Lesion (L4) Epulides (bump on gingiva)	Process	Age	Site	Size	Clinical Appearance	Histological Appearance	Treatment	Prognosis	Other
Fibroepithelial Polyp (aka Reactive fibrous hyperplasia, Traumatic fibroma, Fibroma)	Inflammatory Hyperplasia (from chronic irritation)		- any mucosal surface - trauma assoc areas (buccal mucosa, tongue, gingiva, occlusal plane)	<1.5cm	- asymptomatic	- nodular mass of fibrous CT covered by SSE (usually normal but may be hyperplastic) - NOT encapsulated (blending borders) - occasional inflammation and hyperkeratosis	- excision - will not regress on its own	- recurrence is rare - no malignant risk	- most common soft tissue mass - may be the mature form of a pyogenic granuloma - leaf shaped variety: under dentures
Epulis Fissuratum	Inflammatory Hyperplasia	- middle age/older - F>M	- assoc with ill fitting dentures	- vars	- two folds and a fissure that matches the flanges - linear ulcer at the base - may have papillary component - facial aspect of the ridge	- surface epithelium is often hyperplastic - hyperkeratosis - focal ulceration - mature fibrous CT - may have osseous or chondromatous hyperplasia (reactive)	- surgical removal - prepare new denture/lining - will not regress on its own	- will recur if same denture is used	- variant of FEP?
Pyogenic granuloma	Reactive lesion	- children - young adults - pregnant women	- 75% lesions on gingiva	- may grow to several cm	- large response to irritant - red, ulcerated, friable - grows fast - painless - dome shaped - often pedunculated - red to purple colour	- focal reactive growth of granulation tissue with extensive endothelial proliferation - very vascular lesion (many S/L endothelium lined channels in lobular pattern) - surface ulcerated - mixed inflammatory infiltrate	- excision - remove etiology	- 15% recurrence if cause not removed (may shrink over t) - no malignant potential	- not actually pyogenic (pus) or a granuloma
Peripheral giant cell granuloma	Reactive lesion	- any age - mean: 31-40 - 60% F - Md>Mx - more ant???	- ONLY on gingiva		- red or blue mass - may be ulcerated (50% of cases)	- resorption of underlying bone can occur! - reactive bone in centre of lesion may occur - giant cells - background: plump ovoid and spindle shaped mesenchymal cells - lots of haemorrhage with hemosiderin deposits	- excision down to bone, curettage	- 10% recurrence	- not a true neoplasm - similar to pyogenic granuloma - Diff Dx: CGCG, ABC, brown tumour, cherubism
Peripheral ossifying fibroma	Reactive lesion	- teens/young adults - F>M - ant Mx!	- ONLY on gingiva		- red/pink nodule - often ulcerated	- fibrous proliferation with mineralized products (from periosteum or PDL; bone, cementum like or dystrophic calcifications) - giant cells assoc with mineralized products - may cause tooth displacement/mobility	- excision down to periosteum - cleaning adj teeth	- 8-16% recurrence	- unknown pathogenesis - NOT a soft tissue counterpart of the central ossifying fibroma (DIFFERENT)
Hyperplastic Gingivitis	Inflammatory Hyperplasia		- gingiva		- focal or generalized fibrous hyperplasia of the gingival margin	- see fibrotic tissue surrounded by exudate - see dark areas of inflammatory cells - massive inflammatory response in CT (lymphocytes, plasma cells)	- cleaning/scaling - may need surgery if does not regress		- intensified by pts hormonal status ex pregnancy - hyperplastic response is 2ary to inflammation!
Inflammatory Papillary Hyperplasia	Inflammatory Hyperplasia	- denture wearers	- hard palate		- mult pink/red papules on hard palate - confined to palatal vault (rarely extends onto ridges	- papillary projections covered by hyperplastic, stratified squamous epithelium - elongated rete ridges - hyperplasia involves CT and epithelium - chronic inflam infiltrate - occ signs of acute inflammation	- early lesions: may go with rest from denture - antifungal therapy - remove hyperplastic tissue before making new denture		- generally found under dentures in those who wear them 24 hours/day or have poor OH - also occurs in high vault with partial dentures, mouthbreathers, immunosuppressed
Drug induced gingival hyperplasia	Inflammatory Hyperplasia	- any age - ↑ risk in younger pts - M= F				- increased production of extracellular matrix collagen, NOT cells (i.e. NOT hyperplasia of cells) - histology may show normal tissue or ↑ collagen	- dicontinue meds if possible - Improve OH - folic acid, metronidazole, azithromycin	- recurrence is rare	- medications interfere with intracellular collagen degradation - starts 1-3 months after med use - most common meds: - cyclosporin 25% - phenytoin 50% - nifedipine 25%

General: Diff Dx of bump on the gingiva:

- 5 Ps: polyp (FEP), peripheral giant cell granuloma, pyogenic granuloma, peripheral ossifying fibroma, parulis
- Peripheral odontogenic tumours/cysts (ginigval cyst, peripheral CEOT, peripheral ameloblastoma)
- Soft tissue tumours: schwannoma, neurofibroma
- Granulomatous inflammation

Lesion (L5) Odontogenic cysts	Process	Age	Site	Radiographic Appearance	Histological Appearance	Treatment/ Prognosis	Other
Radicular cyst	Odontogenic inflammatory cyst	- generally adults	- assoc with root of non-vital tooth	- well defined, round, corticated radiolucency	- sources of epithelium: - rests of Malassez (from HERS along PDL) - crevicular epithelium - Sinus lining/fistula tract - lining: non keratinized SSE - neutrophils in epithelium, chronic inflammation in CT - hyaline (Ruston) bodies - mucous cells in epithelium from metaplasia - cholesterol clefts	- RCT - possible excision	- most common cyst - differentiate from apical granuloma - *** rando note: dental lamina: rest of Cerres, reduced enamel epithelium
Residual cyst	Odontogenic inflammatory cyst		- more common in Md - previously extracted tooth	- round corticated radiolucency	- identical to radicular cyst - need Rx to differentiate	- excise - watch	- from leftover radicular cyst (incomplete removal)
Dentigerous cyst	Odontogenic developmental cyst	- <30 yo	- 3rd molars - supernumary teeth - any unerupted tooth	- form around crown of unerupted tooth - epicentre above crown - attachment at CEJ - see expansion of alveolar crest - well defined, round, corticated radiolucency	- accumulation of fluid between REE and crown OR OEE and IEE of the enamel organ - fibrous CT wall (loose or dense) - islands/cords of odontogenic epi rests in the wall - 2-4 layers of epi cells (NKSSE) - not many rete ridges (epi and CT interface is flat) - inflammation causes hyperplasia of SSE	- enucleation of cyst - removal of tooth	- 2nd most common cyst - diff Dx: hyperplastic follicle - look at effects on surrounding structures - hyperplastic follicles often bilateral - follow eruption with poss hyperplastic follicle
Eruption cyst	Odontogenic developmental cyst				- soft, round, blueish swelling - separation of the dental follicle from around the crown of an erupting tooth within the soft tissue - looks the same as dentigerous cyst, but has mucosal part	- self resolve	- soft tissue analogue of dentigerous cyst
Gingival cyst of the newborn	Odontogenic developmental cyst		- from cell rests (rests of Serres) of the dental lamina		- small nodules/cysts on the gingiva superficial to developing teeth - lined by keratinized epi	- no tx - will self resolve	- Epstein pearls ala Bohn's nodules arise from non-odontogenic epi along midpalatal raphe or jxn of soft/hard palate
Keratocystic odontogenic tumour (KOT) previously odontogenic keratocyst (OKC)	Odontogenic developmental cyst		- from cell rests of the dental lamina - post Md > post Mx> other - may be bilateral if assoc with syndrome	- scallopped border, multilocular - expansion is less prominent - may be assoc with a tooth or not - effects on surrounding structures: root resorption, tooth movement	- uniform layer of stratified squamous epithelium with wavy or corrugated parakeratotic epi - cyst has keratin inside! - palisading basal cells - may have hyperplastic epi, chronic inflammation	- remove with large resection since thin lining so enucleation seeds new cysts - assoc tooth must be removed - FU 10-15 yrs - has innate growth potential - 30% recur	- assoc with Basal cell nevus syndrome aka Gorlin Goltz syndrome - calcification of Falx cerebrai - bossing of frontal and parietal bones - hypertelorism - bifid ribs - multiple basal cell carcinomas - planter/palmar pitting - epidermal cysts (whitehead like)
Lateral periodontal cyst	Odontogenic developmental cyst	- >40 yo	- from cell rests of the dental lamina - mostly Md PM region	- unicystic or multicystic (i.e. botyroid odontogenic cysts) - lesions are small and non aggressive - well defined, corticated periphery, radiolucent - may get expansion (rare) and loss of LD	- thin lining of NKSSE (1-3 cells thick) - clear cells: glycogen rich - plaques: focal epi thickenings	- curette out - low recurrence	
Gingival cyst of the adult	Odontogenic developmental cyst		- same loc - Md PM region				- soft tissue analogue of lateral periodontal cyst
Calcifying odontogenic cyst (COC) aka Gorlin cyst	Odontogenic developmental cyst	- any age	- 75% ant to 1st M - may assume pericoronal position	- contain calcified material - floculent radiolucency - well defined, corticated - can cause expansion, displacement	- fibrous capsule NOT epithelium!??? - outlining cyst is odontogenic epithelium similar to ameloblasts with "stellate reticulum" (basal cell layer) - ameloblasts: palasaiding appearance, polarized nuclei - ghost cells (anucleated) - calcifications within ghost cells - see lots of CT: looks "solid" - more tumour like shape vs. cyst/balloon	- conservative enucleation - low recurrence	- uncommon! - may be assoc with other odontogenic tumours ex odontoma - WHO classifies this as an odontogenic tumour
Buccal bifurcation cyst	Odontogenic INFLAMMATORY cyst	Pediatric lesion	- buccal bifurcation of 1>2 Md molars - may occur on Mx molars (trifurcation) - occ bilateral	- see enlarged cortex - tooth roots towards the lingual - crown towards the buccal	- involve vital teeth that are delayed and/or displaced - often has buccal swelling - NKSSE - epi hyperplasia and chronic inflammatory infiltrate - looks like radicular cyst*	- do not remove tooth!	- diff Dx of paradental cyst - a U of T cyst

Lesion (L5) Non- Odontogenic cysts	Process	Age	Site	Radiographic/Clinical Appearance	Histological Appearance	Treatment	Other
Nasopalatine duct cyst	Non- Odontogenic cyst		- arise in nasopalatine canal from epi remnants of nasopalatine duct	- can erode through bone to get fluctuant swelling - may be heart shaped from anterior nasal spine	- ciliated columnar, cuboidal or SSE lining - capsule contains BV and peripheral nn		- most common non-odontogenic cyst - differentiate from nasopalatine foramen
Nasolabial cyst	Non- Odontogenic cyst: soft tissue cyst	- F>M - 4-5th decades	- near alveolar process above apices of Mx teeth	- may cause erosion of underlying bone	- soft tissue cyst - pseudostrat columnar epi (respiratory type) - may have mucous cells, ductal cells (cuboidal), SSE - cyst lining and CT may be folded		- likely derived from remnants of inferior portion of nasolacrimal duct
Dermoid cyst	Non- Odontogenic cyst: soft tissue cyst		- from germinal epi trapped during embryonic development - midline		- orthoker squam epi lining - lumen may be filled with keratin and sebum - dermal appendages (hair follicles, sebaceous glands, sweat glands) may be assoc with this epi		- a form of teratoma
Thyroglossal tract cyst	Non- Odontogenic cyst	- children - young adults	- neck: above thyroid gland, below base of tongue*	- asymptomatic, slow enlarging mobile swelling middle of neck or dorsum of tongue	- cyst lining: SSE or ciliated colunmar epi - capsule contains thyroid tissue and lymph aggregates, sebaceous and mucous glands	- ensure there is a fxning thyroid before extracting!	- 70-80% occur below hyoid bone where embryonic thyroglossal tract makes 2 distinct turns as it descends from the foramen caecum on the dorsum of the tongue to the thyroid gland
Lymphoepithelial cyst	Non- Odontogenic cyst		- extracellular lymph tissue: FOM, post/lat border of tongue	- yellow, tan - asymptomatic - superficial submucosal mass	- epithelial invaginations entrapped w/in lymphoid tissue or minor salivary duct epi transversing the lymphoid tissue - lining: thin paraker SE surrounded by well defined mass of normal lymphoid tissue - cyst lumen may be filled with parakeratin - not many rete ridges		
Simple bone cyst	Pseudocyst	- younger	- post Md	- scalloped radiolucency around roots but LD intact, not moving teeth - multilocular appearance - rarely effects surrounding structures/causes expansion - see thinning of cortex w/o expansion - not as well corticated as other cysts	- empty cavity, may be fluid filled - no real lining - non descript biopsy	- curetting may induce healing	- often occur with PCD and FD - radiographic Dx!
Stafne bone defect	Pseudocyst		- 3 variants: - lingual posterior - 99% of t - lingual anterior - medial ramus - INF to Md canal!	- concavities in the bone surface - thick sclerotic superior border - well defined radiolucency - will increase in size		- no tx - wont break the Md	- 3 variants: - lingual posterior - lingual anterior - medial ramus
Antral pseudocyst Aka mucous retention cyst	Pseudocyst		- Mx sinus	- dome shaped, non-corticated radiopacity	- focal thickening of lining of Mx sinus - contains mucous or serous secretions within CT	- no tx	Diff dx of mucocele, also sal gland tumour
Infected cysts				- loss of uniform cortex - no cortex or thick sclerotic border			
Healing cysts				- fill with granular bone from the periphery inwards			

Lesion (L6) Benign Odontogenic Tumours	Process	Age	Site	Arise From	Clinical Appearance	Radiographic Appearance	Histological Appearance	Treatment	Prognosis	Other
Ameloblastoma	Benign Odontogenic Tumor of Epithelial Origin	M=F >20 yrs	- post Md	- rests of dental lamina - developing enamel organ - epithelial lining of odontogenic cyst - basal cells of oral mucosa	- solid/multilocular - unicystic (15%) - peripheral (1% rare) - locally aggressive - slow growing - painless	- radiolucent - well defined, corticated - soap bubble septa - root resorption - tooth displacement - cortical expansion	- no capsule - fibrous CT with islands of odontogenic epithelium: contain: - central "stellate reticulum" cells with peripheral "ameloblast" cells, palisading and reverse polarity of nuclei - cystic degeneration - 6 histologic patterns	- en block resection w 1 cm margin - long term FU	- high recurrence (15%)	- 6 histologic patterns: - follicular - plexiform* - acanthoic** - granular - basal cell - desmoplastic *islands are connected ** islands are keratinized
Unicystic Ameloblastoma subclass of above	Benign Odontogenic Tumor of Epithelial Origin	10-20 yrs				- cystic appearance - single radiolucent locus - hydraulic expansion - may have dentigerous relationship - may be indistinguishable from cyst	- cyst epithelium looks like ameloblasts (columnar-cuboidal w palisading single layer w nuclei to one side) - focal areas of "ameloblastoma" w/in wall	- local curretage and Carnoy's solution	- 10% recurrence	- 3 variations*: - luminal - intraluminal - mural *vars based on where ameloblastoma cells are i.e. in wall of lumen, inside lumen, in CT wall of lesion
Calcifying epithelial odontogenic tumor (CEOT) aka Pindborg tumour	Benign Odontogenic Tumor of Epithelial Origin	M=F 30-50 yrs	- post Md 2/3 cases		- multilocular	- may be assoc w impacted tooth - internal radiopacities often assoc with tooth crown - floculant calcifications - "honeycomb" pattern - radiolucent - well defined, corticated - soap bubble septa - tooth displacement - cortical expansion	- discrete islands/strands of epithelium in fibrous CT stroma - epithelial cells* have distinct outlines and intercell bridges seen *cells resemble stratum intermedium - amyloid: amorphous, eosinophilic ECM - Liesegang ring calcifications - cells are pleomorphic (but not malignant)	- local resection w rim of surrounding bone	- 15% recurrence	- rare! (<1% benign tumours) - recall: COC is not multilocular and ~anterior
Adenomatoid odontogenic tumor (AOT)	Benign Odontogenic Tumor of Epithelial Origin	2:1 F:M 10-20 yrs (2nd decade)	- Mx>Md - ant		- small sessile mass on facial gingiva - behaves like a cyst	- may have dentigerous relationship w impacted tooth (attaches lower on root vs. at CEJ) - well defined, corticated - internal radiopacities - pebbly calcifications - tooth displacement - root resorption RARE	- thick fibrous capsule - cystic spaces - cellular, epithelioid appearance i.e. spindle shaped cells forming sheets, strands or masses in fibrous stroma - very thin stroma "stellate reticulum" - nuclei away from central space - rosette-structures (characteristic) - duct like structures - calcifications (rings) - amorphous eosinophilic extracellular material	- easy to ressect	- low	- often inhibit eruption - looks very similar to COC (but NO ghost cells)
Calcifying odontogenic cyst/tumor (COC)	*See cyst lecture but classified as a tumour*	M=F 30 yrs	- ant		- mainly intraosseous - may be peripheral: sessile, pedunculated, painless, mucosa coloured	- floculant radiopacities - effects on surrounding structures	- thick fibrous capsule(4-10 cells thick) - basal cells like "ameloblasts" (cuboidal-columnar, palisading, polarized nuclei) - overlying layer resembles "stellate reticulum" - ghost cells (calcifications)			

Lesion (L6) Benign Odontogenic Tumours	Process	Age	Site	Arise From	Clinical Appearance	Radiographic Appearance	Histological Appearance	Treatment	Prognosis	Other
Ameloblastic Fibroma	Benign Odontogenic Tumor of Mixed Origin	M>F 0-20 yrs	- post Md		- well defined, cyst like periphery - coronal to developing teeth near alveolar crest	- radiolucent - well defined, corticated - cortical expansion - inhibit eruption of teeth - looks like dentigerous cyst but doesn't start at CEJ	- solid soft tissue mass +/- capsule - "dental papilla" like mesenchymal tissue with strands/islands of odontogenic epithelium palisading at periphery	- surgical excision	- rare malignant transformation	
Ameloblastic Fibro-Odontoma	Benign Odontogenic Tumor of Mixed Origin				- same as **Ameloblastic Fibroma**	- same as **Ameloblastic Fibroma** but see radiopacities - small lesions may have only 1-2 radiopacities - commonly assoc with unerupted tooth	- same as **Ameloblastic Fibroma** but see enamel and dentin			
Odontoma **1. Compound**	Benign Odontogenic Tumor of Mixed Origin	0-20 yrs	- ant Mx		- multiple tooth shaped radiopacities (denticles) - proper organization (dentin, enamel, pulp)	- well defined, corticated periphery with a thin adjacent radiolucent soft-tissue capsule - can see jaw expansion	- multiple structures resembling small teeth contained in a lose fibrous matrix - enamel matrix is seen	- removal	- may interfere with normal eruption	- most common odontogenic tumour! - considered a harmatoma (focal malformation) vs. true neoplasm
2. Complex			- post Md/Mx		- irregular mass of calcified tissue		- mature tubular dentin mixed with dental pulp and enamel matrix - no order			
Odontogenic Myxoma	Benign Odontogenic Tumor of Mesenchymal Origin	M=F Young adults	-M/PM region - Md>Mx		- causes some expansion	- periphery often ill defined i.e. a subtle lesion (but may be well defined as well) - multilocular - "tennis racket" septa - may scallop between teeth (causes loosening) - rarely resorbs teeth				
Myxoma	Benign Odontogenic Tumor of Mesenchymal Origin				- "snot like"	- no border/capsule - no Rx description?	- haphazardly arranged stellate, spindle-shaped and round cells in loose myxoid stroma/CT - odontogenic epithelial rests may be seen (trapped inside, not part of neoplasm)	- curettage - wide excision	- 25% recurrence	
Odontogenic Fibroma We talk about **central** type Subtypes: - Simple - WHO	Benign Odontogenic Tumor of Mesenchymal Origin	M<F 40 yrs				- unilocular - radiolucent - may have septa/calcifications - may have expansion - may have tooth displacement/resorption - loss of lamina dura	- Simple: stellate fibroblasts with fine collagen fibrils, ground substance +/- small foci of odontogenic epithlium (trapped) - WHO: similar stroma but more prominent odontogenic epithelial islands and strands	- curettage	- low recurrence	- rare! (<1%) - peripheral type also exists (in soft tissue) - *OF is on a spectrum with myxoma but has increased density of mesenchyme background
Cementoblastoma	Benign Odontogenic Tumor of Mesenchymal Origin (cementoblasts)	M=F	- Md -M/PM		- painful expansion of bone in 2/3 of pts - always attached to a tooth (M/PM root) - epicentre at apex	- well defined periphery - radiolucent rim - "wheel spoke" pattern - internally radiopaque - may resorb roots and expand cortices	- gross fusion of lesion to tooth - sheets and thick trabeculae of mineralized material (cementum) with lacunae and reversal lines (basophilic, blue) - cellular fibrous vascular tissue - giant cells	- removal		

Lesion (L7) Benign Non-Odontogenic Tumours	Process	Age	Site	Clinical Appearance	Radiographic Appearance	Histological Appearance	Treatment	Other
Neural Tumours - includes neurilemmoma, neuroma, neurofibroma	Benign Non-Odontogenic Tumor		- IAN canal	- grows from epicentre - looks benign since cortices are intact	- radiolucent - corticated periphery - well defined fusiform expansion of canal cortices			
Atrial-Venous Malformation/Central Hemangioma	Benign Non-Odontogenic Tumor		- post Md (body or nn canal)	- may see soft tissue swelling (check if it blanches) - no growth around epicentre - adjacent teeth/bone affected: may be larger and erupt earlier - tooth may feel depressible w pulse on top - teeth may be tipped out of occlusion	- generally well defined (may be ill defined, mimic malig) - multilocular (b/c multiple vv) - small/large radiolucent spaces - loss of trabeculation - thinned cortices - if canal is involved: widened and serpinginous shape(wave) - may see: - mental/Md foramen enlargement - root resorption/displacement	- phleoliths (soft tissue calcifications due to static blood/dystrophic calc) - bulls eye pattern on scan due to calc		- recall: pts can bleed to death, be aware of these!
Osteoma *Don't confuse with odontoma** 	Benign Non-Odontogenic Tumor		Md (periosteum of ramus or inferior cortex/border of Md)	- may displace soft tissues (mm) causing dysfunction - small osteomas resemble torus/exostosis - exostosis: Mx, grinders	- well defined borders - radiopaque internally - homogenous: compact bone - trabeculated: cancellous	- mature compact or cancellous bone - endosteal or periosteal	- no tx - generally recur if removed	- associated w **Gardner Syndrome** aka intestinal polyposis syndrome - hereditary cancer predisposition syndrome - variant of familial adenomatous polyposis (FAP) - autosomal dom (APC gene) chromosome 5 - Characterized by: - multiple osteomas (inf border of Md) - dense bone islands - multiple supernumary teeth/odontomas - intestinal polyps - epidermoid cysts (keratin filled) - osteomas develop early (before polyps) so note!
Central Giant Cell Granuloma	Benign Non-Odontogenic Tumor	- young <30	- Md - ant to 1st M	- often has soft tissue component - *more aggressive vs. peripheral granuloma ?* - can be red/purple - may bleed!	- well defined, NO cortication - multilocular - faint/whispy septa - hard to see = diagnostic hint - granular bone deposits (occ) - undulating periphery (due to expansion) - may displace/resorb teeth	- identical to peripheral giant cell granuloma - low power of periph looks like fibroepithelial polyp - giant cells, extravasated blood, fibroblasts - need a clear margin to differentiate central vs. peripheral	- surgical	- Note: ameloblastoma, myxoma and CGCG are all multilocular causing expansion and tooth resorption - have different septa (soap bubble, tennis racket, whispy - other giant cell lesions: - brown tumour of hyperparathyroidism - part of ABC or fibrous dysplasia
Cherubism	Benign Non-Odontogenic Tumor	- young (1st decade)	- bilateral - often affects Mx and Md - epicentre: post ramus/tuberosity	- rapid growth - may expose inferior sclera (cherub) - involutes w skeletal maturity	- resemble CGCG - cause significant expansion/tooth displacement - Rx signs may persist after involution - multilocular, septations (whispy/grainy), thinned cortices of Md, sclerosis	- resemble CGCG	- ortho tx - future bone replacement?	- autosomal dom transmission - Dx usually with clinical and Rx criteria alone (NOT biopsy) - often molars are taurodonts (not a Dx feature)
Aneurysmal Bone Cyst	Benign Non-Odontogenic Tumor		- post Md - epicentre ~1st M	- reactive lesion/pseudocyst - rapid and extreme expansion	- multilocular - whispy, R angled septa - large lesion	- CT shows large pools of blood filled spaces (aneurysm) - highly cellular mass of blood-filled spaces w scattered giant cells "blood filled sponge"	- curettage - may recur	- rare - similar Rx appearance to CGCG but in post Md vs. ant
Langerhan's cell histocytosis Aka Histocytosis X	Benign Non-Odontogenic Tumor	- young (but any age possible) - more aggressive in young kids	- Md>Mx - intraosseous and alveolar types	- lesions may affect soft tissue/bone anywhere in body (central and peripheral presentation) - often Dx with skull film as well - often see multiple lesions - in young pts, may get infiltration into follicles which displaces them superiorly (like they're erupting)* - Clinical spectrum: 1. Eosinophilic granuloma - solitary or multifocal - presents as gingival swelling (infection, pain, intraoss presentation) 2. Chronic disseminated histocytosis aka Hans Schuller-Christian disease - multifocal, assoc w diabetes insipidus and expthalmous 3. Acute disseminated histocytosis aka Letterer Siwe disease - considered more malignant (fatal in infants)	- radiolucent - well defined periphery - NO cortication - smooth or irregular - "punched out" lesion - "scooping out" of bone from alveolar crest, epicentre mid root - teeth appear as "floating" - tooth displacement and resorption uncommon - often see periosteal new bone formation (as in inflammatory lesions)	- at low power, don't see CT, just dense inflammatory infiltrate - at high power, see many eosinophils and langerhans cells (pale cytoplasm, indistinct borders, Reniform nucleus (kidney shaped) - can be specifically identified with stains S100, HLA and CD1A - Birbeck granules: tennis racket shaped organelles seen via EM	- long term FU - may need BM transplant	- Langerhan's cells: type of APC - behaves as a malignancy (but not a true malignancy) * 3 L diseases move teeth up like erupting: LCH, lymphoma, leukemia

Lesion (L8)	Process	Age	Site	Clinical Appearance	Radiographic Appearance	Histological Appearance	Treatment	Other
Fibrous dysplasia: 3 Types: **1. Solitary**	Fibro-osseous lesion: Bone dysplasia		- post Mx (most common) - unilateral - anywhere in body!	- most common type - incidental or painless swelling on one side	- ill defined periphery (blending) - internal area: radiolucent/mixed/radiopaque - Radiopaque patterns: - ground glass, orange peel, cotton wool, amorphous (ex Md), fingerprint - see bone enlargement with maintenance of smooth periphery - may encroach on surrounding structures ex Mx sinus - may alter bone pattern of cortical boundaries - loss of lamina dura - interferes with eruption - displacement of teeth - displacement of IAN canal SUPERIORLY	- no capsule - bone is replaced by fibrous CT - see fibroblasts, BV, giant cells - cystic changes - Chinese characters: areas of immature bone forming in CT background - don't see osteoblasts/clasts surrounding bone (i.e. not normal bone formation)	- don't tx while pt is growing - esthetic recontouring - if becomes malignanct, radiotherapy	- stops growing at skeletal maturity
2. Multiple (Jaffe type)		- younger						
3. Multiple FD - associated with McCune Albright syndrome				- polyostic FC - cutaneous pigmentation - hyperfunction of endocrine glands				
Periapical osseous dysplasia (POD/PCD)	Fibro-osseous lesion: Bone dysplasia	- middle age - F - black	- Md ant teeth - epicentre = apical area	- generally no expansion - large lesions may cause expansion - does not cause loss of tooth vitality - usually multiple - solitary = "focal" - more in post teeth - widespread = "florid" (3+ quads) - Mx often progresses slower than Md - if segment gets perio infected, pus can move throughout jaw (severe infection), can get severe disfigurement. Avoid things that cause infection ex implants, biopsy etc	- well defined periphery with radiolucent rim surrounded by sclerotic border - internal area: radiolucent/mixed/radiopaque - matures from the centre outwards - multifocal - matures over t (epicentres merge - CBCT: chunks of amorphous bone with vars thickness radiolucent rim and sclerosis around this - irregular expansion: opaque - radiolucent - opaque - loss of lamina dura, PDL widens - hypercementosis of teeth - root resorption = rare	- no capsule - early phase: lots of CT, small bone chunks (globules, cementum like), looks like FD - mixed phase: dense CT background with regularly positioned chunks of bone (Chinese characters) - see osteoblasts rimming these - mature: see chunks of fibrous CT throughout		- common! - usually an incidental finding - easy to confuse POD with RO - use vitality test to differentiate! - mult teeth suggests POD vs. RO - SBC can also heal with weird border - cementoblastomas have root resorption, pain, thinner rim, not multifocal - odontomas: not PA, younger pts - hard to diff dense bone island and mature POD: look for residual radiolucent rim, don't biopsy
Cemento-ossifying fibroma (COF)	Benign bone neoplasm	- young <35 yrs - F>M	- post Md - superior to IANC	- asymptomatic when small but swells as grows	- well defined, corticated - soft tissue capsule (radiolucent space) - mixed internal structure (calc) i.e. central radiopacity - concentric, space occupying growth pattern (expansion) - tooth displacement - root resorption	- encapsulated - see well defined separation from surrounding bone - no areas of hemorrhage or giant cells as in FD	- excision - don't need wide cut	- these 3 lesions are all Dx based on clinical and Rx info!

Bone Patterns + Conditions (L9)	Process	Age	Site	Clinical Appearance	Radiographic Appearance	Histological Appearance	Treatment	Other
Hyperparathyroidism				- bone pain - stones - stomach ulcers - psychiatric problems	- overall ↓ density - teeth stand out - cortices thin - loss of LD - change in trabecular pattern - short, randomly oriented "ground glass" - may see ↑ density in 2ary hyperparathyroidism	- may be assoc with brown tumour of hyperparathyro		- 1ary cause = parathyroid adenoma - 2ary cause = hypocalcemia - dietary deficiency, poor vit D absorption, vit D metab deficiency
Rickets		- children	- bones	- enamel hypoplasia - delayed eruption	- thin cortices, low trabecular density - large pulp chambers, widened root canals - Thinning/loss of tooth follicle cortices and LD	- abnormal, globular dentin - clefting from long pulp horns - often non-vital		- adults: osteomalacia
Hypophosphatemia Aka vit D res rickets		- infants: fatal - adults			- rarefying osteisis - clefting through the dentin			
Hypophosphatasia				- ricket's like deformities - premature loss of primary teeth - thin enamel - large pulp chambers - generalized low bone density in jaws - alveolar bone loss, abnormal cementum, focal dental resorption				- inherited (↓ alkaline phosphatase) - reqd for osteoid mineralization - inadequate phosphorus - failure of reabsoption in distal renal tubules
Osteopetrosis				- fragile bones - poor vascularity/obliterated BM - prone to osteomyelitis - compression of cranial nn	- "marble bone" disease - bilateral ↑ in bone density - trabecular structure not visible - malformed teeth			- abnormal osteoclases/remodelling
Sickle cell anemia **Thalassemia**				- osteoporosis	- Rx looks dark and hazy - marrow hyperplasia - thinning cortices (look whispy) - jaw enlargement - lack of pneumatization of paranasal sinuses - widening of skull diploe "hair on end" appearance			
Paget's disease		- M>F 2:1 - older >50 yo	- jaws in 20% - mult bones	- enlargement of entire Mx/Md - develop spaces between teeth - buffalo hump + leg bowing - neurologic pain due to bone impingement on foramina	- mixed lesions - altered trabecular - long and linear - short and granular - round patched (cotton wool) - hypercementosis	- puzzle/mosaic bone pattern - may reversal lines - dense bone - few osteoblasts/clasts	Complications: extraction sites heal slow - ↑ incidence osteomyelitis - 1% chance develop osteosarcoma	- osteoclast abnormality - imbalance of bone resorp/apposition - normal serum Ca/P - very elevated alkaline phosphatase! - Great Britain and Australia
Drugs that influence bone density								- corticosteroids - herparin - antiepileptic drugs - cytotoxic drugs - cyclosporin (immunosuppressant)
Cleidocranial Dysplasia				- supernumary teeth - unerupted teeth - severe malocclusion - ramus more linear/parallel				- genetic disease with aplasia/hypoplasia of the clavicles and craniofacial malformations

Benign Tumours (L10, 11)	Process	Age	Site	Clinical Appearance	Histological Appearance	Treatment	Other
Acquired melanocytic nevus		- begin in childhood		Stages of nevus: - junctional (junctional epi i.e. basal cell layer) - compound (junctional epi + CT) - intradermal/intramucosal (all CT)	- benign localized proliferation of neural crest cells - melanocytes initially found in basal layer of epi (jxn of squamous epi and CT) - nests of nevus cells go into CT as they mature	- must biopsy to differentiate from melanoma	- Diff Dx: melanoma
Lipoma	Mesenchymal neoplasm	- adults	- trunk - proximal extremities	- well circumscribed		- conservative excision	- most common Mesenchymal neoplasm
Neurilemmoma (Schwannoma)	Benign peripheral nerve sheath tumours		- can occur in soft tissue and/or bone	- flask shaped radiolucent lesion - well circumscribed, corticated	- encapsulated - Schwann cells = spindle cells (arranged in fascicles) - Two areas: Antoni A and B - Verocay bodies: spindle cells like up like a picket fence (palasaiding)	- should be easy to remove, but grown next to n - conservative excision	- relatively uncommon BUT significant portion occur in H & N
Neurofibroma	Benign peripheral nerve sheath tumours		- skin - Mx ridge	- may be solitary or a component of **Neurofibromatosis type 1** - Hereditary cancer predisposition syndrome - ↑ risk leukemias, brain tumours, rhabdomyosarc, pheochromocytoma, wilm's tumour - one of the most common inherited conditions - autosomal dominant inheritance - mutated tumour suppressor gene that codes for neurofibromin - café au lait macule	- composed of a mixture of cells including Schwann cells - plexiform neurofibroma	- not possible to preserve the n	- most common peripheral nerve sheath tumour
Granular cell tumour	Benign peripheral nerve sheath tumours		- tongue (dorsum)		- thought to be Schwann cell origin - pseudoepitheliomatous hyperplasia	- conservative excision	- uncommon - superficial biopsy may be mistaken for SCC
Congenital epiulis		- newborns	- alveolar ridge - Mx>Md - F>>>M		- NOT of Schwann cell origin, but histologically similar to granular cell tumour		- rare
Hemangioma	Developmental lesion	- infants	- majority: H&N - also skin, organs	- when superficial: red/purple lesions	- localized proliferation of BV	- often spontaneously involute - no tx	- common harmatoma - use diascopy test
Vascular malformations			- Mx and palate	- structural anomalies in BV that present at birth and persist throughout life - Port wine stains (unilateral) - intrabony vascular malformations - nodular appearance, mobile, firm, should blanch - often associated with syndrome: **Sturge-Weber angiomatosis** - vascular malformations along trigeminal n - vascular involvement of meninges on same side - non-hereditary developmental condition - see port wine stain, neuro complications (seizures, learning disabilities), intra-oral involvement		- may lead to severe bleeding (spontaneous or surgery related) - only tx if they want - dental tx in hospital	- difference from hemangioma: this will proliferate during growth then involute
Hereditary hemorrhagic telangiectasia Aka Osler-Weber-Rendu sydrome		- present at birth	- oral mucosa	- see mult red dots all over oral mucosa - hereditary mucocutaneous condition (skin and mucous membranes - autosomal dominant - ALK-1 receptor (endoglin gene) mutation - telangiectatic skin and oral lesions - large arteriovenous malformations in brain, lung, liver - pulmonary HTN, stroke, heart failure, portal HTN, biliary disease - get small clots: thrombosis, embolus			
Lymphangioma		- present at birth	- H&N - cervical - oral - tongue	- large lesion, looks like fish eggs - can be white if close to surface - lymphatic vv tend to occur in interdental papilla close to rete ridges	- very infiltrative border - can infiltrate into skeletal m	- often infiltrative and recur after surgical tx	- harmatomas of lymphatic vv
Osteoid osteoma and osteoblastoma		- young <30		- osteoid osteoma: cortical bone: femur, tibia, small bones in hands/feet, RARE in jaws - nocturnal pain is characteristic - osteoblastoma: spine, femur, tibia, Md - deep pain alleviated by aspirin (PG), swelling - larger, >2cm - **cementoblastoma** is the variant occurring on teeth! :o - both look IDENTICAL	- histologically identical benign tumours of osteoblasts - osteoid looks abnormal (many BV)	- surgical excision	- uncommon
Ossifying fibroma				- swelling on buccal Md	- multi or unilocular radiopacities - well circumscribed, round, smooth border, calcifications	- easy to enucleate - no malignant potential	

Note: Chondrogenic bone tumours are almost always malignant in the H&N (not discussed)

Note: occasionally, you can get fibromas and schwannomas in bone (look the same)

Epithelial	Melanocytic	Soft tissue mesenchymal	Bone
Oral squamous papilloma* Verruca vulgaris* Condyloma acuminatum* *verrucal-papillary lesions caused by human papilloma virus infection	Melanocytic nevus	Lipoma Palisaded encapsulated neuroma Schwannoma Neurofibroma Neurofibromatosis type 1 Granular cell tumour Congenital epulis Hemangioma Vascular malformation Sturge-Weber syndrome Hereditary hemorrhagic telangiectasia Lymphangioma	Osteoma Gardner syndrome Osteoid osteoma and osteoblastoma Ossifying fibroma

Malignant Tumours (L12, 13)	Process	Age	Site	Clinical Appearance	Histological Appearance	Treatment	Prognosis	Other
Cutaneous melanoma	Malignant neoplasm of melanocytic origin		- any site containing melanocytes - 95% skin - 40% extremities	- more common in fair skinned people - 2-8x ↑ risk if fam member has Hx of melanoma - diameter >6 mm - evolving		- wide excision		Types: - superficial spreading - nodular - lentigo maligna - acral lentiginous See alteration in BRAF gene

Oral mucosal melanoma	Malignant neoplasm of melanocytic origin	- older
		- 6-7th decade
			- 25% H&N (gen)
			-50% mucosal melanomas
			- hard palate
			- Mx alveolus
				- much more aggressive in oral cavity
				- start as black/brown macule, become lobulated/exophytic
				- ulceration develops early
				- soft, non-tender
				- underlying bone may appear moth eaten
					- 20% are amelanotic!
					- atypical melanocytes start at epi-CT junction and spread laterally
					- melanoma can mimic many other cell types ex spindle (hard Dx)
					- look for:
					  - upwards migration (usually stay in basal layer)
					  - ulceration
					  - atypia
					  - infiltration
					  - melanin deposits
						- hard to get clear margins	- poor
							- 50% recur
							- 24-44 metasesize
							- 5 yr survival 13-22%
							- pts often die of metast
								- not related to sun exposure
								- EXAM: diff between benign and malignant melanoma!

Rhabdomyosarcoma	Malignant neoplasm of skeletal m origin		- 35% in H&N area		- 3 subtypes: (changes prognosis so imp!)
					  - embryonal (<10yo), most common ST sarcoma in kids, good prog
					  - alveolar (young adults/teens), poor prog
					  - pleomorphic (adults) poor prog
					- small, hyperchromatic (blue) round tumour cells
					- finely granular eosinophilic cytoplasm
					- infrequent cross striations
					- aggregates of tumour cells separated by septa
						- radical surg
						- multiagent chemo
						- radiation
							- 5 yr survival rate
							Embryonal 65-90
							Alveolar: 50%
							Pleomorphic: <30	

Osteosarcoma		- 3-4th decade
		- M>F
			- 1: long bones
			- jaws 6-8%
			- Md: post body/ramus
			- Mx: alv ridge, palate, sinus floor
				- swelling, pain
				- loosens teeth, paraesthesia, nasal obstruction
				Rx:
				- dense sclerosis or radiolucency (vars)
				- widened PDL
				- <u>sunburst/sunray</u> appearance in 25% jaw cases
					- produces osteoid (immature bone)
					- may see chondroid or fibrous tissue
					Subtypes (no changes in prognosis)
					  - osteoblastic, chondroblastic, fibroblastic
					Forms:
					- intramedullary (conventional)
					- peripheral
					  - parosteal (bone surface) good prog
					  - periosteal (beneath periosteum) int prognosis
					  - extraskeletal (anywhere in soft tissue) rare, poor prog
						- complete removal
						- chemo
						- rad?
							- 5 yr survival rate 30-50%	- don't just assume this is an infection and give antibiotics! Do vitality test!

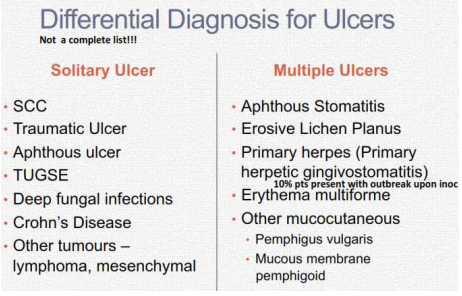
Metastatic disease of the oral cavity		- 40-70 yo	attached gingiva 54% tongue 25% Md 80-90%	- may involve bone and soft tissue - nodular mass - hemorrhagic, exophytic, vascularized - may look like pyogenic granuloma - may be ulcerated, show tooth mobility, bone destruction - paraesthesia, pathological fractures		- surgery, chemo, rad - based on site	- poor	- uncommon - <1% of oral malignancies - common sources: BLuKiPT - Batson's plexus: skips lung involvement
Aggressive fibromatosis	" borderline" malignant disease since doesn't metast			- firm, painless mass - exuberant growth - locally aggressive - can cause facial disfigurement	- hypercellular fibroblast proliferations - infiltrative - elongated cells - streaming fascicles w vars collagen - NO hyperchromasia or pleomorphism	- wide excision - chemo - 23% recur	- high morbidity	- may be assoc w **Gardner syndrome**
Non Hodgkin Lymphomas of the oral cavity (3) 1.Diffuse large B-cell lymphoma		- 6-7th decades	- post hard palate - buccal vestibule - gingiva	- soft tissue lesions: non tender, purplish, boggy swellings +/- ulcerations - bone lesions: vague pain/discomfort (may be mistaken for tooth ache/paraesthesia) - radiographs: ill defined ragged radiolucency. May cause expansion				- most common - aggressive!
2. Bukitt's lymphoma		- children (7yo)	- 50-70% in jaws - Md>Mx 2:1 - post jaws	- pain, tenderness, paraesthesia - marked tooth mobility! - radiographs: ill defined ragged radiolucency	- B cell origin - Endemic/African subtype is highly malignant - sea of strikingly monotonous cells w round nuclei - ↑ cell death and macrophages = "starry sky" pattern	- chemo		- highly aggressive - 90% tumours have EBV and t(8;14) chromosomal translocat (ince c-myc)
3. Extranodal T/NK cell lymphoma, nasal type				- presentation "midline lethal granuloma" - destroys midline sutures and nasal fossa	- see many atypical T lymphocytes, BV infiltration, destruction	- rad - chemo for more disseminated	- good response - favourable	- aggressive
Leukemia **Types (4)**				- thrombocytopenia, palatal peteciae, spontaneous gingival bleed - neutropenia, ulcerations, infections - infiltration of tumour cells, diffuse gingival enlargement, tumour like growth, periapical involvement				

1. Acute lymphocytic leukemia (ALL)		- kids B-ALL = 4 yo T- ALL = teens			- 85% B cell ori	Chemo	95% remission 75-85% cured	- most common cancer of kids
2. Acute myeloblastic leukemia (AML)		- adults - 15-39 yo			- heterogenous	- hard to tx - BM transplant	60% remission 15-30% disease free after 5 yrs	
3. Chronic lymphocytic leukemia (CLL)		- >50 yo - M>F 2:1		- most indolent of all leuks, often asymptomatic - fatigue, weight losss, anorexia			Survival 4-6 yrs	- most common leuk in NA
Multiple myeloma	Plasma cell cancer		- involves whole skeleton - 30% cases jaw	- bone pain, anemia, renal disease, recurrent infections - multifocal destructive bone lesions - amyloidosis (build up of amyloid) may involve oral mucosa (esp tongue), causes enlargement - tooth mobility, paresthesia, swelling, pain Rx: - punched out lesions, soap bubble appearance	- ↑ Igs in blood - Bence jones proteins in urine, proteinuria - see diffuse monotous sheets of vars differentiated plasmacytoid cells	- chemo - poss rad - poss BM transplant	- 50-70% remission Median surv 3 yr	
Plasmacytoma		- M>F 3:1	- bones throughout body		- identical to mult myeloma			- most develop into mult myeloma

General: Non-Hodgkin Lymphoma

- Types
  - Adult
    - Diffuse large B cell lymphoma (aggressive, curable, young)
    - Follicular lymphoma (indolent, older people)
  - Childhood
    - Lymphoblastic lymphoma
    - Burkitt's lymphoma (aggressive, curable, young)
- Dx
  - Lymph node biopsy is gold standard
  - Generally not tx surgically

Lesion (L15) Mucosal Diseases	Size	Age	Site	Clinical Appearance	Histological Appearance	Frequency	Prognosis	Other
Recurrent apthous stomatitis (RAS) Canker sore - 3 variations:		- often children and young adults	- movable, non keratinized mucosa	- painful (not proportional) acute discrete ulcer - shallow or deep - erythmatous border	- central ulcer covered by fibropurulent membrane - epi away from ulcer: spongiosis, lymphocytic exocytosis - superficial CT: band of lymphocytes with histiocytes and plasma cells (mixed inflam infiltrate) - deep CT: ↑ vascularity and mixed inflam infiltrate - see bacteria and granulation tissue	- Dx of exclusion! Based on clinical presentation (rule out other things - biopsy only in Major (rule out SCC		- etiology: unknown - T cell mediated? ↓ CD4:CD8 ratio - 3 main variations: - immunodysregulation - genetics, HLA - stress, neutropenia, AIDS - ↓ mucosal barrier - trauma, nutr deficiencies, smoking CESATION - improve barrier = smoking, hormones ↓preg - ↑ antigenic exposure - toothpaste - meds (NSAIDS, βblocker, INF, methotrexate - micro (strept, H pylori, HSV, VZV, CMV) - foods - assoc with MANY other conditions
1. Minor (80%)	3-10 mm (<1cm)		- ONLY non kerat mucosa -1: buccal/labial mucosa -2: ventral tongue, FOM, soft palate	- shallow, well demarcated, round/oval - prodrome (itch) - yellow/white, removable fibropur membr - erythmatous halo	**Treatment:** - generally no tx - laser tx - chemical cautery (silver nitrate - not done b/c ↑ necrosis) - Tetracycline/Dox/Minocycline rinses	- 1 to many epi/yr	- heal in 7-14 d - no scarring	
2. Major (80%)	1-3 cm (>1cm)	- after puberty	- movable mucosa - soft palate - tonsillar fauces - *any surface	- may have irreg borders - deep extension - longest duration - most SEVERE presentation of all 3		- vars	- heal in 2-6 wks - SCARRING	
3. Herpetiform	Small (1-3 mm - 100s per epi	- F>M - adulthood	- 1: non ker mucosa - ANY mucosal surface may be affected (ker or non ker)	- resembles 1ary HSV infection - pinhead lesions which may coalesce		- FREQUENT	- heal in 7-10 d - no scarring	
Benchet's Syndrome		- Silk route ↑ incidence - M>F - adults	- ocular, orogenital, joints (arthralgia), CNS, CVS - oral: soft palate + oropharynx (RARE RAS sites)*	- classic triad: - oral + genital ulceration, ocular inflam - oral involvement: 1st sign - looks like apthous Major (same dur/freq) - numerous (>6) ulcerations - irreg border - ocular - anterior/post uveitis, conjunctivitis - CVS: vasculitis (pulmonary aa/vv) - CNS: dementia, paralysis - joints: ankle/knee - macular/pustular skin lesions	**Dx:** - lab and pathology findings are NOT diagnostic - International Study group critera: - recurrent oral ulcerations plus 2 of: - recurrent genital ulcers - eye, skin leions - +ve pathergy test (sterile saline leads to nodule) **Treatment:** - generally spontaneous remission - MD prescribed topical/systemic steroids	- vars	- heal in 2-6 wks - SCARRING	- multi systemic disease - etiology: unknown - some HLA assoc (HLA B51) - immunodysreg? (1ary or 2ary to attacks) - enviro antigens: bac (strept), viruses, pesticides, heavy metals
Traumatic ulcers			- tongue, lips, bucc mucosa - gingiva, palate, mucobuccal fold (abraision	- central, removable yellow fibrupur membrane - rolled white border (hyperkeratosis) immed adjacent - well defined - erythema surrounding	- almost ident to RAS (hyperkeratosis w NO dysplasia) - central ulcer covered by fibropurulent membrane - vars thickness - ulcer bed has gran tissue and MIXED inflam infiltrate - epi away from ulcer: normal, hyperplasia or hyperker		- heals within 2 weeks! - if not, biopsy	- differentiate from RAS: RAS occurs where no trauma would occur, NO hyperker rim!
Traumatic ulcerative granuloma with stromal eosinophilia (TUGSE)		- all ages - M>F	- 1ary = tongue - 2ary: gingiva, buccal mucosa, FOM, palate, lip	- ulcer with elevated margins - rolled borders, well circumscribed	- ulcerated mucosa - dense inflam infiltrate involving superficial and deep CT - BOTH! Dx feature - abundant eosinophils! - pointed rete ridges - hyperplastic mucosa	**Tx:** - Incisional biopsy (complete resolution after this)	- last wks-mnths - rare recurrence	- in infants, called **Riga-Fede Disease** - extraction of anterior teeth NOT recomm
White sponge nevus	NOT an ulcer! Inherited disease		- non kerat tissue: - FOM, ventral tongue, buccal mucosa	- white patches on NON-keratinized mucosa - often bilateral - mucosa is spongy upon palpitation	- abnormal tonofilament assembly - hyperkeratosis and vacuolated cytoplasm of spinous cells, no loss of structural integrity	- not usually biopsied	NO assoc w SSC	- autosomal dominant inheritance - mutation in keratin genes 4 and 13 - cytokeratin - forms scaffold
Oral submucosal fibrosis	NOT an ulcer!			- mucosal rigidity (progressive and irrev) - limited vestibular depth - burning sensation - mottled white, brown, red mucosa - gingival recession and tooth staining		- biopsy	↑ risk of SCC	- assoc with betel quid chewing - alkaloids from nut disturb collagen turnover



Lesion (L16) Mucocutaneous Diseases	Process	Age	Site	Clinical Appearance	Histological Appearance	Treatment	Prognosis	Other
Lichen Planus	Chronic inflammatory mucocutaneous disease	- common - older adults UNCOMMON in 1st 2 decades	- anywhere on oral mucosa - #1 = post buccal mucosa - FOM least comm - skin	- multiple, bilateral reticular white lesions - characterized by remissions and relapses - skin lesions: arms and legs - purple flat- topped papules - Wickham's striae - pruritic lesions (itchy) - lesions do not rub off - Types of lesions: - shiny white papules - in linear, annular or reticular patterns - white plaques - atrophic lesions (cause burning) - since surface epi can be thin, looks red - ulcers - white pseudomembrane cover - white striations/erythema on periph - most common: tongue, bucc mucosa - most common presentation: - bilateral reticular white lesions on buccal mucosa - Dx based on clinical features (other forms requires biopsy)	- destruction of basal epithelial cells - low power: - hyperkeratosis - increase in differentiation of suprabasal layers - focal separation of CT and epi (NOT enough to form blisters) - high power - "band like" lymphocytic infiltrate - subepithelial CT - liquefactive degeneration of basal epithelial cells - Colloid/Viatte bodies (ovoid, eospinophillic) - "saw tooth" rete ridges (sharp)	- topical corticosteroids - topical antifungals (for 2ary candidiasis) - maintain good OH - regular FU	- RARE malignant potential to SCC (in atrophic/ulcerative cases)	- etiology: unknown - assoc factors: stress, systemic meds (antihypertensives, oral hyperglycemics, gold salts) - vesicles are RARE in LP
Erythema Multiforme		- young adults	- lips, buccal mucosa - 2nd: palate, tongue - skin	- erythmatous eruption with papules, vesicles and ulcers - vesicles rare b/c rupture quickly - "explosive" onset - skin: symmetrical distr on hands/feet - characteristic "target" appearance - systemic manifestations: - headache, fever, cerv lympadenopathy	- not very specific - inflammation, subepithelial vesicles, degeneration of epithelial cells	- supportive - severe: systemic steroids	- acute, self limiting (cures itself) - spontaneous regression in 2 wks - may recur	- etiology: unknown - Dx based on history and clinical presentation - biopsy may rule out pemphigus vulgaris - may be a hypersensitivity rxn - precipitating factors:50% cases: preceed by 3 weeks - viral (herpes simplex) - medications: sulfonamides, barbituates
Stevens- Johnson Syndrome				- SEVERE variant of **Erythema Multiforme** - extensive hemorrhagic skin lesions - extensive mucous membrane erosions - purulent conjunctivitis		- high dose steroids	- may be fatal	
Pemphigus Vulgaris	Vestibulobullous disease (eruptions of vesicles and bullae)		- anywhere on oral mucosa - 1st: palate bucc mucosa, gingiva	- rapid development of thin walled vesicles which rupture quickly forming ulcerations - skin: ruptured lesions encrust, "fried egg" - scalp, face, axilla, trunk - oral lesions: "first to show, last to go" - irregular, painful ulcerations last weeks - rare to see intact vesicles intraorally	- autoantibodies vs. desmoglien (of desmosomes) - acantholysis: detachment of spinous epithelial cells - Tzank (acantholytic) cells: round, dense dark nucleus - most severe in suprabasal epi layers - intraepi/suprabasal vesicles - "tombstone" appearance - single layer of basal epi cells to CT - severe inflammation	- systemic steroids - immunosupressive agents (azathioprine)	- fatal if untreated - prog vars from well controlled on meds to death	- etiology: unknown - ↑ incidence: Ashkenazi Jews, East Indian - Nikolsky sign: sliding pressure on normal mucosa causes vesicle - NOT specific for pemph - Dx: mult painful persistent ulcers, biopsy perilesional mucosa - biopsy: routine histology (formalin) + direct immunofluor (Michelle's soln, frozen NOT fixed, fluorescein conjugated abs - indirect immunofluor: uses pts serum
Bullous Pemphigoid	Chronic Vestibulobullous disease		- 1ary = skin (extremities, lower ab) - oral: may occur	- skin: large, tense bullae - oral lesions: similar to pemphigoid	- characterized by subepithelial vesicles - autoantibodies vs. component of hemidesmosomes (BP180)	- corticosteroids		- also a Subepithelial blistering disease (i.e. subepithelial vesicles and linear deposits of IgG or complement (C3) along the basement membrane zone with autoantibodies vs. epi-CT attachment complex
Mucous Membrane Pemphigoid (Cicatricial pemphigoid	Subepithelial blistering disease	- 5th - 7th decade	- 1ary = oral mucosa, eye - 90% gingiva - 2ary: palate, bucc mucosa - pharyngeal/esoph! - skin: present in <30%	- oral lesions: erythema, thick walled bullae - desquamative gingivitis: epi can be peeled off at margins of red areas - also found: lichen planus, contact mucositis - pharyngeal/esoph lesions = dysphagia - conjunctivitis	- autoantibodies vs. component of basement membrane (BP180, β4 integrin, laminin 5) - ab bonding to BM leads to complement fixation - subepithelial vesicles - CT has mixed inflammatory infiltrate - not very specific features	- topical systemic steroids - IM injection gold salts, dapsone - opthalmic exams - OH - pharyngeal/esoph lesions = surgical dilation	- rarely fatal - blindness - inability to swallow	- direct immunofluorescence: get line following basement membrane zone
Epidermolysis Bullosa	Subepithelial blistering disease		- skin lesions: hands, feet, elbows, knees	- formation of vesicles/bullae after minor trauma/sontaneously - dystrophic nails - oral lesions: heal with scarring - missing/malformed, hypoplastic teeth		- tx in special clinics		- often inherited - gene mutation in BM component - acquired version in adulthood also exists
Ectodermal Dysplasia	Inherited disease		- oligodontia, anodontia, conical teeth	- prominent supraorbital ridges, protuberant lips, defective sweat glands/nails				- inherited: mult defects of ectodermal origin - mucosal lesions?

Salivary Disease	Process	Age	Site	Gland inv.	Clinical Appearance	Histological Appearance	Tx	Other
Mucocele	Pseudocyst	- young adults - children	- 1 = lower lip - 2 = bucc muc, FOM, retromolar area	Minor salivary gland	- dome shaped, painless swelling, smooth - bluish - fluctuant - fluctuates size	- no epi lining! - 1st neutrophil influx, then macrophages (gran tissue) - adj tissue has non specific inflammatory changes - sialadenitis in adj glands - well circumscribed, surr by compressed gran tissue - foamy histiocytes	- excision - may recur	- severance of minor sal gland excretory duct
Ranula	Pseudocyst		- lateral FOM - neck (if plunging ranula)	- 1 = sublingual - 2 = subMd + minor sal gland	- mucocele on FOM May elevate tongue		- marsupialization - remove assoc gland	
Mucus retention cyst/salivary duct cyst/sialocyst	True cyst	- adults	- FOM, bucc mucosa, lips - RARE lower lip	- major/minor - parotid		- cyst epi (vars appearance: cuboidal/columnar/squam-> oncocytic metaplasia) - LACK of inflammation in surrounding tissue	- excision (cyst + gland)	- less common than mucocele!
Sialolithiasis/ salivary calculi/salivary stones	Obstructive	- young - middle aged		1 subMd gland 2 minor glands	- episodic pain at mealtimes - if palpable: hard yellow stone	- calcifications in duct system - thicker mucous secretions - concentric laminations - sialadenitis	- milking stone out - excision	- Whartons duct has tortuous path, ↑ incidence
Sialadenitis Causes: 1. Infectious a. Viral: mumps, other	Acute viral sialadenitis caused by Paramyxovirus		- bilateral swelling - may involve other sal glands, meninges, pancreas and gonads	Parotid	- fever, malaise, chills, myalgia, anorexia, preauricular pain (bilateral)		- symtomatic - bedrest	- Paramyxovirus: 2-3 wk incubation - adult complications: orchitis (20%), oophoritis, aseptic meningitis, rarely fatal - rare fatalities: viral encephalitis, myocarditis, nephritis - virus replicates in upper respiratory tract
b. Bacterial	Staph aureus infection			Parotid	- swelling, pain, low grade fever, trismus - erythrematous overlying skin - purulent discharge from ducts	- see sialectasia (dialation of ducts/glands) - acute: neutrophils - chronic: lymphocytes/plasma cells, acinar atrophy, ductal dialation, fibrosis	- acute: antibiotics, rehydration - chronic: remove sialolith/gland	- may cause obstructive sialdenitis or decreased sal flow
2. Non-infectious Sjogren syndrome	Inflammation of salivary gland	- F>M 9:1		- lacrimal, major salivary glands	- dry eyes and mouth - arthralgia, myaligia, fatigue	- punctate sialectasia - lymphocytic infiltrate, acinar destruction, epimyoepithelial islands - Dx: minor sal gland biopsy from lower lip - see at least one focal aggregate of 50+ lymphocytes	- multidisciplinary	Other: Sarcoisdosis, Rad tx, allergic rxns - ↓ flow causes retrograde bacterial sialadenitis - ↑ erythrocyte sedimentation rate (ESR) and Ig levels - 75% have rheumatoid factor - +ve antinuclear antibodies (ANA) anti-Ro/La - 40x ↑ risk of lymphoma
Benign lymphoepithelial lesion/ Mickulicz's disease		- 60-80% F - avg age 50		- 80% parotid	- may be a manifestation of Sjogrens - asymmetric enlargement (bilateral, unilateral)	- lymphocytic infiltrate, acinar destruction, epimyoepithelial islands (from squamous metaplasia of remaining ductal elements) (same as Sjorgrens)	- surgical removal - good prog - ↑ risk lymphoma	
Radiation induced salivary gland pathology		- F>M				- early: neutrophil/eosinophil infiltrates, degenerative changes to serous acini - late: degeneration of mucous acini, ↓ vasculature, ↑ fibrosis, vars amts of regeneration	- prevention - saliva substitutes	- no absolute correlation between clinical findings and severity
Adenomatoid hyperplasia of the minor sal glands	Hyperplasia	- M - 4th- 6th decade	- palate		- unilateral swelling of soft and/or hard palate - asymptomatic, sessile, covered by intact normal mucosa	- lobular aggregates of mucous acini (↑ in # and size)	- no tx after confirmed by biopsy	- mimics neoplasm
Necrotizing sialometaplasia			- palate - unilateral		- swelling breaks into an ulcer in 2-3 wks	- pseudomepitheliomatous hyperplasia - preservation of lobular architecture of sal glands - lobular necrosis of sal glands - squamous metaplasia of ducts	- resolves spontan - no tx after confirmed by biopsy	- clinically and histologically mimics malignancy - cause: ischemia, local infarction

Salivary Gland Tumour	Process	Age	Site	Clinical Appearance	Histological Appearance	Treatment	Prognosis	Other
Pleomorphic adenoma	Adenoma (benign tumour)	- 30-50 yrs - (slightly younger) - any age poss	- parotid>palate>upper lip>buccal mucosa	- painless, slow growing, firm mass - movable in major glands, except palate	- named for very variable histo appearance - generally encapsulated - duct like structures, mesenchyme-like background - some myxoid stroma or chondroid matix - may have squamous differentiation	- surgical excision - (not enucleation since may cause recurrence)	- 95% cure rate - <5% risk of malig transformation	- most COMMON sal gland tumour!
Warthin's tumour aka Papillary cystadenoma lymphomatosum	Adenoma (benign tumour)	- 40-70 yrs - M>F 5:1	- parotid - (occ bilateral)	- benign, slow growing, painless	- orderly arrangement of 2 cell layers and lymphocytes - luminal cells: oncocytic, pink - basal cells: flatter, abundant lymphocytic tissue	- surgical excision	- 95% cure rate - 5-10% risk of recur	- ONLY sal tumour assoc with risk factors! - possibly multifocal
Mucoepidermoid carcinoma	Adenocarcinoma (malignant tumour)	- WIDE range - 10-70 yo	parotid>palate>other sites	- on palate: may appear soft or hard - blueish (mucin) - occ intraosseous	- generally non encapsulated - mucous (goblet) and epidermoid (squamous-like) cells + intermediate cells - arranged in cysts/islands - low grade: mostly cystic epi, >50% mucous cells, rare mitosis - int grade: small cysts, mucous cells 10-50%, infreq mitosis - high grade: solid growth pattern, NO cysts, few goblet cells, abundant mitosis/pleomorphism	- surgical resection - radiation if high grade	90% cure low grade 30-90% cure int 30% cure high grade	- most COMMON sal gland malignancy!
Acinic cell carcinoma	Adenocarcinoma (malignant tumour)		- parotid gland	- slow growing - well circumscribed - low grade malignancy	- cells resemble acini - abundant pink/purple cytoplasm - occasionally ducts - usually solid growth pattern, few mitoses	- resection	- good - recurrence <30% - <20% death	
Adenoid cystic carcinoma	Adenocarcinoma (malignant tumour)	- 40-60 yrs	Parotid>subMd gland>palate>other intraoral	- malignant, slow growing, infiltrative - painful	- tumour travels along nn with perineural invasion - islands/nests of small, angular neoplastic epi cells that contain small ducts/pseudocysts - often has cribiform "Swiss cheese" pattern - alternatively: solid and tubular patterns - few mitosis/cellular pleomorphism	- resection - radiation	- 5 yr survival ~70% - poor long term survival (<20%) - lung metastecis	- may see paralysis of facial n
Polymorphous low-grade adenocarcinoma	Adenocarcinoma (malignant tumour)	- 50-80 yrs	- minor sal glands of palate	- slow growing, firm mass	- uniform epi cells in vars patterns (i.e. polymorphous) - tubules, single rows of cells, spindle areas, ducts, glands, cribiform areas - infiltrative margins - perineural invasion	- wide resection	- good - rare to die of disease	
Carcinoma ex pleomorphic adenoma	Adenocarcinoma (malignant tumour)		- parotid		- portions of pleomorphic ademona are identifiable - see invasion, cellular pleomorphism, atypical mitoses	- wide resection - lymph node resection - may bee rad	- guarded - 30-50% cure rate	- rare - poss develop from long standing pleomorphic ademona (mass for years, then rapid growth)

Viral Infections of Oral Mucosa	Virus	Age	Site	Clinical Appearance	Histopath	Treatment	Other
Acute herpetic gingivostomatitis	HSV-1	- young				- supportive - antivirals if Dx early	- initial exposure to virus - may also cause pharyngotonsilitis - may trigger erythema multiform!
Infectious mononucleosis	EBV			- fatigue, fever, malaise, cervical lymphadenopathy, hepatosplenomegaly, rash, cough, palatal petechiae - oropharyngeal tonsillar enlargement, palatal petechiae, necrotizing ulcerative gingivitis			Dx: - clinical presentation - ↑ WBCs (lymphocytes + atypical lymp) - +ve Monospot test
Oral hairy leukoplakia	EBV						Strong assoc with some malignancies: - Burkitt's lymphoma - nasopharyngeal carcinoma - immune-suppression related lymphoma - Hodgkin's disease
Molluscum contagiosum	poxvirus			- oral lesions rare			
Hand foot and mouth disease	Enterovirus			- vesicles on oral mucosa - cutaneous vesicles			- virus also causes acute lymphonodular pharyngitis and herpangia
Oral Squamous papilloma	HPV (6 and 11)	- any age	- tongue, lips, soft palate	- common - benign - low virulence, exact mode of transmission is unknown - usually solitary - soft, painless, pedunculated exophytic lesion with finger like surface projections - white, red or normal colour	- cores of fibrovascular CT covered by SSKE - white due to keratin	- conservative excision	- DNA virus
Verruca vulgaris Common wart	HPV (2)	- children	- skin and mucous membranes - common on skin - uncommon in oral cavity but verm border, labial mucosa, ant tongue	- benign - contagious - often multiple - virus induces hyperplasia - painless white, pedunculated or sessile papule/nodule with papilliferous projections - rough "verruciform" surface	- papillary projections composed of CT cores covered by SSKE - rete ridges converge towards centre of lesion - prominent granular cells (keratohyaline granules) - koilocytic change in epi	Skin: - conser surg excision, liquid nitrogen, cryotherapy, topical keratinolytic agents Oral: - excision - 2/3 disappear spontaneously	- spreads by autoinoculation
Condyloma acuminatum Venereal wart	HPV (6 and 11)	- teens - young adults	- labial mucosa, soft palate, lingual frenum	- sessile, pink, well demarcated exophytic lesion with short blunted surface projections - often multiple	- proliferation of SSE with formation of papillary surface projections - uneven surface, less keratin (pinker) - see koilocytic change: raisionoid nucleus	- conserv surgical excision	- sexually transmitted - frequent co-infection w types 16/18
Multifocal epithelial hyperplasia Aka Heck's disease	HPV						

General Summary: Carcinoma of Oral Mucous Membrane + Lip

- 94% oral malignancies = SCC
- 5 yr survival rate = 60%
- Risk factors:
  - Older (>40 yo)
  - M>F 2:1
  - Any form of tobacco (80% oral cancer pts are smokers) -> related to pack yrs
  - Heavy alc consumption: synergism w smoking
  - Betel quid
  - Infections
    - Tertiary syphillis
    - HPV detected in 10% of cases
    - Only a risk factor for oropharyngeal carcinoma
  - Chronic disease
    - Iron deficiency anemia esp severe i.e. sideropenic *(Fe defic)* dysphagia (Plummer Vinson aka Paterson Kelly Syndrome)
      - Has mucosal webs
- Clinical presentation
  - Tongue 50%
  - FOM 35%
  - Gingiva/alv mucosa 5-10%
  - Buccal mucosa <5%
  - Hard palate rare
  - Oropharyngeal carcinomas include those on soft palate, tonsillar pillars and post 1/3 of tongue (base)
- Appearance:
  - Indurated (hard), non healing ulcer
  - Raised, rolled borders
  - *early SCC often not ulcerated (looks like leuk/erythoplakia)
  - Exophytic (going up) verrucous (wart like) mass
  - Erythematous, granular mass
    - Often looks like pyogenic granuloma or hyperplastic soft tissue in extraction side
  - Fungating ulcerated mass = obvi malignant
  - May see bone loss
- Evidence of invasion
  - Mobility of teeth

    - Limited tongue movement (hot potato mouth)
- Metastasis to lymph nodes
    - Ipsilateral then bilateral
    - Submental, subMd; jugulo-digastric; high-mid-low jugular nodes
    - Fixed nodes
- Absolute Dx = biopsy
    - Well differentiated: large tumour islands, central keratinization (pearls)
    - Moderately differentiated: vars of size of tumour islands, peripheral and central spinous-like cells, few/no keratin pearls
    - Poorly differentiated: small cords/islands of tumour, no evidence of differentiation into spinous cells, no kerat
    - **Verrucous carcinoma**: subtype of SCC that is very well differentiated
        - Stratified structure, minimal atypia, true malignancy but rarely metastasizes
        - Exophytic white portions: hyperkeratosis, papillary surface
        - Endophytic invasive portions: large mass pushing into underlying tissues, wide/long rete ridges
        - Often seen on Md ridge and buccal vestibule
- **Actinic chelitis** *NOT angular chelitis ;) chelitis means inflammation of lip*
    - Slow developing lip condition from excess sun
    - May undergo malignant transformation into SCC
    - Whole lip, thickening, loss of vermil border demarcation
    - Patchy white/red area, fissures, crusting
    - Biopsy: rule out carcinoma
        - See hyperkeratosis, hyperplasia, atrophy, epi dysplasia, basophilic degeneration of collaged (solar elastosis), chronic inflam infiltrate
- **SCC of the lip**
    - Crusted, indurated, nonhealing ulcer
    - White exophytic keratotic lesion
    - Histology: most are well differentiated (keratin pearls)
    - Tx: wedge resection, vermilionectomy (↑ risk paraesthesia)

General Summary: Diseases of blood

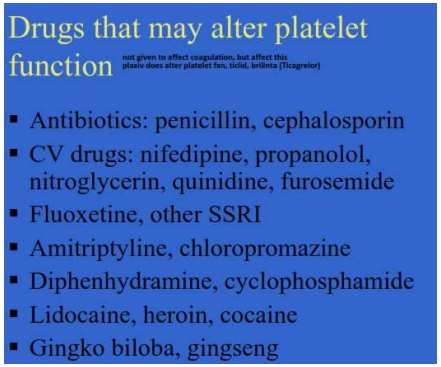
- Hemolytic anemia: ↑ RBC destruction
  - Sickle cell anemia
    - Hb mutation, codominant, can get sickle cell crisis during stress
    - Oral features: hair on end skull rx, reduced trabeculae in Md, pale/yellow mucosa, ↑ osteomyelitis, prolonges paraesthesia, pulpal necrosis (poss asymptomatic)
  - Thalassemia
    - Autosomal recessive, fewer RBCs
    - Alpha thalassemia: 4 presentations:
      - 1 defective genes = no disease
      - 2 defective genes = alpha thal trait (mild anemia)
      - 3 defective genes = Hb H disease (hemolytic anem + splenomegaly)
      - 4 defective genes = hydrops fetalis (lethal)
    - B thalassemia: 2 types
      - Minor: 1 defective gene, asymptomatic
      - Major (Cooley's anemia): 2 defective genes, transfusion dependent anemia, severe
        - Oral features: Large marrow spaces Mx, osteoporosis of Md, malocclusion, enlarged Md/Mx with sinus obliteration, obliterated pulp chambers
  - Valvular heart disease
- Anemia due to impaired cell production
  - 1. Aplastic anemia
    - RBC + WBC maturation problems, BM is acellular
    - Causes: idiopathic, chemical, viral (hepatitis, HIV), drugs, inherited = Faconi Anemia
    - Tendency to bruise and bleed -> gingival bleeding, frequent infectionsm tachycardia
  - 2. Iron deficiency anemia
    - Most common anemia
    - Oral : angular chelitis, atrophic glossitis (sooth tongue)
    - **Plummer-Vinson Syndrome**: rare condition, Fe deficiency with post cricoid esophageal strictures, high inc oral/esophageal carcinoma, F 30-50 yrs, same symptoms + burning oral mucosa
  - 3. Pernicious anemia
    - Poor vit B12 absorption or lack of intrinsic factor
    - Oral symptoms: burning of the tongue (50-60%), lips, buccal mucosa, mucosal erythema and atrophy
- Other blood disorders
  - 1. Neutropenia

        - Oral ulcerations lacking a erythematous periphery (also no pseudomembrane)
    - 2. Agranulocytosis
        - Deficiency of granulocytes, particularly neutrophils
        - Necrotizing deep ulcers of buccal mucosal, tongue and palate (NUG)
    - 3. Cyclic neutropenia
        - Rare periodic reduction in neutrophils, 21 d cycle
        - Severe perio bone loss, premature loss around deciduous teeth
    - 4. Polycythemia vera
        - ↑ mass of RBCs
        - Gingival hemorrhage
- Hemostasis/Bleeding disorders
    - Phases: Vascular (vasoconstr), Platelet (plug), Coagulation (cascade inv thrombin, fibrin), Fibrinolytic (dissolves clot)
    - Clinical Appearance of Bleeding Disorders

	Vascular or Platelet Disorders (pt on antiplatelets: immediate persistend bleeding)	Coagulation Disorders (pt on anticoagulants: delayed bleeding)
Bleeding from cuts	Persistent	minimal
Delayed bleeding	rare	common
Spontaneous gingival bleeding	Characteristic	Less likely
Petechiae (pinpoint red spots - do NOT blanch with pressure)	Characteristic	Less likely
Ecchymoses (larger, more like bruising)	Small, multiple	Large, solitary
Hematomas (large bruises)	Rare	Characteristic
Hemarthroses (bleeding into a joint)	Rare	Characteristic

- **<u>Vascular disorders</u>**: BV wall changes
    - Hereditary
        - Hereditary hemorrhagic telangectasia aka **Osler–Weber–Rendu disease**
        - Ehlers-Danlos disease
            - Abnormal type 3 collagen
    - Acquired
        - Vitamin C deficiency (Scurvy)
            - Spontaneous gingival bleeding and erythema
        - Meds: long term steroids
            - Thins CT, easier to break/bleed

- **Thrombocytopenic (platelet) disorders**
  - Can't clot bleed out
  - ↑ platelet destruction: immunologic, drug induced
  - ↓ platelet production: BM suppression ex by drugs, radiation, viral infections
  - ↑ platelet sequestration: splenomegaly (spleen enlargement)
  - **Bernard-Soulier Disease**
    - Rare, autosomal recessive
    - Platelets lack membrane GP IB that is a receptor for vWF
    - Severe bleeding, spontaneous epistaxis (nosebleed) and gingival bleed



- **Coagulation disorders**
  - These people will bleed out since they can't clot
  - Give DDAVP (desmopressin), pure factor replacements, anti-fibrinolytics
  - 1. Hemophilia A and B
    - A = VIII deficiency
    - B = IX deficiency
    - Spontaneous bleeding
  - Von Willebrand's Disease
    - Most common inherited bleeding disorder
    - Auto dominant
    - Mucosal bleeding/bruising
  - Vit K deficiency
    - Vit K dependent factors: II, VII, IX, X (2, 7, 9, 10)
    - May be due to broad spec antibiotic tx or biliary tract obstruction
  - Liver disease
    - Affects BOTH coagulation and platelet phases
    - Vit K injfections, platelet transfusion?

Fungal Infections

- Candida albicans: yeast and hyphal (invasive) forms
- Classification
    - 1. Pseudomembranous
        - Classic thrush
        - Seen in:
            - Infants
            - Post antibiotics
        - Soft plaques that wipe off (red beneath)
        - Common sites: buccal mucosa, tongue, palate
        - Symptoms: none, tenderness, burning, dysphagia
    - 2. Atrophic (erythematous)
        - After loss of plaques
        - 65% in elderly (due to dentures, underneath)
        - May get mid tongue (median rhomboid glossitis)
        - May get a kissing lesion (from an infected area in the mouth touching another area)
    - 3. Hyperplastic
        - White plaques do NOT rub off
        - Often asymptomatic
        - Anterior buccal mucosa (post to corners of mouth) or dorsum of tongue
        - Resembles speckled leukoplakia
    - 4. Angular chelitis
        - Overclosed pts
        - Combo with Staph aureus
    - 5. Mucocutaneous candidiasis
        - VERY rare
        - Seen in first years of life
        - Mouth, nails, skin
        - 50% pts have endocrinopathy: hypoparathyroidism, Addison's disease
- Dx
    - Clninical appearance
    - Smear looks for pseudohyphae (PAS stain)
    - Don't culture since can grow from most ppls
    - Only biopsy hyperplastic type
- Tx

- Stop abs, improve OH/salivary flow, remove dentures at night, nystatin rinse

- Granulomatous disease
  - Predom cell is activated macrophage with epithelioid appearance
  - Granulomas: foreign body and immune granulomas
  - 1. Infectious
    - A. Bacteria:
      - I. Mycobacterium tuberculosis -> TB or *M bovis -> scrofula*
      - Oral infections = rare (may have ulcers or granular areas on tongue, mucobuccal fold and gingiva)
      - Tx TB:
        - Active: isoniazid, rifampin, pyrazinamide
        - Latent: isoniazid + vit B6
      - II. Mycobacterium leprae
        - Tuberculoid/paucibacillary leprosy
          - Localized skin lesions, elevated margins, hypopigmentation, nerves encased in granulomas
        - Lepromatous/multibacillary leprosy
          - Diffuse lesions, lose hair/sweat glands
          - Oral lesions on tongue, palate; red-yellow papules that enlarge and ulcerate
      - III. Treponema pallidum (Syphillis)
        - Only tertiary is granulomatous
        - 1ary: 3 weeks see a chancre
        - 2ary: diffuse rash at 4-10 weeks, white oral lesions, grey areas (mucosal necrosis)
        - 3ary: organ disease
        - Dx: darkfield microscopy
        - Tx: Penicillin
        - Congenital: mullberry molars, Hutchinson's incisor
    - C. Fungal
      - i. Histoplasmosis
        - mississippi, soil, mild has no tx
  - 2. Idiopathic
  - i. Sarcoidosis
    - 20-40 yo, african amer
    - Gran inflammation of the skin as purple, indurated lesions on face, lips and ears; clinical enlargement of the sal glands, xerostomia
    - May see asteroid bodies in giant cells, Schaumann's body: concentric calcification, ↑ ACE
    - Resolve spontan 60% of time

- II. Orofacial granulomatosis
  - Dx of exclusion
  - Chelitis granulomatosis (lip swelling)
  - Melkersson-Rosenthal syndrome: 3 things: gran enlargement of lips, fissured tongue, face paralysis (may see other oral lesions)
- III. Chron's diease
  - Mucosal linear ulcers esp in buccal mucosa
- IV. Wegner's granulomatosis
  - Triad of granulomas: upper respiratory tract, lung, kidneys
  - "strawberry gingiva" red granular gingival lesions