orally that is coverted by 1st pass metabolism into Acyclovir; tx for HSV-1/2, genital herpes, cold sores & herpes zoster.
  5. **Ganciclovir** – inhibits viral DNA polyermase/viral DNA synthesis; tx Cytomegalic retinitis & CMV prophylaxis in transplant pts; cross BBB;
- **HIV** – depletion of T-cells (CD4); retrovirus w/ RNA as nucleic acid & uses reverse transcriptase to copy genome into DNA of host's chromosomes; DNA segment is permenently incorporated into host.
  → Tx – **Didanosine** (Videx), **Zidovudine** (Retrovir, AZT), **Ritonavir** (Norvir), **Indinavir** (Crixivan).
- Nucleoside Reverse Transcriptase Inhibitors – stops HIV RNA from becoming DNA; drugs converted into AZT-triphosphate analogs in cells to inhibit viral DNA synthesis & replication by inhibiting reverse transcriptase; may cause myelosuppression of bone marrow.
  → Ie – **Didanosine**, **Zalcitabine**, **Zidovudine**, **Stavudine, Lamivudine.**

- Protease Inhibitors – suppresses protease from cleaving viral precursors into peptides; contraindicated w/ pts taking Rifampin.
  → Ie – **Indinavir**, **Nelfinavir**, **Ritonavir**, & **Saquinivir**
- Non-Nucleoside Reverse Transcriptase Inhibitors – non-competitive inhibiting rxn of reverse transcriptase that is independent of nucleotide binding;
  → Ie – **Delavirdine**, **Adefovir**, **Efacirenz** & **Nevirapine**.
- **Interferon** – natural glycoproteins synthesized by recombinant DNA technology to activate host enzymes to block viral RNA translation and intervere w/ virus infecting cells.
  → Tx for chronic Hep B&C, Genital papilloma, Kaposi's sarcoma in HIV pts.
- **Amantadine** & **Rimantadine** – anti-viral that inhibit/block viral membrane matrix protein M2 ion channel; for prophy or tx of Influenza A virus; also enters CNS to tx Parkinson's.
- **Ribavirin** – inhibits viral mRNA synthesis; tx for serious Respiratory Synctial Virus infection for kids, influenza A&B, Hep C, & Sars; ORAL, IV, and Aerosol.

ANTIFUNGALS:

- Mycoses – chronic fungal infections; often superficial and subcutaneous.
- Candida Albicans – inflammatory pruritic infection characterized by white, thick discharge (also causes angular cheilitis); normal inhabitant of oral cavity & vaginal tract; Drug of choice for tx = **Nystatin**.
- List of Antifungals that alter cell membrane by binding to sterol in cell membrane:
  1. **Clotrimazole** – Mycelex Troche/Lozenge – for Oropharyngeal Candida; alters fungal cell membrane.
  2. **Nystatin** – Oral Suspension(swish & swallow)/Ointment – for Oral Candidiasis or Cutaneous; similar structure to **Amphotericin B;** alters fungal cell membrane.
  3. **Amphotericin-B** – Cream/IV inj. – Cutaneous/Systemic Candidiasis; alters fungal cell membrane by binding to ergosterol in fungal membrane; anti-fungal drug of choice for systemic fungal infections;
     → may cause Kidney Toxicity; does not enter CSF.
  4. **Ketoconozol** – Cream/Tablet – Cutaneous/Oral Candidiasis; inhibits Ergosterol synthesis to disrupt fungal membrane; can inhibit/antagonize **Amphotericin B** antifungal effect;
     → Given orally to treat *Histoplasmosis*, *Nonmeningeal coccidiodomycosis*, *Blastomycosis*, *Dermatomycosis*; toxicity may cause ENDOCRINE EFFECTS.
  5. **Fluconozole** – Tablet/Oral - Esophageal Candida; inhibits erogosterol synthesis; crosses BBB and enters CSF; drug of choice for Mucosal Candida;
     → tx for Blastomycosis, Histoplasmosis, & Cyptococcal meningitis in AIDS pts;
  6. **Itraconazole** – inhibits ergosterol synthesis; Broad-Spectrum anti-fungal give ORALLY; Drug of choice fo Blastomycosis & Paracoccidioidomycosis;
  7. **Flucytosine** – a PRODRUG that inhibits fungal DNA & RNA synthesis & cell division; give ORALLY to tx systemic mycosis of Chromoblastomycosis, Candidiasis, & Cryptococcus; enters CSF;
- **Nystatin** & **Clotrimazole** alter fungal cell membrane by binding to sterols in the fungal cell membrane, increasing permeability & permitting the leakage of intracellular components.

SEDATION:

- Tranquilizers; Anti-convulsants; Smooth Muscle relaxant; *Pre-op sedative*; induction agent & supplement for maintaining anesthesia;
- Tranquilizers promote calmness & soothing but w/o sedation or depressant effects;
  - Major Tranquilizers – anti-psychotic agents.
  - Minor Tranquilizers – anti-anxiety agents (benzos)
- Alleviate anxiety & induce sleep & IV causes CONSCIOUS sedation;
- Benzodiazepines, Barbituates, Narcotics all produce sedation & have ability to produce dependence;
- Benzos depresses limbic system & reticular formation thru strengthening GABA (gamma-aminobutyric acid, inhibitory neurotransmitter); NOT used during pregnancy.

- Benzos used for anti-anxiety, sedative, anti-convulsant, & skeletal muscle relaxant; used for IV CONSCIOUS sedation during outpatient surgery.
- Benzos are safer than barbituates; but causes fatigue, slurred speech, dry mouth, nausea, hypotension.
- Most effective oral sedative drug used in dentistry; Benzodiazepines do not provide Anesthesia!
- Oral Benzodiazepines: 1)**Chloridiazepoxide** (librium) – *pre-op sedative*
  2) **Diazepam** (valium) – *pre-op sedative*; anti-anxiety
  3) **Alprazolam** (Xanax) – anti-axiety, good for tx of Agoraphobia.
  4) **Lorazepam** (Ativan) – anti-anxiety.
  5)**Clonazepam** (Rivotril)
  6) **Temazepam** (Restoril)
- Benzodiazepines for *Insomnia*: 1) **Flurazepam** (Dalmane) & 2) **Triazolam** (Halcion)
  - **Triazolam** used as pre-op sedative in dentistry and metabolized in liver by P-450 isoform CYP3A4 enzyme; antifungal agents can increase levels of triazolam b/c they inhibit CYP3A4 isoform for hepatic metabolism of triazolam.
- **Diazepam** – preferred over barbituate as antianxiety; Tx for reversing status epilepticus caused by LA overdose; IV inj. into large vein; contra – glaucoma & psychosis; may cause withdrawal symptoms.
  - *Propylene Glycol* in the IV mix of valium is main cause of thrombophlebitis (vein clot).
  - Also used for muscle spasticity in pts w/ cerebral palsy.
- **Midazolam** – liquid benzo used for pre-op sedation in kids & as injectable for IV **conscious** sedation; very short half life; preferred over diazepam.
- **Flumazenil** (Mazicon)– BENZO ANTAGONIST; reverses benzo in event of overdose.
- **Buspirone** – oral anxiolytic; partial agonist on serotonic receptors (5-hydroxytryptamine) & diminishes serotonergic action; fewer side effects & less sedation than benzos.
  - structurally & physically differ from benzos & barbs b/c not anti-convulsant and doesn't cause sedation and not physically dependent and not hypnotic;
  - slow onset - up to 2weeks; may cause TARDIVE DYSKINESIA (involuntary mvmts);
- Ethyl Alcohol – cuases diuresis by inhibiting production of ADH/Vasopressin; ethanol dilates blood vessels in skin, depresses CNS and may cause coma/death;
  - It is a sedative, a hypnotic drug; alcohol euphoria from removal of inhibitory activity of the cortex;
  - Synergistic w/ **Diazepam**, **Meperidine**, **Pentobarbital**, & **Chlorpromazine**.
- **Disulfiram** (Antabuse) – manages ethanol abuse; inhibits aldehyde dehydrogenase (mitochondrial liver enzyme) so interferes w/ hepatic oxidation of acetaldehyde metabolism from alcohol.
- Metronidazole also inhibits aldehyde dehydrogenase.
- ANTICONVULSANTS:
  1. Phenytoin (Dilantin) (IV)– tx of tonic clonic (grand mal) seizures; may cause phenytoin-induced gingival hyperplasia; produces Na+ channel blockade; most extensively used;
  2. Gabapentin – adjunct to treatment of partial seizures.
  3. Carbamazepine (Tegretol)– prophy for partial seizures (psychomotor) & temporal lobe seizures & tx for tonic clonic seizures & trigeminal neuralgia; produces Na channel blockade in order to treat trigeminal neuralgia; **rare but may cause aplastic anemia.**
     a. Adverse effects – diploma, ataxia, enzyme induction, blood dyscrasias.
  4. Diazepam (Valium) – tx for staticus epilepticus & emergency treatment for seizures.
     a. Adverse effects – drowsiness, dizziness, & ataxia.
  5. Valproic Acid (Depakene)– causes neuronal membrane hyperpolarization; prefered tx for complex partical seizures, absence seizures, & multiple seizure types;
     a. Adverse effects - hepatotoxicity & dyscrasias, GI distress, lethargy, headache.
  6. Ethosuximide (Zarontin)– tx for absence seizures b/c causes minimal sedation by blocking Ca+ channels; adverse effects – GI distress, lethargy, & headache.
  - Most common anticonvulsants are CNS depressants; may cause respiratory depression.
- BARBITUATES: depress neuronal activity in the midbrain reticular formation by ↑membrane ion conductance (Cl^-) & ↓glutamate-induced depolarization & ↑inhibitory effects of GABA;

→ may develop serious drug dependency; anti-convulsant but NOT ANALGESIC!
→ Barbituates are well-absorbed orally; CNS depressant; metabolized in liver;
→ cause of death – resp. failure due but reversed w/ O_2 under positive pressue; so most important therapeutic measure taken in event of barb poisoning is to assure ADEQUATE RESPIRATION.
→ Barbs exhibit steeper dose-response relationships than benzos;
→ ↓ ½ life of drug metabolized in liver b/c induce formation of liver microsomal enzymes that metabolize in drugs; 4 types classed by DOA:
1. *Ultra-Short Acting* – IV for GA induction & Stage III surgical anesthesia.
   i. 5-20min; **thiopental** (MOST COMMON, for anesthesia), **methohexital**, **thiamylal**; contra-PROPHYRIA, liver dysfunction, emphysema, drug addiction.
2. *Short Acting* – oral for calming effect for pre-op appts & insomnia;
   i. 1-3hrs; **secobarbital** & **pentobarbital**; good for kids.
3. *Intermediate Acting* – relieve dental anxiety w/ daytime sedation & tx for insomnia.
   i. 3-6 hrs; amobarbital & butabarbital.
4. *Long Acting* – tx of daytime sedation & epilepsy.
   i. 6-10 hrs; **phenobarbital**(anti-convulsant), **mephobarbital**, **primadone**.

→ As decrease in DOA, increase in lipid solubility so Ultra-short acting has highest lipid solubility and rapidly leaves brain for other tissues due to increased solubility (reason for short DOA);

- Main target of INHALATION ANESTHETIC is brain; Lipophilic molecules; administration of anesthetic preceded by IV/IM barbituate w/ endotracheal intubation; 5 volatile liquids that require vaporization & may irritate respiratory tract & cause *malignant hyperthermial;* they cause ↓in arterial pressure.
  1. **Enflurane** – less potent but rapid onset with risks of seizures; CNS irritant effect.
  2. **Halothane** – powerful but toxin in adult liver; sensitizes heart to catecholamines.
  3. **Isoflurane** – combo with IV anesthetics; can cause heart irregularities.
  4. **Sevoflurane** – good for kids, less irritating with rapid awakening.
  5. **Desflurane** – heating component; irritating so used w/ IV agents but awaken faster than any other inhalant; has low blood:gas partition coefficient, but not used to induce anesthesia.

NARCOTICS:

- Opoids are analgesics, antitussives, antidiarrheals, & preanesthetic meds; DEA schedule II & III; opoid alkaloids = morphine & codeine; opoids raise pain threshold & tolerance;
  → Opiods are most powerful drugs for pain relief; reduces amt of GA required for surgical anesthesia; strongest opioids – **Morphine**, **Meperidine**, **Fentanyl**, & **Methadone**.
  → Side effects – sedation, drowsiness, dizziness, nausea (MOST COMMON SIDE EFFECT).
  → Respiratory Depression is major disadv. Of opioids & most significant adverse rxn.
  → Opoid Receptors:
    1. *Mu* – for morphine; the supraspinal analgesic activity of morphine is mediated primarily thru its influence on the Mu opioid receptor.
    2. *Delta* – for enkephalins
    3. *Kappa* – for dynorphins
    - Opoids bind to these receptors in brain to increase pain threshold.
- 3 types of Endogenous (produce naturally in body) Chemicals (produce morphine-like effects to reduce pain):
  1. β-endorphines – bind to opoid receptors and have potent analgesic activity.
  2. Enkephalins – bind to OPIOID DELTA receptors & more distributed than endorphines; role in pain, mvmt, & mood preception.
  3. Dynorphins - most POWERFUL opioid found throughout CNS & PNS that bind to Kappa receptors; regulates pain at spinal cord level, influences behavior at the hypothalamic level, & regulate CV system.

- **Morphine**(Opiates) **-** is the primary active agent in opium, an opium alkaloid; causes analgesia, drowsiness, euphoria, mental clouding, miosis, constipation, nausea, vomiting, & resp. depression.
  → IV or IM (2-3 hrs), oral (3-4 hrs), sustained release is 8-12 hurs;
  → NOT used in dentistry due to its addictive liability.
- Narcotic analgesics = effectively reduce pain (not-inflammation) by working in brain to block ascending pain impulses that travel from perphery (PNS) into brain (CNS); opoids – common in dentistry is HYDROCODONE (simlar potency as morphine); ok w/ coumadin/warfarin.
  → Hydrocodone + Acetaminophen = **Vicodin**, **Lorcet**, **Lortab**, **Maxidone**, **Zydone**.
  → Hydrocodone + Ibuprofen = **Vicoprofen;** good for mod. to severe pain, good anti-inflammatory.
  → Oxycodone + Acetominophen = **Roxicent**, **Percocet**, **Tylox**.
  → Oxycodone + Aspirin = **Combunox**, **Percodan** **strongest pain med for outpatient basis.
  → Oxycodone (similar potency as morphine) = **Oxycontin**
  → Meperidine + Promethazine = **Meperganfortis**;
  → Codeine + Acetaminophen = **Tylenol #3**; better than Empirin but poor anti-inflammatory.
  → Codeine + Aspirin = **Empirin**; avoid in asthmatics b/c codeine precipitates acute asthma attacks.
- Most common side effect of opoids = nausea; also constipation, resp. depression, drowsiness, sedation, miosis, & euphoria.
- Narcotics work in the brain(CNS) while ibuprofen & NSAIDS work in peripheral tissues (PNS); can be given in combination b/c 2 different mechanism completment each other for effective pain reduction;
- **Hydrocodone** – Synthetic codeine derivative but more efficacious than codeine; poor anti-inflammatory & avoid in asthmatics.
- **Oxycodone** – Synthetic codeine derivative but more efficacious than codeine; avoid in asthmatics; Highest dependency liability;
- **Codeine** – less efficascous opium alkaloid analgesic; also antituissive that is weaker than morphine, less addictive, and less constipating; given ORALLY (3-4 hrs).
- **Meperidine** (Demerol) – synthetic opioid but less potent than morphine & short DOA & doesn't cause miosis & cough suppressant; most abused drug by doctors; IV (for conscious sedation) or oral (3hrs) ; demerol tx for mod. to severe dental pain and may be used as pre-op pain/anxiety reliever.
  → Can cause seizures, tremors, & muscle spasms.
- **Fentanyl** – transmucosal prep/lollipop lozenge (Actiq), patch (Duragesic), IV (Sublimaze); 100x more potent than morphine; IV for conscious/general anesthesia;
- **Pentazocine** (Talwin)– chemically related to morphine but less potent; as strong as codeine; given ORALLY and lasts 4 hrs; blocks painkilling action of other opioids;
- **Propoxyphene** – propoxyphene napsylate + acetaminophen; oral syntehtic opioid analgesic structurally similar to Methadone;
- **Darvocet-N 100 =** acetaminophen + propoxyphene; for pain control after dental surgery.
- **Darvon compound-65** = aspirin + caffeine + propoxyphene.
- **Naloxone/Narcan**, **Nalmefine** & **Naltrexone** (also for alcohol dependency)– all narcotic antagonist for narcotic overdose.
- **Methadone** – also tx Heroin withdrawal.

ANTIDEPRESSANTS & ANTIPSYCHOTICS:

- Tricyclic Antidepressants – tx for unipolar disease (depression); inhibits neuronal re-uptake of NE & serotonin so increase potentiation of neuotransmitter action;
  - Ie – best drug is **Amitriptyline** (Elavil) - greatest *anticholinergic*; **Desiparmine** (Norpramin) has least anti-chol effects; also **Doxepin** & **Imipramine**.
  - Side effects – drowsiness, xerostomia, constipation, blurred vision & tachycardia.
  - Highest incidence of DRY MOUTH w/ 75% of pts, due to secondary anti-chol effect.
- Selective Serotonin Reuptake Inhibitors (SSRI) = tx for depression; very high specificity for blocking re-uptake of serotonin into pre-synaptic cell so increasing time for attachement to post-synaptic cell.
  - Ie – **Fluoxetine** (*Prozac*) – SSRI prototype & longest ½ life.

    - Ie – **Paroxetine**, **Sertraline**, **Fluvoxamine** – all tx for panic attacks, depression, & OCD.
    - Ie – **Citalopram** (Celexa) & **Escitalopram** (Lexapro) – tx for depression & anxiety.
    - Side effects – nausea, headaches, anxiety, agitation & SD.
- LA & Tricyclic antidepressants & SSRI all increase NE in tissues so not good with LA & EPI due to ↑BP.
- Tricyclic Antidepressants & SSRI are NE reuptake inhibitors so cause xerostomia in 75% of pts (secondary anticholinergic effects).
- Lithium – tx for bipolar disorder (cyclical changes b/w manic & depressive phases of behavior); *supresses MANIC phase;* s-times administered w/ anti-depressants b/c cant handle depressive state alone; not used for acute manic episodes.
- Monoamine Oxidase Inhibitors – tx for depression & parkinson's; antagonizes monoamine oxidase which degrades naturally occuring monoamines(like EPI, NE, DOPAMINE, SEROTONIN);
    - Contraindicated w/ LA.
    - Interacts w/ Meperidine (demerol), EPI, EPHEDRINE (food w/ large amts of TYRAMINE).
    - Ie – **Isocarboxazid**(Morplan), **Phenelzine**(Nardil), **Tranylcypromine**(Parnate), **Selegiline**(Eldepryl).
- ANTI-PSYCHOTICS – tx of psychosis w/ schizophenia, paranoia, & manic-depressive illness;
    1. *Phenothiazines* – block dopaminergic sites in brain; most effective antiemetic b/c depress chemoreceptor trigger zone to reduce nausea & vomiting; not for pts w/ CNS depression & epilepsy; may cause liver toxicity, hypotension, dry mouth; NOT anti-convulsant!
        → *TARDIVE DYSKINESIA* – involuntary motion of facial muscles, limbs, & trunck; effects basal ganglia; irreversible effect of phenothiazine; effects 20% of pts on drug>1yr;
        → **Extrapyramidal Syndrome** – muscle spasms of oral-facial region; results from blockade of dopamine receptors in brain; stop drug immediately.
        → **Chloropromazine** & **Thioridazine** are phenothiazine prototypes that cause sedation, antiemetic (prevents nausea), α-adrenergic blocker & potentiation of narcotics.
        → Contra – severe CNS depression/epilepsy; caution in pts w/ liver disease.
        → Adverse effects – hypotension, liver toxicity, xerostomia, tardive dyskinesia.
        → These drugs will potentiate action of sedative drugs so use caution w/ sedation.
    2. *Butyrophenones* – **Haloperidol** (potent dopamine antagonist) & **Droperidol**; tx for schizo & Tourette's.
    3. *Thioxanthenes* – less potent; **Cloroprothixene** & **Thiothixene**; tx for schizo.
    4. *Diverse Heterocyclic Antipsychotics* – antagonize dopamine & seratonin; more effective and less toxic than older Rx; effectively tx Schizophrenia but more expensive!
        → ie- **Molindone**, **Clozapine**, **Loxapine**, **Olanzapine**, **Risperidone**, **Quetiapine**.
- Neuroleptic Agents(anti-psychotic) – tx of ACUTE manic episodes of bipolar disorder.
    - Ie – **Chlorpormazine** (phenothiazine) & **Haloperidol** – effect in extreme psychotic behavior.
- Neuroleptanalgesics – neuroleptic-opoid combinations that combine **Fentanyl** & **Droperidol**; Opoids provide analgesia & anesthesia;
    - **Fentanyl** – highly potent opoid used as premed/adjunct to inhalation agents; used w/ Droperidal & Nitrous to provide balanced anesthesia; Fentanyl come in transmucosal prep, transdermal patch, or as IV prep.
    - **Innovar** = Fentanyl + Droperidol; produces neurolepanalgesia w/ tranquilizing from Droperidole and analgesia from Fentanyl.
- **Propofol** (Diprivan) – IV anesthetic w/ rapid onset/recovery (more rapid than barbs) & better tolerated; respiratory depressant but doesn't produce vomiting/nausea and doesn't increase intracranial pressure; safer for pregnant women but contra for kids!
- **Etimodate** (Amidate) – advantage over other IV drugs is minimal resp/CV depressant effects; rapid induction/recovery; often used w/ opoids; maintains CV stability but high incidence of vomiting.
- **Ketamine** – drug of choice for DISSOCIATIVE ANESTHESIA; causes catatonia amnesia & analgesia w/o loss of consciousness by blocking NMDA receptor & blocking excitatory effects; ONLY anesthetic that acts as CV stimulant; increase crebral blood flow & intracranial pressure; no bronchospasms;

ANTIHISTAMINES:

- Two types of Histamines:
  1. H1 receptors – allergic rxns.
  2. H2 receptors – gastric acid secretions; histamine stimulates parietal cells to produce HCL.
- Antihistamines compete for receptor sites w/ natural histomine (found in all tissues); histamine is stored in preformed **mast cells & basophils**;
  - Histamine is released after response to IgE allergic rxns – role in hay fever, uticaria, angioneurotic edema; also controls acid secretion (HCl) in stomach.
  - **H1 Blockers** – both stimulate & depress CNS: two generations
    1. 1st Gen. – **Diphenyhydramine** (benedryl), **Chloropheniramine**, **Tripelennamine** (PBZ); broad action = antihistamine, anticholinergic, antiserotonergic, antibradykinin & sedative.
    2. 2nd Gen.– **Cetirizine**(Zyrtec), **Fexofenadine**(allegra), **Loratadine**(claritin), **Desloratidine** (Clarinex); they have poor CNS penetration so less drowsiness.
  - All H1 receptor antagonist block vasodilation, bronchicontriction, & capillary permeability.
  - **H2 Blockers** – compete w/ H2 receptors so only compete w/ histamine in GI tract; interferes w/ acid secretion in GI; all reversible COMPETITVE antagonists of H2 receptors w/ DOA = 12-24 hrs.
    → Block stomach acid secretions & treat duodenal ulcers by inhibiting histamine at parietal cells.
    → Ie: **Cimetidine**(Tagamet-may interact w/ hepatic metabolized drugs, may cause gynecomastia), **Ranitidine**(Zantac – for GERD), **Famotidine**(Pepcid), **Nizatidine**(Axid).
    → Tx for acid-peptic diseases, ulcers, Zollinger-Ellison Syndrome (Hypersecretory disease) & GERD (but **Omeprazole** (Prilosec) is more effective which is a "proton-pump" inhibitor).
- HCl – produced by parietal cells of stomach thru pump w/in each cell which pumps protons into stomach; used for food digestion; H+ is pumped into stomach contents to make HCl for digestion.
  → H+/K+ATPase pump which is inhibited by **Omeprazole** (Prilosec), **Lansoprazole** (Prevacid); so they reduce stomach acid formation by inhibiting proton-pump of stomach's parietal cells;
  → Also reduced by inhibiting histamine is stomach at histamine type 2 receptors; ie- **Ranitidine**, **Cimetidine**, & **Fomatidine**.

NSAIDS:

→ Cyclooxygenase (COX) – enzyme produces prostaglandins; Prostaglandins derived from unsaturated fatty acids in cell membranes; 2 forms of COX enzymes:

1. *COX-1*: enzyme produces prostaglandins in GI tract and protects against ulcers;
   a. Ie – NSAIDS inhibit COX 1 & 2 so non-selective COX inhibitors.
2. *COX-2*: enzymes produces prostaglandins at sites of surgery, infection, inflammation; no GI ulcers.
   a. Doesn't affect clotting/platelet aggregation.
   b. **Rofecoxib**(Vioxx), **Celecoxib**(celebrex), **Valdecoxib**(Bextra) – COX-2 inhibitor (not salicylates, not opiates, not NSAIDS); tx of rheumatorid & osteoarthritis & pain from dysmenorrhea.
   c. **Piroxicam** – NSAID for tx of rheumatoid & osteoarthritis;

- Acetaminophen (**TYLENOL**) – weak COX inhibitor but also inhibits prostaglandin synthesis in CNS but reduces pain & doesn't effect coagulation; Analgesic & Anti-Pyretic, NOT ANTI-INFLAMMATORY; categorized w/ NSAIDS but not necessarily one; good for pts w/ GI, bleeding disorders, asthma, young children, and pregnancy; less drug interactions but can cause hepatic necrosis.
  → Drug of choice to relieve mild to mod. pain in pts taking anti-coagulant b/c no platelet problems!
  → Only OTC non-inflammatory analgesic in the US.
- Analgesic efficacy of combining acetaminophen & ibuprofen is greater than either acetaminophen or ibuprofen alone.
- NSAIDS (COX Inhibitor)– inactivate enzyme prostaglandin endoperoxide synthase (cyclooxygenase) so decreases prostaglandin sythesis; ANALGESIC, ANTI-PYRETIC, ANTI-INFLAMMATORY; 3 types:
  1. *Proprionic Acid Derivatives*: **Ibuprofen** (motrin-400mg of Ibuprofen, advil, rufen), **Fenoprofen**, **Ketoprofen**, **Naproxen**, **Naproxen Sodium**; all NON-SELECTIVE COX inhibotors.

→ **Ibuprofen** may interact w/ Warfarin(Coumadin) to cause unnecessary bleeding.
→ **Naproxen** – anti-inflammatory & analgesic and longer acting than ibuprofen but inhibits platelet aggregation; better w/ Type II diabetes pts.

2. *Acetic Acid Derivatives:* **Indomethacin**, **Sulindac**, **Tolmetin**;
3. *Fenamic Acid Derivatives:* **Meclofenamate**, **Mefenamic Acid**.
4. **Ketorolac** (Toradol) – more effective analgesic than aspirin; used for mod to severe pain after dental surgery but suggested for no > 5 days;

→ Side effects of NSAIDS: GI ulcers, ↑bleeding time, impaired renal fct, contra - pregnancy in 3rd trim.
→ NSAIDS reversibly reduce platelet adhesives; works best for mild to moderate pain; "ceiling effect"

- Salicylate/Salicylic Acid(**Aspirin**) – non-selective COX inhibitor; interferes w/ clotting irreversibly reducing platelet adhesives but doesn't affect coagulation pathway;
  - discontinue 5-7 days for normal clotting time to reappear.
  - if given w/ ibuprofen, analgesic efficacy < aspirin/ibuprofen alone.
  - Antipyretic action explayed by cutaneous vasodilation leading to increased heat loss.
  - Salicylism – overdoes of aspirin; not for kids w/ viral infection (REYE'S SYNDROME); headache, confusion, vertigo, tinnitus, nausea, sweating, vomiting; also contra for pregnancy in 3rd trimester.
  - Low doses of aspirin has cardioprotetive effects b/c reduce thromboxane production in platelets causing inhibition of platelet aggregation and can't form thrombi (clots).

CORTICOSTEROIDS:

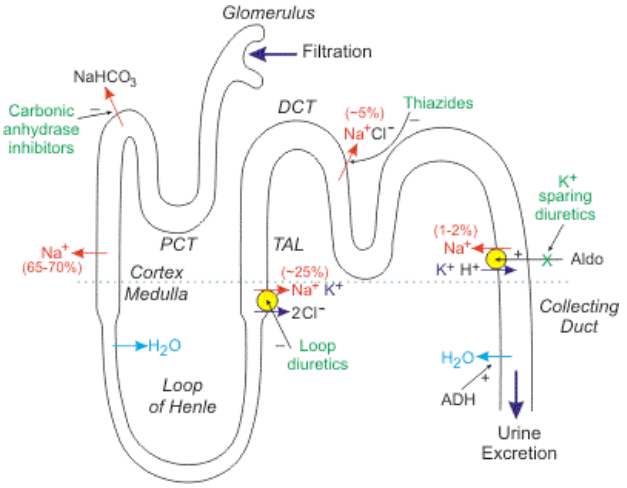
- Corticosteroids: (don't cure diseases) – produced by ADRENAL CORTEX but don't CURE any disease.
  1. Glucocorticoids – affect carbs, lipids,& protein metabolism; used as **anti-inflammatories.**
  2. Mineralcorticoids – regulate Na+ (at collecting duct) & K+ metabolism in the COLLECTING TUBULES; tx for asthma, arthritis, allergies, stomatitis, erythematosis, & TMJ disorders.
- Contraindication – any infections (bacterial, viral, fungal), CHF, or ulcers;
- Adverse rxns – Cushing's syndrome, Hyperglycemica Osteoporosis, ulcers & increase risk of infection; they represent replacement in Addison's Disease (deficiency in steroids).
- **Addison's Disease** – hyposecretion of aldosterone & cortisol; tx w/ 2ml of cortisol; corticosteroids only REPLACEMENT therapy for addison's, not treatment;
- Inhaled Corticosteroids (for asthma) – ↓airway inflammation in asthma enhancing bonchodilating effects of β_2 adrenergic agonists; ↓ blood levels but can cause candidiasis of mouth & pharynx;
  - Ie- Triamcinolone, Beclomethasone, Fluticasone, & Budesonide.
- **Glucocorticoids** – act on arachidonic acid metabolism which induces synthesis of protein that inhibits phospholipase A2, thus ↓**prostaglandin** & leukotriene production; may cause ULCERS!
  - creates anti-inflammatory & immunosuppressive actions.
  - ↑gluconeogenesis, ↓use of glucose,↑protein sythesis,↑protein catabolism, impair wound healing, and ↑chance of infections.
  - Ie – **Prednisone**, **Prednisolone**, **Dexamethasone**, & **Triamcinolone**.
  - Ie – **Beclomethasone**, **Budesonide**, & **Flunisolide** – special glucocorticoids (INHALERS) used to tx chronic asthma & bronchial disease.
  - Fluticasone (Flonase/Flovent) – corticosteroid administered by inhalation to treat asthma by decreasing inflammation in the airway of asthmatics.
  - Inhaled corticosteroids often cause fungal infections (candidiasis).
  - Nasal spray cortico. used for seasonal allergies: **Triamcinolone**, **Fluticasone**, **Budesonide**.
  - Toxic effects – growth inhibition, hyperglycemia, osteoporosis, psychosis, & salt retention.
- Prednisone – corticosteroid w/ anti-inflammatory actions; tx for rheumatoid & osteoarthritis; side effects – insomnia, ingestion, arthalgia, edema, peptic ulscers, osteoporosis, muscle weakness.
- Cortisol – major natural corticosteroid produced by adrenal cortex; mainly glucocorticoid.
- **Mineralcorticoids** - ↑ Na retention,↑Potassium depletion (can cause edema & ↑BP if excessive).
  - Ie – **Aldosterone** (natural), **Deoxycorticosterone**, **Fludrocortisone**;

- Aldsterone – secreted by cells in Zone Glomerulosa of adrenal cortex; regulated by ACTH & renin-angiotensin system (regulates blood volume & pressure);
    - promotes reabsorption of Na into blood from glomerular filtrate;
    - so ↑aldosterone = ↑Na & ↓K in blood; so ↓Na in blood causes ↑BP/blood volume.
    - ↓Na = juxtaglomerular cells secrete renin which converts angeiotensinogen to angiotensin 1 which is converted to angiotensin 2 which stimulates adrenal cortex to release aldosterone.
- ADH (Vasopressin) - ↓urine by ↑reabsorption of water by tubules; ↑ADH causes arterioles to constrict = ↑BP; ↓ADH = ↓water; alcohol inhibits ADH production so extreme loss of water.

CV DRUGS:

- ANTIARRHYTHMIC AGENTS (classified via Vaughan-Williams Classification System)
    1. Group I – Na channel blockers; further classified based on action potential duration.
        1. IA – *Prolong action potential*:
            a. **Procainamide** –anti-A agent; tx of cardiac arrhythmias; derivative of ester LA procaine; ↓myocardial conduction velocity, excitability, & contractibility by inhibiting influx of Na thru myocardial cell membrane so increase recovery period after repolarization.
            b. Similar to **Quinidine** (atrial fibrillation, tx for *supraventricular tachyarrhythmia, PROTOTYPE for anti-A*) & **Disopyramide** – converts atrial arrhythmias to normal sinus rhythm.
        2. IB – *Shorten action potential* – **Lidocaine**(used for emergency ventricular arrythmias & decrease cardiac excitability, IV), **Mexiletine**, & **Tocainide**.
        3. IC – *No action potential* – **Flecainide**, **Moricizine**, & **Propafenone**(tx for ventricular arrhythmias & supraventricular tachycardias).
    2. Group II – Betablockers – for controlling ventricular rate during atrial tachyarrhythmias.
        1. Propranolol & Esmolo are prototypes! Side effects – bradycardia & hypotension.
    3. Group III – Potassium Blockers - **Amiodarone** (Cordarone)– most potent & *broad spectrum* anti-A compound; blocks Na, K, Ca channels & β receptor; tx for suppressing supraventricular & ventricular arrhythmias.
    4. Group IV – Ca Channel Blockers - **Verampamil** –anti-A agent that inhibits intracellular entry of Ca; ***drug of choice for suppression of supraventricular tachycardias stemming from AV node.
        → Ca channel blockers are good antianginal agent, esp. chronic angina;
        → Cause peripheral arterioles to dilate & total peripheral resistance decrease.
        → Also cause increase in oxygen delivery to myocardium; nitrates relieve acute angina.
        → Ie – **Verampamil** (prototype), **Ditiazem**, **Nifedipine**
- Other Anti-arrythemics – **Adenosine** & **Digitalis** (cardiac glycoside).
- Cardiac Glycosides: called digitalis b/c from digitalis plant;
    - helps heart beat strongly, slowly, & efficiently; tx of *supraventricular arrythmias,* shock, & CHF.
    - **Inhibits Na-K-ATPase** membrane pump by inhbiting adenosine triphosphate enzymes (ATPase/Na-K-ATPase); inhibiting Na-K-ATPase leads to increase **CALCIUM** ion influx which causes ionotropic effect of glycosides.
    - **Digoxin** (Lanoxin) – anti-A that directly increases myocardial contraction force; most common & versatile; creates positive ionotropic effect (help heart beat stronger); may cause appetite loss & diarrhea; contra – ventricular fibrillation & ventricular tachycardia.
- Most drugs of cardiac arrhythmias act primarily by ↑refractory period of cardiac muscle.
- ANTICOAGULANTS:
- *Prothrombin Time* – detects plasma coagulation defects (factors V, VII, X); thrombin – prothrombin in presence of Ca, thromboplastin, or other factors.
- International Normalized Ratio – prothrombin time expressed in INR values; fibrin=blood clot.
    - INR = PTT/standard PT time X constant (INR = 1, then normal PT time of 12 sec).
    - INR>1 = anticoagulant effect; incr. INR = inc. in anticoagulant effect.
    - No oral surgery if >5; very effective is <4.

- **Wafarin/Coumadin** & **Dicumarol**– anticoagulant; antagonized vit K to prolong clotting time so decreasing liver synthesis of factors II, VII, IX, X so cant for fibrin;
  → used after MI to prevent coronary occlusion, pulmonary embolism, and venous thrombosis.
- Glycoprotein IIB/IIA Inhibitors – reversible anti-platelet agents used to prevent acute cardiac ischemic complications; the block platelet glycoprotein IIB/IIA receptor (binding site for fibrinogen, von Willebrand factor, and other ligands);
  → **Abciximab** (Reopro), **Eptifibatide** (Integrilin), **Tirofiban** (Aggrastat).
- **Enoxaparin**, **Dalteparin**, **Tinzaparin** – low molecular weight heparin type anticoagulants that prevent deep vein thrombosis; Heparin inhibits rate of clotting proteases by antithrombin III imparing normal hemostasis & inhibiting factor Xa.
- **Heparin** creates potentiation of antithrombin III inactivating thrombin/prothrombin (factor II) & prevents fibrinogen conversion to fibrin; contained in mast cells& basophils;
  → high MW heterpolysaccharide found in the LUNGS;
  → neutralizes tissue thromboplastin and blocks thromboplastin generation so affects coagulation pathway and prevents fibrin formation.
  → Small effect on PTT but strongly inhibit factor Xa.
  → Used for prophy/tx for thromboembolic disorders; administered subcutaneously.
- **Vitamin K** – group of fat soluble vitamins for synthesis of factors II, VII, IX, & X & prothrombin in liver.
- **Clopidogrel** (Plavix) – inhibits blood clotting by inhibiting platelet aggregation; no ulcer side effect like aspirin so antiplatelet drug of choice for pts w/ history of ulcers.
- **Abciximab**, **Eptifibatide**, **Tirofiban** – glycoprotein IIb/IIa inhibitor type of antiplatelet agent; reversible anti-platelet agents to prevent cardiac ischemic complications;
- **Lepirudin**, **Argatroban**, **Danaparoid** – thrombin-inhibitor type anticoagulants; inhibits fibrin formation; tx for post-op deep vein thrombosis.
- Conditions Managed by Anticoagulants:
  1. Coronary Artery Disease
  2. Angina Pectoris – prevent thrombus from forming.
  3. MI
  4. Stroke – prevents thrombus from forming.
- ANTICHOLESTEROLS:
- HMG-CoA Reductase = hydroxymethyglutaryl coenzyme A reductase, which is key step in synthesizing cholesterol; inhibited by "statin" drugs – **Atorvastatin** (lipitor), **Simvastatin** (zocor), **Fluvastatin**(lescol), **Lovastatin** (mevacor), **Pravastatin** (pravachol), **Rosuvastatin** (Restor);
  → When statin drugs inhibit this enzyme, cholesterol isnt produced in liver, so decreases blood cholesterol levels. Tx for coronary artery disease;
  → **do not prescribe statin drugs w/ ERTHROMYCIN drugs, may cause renal failure.
- Coronary Artery Disease – narrowing of blood vessels of heart restricting O_2 flow to heart muscles.
- Mechanism of Action of ANTIHYPERTENSIVES:
  1. Diuretics: 3 types
     a. *Thiazides* – inhibit Na reabsorption in DISTAL OF RENAL TUBULE causing increased excretion of sodium & water;
        i. **Hydrochlorothiazide** (HCTZ) – most widely used diuretic for hypertension but may require K+ supplementation;
        ii. **Dyazide** = Triamterine + HCTZ;
        iii. **Metolazone** – oral quinazoline & sulfonamide diuretic to manage edema & hypertension;
        iv. **Indapamide** - first new class of antihypertensives/diuretics; used in



advanced renal failure;
   v. Thiazides tx = hypertension, edema of CHF, renal edema, Hypercalciuria, Nephrotic diabetes insipidus; adverse effects: *Hypokalemia* (can predispose pt to digitalis to ventricular arrhythmias), *Hyperuricemia*, *Hypercalcemia*.

b. *Loop Diuretics* – inhibit reabsorption of Cl- & Na in ASCENDING LOOP OF HENLE causing ↑ secretion of Na, water, & Cl;
   i. ie – **Furosemide** (Lasix) – prototype, **Bemtanide**, **Torsemide**, **Ethacrynic Acid**.
   ii. MOA – ↑Ca content of urine which cuases ↓renal vascular resistance & ↑renal blood flow.
   iii. Drug of choice with Acute Pulmonary Edema of CHF; adverse effects – ear problems.

c. *Potassium-Sparing Diuretics* – act in COLLECTING TUBULE & conserve K^+; most toxic effect = hyperkalemia; ie:
   i. **Spironolactone** (Aldactone) – competes w/ aldosterone receptor sites causing increased secretion of Na, Cl, & water; tx for aldosteronism & CHF.
   ii. **Triamterine** (Dyrenium) – promotes Na & water excretion but retains K^+; blocks Na channels; **Dyazide** = HCTZ + Triamterine.
   iii. **Amiloride** (Midamore) blocks Na channels in late distal tubule & collecting duct which decreases K+ excretion;

- Osmotic Diuretics – highly filtered by glomerulus; reduce edema from neurosurgery or trauma to the CNS; ie – **Manitol**, **Glycerin**, **Isosorbide**, & **Urea**; given via injection.

2. β - adrenergic blockers – decrease peripheral pressure by increase cardiac output.
   a. Cardioselective β blockers -block β1 receptor; **Atenolol** (tenormin) & **Metoprolol** (lopressor).
   b. Nonselective β blockers – **Nadolol** (Corgard) & **Propanolol** (Inderal).
   c. Both Atenolol & Propanol good for angina too.
3. Angiotensin-converting Enzyme Inhibitors – inhibit conversion of angiotensin 1 to 2 by inhibiting angiotensin converting enzyme, causing vasodilation & increased urinary volume excretion because Angiotensin II stimulates release of Aldosterone which promotes Na & H20 retention;
   a. Ie – **Lisinopril**, **Ramipril**, **Enalapril**, **Captopril**, **Benazepril**, **Ramipril**, **Fosinopril**, **Quinapril**, & **Perindopril;** used to treat hypertension & CHF.
   b. *Angiotensin II* (stimulate release of ADH – sodium & water retention) *receptor blockers* – **Losartan**, **Valsartan**, **Candestartan**, & **Irbesartan**.
   c. ACE inhibitors & Angiotensin II receptor blockers indirectly inhibit fluid volume increases.
   d. **Renin** – proteolytic enzyme of kidney & stored in juxtaglomerular apparatus and converts angiotensinogen to angiotensin 1.
   e. **Angiotensin II** – vasopressor; ↑ peripheral resistance & ADH release causing ↑cardiac ouput.
4. Ca-channel Blockers – **Nifedipine** & **Ditiazem** – both for angina; **may cause gingival hyperplasia.

- Other vasodilators (DIRECT VASODILATORS) – **Minoxidil** (severe ↑BP), **Nitroprusside** (ER BP), **Diazoxide** (ER BP), **Hydralazine** (Apresoline); direct vasodilator action on smooth muscle of arterioles.
- Angina – chest pain from occlusion of coronary arteries; Treatment:
  1. **Nitroglycerin** (Nitrates) – coronary artery vasodilator; administered SUBLINGUALLY w/ onset 2-4min; side effects – hypotension & headache; single most effective anti-anginal agent for acute angina episodes.
  2. Non-nitrate vasodilator = **Dipyridamol** (persantine)
  3. β blocker – **Propanol**, **Nadolol**, **Atenolol**.
  4. Ca channel blockers – **Verapamil**, **Nifedipine**, **Diltiazem**; they are INDIRECT vasodilators.
  5. **Amyl Nitrite** – inhalation agent; oxidizes hemoglobin to methemoglobin which binds cyanide tightly keeping it in circulation & away from tissues; used for emergency tx for cyanide poisoning;
     - vasodilator & highly volatile & extremely potent so rarely prescribed and not drug of choice for angina; side effects – orthostatic hypotension &
     - Most rapid antianginal drug (10sec) w/ DOA = 3-5 min;

- Abused to produce euphoria and as sexual stimulant;

ANTIDIABETICS:

- Insulin: secreted by pancreatic β cells of islets of Langerhans & essential for glucose metabolism; subcutaneous injection;
  a. ↑protein synthesis,↓gluconeogenesis, ↑glycogen synthesis, ↑triglyceride storage.
- Antidibetic/Oral Hypoglycemic agents for Type 2 diabetes;
  1. **Glyburide** & **Chloropropamide** – stimulate insulin release from pancreas & reducing glucose out from liver.
  2. **Metformin** & **Pioglitazone** – increase insulin sensitivity at peripheral taget sites;
  3. **Tolbutaminde** – *sulfonylurea*; stimulates synthesis & release of insulin from pancreas and increases sensitivity of insulin receptors & utilization of insulin.
- Humulin 70/30 – brand name for human form of insulin; an insulin mixture of insulin (30%, fast onset) & isophane insulin suspension component (70%, long duration).
- Insulin Zinc Suspension (lente insulin): DOA = 18-24 hrs & an intermediate acting insulin.
- Insulin preps mimic endogenous insulin for type 1 & 2 diabetes:
  1. Ultra-rapid acting insulin – Onset - .25-.5hrs; DOA = 3-4 hrs; **Insulin Lispro**
  2. Short-acting insulins: onset = .5-3hrs; DOA = 8-12 hrs;
     a. **Regular Insulin** or **Prompt Insulin Suspension.**
  3. Intermediate-acting insulin = onset = 8-12 hrs; DOA= 18-24 hrs;
     a. **Lente insulin** & **Isophane insulin**.
  4. Long-acting insulins = DOA >36 hrs, **Protamine zinc insulin** & **Ultralente insulin**.
- Insulins differ in their onset & DOA.
- *Hypoglycemia* is most serious and most common complication of insulin therapy;

DRUGS:

- Onset of action (Rate of Absorption) for different drug administration:
  1. *Oral* – 30 min (safest & easiest route but unpredictable & least effective; many different dosage forms); oral route most known for its significant hepatic "first pass" metabolism;
     a. generally absorbed best from duodenum; disadvan-1st absorbed in intestines & blood from intestines then filtered in liver (hepatic filter); emotional stress decreases rate of absorption of a drug when given orally.
  2. *IM* – 5 min; not bicepts; for child = ant. thigh & ¼" of needle; adult = butt/deltoid & 1"of needle; never go deeper than 2/3rd of needle length.
  3. *Subcutaneous* – 15 min; injection under the skin so absorption less rapid.
  4. *Inhalation* – 5 min; MOST utilized route of administration w/ NO to sedate peds patients.
  5. *Patch* – 12-24 hrs; systemic effect.
  6. *Intra-arterial Injection* – injected into specific artery; may cause burning.
  7. *Intravenous Injection* – most rapid onset; allows for titration of individual dosages of drug but difficult to reverse; disadv – such rapid onset that overdose is difficult to reverse.
  - Parenteral Administration(not GI) – IV, IA, IM (uniform admin) & Subcutaneous.
  - Enteral Administration (GI) – buccal, sublingual, rectal, or oral
  - Topical – local effect.
  - Transdermal – systemic effect.
- Drug's onset of action primarily determined by rate of absorption.
- Major effect of a drug is determined by how much of the drug is free in plasma.
- Additive Effect – no greater effects!; when 2 drugs given & result is sum of their individual actions when given alone.
- Synergistic – combine 2 drugs & sum of action > sum of individual actions.
- Competitive Antagonism – when response achieved by increase dose of agonist in presence of antagonist; cant respond in presence of noncompetitive antagonism.

- Cumulative Action – excessive accumulation effect that occurs if a drug is administered repeated and higher conc. of drug is desired may be achieved.
- Four types of binding to receptors:
  1. *Ionic Bonds* – electrostatic attraction b/w ions; NOT covalent bonds or nitrogen bonding!
  2. *H^+ Bonds* – b/w polar molecules.
  3. *Van der Walls* – weak interactions occur b/c close proximity.
  4. *Hydrophobic Interactions* – b/w drug, receptor & env't.
- Four Physiological receptors that drugs bind to:
  1. *Receptors as enzymes:* phosphorylating proteins in cell which alters cellular biochemical activities.
  2. *Ion Channels:* bind to ion channels & alter cell permeability.
  3. *G-Protein Coupled Receptors:* when bind to receptor, secondary messengers (cyclic AMP) produce to affect cells;
  4. *Receptors in cell nucleus:* modify transcriptions of specific genes.
- Drugs transfer across cell membranes through…
  1. Passive transfer – simple diffusion (lipid soluble drugs – only NON-IONIZED drugs are soluble in lipids) & filtration (MV<60,000) & osmosis.
  2. Active trasnfer – lipid insoluble drugs (glucose) shuttled across membranes w/ carrier molecules that *provide energy* for transporting drugs to regions of higher concentration.
- Facilitated diffusion – carrier-based transfer; driving force = concentration difference; MOST DRUGS absorbed by facilitated diffusion.
- Osmosis – pure solvent transfers thru semi-permeable membrane from low to high solute concentration; impermeable membrane to solute but permeable to solvent.
- Drugs that cause **Orthostatic Hypotension**: (abnormally low BP when pt assumes standing position)
  1. Antihypertensives – Guanethidine (Ismelin)
  2. Phenothiazine – Chlorpromazine & thioridazine (anti-psychotics)
  3. Tricyclic Antidepressants
  4. Narcotics – Demerol/Morphine
  5. Anti-parkinson's drugs – Levodopa, Carbidopa, Levidopa.
  6. NSAIDS.
- After vasovagal syncope, orthostatic hypotention is 2nd most likely cause of transient unconsciousness in dentist office; **Sny-Drager Syndrome** – chronic orthostatic hypotension.
- *Phantom Pain* – pain w/ no basis but fixed on some anatomy.
- *Intractable pain* – pain resistant/refractory to analgesics.
- *Referred pain* – pain in area other than site of origin.
- *Psychologenic pain* – pain caused by psychic/mental factors.
- *Pain Threshold* – lowest level of pain a pt. can detect.
- Schedule of drugs criteria based on(Controlled Substance Act of 1970):
  1. Potential for abuse, 2. Medical usefullness, 3. Physiological Dependence, 4. Physical Dependence.
- Schedule of Drugs:
  I. Not considered legitamate for medicine; no Rx; ie – **Marijuana**, **Crack cocaine**, **Heroin**.
  II. ↑abuse potential but legitamate for medicine; no refills, cant call in; ie – **Morphine**, **Oxycodone**, **Ritalin**, **Cocaine**, straight **Codiene**.
  III. Less abuse potential; can call in Rx & refills ok; ie – **Codiene**, **Vicodin, Tylenol #3, Hydrocodone**.
  IV. Less abuse potential; ie – **Diazepam (Valium), Lorazepam (Ativan), Alprazolam (Xanax)**.
  V. Small abuse; common Rxs, may have small amount of Codeine. .
- ↑LD50/↓ED50 = ↑therapeutic index = ↑safety. (LD = lethal dose, ED = effective does).
  - Ideal = therapeutic index of 100; ratio measures drug's SAFETY.
- *Bioavailability* of a drug – measurement of rate & amount of therapeutically active drug that reaches systemic circulation = 100% when IV; affected by dissolution (GI tract) & distruction (liver).
- *Habituation* – acquired tolerance from repeated exposure to drug;
- For all drugs but IV & IA, drugs absorbed systemically prior to receptors.

- ***Initial distribution of drug into tissues is determined by rate of blood flow in tissues.
- *Cummulative action* – increase concentration of drug desired when administered repeated.
- *Idiosyncrasy* – response to drug that is unusual/abnormal.
- Factors Affecting **Hepatic drug Metabolism**:
  1. Microsomal enzyme inhibition – drugs inhibit CYP isoforms of P-450.
  2. Microsomal enzyme induction -↑metabolism and↓ drug blood levels.
  3. Plasma protein binding – drugs wont enter liver if highly bound to plasma proteins.
  4. Genetic factors & Pathological factors.
- Urinary Elimination of Drug:
  1. Glomerular filtration – **all** drugs filter thru this b/f enter renal tubules.
  2. Tubular reabsorption – reabsorbed back into blood (highly lipid agents).
  3. Active transport.
- Other excretory pathways for drugs: GI, Lungs, Sweat.
- **Efficacy** of drug = intrinsic ability = ceiling effect = maximal; regardless of dose.
- **Potency** – conc. of 2/more drugs that produce the same drug effect; the effect that usually is chosen is 50% of max. effect & dose causing this is EC50; determined by affinity of receptor for the drug.
- Most important enzyme systems for biotransformation of drugs is in the LIVER!
- *Phase 1 Reactions:* in liver microsomal enzyme systems (mixed fct oxidase system or P-450 system); 3 patterns of drug metabolism.
  1. Active parent drug converted to inactive metabolite.
  2. Active parent drug → 2nd active compound→ inactive compound
  3. Inactive parent drug converted to active compound.
- Most common rx in metabolism is OXIDATION RXN of when hydroxyl group attaches to drug molecule; 5 cytochromes (drug metabolism familes); ie *CYP-1A2* (convert to oxidizing product).
- *Phase 2 Reactions:* parent drug rendered inactive & excreted in urine thru conjugation rxns – coupling drug w/ acid (glucuronic acid) & results in metabolite glucuronide; in liver, kidneys, & other tissues.
    - Conjugation results in polar-water soluble compounds so excreted in urine.

CHEMOTHERAPY:
- 8 classes of chemotherapy:
  1. *Alkylating Agents* – form covalent bonds to nucleic acids so alkylate DNA so it doesn't replicate; good for leukemia, lymphoma, myeloma, & carcinoma; common bonding site = N-7 position of Guanine.
     a. **Cisplatin** - side effects: nausea, alopecia, xerostomic, & mucositis.
     b. Nitrogen Mustards **– Mechlorethamine, Cyclophosphamide, Chlorambucil, & Melphalan**.
     c. Nitrosureas – **Carmustine**, **Lomustine**, **Semustine**;
     d. **Bisulfan** – tx for chronic granulocytic leukemia.
  2. *Anthracyclines* – destroys DNA; **Daunarubicin** & **Doxorubicin**; Mucositis is common!
  3. *Antibiotics* – **Dactinomycin**
  4. *Antimetabolites* – interferes w/ biochemical rxn, so interferes w/ S phase of reproduction cycle; oldest & most important chemo.; **Methotrexate**, **5-Fluorouracil** (5-FU), 6-**Mercaptopurine**.
     a. Folid Acid Analogs – **Methotrexate** (may cause oral ulcers);
     b. Pyrimidine Analogs– **5-FU**, **Floxuridine**, **Cystosine Arabinsoide**, **6-Merpatopurine.**
     c. Purine Analogs **– Mercaptopurine, Thioguanine;**
  5. *Antimicrotubular* – inhibits cell mitosis; **Paclitaxel** (taxol).
  6. *Antiestrogen* – blocks estrogenic tumors, like breast cancer; **Tamoxifine** (nolvadex)
  7. *Vinca Alkaloids* – mitotic spindle poisons; **Vinblastin** & **Vincristin**.
  8. *Gonadotropin Hormone-Releasing Antigen* – inhibit GDTH; **Leuprolide**.
- **Asparinigase** – deprives tumors of amino acids for protein synthesis; **Interferons** – boost immune system; both don't fall in chemo category but are used to tx cancers!
- **Interferons** – inhibit cell growth, induce gene transcription & alter state of cell differentiation; types:

    - Interferon α2a – hair cell leukemia.
    - Interferon α2b – chronic hepatitis B
    - Interferon αn3 – recurring genital warts
    - Interferon β1a – tx for MS.
- Mucositis – common rxn to chemotherapy involving inflammation of mucous membranes; use 5-fluorouracil, Methotrexate, & Doxorubicin.
- Alopecia is most common chemo side effect ; occurs 1-2 weeks after tx; also increase in infections like candida and degeneration of lymphatic tissue;
- Most chemo drugs are teratogenic and need to be avoided in pregnant women.
- Colony Stimulating Factors:
    1. **Darbepoetin Alpha** – induces erythropoiesis; tx for anemia from renal failure.
    2. **Pegfilgrastim** – stimulates neutrophils and decreases infections.
    3. **Sargramostin** – myeloid reconstitution after bone marrow transplants.
- Aromatose Inhibitors –
    1. **Exemestane** – prevents conversion of androgens to estrogen by tying up enzyme aromatous; tx for breast cancer.
    2. **Letrozole** – first line of treatment for hormone receptor positive or metastatic breast cancer in post menopausal women.
- 5-Hydroxytryptamin type 3 Receptor ($5\text{-}HT_3$) – seratonin receptor activated during chemo causing emesis(vomiting); antagonist for this receptor: **Granisetron** & **Ondansetron** (prophy for chemo).
- Immunosuppressants:
    1. **Pimecrolimus** (Elidel) – tx for mild to moderate dermitis.
    2. **Sirolimus** (Rapamune) – prophy for organ rejection patients.
    3. **Tacrolimus** – tx for moderate to severe dermatitis.
- **Adalimumab** (Humira) – monoclonal antibody binds to human tumor necrosis factor alpha receptors; tx for rheumatoid arthritis.
- **Alefacept** (Amevive) – monoclonal antibody, tx of moderate to severe psoriasis.
- **Infliximab** (Remicade) – monoclonal antibody binds to TNF alpha; tx for ankylosing spondylitis, Crohn's disease, & Rheumatoid arthritis.
- **Trastuzumab** – monoclonal antibody binds to human epidermal growth factor receptor 2 protein (HER-2); tx for metastatic breast cancer;
- Modafinil (Provigil) – CNS stimulant to improve wakefullness in pts w/ excessive daytime sleepines & ADHD; decreases GABA mediated neurotransmission.

MISCELLANEOUS:

- Rx – p.c. = after meals; h.s. = at bedtimes, a.c. = before meals.
    - *Superscription* = pt's info; *Inscription* = drug & drug strength; *Subscription* = directions to pharmacist; *Transcription* = directions to pt.
- **Glaucoma** – increase in intraocular pressure; poor drainage of aqueous humor(fluid in eye) and can cause blindness; tx:
    1. **Pilocarpine** (Isopto-carpine) – cholinergic agonist; eye drops causing papillary constriction.
    2. **Latanoprost** (Xalatan) – prostaglandin analog; eye drops reduce intraocular pressure.
    3. **Betaxolol** (Betoptic) – β-blocker; eye drops ↓pressure by ↓production of aqueous humor.
    4. **Bimatoprost** (Lumigan) – same as latanoprost.
- Drugs that produce REVERSIBLE *Xerostomia*:
    a. **Amitriptyline** (elavil) – tricyclic antidepressant; highest incidence of xerostomia!
    b. **Diphenhydramine** (benadryl) – sedating type anti-histamine
    c. **Atropine** – powerful anticholinergic, blocks saliva production.
    d. **Diazepam** (Valium) – benzodiazepine tranquilizer.

- Rheumatoid Arthritis(RA) – chronic inflammation of synovium that lines joints causing pain, swelling, & destruction; treatment:
  1. **Prednisone** – decreases inflammatory response.
  2. **Gold injection** – decreases prostaglandin production.
  3. **Methotrexate** – affect immune function.
  4. **Nabumetone** (relafen) – NSAID that inhibits prostaglandin sythensis.
  5. **Piroxicam** (feldene) – NSAID that inhibits prostaglandin synthesis; may cause gastric irritation, heart burn, & nausea.
- All of these also work for *OSTEOARTHRITIS* (except gold injections) - the progessive loss of articular cartilage due to excessive loads; drugs for OA provide analgesic & anti-inflammatory action.
- Anti-Rheumatic Agents:
  1. **Etanercept** (enbrel) – decreases S&S of rheumatoid arthritis; recombinant DNA-derived protein which binds to TNF – which plays important role in RA causing increased inflammation in RA.
  2. **Infliximab** – treatment for Crohn's Disease(inflammation of GI tract) & RA; monoclonal antibody that binds TNF so decreases inflammation.
- Parkinson's Disease – deficiency of neurotransmitter dopamine in brain due to nerve cells in basal ganglia degeneration; slow progressing & degenerative disorder; distinguishing features: tremors at rest, sluggish initiation of mvmts, & muscle rigidity; Treatment -
  - **Levodopa** – precursor for dopamine.
  - **Carbidopa** w/ Levodopa (Sinemet) reduces required dose of levodopa by 75% w/o side effects; Carbidopa inhibits peripheral decarboxylation of levadopa; Carbidopa doesn't cross the BBB, so levodopa converts into dopamine in the brain.
  - **Bromocriptine/Pergolide** – dopamine agonists & often given to enhance Levodopa's action.
  - **Selegine** –inhibitor of MAO Type B: enzyme causing oxidative deamination of dopamine in brain.
  - **Amantadine** – anti-viral agent that potentiates dopaminergic responses
  - Anticholinergic drugs also tx parkinson's – like **Benztropine** & **Trihexyphenidyl**.
- Drugs that causes OSTEONECROSIS of the Jaw: temp/perm. loss of blood to bone & bone dies; non-healing of extraction socket or exposed jaw bone are symptoms.
  1. **Zolendrionic Acid** (Zometa), 2. **Palmidronate** (Aredia), 3. **Alendronate** (Fosamax)
- Gastric Antacids – directly neutralized gastric acid (HCl) from stomach; decrease conc. & total load of gastric acid; *DYSPEPSIA* – impairment of the power/function of digestion; antacids:
  1. **Sodium Bicarbonate** (only systemic antacid) – Alka-Seltzer.
  2. **Calcium Carbonate** – Amitone, Tums.
  3. **Aluminum Hydroxide** (most potent but less neutralizing) – Alterna gel & Amphojel.
  4. **Magnesium Hydroxide** – milk of magnesia
  5. **Bismuth Salts** – Pepto-Bismol.
  6. **Magnesium** & **Aluminum** – Maalox & Mylanta.
- Growth Hormone **– Somatotropin –** secreted from anterior pituitary gland; ↑protein synthesis rate, ↓carbohydrate utilization rate, & ↑mobilization of fats for energy; subcutaneous/IM for 3x/week.
  → Human Growth Hormone- prepared commercially as purified polypeptide hormone of recombinant DNA origin; used as replacement therapy for pts with HGH deficiency.
- Gout – elevated levels of uric acid in blood stream; Treatment:
  1. **Colchicin** – impairs leukocytic migration to inflammation areas & disrupts urate deposition; not IM or subcutaneous b/c causes tissue irritation; kidney & liver damage & bone marrow depression are side effects; NSAIDS are also used like **Indomethacin** for acute gouty arthritis.
  2. **Allopurinol** – ↓uric acid production; inhibits xanthine oxidase which is an enzyme that coverts hypoxanthine to xanthine and xanthine to uric acid; drug of choice for CHRONIC GOUT.
  3. **Probenecid**(benemid) & **Sulfinpyrazone**(anturane) – enhance uric acid clearance; both in kidnesy & inhibit reabsorption of uric acid; slows secretion of PCNS & cephalosporins.

- Caffeinism – 600-750 mg of caffeine/day (more than 10 cups/day) w/ >1000mg in the toxic range; caffeine stimulates CNS unequally w/ cortex most and spinal cord least.
- **Mercury** – prescense in body determined by urine test; average ½ life = 55 days; mercury accumulates in brain, liver & kidney.
  - Can cause irritability, excessive saliva, loose teeth, gum disorders, slurred speech, & tremors; these symptoms are chronic; higher than avg. accumulations occur in brain, liver, & kidney..
  - tx – gastric lavage and fluid therapy and British Anti-Lewisite (BAL)/ Dimercaprol – complex w/ mercury & allow to be excreted as inactive compound.
- Analeptic – not safe/recommended; CNS stimulant that overcome drug-induced resp. depression & hypnosis; ie – **Pentylenetetrazol**, **Nikethamide**, **Doxapram**, **Picrotoxin**, & **Strychnine**.
- Xanthines – for mental allertness, decrease sleep, and increase mood; ie – **Caffeine** (only OTC), **Theophylline** (for asthma), & **Theobromine**;
  - Theophylline & Theobromine weaker CNS stimulants than caffeine.
- Loperamide (**Imodium**): 1) Anti-Diarrheal which inhibits perstalsis.
  2) Opoid family but doesn't penetrate CNS so OTC.
  3) No drug abuse/dependence.
- Diphenoxylate (**Lomatil**): antidiarrheal & inhibits GI tract motility & propulsion; Diphenoxylate & Atropine together require prescription;
- Laxatives act in reverse manner of anti-diarrheals b/c increase GI motility to treat constipation; Ie- Magnesium Hydroxide, Caster Oil, Metamucil, & Methylcellulose.
- Oral Contraceptives block ovulation by inhibiting anterior pituitary hormones FSH & LH; both estrogenic & progestational agents; increase risk of thromboembolism and heart disease in smokers.
  → Contains both estrogenic agent & progestational agent.
  → Highest risk associated w/ BCP is thromboembolism.
- Drugs travel thru bloodstream by binding to albumin protein, which is abundant in plasma and enables drug to be carried to all tissues and organs.
- Virtually any drug can cross placenta of pregnant women & enter fetal circulation so check w/ DR.
- Habituation – acquired tolerance form repeated exposure to particular stimulus but w/o the addictive, physiological need to increase dosage.
- Tolerance – decreased responsiveness to a drug after chronic administration; dosage required to produce usual effect is increased.
- Toxicity is both dose-depenedent & time-dependent;
- Dyesthesia – uncomfortable/painful sensation; in dentistry, manifests as post-op sequela to regional administration to LA.

PROSTHODONTICS

FIXED:

- **Gold Crown Preparation** = .5 – 1.0 mm;
- **PFM:** metal = .5mm, porc. = 1-1.5mm, total = 1.5-2mm; labial shoulder = 1.5mm; supporting cusp reduction = 2.0mm & opposing walls no more than 10^{o}.
- Absolute minimum required thickness of porcelain = .7mm & metal coping thickness = .3-.5mm for high noble & .2mm for base metal;
- Proper thickness need to prevent distortion during firing of porcelain;
- PFM Alloys -
  1. **High Noble** – 98% Au/Pl/Pt; doesn't oxidize during casting; BEST!
  2. **Noble** – 50-60% Pl & 30-40% Silver; Palladium-silver alloy; not nobel metal so oxidizes on casting.
  3. **Base-Metal** – 70-80% Ni & 15% Chromium; Nickel-Chromium alloy; oxidizes & causes PFM interface problems; less resistant to corrosion; stronger & lower density than noble metal;
     a. Alloys w/ less than 25% noble elements;
     b. Another example is Chromium Cobalt used for RPDs;
     c. ↑resistance, modulous elasticity, melting temperature; compared to type 4 gold.
     d. ↓density, specific gravity, & yield strength; all compared to type 4 gold.
     e. The low density makes casting more difficult;
- ADA Classifies Alloys as follows:
  1) Type I – used for small inlays
  2) Type II – larger inlays & onlays
  3) Type III – onlays, crowns, & short-span FPDs
  4) Type IV – thin veneer crowns, long-span FPDs & RPDs
- Porcelain adheres to metal primarily by chemical bond (COVALENT BOND); since true chemical bond, failure/fracture will occur in porcelain rather than porcelain-metal interface;
- Repeated fracture of PFM is due to INADEQUATE FRAMEWORK DESIGN;
- **All-Ceramic Crowns** – have *low flexural strength* and tendency to fracture at minimum deformation;
- PFM & All cermaic crown require the SAME amount of overall tooth reduction = 1.5-2.0mm;
- PFM prep must have all surfaces smooth & rounded in order to prevent fractures;
- Outer junction of porcelain to metal should be at right angle = 90^{o};
- **Butt Joint** – poorest type of finish line; optimum margin is ACUTE EDGE; main disadvantage is any inaccuracies in the crown fit are reproduced at the margin, causing an increased thickness of cement;
- **Best finish margin but least marginal strength** = bevel/feather edge; may causes inacurrate extension & distortion of wax pattern; optimum margin for casting b/c easily BURNISHED;
- Chamfer is preferred finish line on cast gold restorations; a well prepared chamfer combines the advantage of easily definable margin on the impression & die, with minimal tooth preparation;
  → Reduces thickness of cement;
- **Shoulder Margin w/ a Bevel** – this margin allows a sliding fit to occur at the margin, thus maybe used on proximal box of inlays or occlusal shoulder of mand. ¾ crowns;
- Margins for different materials:
  --All Ceramic = Shoulder
  --PFM w/ porcelain to margin edge = Shoulder
  --PFM w/ metal collar = Shoulder bevel or Chamfer
  --Full Gold Crown = Bevel or Chamfer;
- If margins extends into biologic width, constant gingival irritant occurs and crown fails; so crown lengthening needs to be performed before FIRST crown preparation;
- Advantages of Partial Veneer Restorations (¾ or 7/8 crowns):
  → Great deal of margin is accessible to dentist & patient
  → Less of restoration margin is in close proximity to gingival crevice (less perio irritation);
  → More easily seated during cemetation
  → Portion is accessible if pulp vitality ever needs to be tested;
- Reverse ¾ crown – common on mandibular molars to perserve LINGUAL area.

- Standard ¾ crown – preserves buccal area; MOST COMMON type of partial veneer crown;
- 7/8th crown (all metal) is a ¾th crown (all metal) whose vertical distal buccal margin is position slightly mesial to middle of buccal surface; *advantages*:
  → esthetics, DB finish line easy to access, provides more coverage, excellent abutment for bridge;
- The path of insertion of anterior ¾ crown should parallel the incisal ½ - 2/3 of labial tooth not tooth's long axis; if parallel to long axis, will cause more gold to be displayed;
- A pin modified ¾ crown can preserve the facial surface & 1 proimal surface; preferred in cases which require repairing of sever lingual abrasion on incisors & canines;
- Gold Crown Occlusion – check w/ silver plastic shim shock;
- ALUM – **aluminum potassium sulfate;** for cords for patients w/ ↑ BP; ZnCl delays healing so don't use; ↑ BP w/ epi cords when applied to severly lacerated gingival sulcus but minimual changes when place in an intact gingival sulcus;
- Mechanical Properties of RESINS influenced by -
  1) MW of polymer
  2) Degree of cross-linking
  3) Composition of monomers used to prepare polymer
  4) Acrylic resins EXPAND when immersed in water & become DISTORTED when dried out;
- **Methyl Methacrylate**(MMA) = liquid monomer; hydroquinone inhibitor, cross-linking agents, & chemical activator (*dimethyl-p-toluidine*) which is only present in self-cured resins; EXOTHERMIC;
  → Other monomers – **ethyl methacrylate, vinyl ethyl methacrylate, & epimine resins;** all less irritating to the pulp; MMA is most frequently used!
  → Excessive shrinkage may occur if too much monomer is added to the polymer;
- **Polymethyl Methacrylate**(PMMA) = powder polymer; benzyol peroxide is initiator; Cross-linking agents contribute greatly to STRENGTH of polymer;
- Heat Cured Resin – stronger & superior color stability because they contain less residual monomer & higher MW than self-cured resins;
  → *heat* (accelerator) decomposes benzoyl peroxide (initiator) into free radicals which initiate polymerization of MMA to PMMA;
- Self Cured Resin – *dimethy – p – toluidine* (activator – tertiary amine) added to MMA causing decomposition of benzoyl peroxide into free radicals which initate polymerization of MMA to PMMA;
  → Generally used for repairs;
- **Polymerization Range** = temp of 60°C – 77°C (140°F – 170°F);
- *Porcelain Veneer Contraindications*:
  1) severe imbrication (overlapping) of teeth
  2) traumatic occlusal contacts
  3) unfavorable morphology
  4) insufficient tooth structure & enamel
  5) high caries index
  6) short clinical crown
  7) minimal horizontal overlap;
- Some techniques to remember w/ veneers – should be tried in WET; fit surface is treated w/ silane & protected w/ light cured unfilled resin; enamel surface cleaned w/ pumis & water;
- Most common causes of crown failures **– lack of attention to tooth shape, position, & contacts**;
- Greatest potential for wear exists b/w porcelain & tooth b/c porcelain causes accelerated wear of opposing dentition – 40x more wear than gold; so gold preferred for bruxism pts;
- The best measure of the potential clinical performance of a casting alloy is its ADA CERTIFICATION;

PONTICS & FPDs:

- Portion of pontic approximating ridge should be as convex as possible!
- 6 Types:

1. *Sanitary* – nonesthetic zone (convex everywhere); **most commonly used where esthetics is not important**;
2. *Saddle* – don't use due to hygiene, looks most like a tooth;
3. *Modified Ridge Lap* – illusion of tooth but all convex; **BEST for esthetics**;
4. *Conical* – rounded; for mandibular thin ridges;
5. *Ovate* – sanitary version of saddle; sits in concavity of ridge.

- Facial lingual dimension of pontic determined by opposing FL contacts;
- Pontics shouldn't be in contact during non-working movement; may be in CO contact & may/may not be in working-side contact;
- Pontics must have passive pinpoint contact w/ gingiva; excessive tissue contact is one of the major causes of failure of fixed bridges;
- Pontics must not be concave in 2 directions; they should be convex MD & concave FL;
- Pontic design is more important than pontic material;
- Multiple adjacent pontics on anterior FPD have reduced FACIAL EMBRASURES to enhance esthetics;
- **Solder joints** – connectors of CHOICE when abutment teeth are in normal alignment & good bone support; strength of solder connector is ↑ with ↑ height w/ circular form preferred;
  → solder must have much lower fusion temp. the metal it is joining;
  → CLEANLINESS is most important prerequisite of soldering since the soldering process depends on WETTING the surfaces to achieve bonding;
  → **Flux** (often BORAX) displaces gases & removes corrosion products by combining w/ them or reducing them;
- Failed bridge is more detrimental to dental health than failed RPD but fixed restorations are ALWAYS the tx of choice, unless contraindicated; Success/Failure of RPD depends mostly on PONTIC DESIGN;
- **Factors that Determine a FPD Design:**
  1. *Root Configuration* – roots that are broader labiolingually than mesiodistally are preferred to rooths that have round cross-section;
  2. *Crown to Root Ratio* – ideal ratio is 1:2 but 2:3 is more realistic and 1:1 is minimum!
  3. *Axial Alignment of teeth* – parallelism of abutment preps is best determined by long axis of preps;
  4. *Length of Lever Arm (span)* – replacing 3 teeth is MAXIMUM, more than 2 is high risk;
     a. Edentulous space involving 4 adjacent teeth other than 4 inciros is best treated w/ RPD;
- The MOST LIKELY indication for splinting is tooth mobility w/ pt discomfort;
- DO NOT splint natural teeth & implants in a FPD b/c implants lack PDL;
- Nonrigid Connector – mechanical union of retainer & pontic rather than solder joint (T-shaped key & dovetail); restricted to SHORT-SPAN bridge that is replacing 1 tooth;
  - Used when retainers CANNOT be prepared to draw together w/o excessive tooth reduction;
  - Path of insertion of key into keyway should be parallel to pathway of retainer;
- When stress breaker on distal of pontic, occlusion unseats key from key;

PORCELAIN:

- Porcelain shade in order – value (brightness), chroma (saturation), hue (color).
- **Value** – brightness; MOST CRITICAL characteristic that is matched FIRST; relative amount of lightness or darkness in a color; intensitiy of a color; Impossible to increase value; staining reduces value;
- **Chroma** – saturation; **single most important factor in shade matching**; CAN be ↑ using stains;
- **Hue** – basic color; drastic changes of hue are often impossible but ORANGE STAIN is most often used to change hue;
- Some Facts for *Shade Selection*: 1) quick rubber cup/prophy to make shade selection more accurate;
  2) do not gaze for more than 5 seconds
  3) proceed by process of elimination
  4) half-closed eyes can increase sensitivity of rentinal rods ot better select the color's VALUE;

- Porcelain - rusts at temp > 2000°F; has good biocompatibility; should be under slight compressive stress; Porcelain substrate alloys melt at high temperatures;
- In all ceramic crown, high strength sintered ceramic is core material;
- *Opaque Porcelain* – 1st layer; masks metal color, creates CHEMICAL bonds w/ metal;
  - → it will show thru facial surface of crown if inadequate tooth reduction, too thick metal, too thick porcelain, or inadequate thickness of body porcelain;
- *Body Porcelain* – bulk of restoration; most of color & shade;
- *Incisal Porcelain* – translucent layer;
- Porcelain bulked out to compensate for it 20% shrinkage;
- Porcelain stains are Metallic Oxides;
- Smooth porcelain gives impression of larger size & changes in contour are used to alter the apparent long axis inclination of a tooth;
- Metamerism – different color match under 2 different light sources; staining porcelain decreases value and increases metameric responses; ;
- Flourescence – material reflect UV radiation; teeth fluoresce mainly blue-white hues (400-450nm); makes a definite contribution to the brightness & vital appearance of natural tooth;
  - → **Blue fatigue accelerates yellow sensitivity**: means if you look at blue color object while selecting the shade, it helps accentuate the ability to discriminate b/w yellow shades;
- Color of a pigment is determined by selective absorption & selective radiation/scattering;
- Natural Glace (glaze firing) – when porcelain itself is glazed by separate firing; more permanent than overglazes;
- Glazed porcelain least irritating to gingiva compared to other restorations and resists abrasion;
- Overglazes (applied glazes) – ceramic powders that maybe added to a porcelain restoration after it has been fired; **erosion may occur in a month creating rough & porous surface**;
- Classes of Porcelain: --**High-fusing** → denture teeth
  --**Medium-fusing** → all ceramic crowns
  --**Low-fusing** → metal ceramic crowns; contains *aluminum oxide* (↑ its resistance to "slumping down" during firing) + *calcium oxide* + *oxides of potassium, sodium, & chromium* (help reduce cross-linkage b/w oxygen & silicone to lower porcelain's fusing temperature;
- Porcelain = feldspar (main) + quartz (strengthener) + metal oxides (impart shade of porcelain); amorphous structure (not crystalline);
  - → **Kaolin** (clay) → binds particles of porcelain together; more in house porcelain;
  - → Compressive strength of porcelain GREATER than tensile strength;
  - → Porcelain is BRITTLE & not capable of plastic deformation;
  - → Constituents of Porcelain:
    1) Silicone Dioxide (64-69%)
    2) Aluminum Oxide (8-19%)
    3) Potassium Oxide (8%)
    4) Sodium Oxide (2-5%)
- *Aluminous porcelain* uses alumina, not quartz as strengthener; it is considerably stronger than conventional porcelains;
- **Degassing** – process of heating (980°C) a casting to burn off impurities prior to porcelain adding; necessary for all gold-porcelain systems; degassing metal at too low temp will effect formation of oxide layer and it will decrease the bond;
- **Pickling** – reduces surface oxides; 50% HCl; frequently the surface of gold casting is dark due to formation of surface oxide film;
- Causes of Porcelain Fracture:
  1) Poor metal framework (main cause);
  2) Degas too low temperature
  3) Contaminate metal prior to opaque application
  4) Fusing opaque at too low a temp or too short a time;

- **Sintering** – changes powder porcelain to solid; ↑ dentisity; shape maintained.
- Metal & Ceramic must have closely matched COEFFICIENTS OF THERMAL EXPANSION to avoid porcelain fractures;
- Alloys should have high proportional limit & high modulus of elasticity to reduce stress on porcelain;
- 3 Stages in *Firing Porcelain*: 1) Low bisque firing, 2) Medium bisque firing, 3) High bisque firing;
- Porcelain must have: 1) Low Fusing Temperature (if fired too much, it devitrifies/milky);
  2) High Viscosity
  3) Resistance to devitrivation (crystallization);
- Most common cause of POROSITY in porcelain is **inadequate condensation of porcelain;**

REMOVABLE PARTIAL DENTURES:

- Total occlusal load applied to RPD is enhanced by: 1) occlusal surface area
  2) occlusal efficiency
  3) number of existing teeth
- Kennedy Classification – based on MOST POSTERIOR edentulous area to be restored; periodontal damage to abutment teeth is avoided w/ firm tissue support; 4 Classes:
  1. *Class I* - bilateral distal extension;
  2. *Class II* – unilateral distal extension;
  3. *Class III* – unilateral edentulous space bound by teeth; it is a tooth-borne RPD b/c it **depends entirely on abutment teeth for support**;
  4. *Classs IV* – anterior teeth are missing and across the midline; it is a tooth-borne RPD b/c it **depends entirely on abutment teeth for support**; NO MODIFICATIONS!

  → Classifications are done after NOT BEFORE extractions are done;
  → If 3rd molar is present & not to be used as abutment, it's not considered in the classification;
  → If 2nd molar is missing & will NOT be relplaced, it's NOT considered in the classification;
- Craddock Classification – based on denture type; 3 types: 1) Type I – mucosa borne
  2) Type II – tooth borne
  3) Type III – mucosa & tooth borne
- Major & Minor connectors MUST BE RIGID for functional stresses applied to RPD to be evenly distributed throughout the mouth;
- Major Connector – the unit of RPD that connects the parts of the prosthesis located on one side of the arch to parts on the opposite side of the arch;
  → should be free of movable tissues & shouldn't impinge gingival tissues;
  → most frequently encounter interferences from LINGUALLY INCLINED MAND. PREMOLARS;
- Maxillary Palatal major connectors **may be beaded** to produce a positive contact w/ the tissue;
- **Single Palatal Bar –** lacks rigidity so for bilateral short span edentulous areas; connected to 1st molars;
- **Palatal Horseshoe-shaped plate** – used when large, inoperable torus prevent using other designs;
- **Anteroposterior palatal bar** for RPD – MOST RIGID palatal major connector; used in almost any maxillary partial denture;
  → both ant. & post. connectors cross the midline at RIGHT ANGLES rather than diagonal;
- **Palatal Plate connector** – think broad connector that can be used for simple edentulous areas and full palatal coverage;
- **Lingual Bar** needs 7mm of height = 3mm below gingival margin + 4mm of vertical height;
- **Lingual Plate** should cover middle 1/3 of lingual surface of teeth; Indications:
  1. High lingual frenum or when there is NO SPACE in the floor of the mouth
  2. If vestibule is <5mm;
  3. Mandibular tori can't be removed
  4. To support/stabilize periodontically weakned teeth;
     - Severe anterior crowding is CONTRAINDICATED for using lingual plate;

- **Labial Bar –** should be 3mm below gingival margin; used with lingaully inclined mand. anterior teeth or w/ large lingual tori;
- Stress Breaker – device that relieves the abutment teeth to which an FPD/RPD is attached, of all /part of the forces generated by occlusal function; *2 types*:
    1. **Wrough-Wire Retentive Clasp** – simplest form of stress relief; Wrought metal is stronger w/ greater flexibility than cast metal; 25% greater strength & hardness;
        a. Yield strength can be drastically reduced if exposed to too much heat causing recrystallization or gain growth;
        b. Terminal end of retentive arm is placed in middle of gingival 1/3 of crown;
        c. 20-gauge wrought wire is 2x more flexible than an 18-gauge wire;
        d. 20-gauge cast clasp into .010 undercut is alternative to wrought wire;
    2. **Split-bar Major connector ("hidden lock")** – flexible connection b/w direct retainer & denture base; stress-breakers with a moveable join;
- Shorter clasps need finer gauge of wire (higher # = finer) because need optimum flexibility;
- Round Cross-section of clasps = ↑ Flexibility of Clasps
  = ↑ length & taper
  = ↓ cube ratio/thickness & width;
- Indirect Retainers – RESTS, MINOR CONNECTORS, & PROXIMAL PLATES; function to counteract/prevent VERTICAL/UPWARD DISLODGEMENT of the distal extension base;
  → anti-rotational device; also prevents DOWNWARD movement so protects soft tissue;
  → Serves at 3rd reference for seating framework & making altered cast impressions;
  → Indirect retainer for distal extension are placed as far away from edentulous space while rests are placed on abutment teeth next to edentulous areas for max support for tooth borne partials (class 3 & 4);
  → The greater distance b/w fulcrum line & IR, the more effective the IR;
- No indirect retainer for kennedy class 3 – no fulcrum line;
- As denture base moves upward, the most anterior rest (direct retainer) resists downard movement;
- Direct retainers must be effect for an indirect retainer to function;
- Direct retainers – Intracoronal attachment & Clasps;
- Intracoronal Retainers - MOST ESTHETIC direct retainer for RPD; built into contour of a crown to produce mechanical & frictional retention; not used when RPD depends on edentulous area for support (class 1 or 2);
- Clasps – extracoronal retainers; most common direct retainer for RPD; *2 types*:
    1. **Suprabulge clasps** originate above the height of contour or survey line, usually from occlusal rest;
        a. *Circumferential Clasp* – composed of retentive arm & bracing arm; engages undercut on side OPPOSITE of site of rest.
        b. *Ring Clasp* – engages undercut located on same side of rest;
        c. *Embrasure Clasp* – when no edentulous space exists
        d. *Reverse-action Clasp* – hairpin clasp; enagages undercut located on same side as rest or on any posterior tooth;
        e. *Extended Arm Clasp* – circumferential clasp that extends to neighboring teeth;
        f. ½ & ½ *Clasp* – consists of 1 circumfertial clasp emanating from rest and another amr from minor connector on opposite side;
    2. **Infrabulge retainers** – I, J, U, L, T Bar clasps; approaches crown undercut from BELOW the tooth's height of contour; the provide retions by resistance of metal to deformation;
        a. Must NOT be placed into tissue undercuts nor contact abutment of any places except specified undercut;
        b. Advantages – more efficient retention, less distortion, less caries, & greater adjustability;

- Each clasp must be designed to encircle more than 180° (more than ½ the circumference of tooth);
- **Elongation** – most important mechanical property of clasps of RPD;
- Faiure of partial dentures due to poor clasp design is best avoided by altering tooth contours; premolars & molars most often need to be altered;
    - → GUIDING PLANES serve to ensure predictable clasp retention;
- Primary purpose of **rests** – VERTICAL SUPPORT for RPD & resist VERTICAL FORCES of occlusion;
- Occlusal Rest - **Positive Rest** – form acute angles w/ minor connectors that connect them to the major connectors; Rest = 2.5mm & < 90° angle to minor connector; reduce marginal ridge by 1.5mm;
    - → the rest occupies the middle 1/3 of the occlusal surface;
- Cingulum Rest – vertical stop on Anterior tooth; confined to maxillary canines, but sometimes maxillary centrals; less torquing stress than incisal rest (not esthetic);
- Reciprocating arm = lingual arm; Retentive arm = buccal arm;
- Fuction of reciprocal clasp arm:
    1) Reciprocation
    2) Stabilization
    3) Bracing (auxillary indirect retainer)
- In RPD, stability insured by occlusion;
- Design characteristics for RPD – 1) Support, 2) Retention, 3) Bracing, 4) Guidance;
- For RPD, minimal functional stress on abutement teeth; **most of stress on residual ridge causing resorption**;
- Precision Attachements – Male & female preconstructed parts; little tolerance;
    - → adv – provide retention w/out a lot of metal displayed; excellent bilateral stabilization;
    - → disadv – difficult to repair; never to be used with distal extension RPD w/o stress breaker;
    - → primary indication are when teeth are present on both ends of the edentulous area;
    - → cast crowns must be provided on all abutments;
- **Semi-precision** has more tolerance & less retention; it is a cast into the crown & RPD; male portion is cast into the RPD;
- Surverying: 1) Path of Insertion, 2) Position of Survey Lines, 3) Locate Undercut & Nonundercut areas.
- Dental Surveyer – an instrument used to determine the relative parallelism of oral anatomy; areas used for support CANNOT be determined by surveying;
- When selecting teeth for RPD, the most important factor is **available interarch space**;
    - → MD width – from distal of lower canine to beginning of slope of ridge;
    - → BL width – narrower than natural teeth b/c decreases stress transferred to denture support area during food bolus penetration; also increases tongue space;
- **Chromium Cobalt** is inflexible but best for RPDs; adv – corrosion resistance, high strength, & low specific gravity; low density and high modulus of elasticity (stiffness); low cost;
    - → Chromium – for corrosion & tarnish resistance due to SURFACE OXIDE LAYER;
    - → Cobalt - ↑rigidity, strength, & hardness;
    - → Nickel - ↑ ductility; measured as percentage of elongation; metallic component of RPD w/ the greatest potential for allergic reactions in the mouth;
- When recording CR for RPD, the occlusal rim is attached to the completed partial denture metal framework, instead of record base for complete denture;
- Most important factor in determining the succes of distal extension RPD is proper COVERAGE over residual ridge;
- If the indirect retainers are not seated as extension base are depressed, the bases need **relining**;
- If pt complains of **sensitivity to percussion** on an abutment tooth of distal extension, most likely causes is the occlusion on this abutment;
- Defective occlusal contacts can also cause a feeling of **looseness** to the denture;
- Altered Cast Technique – purpose is to recored the form of the edentulous segment w/o tissue displacement & to accrately relate the edentulous segment of the teeth via metal framework;
    - → Helps obtain soft tissue suppor to aide abutments in resisting functional stress;

→ It is a secondary impression system that uses metal framework to hold customized impression trays for the edentulous areas;
→ Impression records of edentulous ridge tissues in the exact form that they will assume the finished RPD is in place on the teeth;

- Considerations when preparing an RPD abutment to receive a crown:
  1. Path of Draw
  2. Location of rests
  3. Orientation of guiding planes
  4. Placement of porcelain metal finish lines
- When RPD preferred over FPD: 1) loss of 4 maxillary incisors
  2) distal extension
  3) long span edentulous area
  4) periodontally involved abutment teeth
  5) after recent extractions
  6) economics

COMPLETE DENTURES:

- If denture falls out when smiling, buccal notch & flange overextended; when yawning, distobuccal flange overextended;
- Sore gums & aching muscles = reduce VDO; generalized soreness after 1st appointment of denture insertion is most likely due to improper occlusion;
  → To identify prematurities, the best method in mouth is to use warm disclosing wax by inserting the wax bilaterally & have pt close into CR;
- Tingling/numbing in corner of mouth/lip, excessive pressure from lower buccal flange near mental foramen;
- Mandibular Denture - --Distal Buccal Extension = Masseter Muscle
  --Distal Lingual Extension = Superior Constrictor Muscle
  --Lingual Border = 1) Palatoglossus Muscle
  2) Superior Pharyngeal Constrictor Muscle
  3) Mylohyoid Muscle
  4) Genioglossus Muscle
- Healing of ridge post-extraction = 4-6 months (reline at 5 & 10 months);
- Reline CONTRAINDICATED for decreasing VDO; if decreased VDO, then new dentures are indicated;
- After relining a denture, if a pt constantly returns for adjustments due to sore spots on ridge, check occlusion b/c relining may have changed CR contacts, loss of CR contacts;
- Recording CR is an essential starting point in design of denture; for complete dentures, MIC of teeth in CO is established to coincide w/ pt's CR, so CO=CR;
- Flabby Max. anterior ridge when max. complete opposes 6 mand. anterior teeth;
- Setting denture teeth edge to edge = cheek biting; tx = reduce facial of mandibular molars & create proper horizontal overlap; cheek biting also caused by ↓ VDO;
- Primary reason to use plastic teeth in denture is b/c plastic teeth are retained well in acrylic resin; plastic teeth are retained better than porcelain teeth; porcelain teeth also cause denture clicking;
- Biting corner of the mouth – reset canines & PMs.
- When Pt has Complete Max. Denture but **lacking posterior support**, the following occurs:
  1. Excessive amts of hyperplastic tissue on anterior portion of maxilla;
  2. Poor bone structure in anterior maxilla
  3. Fibrous tuberosities
  4. Pt complains of looseness of denture and they can no longer see their upper teeth;
- Central Incisors should be 8mm anterior to center of incisive papilla; if placed to far superior & anterior, effects "F" & "V" sounds; Primary role of anterior teeth on denture is ESTHETICs;
- Max & Mand. anterior teeth should NOT contact in CR;

- Most common error that contributes to poor esthetics is placing Max. anterior teeth directly over edentulous ridge; Maxillary teeth should be placed FACIAL to the ridge;
- Max. centrals are most important teeth for esthetics. duh
- If burning sensation of complete max. denture then pressure on INCISIVE FORAMEN;
- Position of Lips for Complete Dentures corrected by: 1) Correct VDO
  2) Thickness of anterior border
  3) Teeth position
- "S" Sound – tip of tongue w/ anterior palate & lingual of Max. ant. teeth; sound that brings the mandibule CLOSEST to the maxilla;
- "Th" Sound – tongue protrude b/w max & mand anterior teeth (2-4mm).
- "F" & "V" Sound – incisal edge of maxillary teeth & lower lip;
- "P" & "B" Sound – formed TOTALLY by lips;
- Palate too think & incisors are too far palatal if saying "S" but sounds like "Th";
- If teeth set too far lingually, the T will sound like a D; if set too far labially, the D will sound like a T;
- High palatal vault or constricted palate can cause WHISTLING sound; whistling during speech with dentures can cause: 1) insufficient vertical overlap
  2) excessive horizontal overlap
  3) area palatal to incisors are improperly contoured;
- A pt having difficulty swallowing may have insufficient interocclusal space caused by excessive VDO;
- Learning to chew food satisfactory with new dentures requires at least **6-8 weeks** to establish new memory patters;
- Most effective time to test phonetics → wax try-in.
- Longer time pts is edentulous then greater difficulty w/ phonetics than short time pts;
- Most important factor for retention of completes is PERIPHERAL SEAL;
- **Mucobuccal Fold** is most important factor for Maxillary complete RETENTION;
- Maxillary Complete & Mand. bilatateral Distal extension may show:
  1. Decreased VDO
  2. Prognathic Facial Appearance (associated w/ edentulous state).
- Maxillary Denture – Primary Support = Residual Ridge
  Secondary Support = Palatal Rugae
- Mandibular Denture - Primary Support = Buccal Shelf & Residual Ridge
  Secondary Support = Anterior Lingual Border
- **Coronoid Process** interferes w/ denture opening when Max. buccal space filled w/ denture flange; so coronoid process can limit the thickness of denture flange;
- Camper's Line – parallel to maxillary occlusal rim; line running from **inferior border of ala nose to superior border of tragus of ear;**
- To determine maxilla occlusal rim vertical length = 2mm below upper lip.
- Acrylic Resin for denture repairs → pressure = 20-30 psi; MOST COMMON cause of porosities in denture is due to insufficient pressure on flask during processing;
  - → Porosities also occur if packing & processing of power & liquid resin is too plastic (stringy/sandy);
- **Palatal Seal** – Posterior Outline → formed by "ah" line or vibrating line (foveal palatini) connecting pterygomaxillary notches; hamular notch is on posterior border;
  Anterior Outline → formed by "blow" line & located at distal extent of hard palate;
  - → Width = 6 mm on left & right & 3 mm at the center;
  - → Depth = 1.5mm on left & right & .5mm at the center;
  - → Outline & depth of seal is different for every pt, determined by palatal form on each pt;
  - → Palatal seal should NEVER be removed; placement of seal ALWAYS done by dentist, not lab!
  - → Excessive depth of seal usually results in unseatin gof denture;
  - → Functions: 1) Completes border seal of max. denture

2) Prevents food impaction
3) Improves denture's physiologic retention
4) The seal compensates for polymerization & cooling shrinkage of denture resin during processing;

- **Vibrating Line** – 2mm in front of fovea palatini; extends from 1 hamular notch to the other;
- **Hamulus** – superior attachement *of pterygomandibular raphe* (tendon) which is b/w buccinator & superior constrictor muscles; extension of MEDIAL PTERYGOID PLATE of sphenoid bone;
- Increased VDO causes clicking of teeth, effects phonetics, & esthetics; need to remount or new CD/CR;
- Compensating Curve – anteroposterior curvature & mediolateral curvature in the alignment of occluding surfaces & incisal edges of artificial teeth used to developed balanced occlusion;
  → Entirely in DENTIST's control
  → Allows dentist to alter the effective cusp angulation w/o changing form of manufactured denture teeth;
- **Average Interocclusal Space** at REST = 3mm;
- VDO + Interocclusal Space = VDR; VDR > VDO (always!); ↓VDO = ↑ interocclusal distance;
- Correct VDO is evaluated using 4 methods:
  1. Evaluating the overall appearance of facial support;
  2. Visual observation of space b/w occlusal rims at rest
  3. Measurement of dots on face (placed on tip of nose & chin)
  4. Observation when "s" sound is enunciated accurately;
- Excessive VDO = ↓ freeway space; Decreased VDO = ↑ freeway space;
- For complete dentures, path of condyle determined by: 1) Shape of fossa
  2) Meniscus
  3) Muscular Influence
- Submucosal Vestibuloplasty – usually performed on maxillary arch to improve available denture base; procedure is favored b/c no raw tissue surface remains to granulate & re-epithelialize;
- Underlying BASAL BONE (under the retromolar pad) resists resorption; marked resorption of ridge occurs if mandibular complete denture base terminates short of retromolar pad;
- For the 1st few days after pt receives new dentures, they will have some difficulty eating & EXCESSIVE SALIVA due to reflex PARASYMPATHETIC stimulation of salivary glands;
- Balanced Occlusion is objective of complete dentures;

OVERDENTURE & IMMEDIATE DENTURES:

- Overdenture – denture whose base is constructed to cover all of an existing residual ridge & selected roots; most important is preventing ridge resorption;
  → retained roots help PREVENT RESORPTION of alveolar ridge, improve denture rentention & allow pt some sense of "naturalness" in function of the dentures;
  → not always necessary to cover root beneath overdenture but if a root is not covered, the exposed surfaces are highly susceptible to decay;
- Immediate Dentures: ideal to fabricate max & mand. dentures at same time; Complete in 2 steps
  1. Extract all posterior teeth EXCEPT max. 1st PM & its opposing tooth so leaves posterior stop to maintain VDO;
  2. After healing of posterior area, denture fabrication can begin; Anterior teeth extracted at time of denture insertion;
  → For the 1st 24 hours, do not remove dentures, eat soft foods, & return in 24 hrs to dentist;
  → Advantage = duplicate position of natural teeth; they are esthetically advantageous in that the pt is never w/o either natural or artificial teeth;
  → Major disadvantage is Anterior teeth try-in for esthetics;
  → Prevents tongue enlargement b/c when natural teeth are lost & not replaced, the tongue expands into the available space;

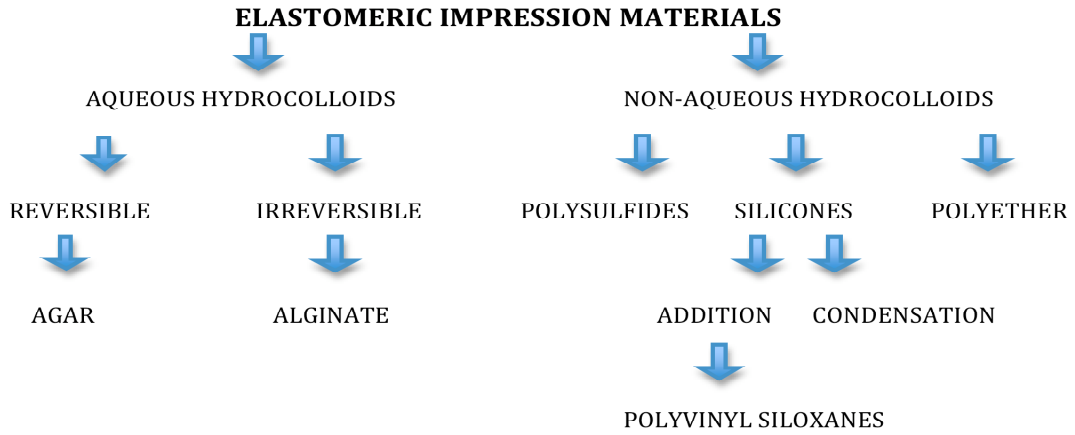
→ Relining/Rebasing the denture is REQUIRED in 8-12 months! Schedule relines at 5 months & 10 months post-extraction;

DENTURE DESIGN CHARACTERISTICS:

- **Stability** – the relationship of the denture base to bone that resist dislodgement of the denture in HORIZONTAL diretion; involves resistance to horizontal, lateral & torsional forces (most important);
  → All components of RPD, except retentive clasp tip, contribute to stability;
- **Support** – resistance to VERTICAL SEATING forces; provided by rests & denture bases; MOST IMPORTANT design characteristic for oral health; for RPD, support given by rests & edentulous areas;
- **Retention** - quality in restoration that resists the force of gravity, sticky foods, & forces associated w/ mandibular movement; direct & indirect retainers provide retention;
  → clasps placed in undercut areas of abutment teeth provide retention;
- **Reciprocation** – the means by which one part of the metal framework opposes the action of the retainer in function; reciprocating element must be placed OPPOSITE the direct retainer;
  → Must contact the abutment as the retentive tip passes OVER the tooth's height of contour;
  → refers to function of reciprocal clasp arm to counteract forces exerted by retentive clasp arm;
- **Bracing** - horizontal force transmission by placing rigid portions of clasps or other parts of the RPD in non-undercut areas of abutment teeth;
- **Guidance** – during insertion & removal obtained by contact of rigid parts of the framework with areas on axial tooth surfaces parallel to the path of insertion;

IMPRESSION MATERIALS:

- Rinse & Disinfect prior to pour of impressions or sending to lab; spray/soak for 10 minutes;
- Bite Registration Material → **Addition-reaction silicone** impression material; very low flow and minimum resistance to the patient's jaw closure;
  → Technique – Have pt bite teeth tightly in CO & inject material b/w max. & mand teeth ONLY into areas where teeth have been prepared;
- Ideal Material for Recording CR (not wax!) - 1) Rapid setting plaster
  2) ZOE Pastes
  3) Modeling Plaster
- Best impressurion technique for pt w/ loose hyperplastic tissue is to register tissue in PASSIVE position;
- The primary indicator of accuracy of border molding is the stability & lack of displacement of the tray in the mouth; modeling compund has LOW thermal conductivity;
- Border Molding: *2 stages*: 1st stage, the molding should approximate the borders & be slightly OVEREXTENDED; excess trimmed & 2nd stage is refining remaining molding by repeating process;
  → Most critical area on MAX denture = MUCOGINGIVAL FOLD above max. tuberosity area;
  → For MAND. denture, **distofacial** extension determined by MASSETER MUSCLE & **distolingual** extension limited by SUPERIOR CONSTRICTOR MUSCLE;
  → Dislodgement indicates overextension; very common area of overextension is the distobuccal corner of mand. denture pushing against Masseter muscle;
- Ease & Accuracy of Border Molding: 1) Accurate fit of custom tray
  2) Control of bulk & temp of modeling compound
  3) Dried Tray



- Polymerization – **changing elastomeric materials from pastes to rubber-like materials**;
  - → *Addition Polymerization* (no ionic forms) – adding of units on each side of C-C double bond; forms polymer w/o forming any other chemical;
  - → *Condensation Polymerization* – involves ionic species & produces small molecule by-products of each step of rxn; when other chemical or by-produces are produced that arent the polymer;
- Reversible Hydrocolloids, like Agar, are 85% water and can change physical state by adding or removing heat; expesive equipment & difficult to disinfect;
  - → dimensionally unstable (single & immediate pour); LONGEST SHELF-LIFE;
- Agar – needs special equipement; good for crowns; physical state can be changed from GEL SOL by applying heat & reversed back by removing heat;
  - → only elastomeric that doesn't involve a chemical reaction to set;
- Alginate - ↑ temp = ↓ gelation time; too much/little water weakens gel; Reactor = **Calcium Sulfate**; very limited dimensional stability; want 3mm b/w tray & tissue;
  - → **Sodium Alginate** – tendency to give up water (**synerisis -** shrinks impression) or gain water (**imbibition** – expands impression); CONTROLS SETTING TIME of alginate b/c it's the retarder;
  - → ↓ Water/Powder Ratio = ↑ setting of gel; once all the $NaPO_4$ has reacted, the Na Alginate reacts w/ remaining calcium ions & forms **calcium alginate**;
  - → Fast removal of impression from mouth ↑ compressive & tensile strength of impression;
  - → It is a **double decomposition reaction** b/w sodium alginate + calcium phosphate;
  - → Best method to control gelation time of alginate is to alter water temperature;
  - → If impression is grainy, may be caused by *improper mixing, prolonged mixing, or to low water:powder ratio*;
  - → *ALGINATE CONSTITUENTS*:
    1. Diatomaceous (silica) = 50% (FILLER)
    2. Potassium Alginate = 20% (forms SOL)
    3. Calcium Sulfate = 16% (REACTOR)
    4. Zinc Oxide = 7% (PLASTICIZER)
    5. Potassium Fluoride = 6% (improves GYPSUM)
    6. Sodium Phosphate = 1% (RETARDER, controls setting time)
- Polyethers – **hydrophilic** so unstable if moisture but tolerates moisture better than any other elastomer; rubber formed by cationic polymerization – cation but no free radicals;
  - → **SHORTEST WORKING & SETTING TIMES** (5-6min); contracts slightly during setting;
  - → Custom trays needed since elastomers are more accurate in uniform thin layers that are 2-4mm thick;
  - → excellent dimensional stability; can be poured up to 1 wk; *2 Components*:
    1. *Base* – polyether (polymer), silica filler & plasticizer
    2. *Accelerator* – crosslinking agent called **aromatic sulfonic acid ester** which produces cross-linking by *cationic polymerization;*
- **Hysteresis** – when material has melting temperature difference from its gelling temperature;

- Polysulfide – WATER is by-product; exothermic & accelerated by temperature; strongest resistance to tearing & high flexibility but causes distortion; **Longest Setting time = 12-14 min.**
  - → requires custom tray for impression to control polymerization shrinkage; 2 components:
    1. White BASE – contains low weight polysulfide polymer;
    2. Brown ACCELERATOR – contains **LEAD DIOXIDE** & sulfur; lead dioxide accelerator is responsible for brown color that is difficult to clean off clothes!
- Silicones – ETHYL ALCOHOL is by-product (causes shrinkage); for complete dentures/crowns; don't mix initially by hand; less expensive, easy cleanup; 1 year shelf life;
  - → low tear strength & poor moisture tolerance; must be poured immediately;
  - → **poor dimensional stability** because principal rx occurs during setting time is a condensation reaction via elimination/evaporation of ethyl/methyl alchol; *2 components*:
    1. Base – liquid silicone polymer (dimethylsiloxane)
    2. Reactor – cross-linking agent **ethyl ortho-silicate** (metal organic ester) w/ activator = **tin octoate**;
- Polyvinyl Siloxanes – NO BY-PRODUCT; Silicone (silane H+ groups) & Vinyl Silicone (vinyl groups, catalyst); ↑ temp = ↓ setting time; can be poured up to 1 week;
  - → Excellent dimensional stability & very low permanent deformation;
  - → Poor tear strength, lowest temp rises, stiff, poor wettibility by gypsum;
  - → MOST WIDELY USED & MOST ACCURATE;
- ZOE Impression Paste – sets as hard, brittle mass; ↑water = ↑setting time; **↓setting by adding oil**;
  - → **Chelate** – forms in typical acid-base reactions;
  - → Setting time accelerated by ADDING a drop of WATER to the mix; **MESSY** & not recommened for gagging pt; dimensional stability affected if custom tray is NOT used;
  - → Difference b/w ZOE paste & modeling compound, ZOE must be done in 1 insertion while modeling compound is done in 2;
  - → can record soft tissue at rest, sets in 5 min, stable, & less expensive than polysulfides;
  - → Needs no undercuts of ridges; paste need to be uniform in color; 5 Components:
    1. Calcium Chloride – accelerator
    2. Oil of Cloves (70-85% eugenol) – reduces burning
    3. Vegetable oil – plasticizer
    4. Resinous Balsam – increases flow.
    5. Rosin - ↑ speed of reaction & makes smoother product;
- SULFER in latex gloves retards PVS setting times;
- Elastomers are more accurate in uniform → 2-4mm thick w/ thin layers;
- Longest to Shortest Working time = Agar > Polysulfide > Silicones > Alginate = Polyether
- Best to Worst Dimensional Stability = Add'n Silicones > Polyether > Polysulfide > Condition Silicones

DENTAL CASTING & GYPSUM:

- *3 types of Investment Materials*:
  1. **Gypsum-Bonded** – binder is gypson (calcium sulfate HEMIhydrate); for convetional gold alloys, Type 1, 2, & 3 gold alloys;
     i. Strength of investment for gold is dependent on amt of GYPSUM;
  2. **Phosphate-Bonded** – binder is metallic oxide & phosphate; for base metal alloys for PFMs & Type 4 gold; chosen for silver-palladium, gold-platinum, & nickel-chromium alloys;
     i. Any allow w/ casting temp >2100^{o}F/1150^{o}C, should cast with binder OTHER than gypsum;
  3. **Silica Bonded –** binder is silica gel; for base metals for RPD framework;
- the expansion of investment provides larger mold to compensate for subsequent contraction of alloy.
- 4 Mechanism Compensate for Solidification Shrinkage of Alloy during Casting: (they play a role in producing expanding mold): 1) Setting Expansion of the investment
  2) Hygroscoping expansion of investment (presence of water)

3) Thermal expansion of investment
4) Wax pattern expansion

- **Quartz** or **Cristobalite –** refractory materials used for these investments to provide thermal expansion for the investment;
- **Potassium fluoride** added to flux to dissolve passivating film (supplied by chromium) that may prevent wetting of the metal with the solder; Potassium fluoride is most common agent in flux;
- ↑ strength of solder joint (circular) is increasing height of it;
- **Antiflux** – restricts flow of solder; soft graphite pencil.
- Casting alloys – Type 1 to 4 from weakest to strongest;
- Gypsum Products – different HEMIHYDRATE particles in each product so different amount of water; main constituent = **Calcium Sulfate Hemihydrate** →all products form this reaction product; *4 Types*:
    1. **Type 1** – Impression Plaster; **β - hemihydrate**;
    2. **Type 2 -** Plaster (model); **β - hemihydrate**; for ortho – 2x of water than stone; higher setting expansion than stone;
        - **a.** Heating gypsum in open vessel at 150-160°C = PLASTER;
    3. **Type 3** – Dental Stone; **α - hemihydrate**; for dentures;
        - **a.** Heating gypsum under pressure at 120-150°C = STONE;
    4. **Type 4** – Dental Stone (die stone); **α - hemihydrate**; for die-work; increased strength & expansion;
        - **a.** Boiling gypsum in 30% CaCl & MgCl = DIE STONE;

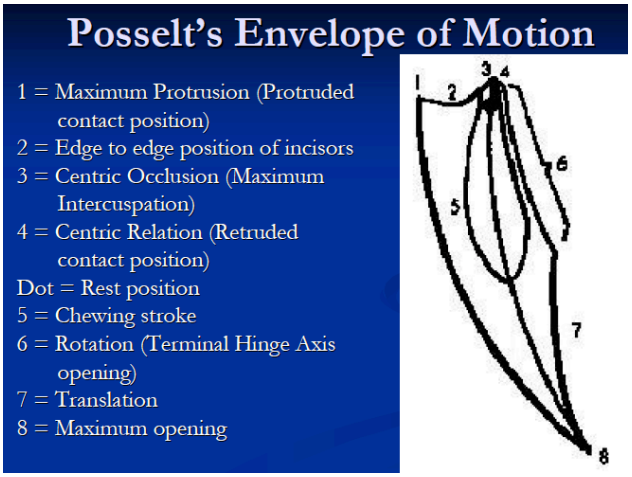
    → β - hemihydrate requires more water b/c crystals are sponginess & irregular shaped & more porous than α - hemihydrate (more dense crystals);
    → main differences b/w dental plaster & stone powders is PARTICLE SIZE & SHAPE & POROSITY;
    → more water used → less expansion & ↓ setting time & ↓ strength;
    → when water removed, it forms Calcium Sulfate HEMIHYDRATE, but when water is added, it forms Calcium Sulfate DIHYDRATE; **Starting gypsum is dihydrate**;
    → gypsum + water = heat (exothermic);
    → All gypsum products are weaker in tensile strength than compressive strength;
    → Gypsum *Accelerators* – potassium sulfate, sodium chloride, & aluminum;
    → Gypsum *Retarders* – borax, sodium citrate;
- Gypsum sets faster when → 1) ↑ spatulation
  2) lower water:powder ratio
  3) use mix of water & ground up gypsum particle
- To prevent air entrapment is to place the proper amount of water in the mixing bowl first then sift the model plaster/stone into the bowl;
- Maxillary sinus appears to ENLARGE throougout life if it is not restricted w/ natural teeth/dentures; as the sinus enlarges, the tuberosities move downward;
- If low tuberosity is not removed, accidentally underextended mand. denture will be made causing limited space for teeth;
- When the casting is COLD-worked to provide required article/appliance, it is called wrought metal in contrast to cast metal;
- **Brittle** – material w/ high compressive strength but low tensile strength;
- **Specific Gravity –** property of gold alloys that exceeds a base-metal alloy in numerical value;
- **Sprue** – small diameter >1.5mm (10-12 gauge) PIN made of wax/plastic; sprue should be equal/greater than thickest portion of the wax/plastic pattern;
    → sprue attached to wax pattern at 45° angle;
    → Spruing at a thin area can produce the same result as usuing a sprue that is too smal causing ***shrink back porosity*****, causing turbulence in the flow of the molten metal;**

CEMENTS:

- The type of cement used does NOT affect or increase crown retention;
- Tooth must be WIPED DRY, not air dried or dried w/ alcohol, before cementation;
- Always apply cement to both restoration & tooth;
- Composite Resin – luting material of choice to cement a ceramic crown & can provide STRONGEST BOND;
- Zinc-Phosphate Cement – also can be used to cement ceramic crowns; good compressive strength but high pH so need 2 layers of varnish to protect the pulp;
- Zinc Polycarboxylate or ZOE – biologically compatible cements; used when preps have adequate length & retentive features or when prep is deep and pulp vitality is a concern;
  - Zinc Polycarboxylate & GI cements adhere to calcified dental tissue and have SUPERIOR biologic compatibility than zinc phosphate cements;

ANATOMY/OCCLUSION:

- CR = bone to bone relation (no tooth contact) – most unstrained retruded anatomic & functional position; cannot be fored into CR from rest position, mand must be relaxed and then guided into CR;
  - → condyle in most SUPEROANTERIOR POSITION w/ the articular disc interposed b/w condyle & eminence;
- *Rest Position* → **Muscle Guided** (Freeway space); tonic stretch reflex; average = **2-6mm**;
- *CR* → **Ligament Guided** (retruded position); bone to bone; REPEATABLE reference point;
- *CO* → **Tooth guided** (intercuspal position); determined by cusps of teeth; during "empty mouth swallowing", the mandible is braced in intercuspal position; tooth contacts longer in swallowing than chewing;



- 1-7 = Anterior Border Movement – MAX. OPENING;
- 4-8 = Posterior Border Movement;

- Jaw relationship most commonly used in ACTUAL design of restorations is the AQUIRED centric occlusion;
- **Non-working Side Interferences** (Balanced Side) – facial cusps of mandibular molars;
- **Working Side Interferences** – Lingual cusps (inner aspect) of Maxillary molars;
- Protrusive Interference – b/w distal inclines of facial cusp of maxillary teeth & mesial inclines of facial cusps of mandibular teeth;
- Protrusive record made to register condylar path; when restoring entire mouth w/ crowns/ protrusive condylar path inclination influences *mesial inclines of mandibular cusps*;
- Centric Interference (forward slide) – correct by grinding mesial inclines of maxillary teeth & distal inclines of mandibular teeth;
- Mandibular Movements - Protrusive (anteriorly) = 9-10mm; Laterally = 10mm
  Inferiorly (opening) = 50-60mm; Posteriorly = 1mm
- Frankfort Horizontal Plane – outer canthus of eye to tragus of ear;
- Class II occlusion not good for canine guidance or group function;

- Mandibular Condylar Movement → --Retrusive Mvmt = move back & up
  --Protrusive Mvmt = move down & forward
  --Lateral & Working Mvmt = down, forward, & laterally
  --Lateral & Non-working Mvmt = down, forward & medially.
- **Masseter Muscle** – contracts during swallowing;
- **Functional Cusps:** UL & LB; Also called Supporting, Working, Stamp, or Centric Cusps; Contact centric stops; they are broader & more rounded cusp ridges; used to CRUSH food;
- **BULL RULE** – for Non-supporting, Balanced, Non-working, & Guiding Cusps;
  → inner occlusal incline leading to these cusps are *Guiding Inclines* – b/c in contact mvmts, they guide supporting cusps away from midline;
  → narrower & sharper cusp ridges to SHEAR food;
- In **posterior crossbite**, supporting cusps & guiding cusps are opposite; so BULL RULE for working cusps;
- **Non-Working** (balancing) Interferences occur on INNER inclines of FACIAL cusps of Mand. molars;
- **Working side**(non-balancing) Interferences occur on inner aspects of LINGUAL cusps of Max. molars;
- During lateral excursions, the opposing cusps contact on WORKING side;
- During later excurions, on the balancing/non-working side, the maxillary lingual cusps contact the mandibular facial cusps;
- Selective Grinding - 1) Never Grind Max. Lingual Cusps (Primary centric holding cusps);
  2) Grind Mand. Buccal Cusps if needed (Secondary centric holding cusps);
  → Only grind cusps if premature contacts;
  → Purpose of selective grinding is to remove all interferences w/o destroying cusp height; so instead of grinding cusps, fossa or marginal ridges opposing premature cusp is deepened;
- **Centric Interferences** (forward slide) is corrected by grinding MESIAL inclines of maxillary teeth & DISTAL inclines of mandibular teeth;
- Bennett Movement → aka Lateral Shift/Immediate Side Shift; working side of condyle only; this mvment influences MD position of posterior teeth cusps;
- Bennett Angle → sagittal plane & path of Non-working condyle during lateral movement;
- Eccentric Occlusion – a protrusive & right & left lateral contacts of the teeth's inclined planes when the mandible is not moving;
- Bilateral Eccentric Occlusion – not an objective in RPD construction, unless opposing a complete denture; is an objective in complete dentures;
- Bilateral Balanced Occlusion – dictates a MAXIMUM number of teeth that should contactduring mandibular lateral excursive movements;
- Mutually Protected Occlusion **(Canine Guided/Organic Occlusion)** – most widely accepted arrangement of occlusion; when anterior teeth protect posterior teeth in all mand. excursions;
  → Canines provide predominant guidance thru full range of mvmt in lateral mand. excursions;
  → When placing crown on max. canine, if you cange canine guided occlusion to group function, you increase the chance of non-working side interferences to occur;
- Anterior Guidance - result of horizontal & vertical overlap of anterior teeth; produces disclusion of posterior teeth when mand. protrudes & moves in lateral excursion;
  → the greater the overlap, the longer cusp height;
- Incisal Guidance – measure of the amount of mvmt & angle at which the lower incisors & mand. must move from overlapping position of centric occlusion to an edge to edge relationship w/ max. incisors;
  → Second end-controlling factor in articulator mvmt & is to some degree, under the dentist control; other end-controlling factor is RIGHT & LEFT CONDYLAR mechanisms;
  → Mechanical equivalent of horizontal & vertical overlap;
- 4 Determinants for restoring complete & functional occlusion:
  1. Vertical Overlap of Anterior teeth
  2. Contour of Articular Eminence

3. Lateral Shift of Working Condyle
4. Position of Tooth in Arch

- Determinants of Occlusion – 1) TMJ, 2) Occlusal Surface of teeth, & 3) Neuromuscular System;
- Group Function Occlusion **(Unilateral Balanced Occlusion)** – characterized by NO non-working side contacts in a natural dentition;
  → when ALL posterior teeth on side contact evenly as jaw moves toward WORKING side;
- End-Controlling factors of Articular Movement: 1) R & L Condylar Mechanisms
  2) Incisal Guidance
- **Condylar guidance** is totally dictated by patient, not by dentist at all; *inclination of condylar guidance depends on:* 1) shape & size of bony contour of TMJ
  2) Muscle actions attached to mandible
  3) limiting effects of ligaments
  4) method used for registration;
- In complete dentures, the condyle path during free mand. mvmt is governed mainly by shape of fossa & meniscus & muscular influence;
- Inclination of condylar path during protrusive mvmt varies from steep to shallow in different pts, which is the most important factor that affects selection of post. teeth w/ appropriate cusp height;
- **Protrusive record is probably the LEAST reproducible maxillomandibular record;**
- Functionally Generated Pathway Technique (FGP) – records movements in wax intra-orally & transferred to articulator in form of a static plastic cast (functional index);
- TMJ – **ginglymoarthrodial joint** - slides/glides & rotates; 2 compartments:
  1. *Lower Compartment* – Condyle-Articular Disc; Hinge type or ROTARY movement;
  2. *Upper Compartment* – Mandibular Fossa-Articular Disc; SLIDING/TRANSLATORY movment; Lateral pterygoid muscle contract so condyle slides FORWARD;
- **Terminal Hinge Position** (Transverse Horizontal Axis) – the one relation of the condyles to the fossae in which a pure hinging movement is possible;
- Closes Mandible - 1) Masseter
  2) Medial Pterygoid
  3) Temporalis anterior fibers (posterior fibers retract the mandible)
- Opens Mandible - 1) Lateral Pterygoid (also PROTRUDES & LATERAL mvmt)
  2) Anterior Digastric
  3) Omohyoid
- **Lateral Pterygoids** are mainly responsible for positioning & translating the condyles;
- Cusp Inclination - angle made by slopes of a cusp w/ a perpendicular line bisecting the cusp, measured MD or BL; under the DENTIST's control;
- Functionally Generated Pathway Technique – prerequisite is optimal occlusion; allows cuspal mvmts of the dentition to be recorded in wax intra-orally then transferred to articulator in form of a static plastic cast (**functional index**); use low-fusing hi-fi wax;
  → all mandibular motion must be directed from an eccentric centric position (never the reverse);
- When surface to surface contact of flat cusps occur, it should be change to a point to surface contact;
- When centric occlusion is established, NEVER take the teeth out of centric occlusion;

OCCLUSAL CONTACTS FACTS:

- DL cusp of **mand. 1st molar** opposes lingual groove of max. 1st molar (same as mand. 2nd molar); its DB cusp opposes max 1st molar central fossa & its D cusp occludes w/ distal triangular fossa of max 1st molar.
- MB & DB cusps of max 1st molar oppose MB & DB grooves of mand. 1st molar;
- Oblique ridge on Max. 1st molar opposes developmental groove b/w DB & D cusps of mand. 1st molar;
- Lingual cusps of mandibular 1st PMs don't occlude anything!
- Lingual cusps of max PMs occlude the distal triangular fossa of their opposing counterpart;
- Outer aspects of lingual cusps of mandibular molar don't contact maxillary teeth; duh.

- ML cusps of permanent mandibular molars occlude w/ the lingual embrasures b/w their class counterpart & tooth mesial to it;
- Buccal cusp tips of max. PMs oppose facial embrasure b/w their counterpart & tooth distal to it;
- Max & Mand. canine cusp tips do NOT contact any other tooth;

PATHOLOGY:

- Palatal Tori – more Females than Males; max size at 30's or 40's; may act as fulcrum & causing rocking of MAX. denture; post-op healing slow if removed due to poor blood supply of thin tissues over tori;
  → Thin mucosa is found over palatal & mandibular tori;
  → Palatal tori is not usually removed but MAND. tori is usually removed prior to making dentures;
- Inflammatory Papillary Hyperplasia – denture irritation & food impaction; hard palate; red, firm & painless; Candida Albicans may contribute to inflammation; most pts are unaware of lesions;
- Denture-Induced Fibrous Hyperplasia – **Epulis Fissuratum;** vestibular mucosa; trauma from bad denture; painless folds of fibrous tissue; often overextension of denture;
  → Traumatic occlusion of natural teeth opposing an artificial denture may also cause epulis fissuratum;
- Paget's Disease – **Osteitis Deformans;** bone disorder in which bone becomes enlarged but weakened w/ heavy calcifications; often discovered in dental office b/c pts dentures don't fit due to widening of alveolar ridge;
- Diabetes – impairs WBC; delays healing, ↑ progress of periodontitis, ↑ calculus, & ↑ PA lesions; not associated with mucosal bleeding/bleeding disorders;
- Denture Stomatitis – localized or generalized chronic inflammation of the denture-bearing mucosa; presents as redness & burning; trauma & secondary fungal infection are most likely causes;
- Children who wear dentures & acromegaly pts w/ dentures often need their dentures relined or remade often to allow for bone growth;
- Osteoporosis – most common cahnge associated w/ systemic disease;

MISCELLANEOUS:

- Excessive wear on occluding surfaces of teeth is usually caused by disharmony between CO & CR;
- Solder must melt at **least 150°F** below fusion temperature of metals; Gold solder used for FPD & Silver solder used for ortho appliances;
- Horizontal Forces – most destructive to periodontium;
- Ante's Law – root surface area of abutement teeth supported by bone must equal/surpass the root surface area of teeth being replaced w/ pontics;
- Strain/Work Hardening – hardening/deformation at room temp; ultimate result is fracture; ↑hardness, strength, & proportional limit; ↓ ductility & resistance;
  → ie – bending wire back & forth rapidly between the fingers;
  → done at room temp in contrast to **forging** which is working at higher temperatures;
  → under microscope, elongated grains in microstructure of wrought wire indicated worked/strained hardening;
- Quenching – metal cooled from ↑ temp to room temp; To achieve softened condition for type 3 gold, quench in water 30-40 sec; *advtages* – maintains the metal's malleability & ductility and the casting is more easily cleaned cuz investment becomes soft & granular;
- Annealing – softening a metal by controlled cooling of material to ↑ ductility & strength & less brittle;
  → 3 stages – recovery, recrystallization, & grain growth;
  → gold foil is annealed to remove volatile surface impuruities prior to placement in prep;
- Fritting – process for manufacturing low & medium fusing porcelains; creates fine porcelain powder (frit) that can be added over by other metallic substances to produce color in porcelain;
- High Sag Factor(Distortion) – leads to distortion of bridge spans when porcelain is fired;
- X-ray Signs of Occlusal Trauma: 1) Hypercementosis

2) Root Resorption
3) Alteration of Lamina Dura
4) Alteration of Periodontal Space

- Facebow – caliper device records pts. maxilla/hinge axis relationship = open/close axis;
  - → Record used to orient the maxillary cast to the hinge axis on the articulator;
  - → Hinge-axis face bow transfer enables the dentist to ALTER VDO on articulator;
  - → Hinge-axis face bow is used to record opening & closing of the mandible;
- The preferred method to **preserve the face-bow transfer** is TAKING A PLASTIC INDEX;
- When alter VDO, casts should be mounted on Hinge axis;
  - → Facebow/hinge axis yield error of 2mm or less on most patients;
- Pantograph – precise tracing of paths followed by the condyle; need 2 facebows & fully adjustable articulator;
- Arcon Articulator – condylar element on LOWER MEMBRANE of articulator; FIXED condyle angle; like panadent – for CROWNS & DIAGNOSTIC CASTS;
- Non-Arcon Articulator – condylar element on UPPER MEMBRANE of articulator; NON-FIXED condyle angle; for DENTURES;
- Prolonged sensitivity to heat, cold, & pressure after crown cementation is usually related to OCCLUSAL TRAUMA; if CR occlusion is high, pt complains of cold sensitivity & pain on biting hard;
- Excursive movements must also be checked b/c if pt complains of pain when chewing soft foods, this indicates improper balancing or working contacts;
- Initial sensitivity can be caused by acid irritation accentuated by dehydrated dentin from prolonged drying of tooth b/f cementation or incorrect liquid/powder ratio of cement;
- If marginal ridge is left higher than adjacent marginal ridge, a RETRUSIVE interference movement may occur;
- Advantages of Post & Core:
  1) Marginal adaptation & fit of restoration independent of fit of post;
  2) Restoration can be replaced without disturbing post & core;
  3) Can be treated as an independent abutment;
- A post & core must have roots w/ adequate length, bulk, and straightness; if root configurations not favorable, then use pin-retained amalgma or composite core;
- Glazed porcelain, polished gold, unglazed porcelain, & polished acrylic are preferred in that order of their acceptability to soft tissue;
- Electrosurgery – passing small current of electricity thru the gingival tissues, causing cells to desiccate or scorch; results in some delayed healing b/c lack of proper clot formation;
  - → very good at stopping hemorrhage;
  - → too low a current can be detected by tissue drag;
  - → objectives – coagulation, hemostasis, access to margins, & reduce inner wall of sulcus;
  - → potential serious damage to PDL & surrounding bone, causing loss of attachment;
- *Human Dentition Features the Effect PDL Health & Hard Tissue to resist occlusal Force:*
  - → Anterior teeth have slight/no contact in MIP
  - → Occlusal table is <60% of overal FL width of tooth
  - → Occlusal table is at right angles to tooth's long axis
  - → Mand. molar crowns are inclined 15-20^{o} toward the lingual

RADIOLOGY

MISCELLANEOUS:

- For radiopaque structures, less radiation penetrates the structure & reaches the film so more radiation absorbed in structure;
- For radiolucent structures, less dense materials ALLOW radiation to pass thru by absorbing very little radiation;
- Most benign lesions are unilocular and well-defined;
- 90% of diffuse radiolucent structures are cancer; if loss of cortical places, the 1st diagnosis is cancer;
- Osteoradionecrosis is necrosis of bone produced by ionizing radiation; more common in the mandible than maxilla due to richer vascular supply in maxilla & b/c mandible is more often irradiated;
  - → Most common precipitating factors are pre & post irradiation & periodontal disease; damage to blood vessels predisposes a pt to developing this;
  - → don't heat bone >116°F/47°C.
- dental radiographs should be retained indefinitely; legally they are the property of the DENTIST but pts have right to reasonable access to radiographs;
- pts may refuse radiographs but no document can be signed by the pt that releases the dentist from liability;
- Digital Radiography – requires LESS radiationg than traditional x-rays b/c the sensor is more sensitive to xrays; radiation exposure to pt is reduced by 50-80%; sensor is used in place of film;
  - → Superior gray scale resolution, increase speed of image viewing, decreased cost of equipment & film, image enhancement, & superior pt education;
- Storage Phosphor Imaging System – type of digital imaging system that uses a reversible imaging plate rather than a sensor to record image; plates are more flexible thus more comfortable for pt;
- Direct Digital Imaging System –uses an intraoral sensor attached to a fiberoptic cable;
- Indirect Digital Imaging System – scans an existing xray and digitizes the image;
- Charge-Coupled Device – the MOST COMMON digital image receptor; solid state detector w/ a silicon chip embedded in it; used in home video cameras, fax machines, & telescopes;
- Primary Radiation – radiation generated at the ANODE of the xray tube that is attenuated by the filter & object;
- Scondary Radiation (**Scattered Radiation) –** arises from interactions of the primary radiation beam w/ atoms in the object being imaged; a LEADED RECTANGULAR cone best ↓ amt of scatter radiation;
  - → major source of image degradation in both xray & nucelar medicine imaging techniques;
  - → operator recieves greates hazard from secondary radiation;
- **Collimation –** control of size & shape of xray beam using metal plates & slots to confine & direct radiation;
- Radiation beam should be as small as practical; diameter of circular beam of radiation at pt's skin can't be larger than **2.75 inches**;
- Xray beam composed of rays of different wavelengths & penetrating power (**polychromatic)** b/c the potential across the xray tube constantly changes at the kilovoltage changes;
  - → Short wavelength xrays = high energy; produced at high kVp & penetrates object more readily;
  - → Long wavelength xrays = low energy; produced at lower kVp thus ↓ penetrating power;
  - → **Aluminum discs are used to filter out these useless long wave rays to ↑ quality of xray;**
- Filtration – removal of parts of xray spectrum using absorbing materials in the xray beam; reduces pt dose, contrast, & film density; 3 types of Filtration:
  1. *Inherent Filtration* – parts include glass envelope of the xray tube & oil surrounds xray tube to cool the tube to dissipate heat; corresponds to ~0.51mm of aluminum;
  2. *Added Filtration* – obtained by placing thin sheets of aluminum in cone to filter the useful beam futher;
  3. *Total Filtration* – consist of inherent filtration + added filtration; .5mm & 2.5mm of aluminum;

- Operator should never remain in room holding xray in place for pt; if child needs help, have parent hold film with lead vest draped on them;
- Operator must avoid primary beam by positioning themselves at 90°-135° angle to the beam;
- **EKTA-Speed Film** – provides the MOST EFFECTIVE way to REDUCE exposure time, amoutn of radiation reaching pt & amount of scatter radiation;
- Other factors that ↓ Pt Radiation:
  1) Lead apron is MOST EFFECTIVE way to stop xrays
  2) ↑ filtration using aluminum disk
  3) lead diaphragms placed w/in cone of xray tubehead
  4) collimating an xray beam
  5) ↑ source-film distance
  6) intensifying screens (used with pano & ceph)
- *Committee on Radiation Protection of National Bureau of Standards* – recommends person who works near radiation be exposed in 1 yr to max dose of 5 REM (.1 REM/week);
  → **Maximum Permissible Dose** = .5 REM for non-occupationally exposed persion;
- Sequence of Radiation Injury: 1. Latent Period, 2. Period of Cell Injury, 3. Recovery Period;
- Effects of radiation exposure are ADDITIVE, & the damage that remains non-repaired accumulates in tissues;
- The greater the rate of potential for mitosis & more immature the cells & tissues, the more susceptible or sensitive these cells are to radiation;
  → **Radiosensitive cells:** immature blood cells (small lymphocytes), bone marrow, reproductive cells, & immature bonce cells; Prostate gland is very sensitive to radiation;
    - Hemopoietic tissuse is most sensitive to radiation.
  → **Radioresistant cells**: mature bone, muscle, & nerves (pulp); Muscle cells are most radioresistant;
- Radiation Absorbed Dose – measure of the energy imparted by any type of ionizing radiation to a mass of any type of matter; unit of absorbed dose = **rad;**
- Equivalent Dose – correct unit of measurement used by dentist to compare the biologic-risk effects/estimates of different types of radiation damage to tissue/organ;
- Effective Dose – used to estimate the risk in humans;

Conventional Measurement	SI, Metric Equivalent
Rem	Sievert (Sv)
Rad	Gray (Gy)
Roentgen	Coulomb

- Exposure – measure of radiation quantity, the capacity of the radiation to ionize air; Roentgen is tranditional unit of radation expsure measured in air; Roetgen only applies to xrays & gamma rays;
  → Xrays have more energy than line; ~1% of energy released in xray tube is released as xrays;
- Electromagnetic Radiation – includes microwave, x-radation, visible light, & gamma radiation; Xrays & gamma rays are type of **non-particulate** radiation energy;
- **Submandibular gland fossa** – large radiolucent space ~5mm below MB rooth of mand. 1st molar;

RADIOGRAPHIC SOLUTION & ERRORS:

- Developer Solution – solution that converts the invisible image on a film into a visible image composed of minute masses of black metallic silver;
  → Films keep getting lighter & lighter after each development, to correct this problem simply replenish the developing solution; so as developing solution gets weaker, film gets lighter;
  → Function is to **reduce silver halide crystals to black metallic silver;** 4 Chemicals:
    1. Developing Agent – hydroquinone
    2. Antioxidant preservative – sodium sulfite
    3. Accelerator – sodium carbonate
    4. Restrainer – potassium bromide
- Fixer Solution – chemical solution whose function is to stop development & **remove remaining unexposed crystals;** fixing time is at least twice as long as developing time; 4 chemicals:
    1. Clearing Agent – sodium/ammonium thiosulfate; commonly called **hypo** dissolves & removes underdeveloped silverhalide crystals from emulsion;

2. Antioxidant preservative – sodium sulfite
3. Acidifier – acetic acid
4. Hardener – potassium alum

- If a dried xray were processed a 2nd time, there would be no change in contrast/density;
- **Yellowish brown film** is caused by insufficient fixing or rinsing;
- **Fogged film** may result from improper film storage or outdated films; or due to faulty safelight in darkroom with white light leaking; or b/c exposed to radiation other than from primary beam;
- Low solution levels will appear as developer cut-off (straight CLEAR border) or fixer cut-off (straight BLACK border);
- **Static Marks** (multiple black lines) - due to friction when opening film packets causing static electricity;
- **Torn Emulsion** – films were allowed to touch or overlap while drying;
- **Clear Films** – emulsion washed away b/c film left in water over 24 hrs; or weren't exposed to radiation;
- **Light Films** – underexposed/image not dense enough; due to…
  - → Incorrect mA (too low) or exposure (too short)
  - → Incorrect focal-film distance
  - → Cone too far from pt's face
  - → Film place backwards;
- **Dark Films** – overexposed/image too dense; due to…
  - → Incorrect mA (too high)
  - → Exposure too long
  - → Incorrect kVp (too high)
- **Poor Contrast** (very dark/very light areas) – incorrect kVp (too high);
- Herringbone (Diamond Effect) – a zig zag pattern appears on the processed film when film is placed backwards in mouth;

TYPES OF RADIOGRAPHS:

- Pano is the screening xray for pathology of the jaws; Excellent in **Sialography** – techniqued used in radiology that films the salivary gland after an opaque substance is injected into duct;
  - → Disadv. → ↑ object-film distance causing image distortion & proximal overlapping;
  - → **If Chin tilted too far UPWARD** = **Reverse Occlusal Plane Curve** (frown) – where mand structures look narrower & max structures look wider;
  - → **If Chin tilted too far DOWNWARD = occlusal place shows excessive upward curve** (big smile); also severe interproximal overlapping & anterior teeth appear highly distored;
- Ceph – useful to assess tooth-to-tooth, bone-to-bone, & tooth-to-bone relationships; serial cephs can show amount & direction of growth;
  - → Most stable area from which to evaluate craniofacial growth is **anterior cranial base** due to its early cessation of growth;
- BWs – does NOT show root apices; vertical BW angulation = +8º-10º; a fuzzy/indistinct image of crestal bone is often associated w/ early periodontitis;
  - → Adjust HORIZONTAL ANGULATION to direct the central ray toward center of film;
  - → Child w/ primary teeth, use #0 film.
  - → Child w/ mixed dention, us #1 film
  - → Child with 2nd molars, use 2 to 4 #2 films; sometimes 2 long #3 films but not recommended;
- Submental-Vertical (Submentovertex) – xray for diagnosing BASILAR SKULL FRACTURES & provides some info about zygoma, zygomatic arches, & mandible; use when suspect fracture of zygomatic arch;
  - → source below mandible & film about the head;
- Water's View – standard xray of choice for showing an ANTERIOR view of the **paranasal sinuses** & mid-face & orbits; face lying against film & x-ray source behind the pt's head;

→ BEST film for radiographic diagnosis of **mid-facial fractures**, sinus infections, & its view best demonstrates lesions of the max. sinus;

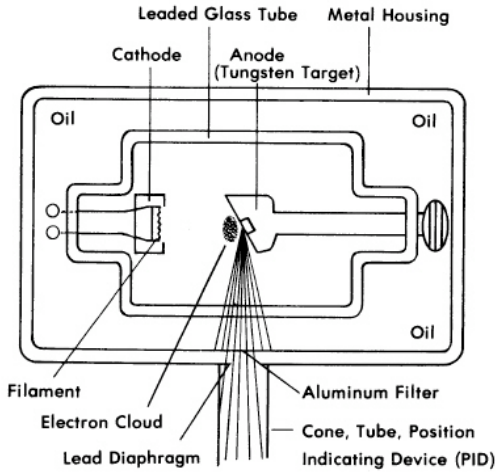
- Towne's View – best film to visualize the CONDYLES & neck of mandible from AP projection; film under head & source is from the front & rotated 30° from frankfort plane & directed at condyles;
  → towne's view eliminates the superimposition of the mastoid & zygoma over the condylar neck in the straight postero-anterior projection which often makes interpretation difficult;
  → **Reverse Towne's View** - used to identify fractures of the condylar neck & ramus area;

RADIOGRAPHIC TECHNIQUES:

$$\frac{\text{Original Intensity}}{\text{New Intensity}} = \frac{\text{Original Distance}^2}{\text{New Distance}^2}$$

- Inverse Square Law – the intensity of the film exposure decreases as a squared ratio as the distance b/w the object & source of xrays increases; meaning intensity ↑ or ↓ exponentially as the source & object are moved while the distance b/w object & film remains the same;
- Half-Value Layer – amt of material required to reduce the intensity of an xray beam to half; normally expressed in aluminum or copper thickness; HVL is indicator of QUALITY of an xray beam;
  → Strickly defined for different quantities – photon fluence, energy fluence, or absorbed dose;
  → Not constant!! When measuring mutliple HVLs, the 2nd HVL is greater than the 1st HVL;
  → The HVL of a beam is ~2mm of aluminum (this means 50% of the xrays exiting the vacumme tube are absorbed by 2mm aluminum; doubling the thickness of aluminum will NOT absorb all the xrays, but one half of the remaining xrays;
- Intensifying Screens – used in extra-oral xrays that convert xray energy into visible light which then exposes the screen film; radiation a pt receives is ↓; used for all extra-oral xrays (pano, ceph);
- **Kilovoltage** – quality or penetrating power of the xray beam that controls the speed of electrons;
  → Suitable ranges are 65-100 kVp;
  → Influences the xray beam & radiograph by altering contrast quality (for pts w/ thick jaws, ↑kVp), determining the quality of xrays produced, & determining velocity of electrons to anode;
- **Milliamperage** - the number of electrons (which determines the quantity of xrays produced) is controlled by the TEMPERATURE of the tungsten filament (mA setting); the hotter the filament, the electrodes are emitted & available to form the electron stream; suitable range = 7-15 mA;
  → Controls the # of xrays produced;
  → the intesity of xrays produced a particular kVp depends on that number;
  → setting the xray machine for specific mA means adjusting the former temp to yield the current flow indicated;
  → to ↑ film density = ↑ mA, kVp, & time & ↓ source-object distance;
- **Exposure Time** – length of time xrays are produced & the time the pt is exposed to them;
- **Contrast** – only one exposure factor affects contrast → kVp; filtration also plays a role;
  → ↑kP = more shades of gray = low contrast; so ↑kVp causes the resultant xray to have a LONGER SCALE of CONTRAST and ↓kVP causes ↑ subject contrast w/ SHORTER SCALE OF CONTRAST;
  → high contrast = very dark & very light areas
  → low contrast = many shades of gray; preferred in dentistry;
- **Density** – overall DARKNESS of a xray that ↑ as mA, kVp, or exposure time ↑;
- **Focal Spot** – small area of tungsten on the anode (target) from which the xrays emanates & receives the impact of the speeding electrons; it is 1 of 3 factors that influence image sharpness; Size of xray tube focal spot influences radigraphic DEFINITION;
  → **Target**(tungsten target) – tungsten wafer embedded in anode face at the point of electron bombardment;
  → Target Film distance is determined by length of cone:
    - 20cm (8 inches) – short cone that exposes more tissue by producing more divergent beam.
    - 41cm (16 inches) – long cone that ↓ amt of exposed tissue by producing a less divergent beam & sharper image;

- Xrays are generated when a stream of electrons (produced by filament) travels from Cathod & is suddenly stopped by its impace at tungsten target;
- Filament located in the cathode and is made of tungsten wire;
- The small area on the target that the electrons strike is the **focal spot** (the xray source);
- <u>Dental X-Ray Tube Parts:</u>
  1. *Filament* – coiled **tungsten** wire in cathod that when heated to incandescence, emits/produces stream of electrons;
  2. *Molybdenum cup* – houses the tungsten filament;
  3. *Electron Stream* – travels from filament in the cathode to the tungsten target;
  4. *Tungsten Target* – located in anode to stop stream of electrons;
  5. *Focal spot* – portion of tungsten target struck by electron beam;
  6. *Copper Sleeve* – located in the cathode;
  7. *Vacuum*
  8. *X-ray Beam* – produced when electron stream bounces off focal spot on tungsten target;
  9. *Leaded glass housing* – houses entire xray tube;



- <u>Vertical Angulation</u> – foreshortening & elongation are produced by incorrect vertical angulation;
  → **Foreshortening** – shortened image caused by EXCESSIVE vertical angulation; teeth appear short due to too much angulation or poor chair position;
  → **Elongation –** elongated image caused by too LITTLE vertical angulation; MOST COMMON error when taking xrays where teeth appear too long due to either too little vertical angulation or film not being parallel to long axis of teeth of the occlusal plane not being parallel to the floor;
- <u>Horizontal Angulation</u> – maintaining central ray at 0^{o}; central ray should be perpendicular to mean antero-posterior plane of teeth being xrayed;
  → Overlapping-interproximal areas are overlapped due to incorrect horizontal tube angulation;
- <u>Bisecting Angle Technique</u> – image on the film is equal to length of tooth whne the central ray is directed at 90^{o} to the imaginary bisector;
  → Tooth & radiographic image are equal in length when 2 equal triangles are formed that share a common side (imaginary bisector);
  → **Decreases exposure time**; xray film may be distored b/c image is not true reproduction of the object (due to use of short cone);
- <u>Paralleling Technique</u> – based on concept of parallelism since film is placed parallel to the long axis of tooth being xrayed & central xray beam is directed perpendicular to long axis of teeth & plane of film;
  → Film holder MUST be used;
  → Disadv – film placement difficult, ↑ exposure time required b/c use of long cone, & object-film distance is ↑ to keep film parallel so image magnificaiton & loss of definition;
  → AKA – XCP (extension cone paralleling technique), Right-Angle technique, & Long-cone technique;
- <u>SLOB</u> – if object in question appears to move in SAME direction as xray tube, then it is on the LINGUAL aspect; if it appears to move in OPPOSITE direction as xray tube, then it is on BUCCAL aspect;
- **Cervical Burnout** – phenomenon caused by relatively low x-ray absorption on the mesial/distal surfaces of teeth, b/w the edges of the enamel & adjacent crest of alveolar ridge;
- 5 Rules to Create Accurate Image on Xray:
  1. Use smallest focal spot that is practical; as focal spot ↓, image sharpness ↑;
  2. Use longest source-film distance that is practical;
  3. Place film as close as possible to structure being radiographed
  4. Direct central ray at a close to a right angle to the film
  5. Keep film parallel to the structure being radiographed;