### Descriptions

- **Size**
- **Color**: Specific Color, Blanching, Uniform vs Varigate
- **Other** (if important)
  - Margins: Well/Ill Defined
  - Consistency: hard/soft, solid/fluctuant
  - Shape: round, oval, polygonal, annular, serpiginous
  - Arrangement: grouped (confluent, herpetiform, arciform, annular, reticulated aka net like, linear, serpiginous) vs disseminated
  - Distribution: extent (generalized, isolated, localized, regional), pattern (symmetric, exposed areas, sites of pressure, intertriginous, random, dermatomal)
  - Number: Single/Multiple
  - Mobility: Fixed/Mobile
  - Estimate of Depth: Epidermis → Dermis → Subcutaneous
  - Temp: NI/Hot/Cold

- **Primary Lesion**
  - Solid
    - Flat: **Macule** (<0.5-1cm) vs **Patch** (>0.5-1cm)
      - Diameter > Elevation: **Plaque** (any size)
      - Elevation > Diameter: **Papule** (<0.5-1cm) vs **Nodule** (>0.5-1cm)
  - Liquid
    - Superficial: **Vesicle** aka **Small Blister** (<0.5cm, fluid filled) vs **Bulla** aka **Large Blister** (>0.5cm, fluid filled) vs **Pustule** (pus filled)
    - Deep: **cyst** (fluid filled) vs **abscess** (pus filled)
  - Blood
    - **Petechiae** (non-blanching)
    - **Purpura** (non-blanching)
    - **Telangiectasia** (blanching)
  - Urticaria/Wheals/Wells/Hives (fluid, well demarcated b/c superficial, rapidly changing in shape, localized or generalized to trunk/extremities, pluralic) vs **Angioedema** (severe, poorly demarcated b/c deeper, slowly changing in shape, localized to mainly lip, face, hands, feet, penis, scrotum). Tx: two high dose H1B, H2B, Epi, IVIG, Plasmapheresis. **Cyclosporine Erosion** (superficial loss into epidermis w/o scar formation) vs **Ulcer** (deeper loss into dermis w/ scar formation) — Pressure ulcer over bony prominences, RFs: incontinence, poor nutrition, elderly, immobile, neuro deficits, Tx: change positions Q2hrs, pressure reduction beds, balanced nutrition, keep skin clean/dry, plastic surgery (debridement then flaps/grafts)
    - Stage 1 (skin intact there is evidence of inflammation, etc)
    - Stage 2 (partial thickness skin loss)
    - Stage 3 (full thickness skin extending to subcutaneous fat)
    - Stage 4 (exposed muscle/bone)

- **Secondary Lesion**
  - Scale (abnormal accumulation of dead exfoliating epidermal cells)
  - Crust/Scab (dried serum, blood, purulent exudates) thin/friable/delicate vs thick/adherent/tough
  - Scar (soft/atrophic vs hard/hypertrophied vs keloid)
  - Excoriation (2/2 scratching)
  - Atrophy (thinning of epidermis) vs **Lichenification** (thickening of epidermis)
  - Ichthyosis (fish like scales w/ xerosis aka dry skin) DDx: rare hereditary disorders varying from mild to life-threatening diseases, eg. Ichthyosis Vulgaris, X-Linked Ichthyosis, Lamellar Ichthyosis, Epoidermolytic hyperkeratosis, etc
  - Surface: smooth, dome, cone, flat, vegetative aka warty
  - Livedo Reticularis (seen in vascular diseases, viscosity diseases and certain drugs (amantadine, quinine, etc)

- **Anatomy & Histology**
  - Skin
    - Epidermis: stratum basalis/spinosa/granulosa/corneum
  - Glands
    - Eccrine Sweat Gland (whole body, main sweat gland, coiled tube in dermis directly draining into skin)
    - Apocrine Sweat Gland (just axilla/groin, minor sweat gland, coiled tube in dermis draining into hair follicle)
    - Sebaceous Gland (just face/neck/back, sebum gland, gland in dermis that drains into hair follicle)
  - Hair
    - Vellus (fine nonpigmented hair aka peach fuzz that covers entire body and is not affected by hormones, hair that is similar to this on fetus is called lanugo) vs Intermediate (in b/t) vs Terminal (thick pigmented hair on scalp/beard/axilla/pubic area and is affected by hormones)
- Anagen (normal active growth) vs Catagen (in b/t anagen and telogen) vs Telogen (resting phase) vs Exogen (hair shedding)
- DHT (causes growth of terminal hair but also androgenetic alopecia) vs Testosterone (causes growth of axillary/pubic hair)
- Evaluation: hair pull (normally w/ gentle pull 3-5 hairs can be dislodged anymore is pathologic), trichogram (pull 50 hairs and determine the proportion of phases), scalp biopsy

### Historical Facts
- Onset & Evolution
- Alleviating/Exacerbating Factors
- Associated Sx
  - Pain
  - Pruritus

### Drug Reactions
- Drug eruptions can look like any dermatologic disease therefore with any acute rash think of drug reaction but the most common are: (1) Exanthematous/Morbilliform Reaction = multiple diffuse confluent <1cm pruritic erythematous macules/papules, 1st 2-3wks of Tx, begins chest/chest and then generalizes over 1-2d (Beta-Lactams, Carbamazepine, Allopurinol, Sulfas, NSAIDs, etc but can also be due to virus, bacteria, rickettsial, parasitic infections), (2) Urticaria/Angiodema to full Systemic Anaphylaxis (Contrast, Beta-Lactams, Sulfas, Amio, Immunologics, Cytotoxics, ACE-1, CBs, Opiates, etc), (3) Pigmentation (Amio, Antimalarials, Minocycline, Cytotoxics, etc), (4) EM/SJS/TEN (Sulfa, Allopurinol, NSAIDs) (5) Photosensitivity (tetracycline, etc)

### Tx
- Hold all nonessential meds
- Check which meds were started recently
- Note improvement after withdrawal
- Consider drug challenge w/ readministration
- Start Complete Histamine Blockade Then if no improvement Steroids and if still no improvement then epi and derm consult
- Tx pruritus (above)

### ICU Rash
- Drug Reaction, Serum Sickness, Sweet’s Syndrome, Viral, Bacterial, EM/SJS/TEN, etc
- Dx: Tzanck Test (unroof vesicle, scrape base, smear on slide, air dry, stain w/ Wright’s stain, examine for acantholytic cells and multinucleated giant cell), Viral Cx/POR, Bacterial Cx/GS, BX

### Tests/Procedure
- Dermoscopy (magnifying glass w/ light source)
- Diascopy (pressing glass slide against red lesion to see if blanchable (capillary dilatation) vs non-blanchable (extravasation of blood))
- GS & Cx (bacterial), KOH Prep, Wood’s Lamp (360nm black UV light, exposes fluorescent pigments, used in seeing erythrasma), Sambourd Cx (fungi), Tzanck Prep (viral)
- If lesion is concerning for a BCC/SCC then do a Shave Bx: iodine, anesthetic w/ epi, scalpel and cut out lesion, silver nitrate or suture
- If lesion is concerning for melanoma or some weird dermatologic condition then do a Skin Bx: 4mm Punch Bx Kit, sterilize w/ just an alcohol swab, 1% lidocaine, 8x, cut base w/ scissors, apply pressure, close w/ suture, call dermatopathologist about how to send specimen in cap (usually sent soaked in saline in specimen cup)
- If lesion is not concerning for cancer at all then just Freeze: place narrow end of an otoscope cap over lesion then spray liquid nitrogen (Verruca-Freeze) into cap a few times, tell pt that it will turn into a blister over a course of 5d and then it will fall off, just keep area clean
- Fusiform Excision: fusiform excision lengthening lesion 3:1 which eliminates “dog-ears” w/ long axis along Langer’s lines

### Medicines
- General Skin Friendly Products
  - Soaps (Dove, Aveeno, Oil of Olay)
  - Lotion (Eucerin, Aveeno, Aquaphor, Burts Bees)
  - Detergents (Arm & Hammer Perfume and Dye Free) NB use double rinse cycles
- Topical Steroids
  - General Rx: “XXX” cream apply to affected area BID dispense as increments of 15g w/ 15g = small area vs 120g = very large area, take x1wk but if longer than M-F and weekends off
  - Sx: thin lightened skin w/ telengectasia and stria
  - NB use creams b/c best properties overall when compared to ointments, gels, lotions, etc, don’t “occlude” creams like with gloves, band aids, etc b/c increases potency, never use combo antifungals and steroids always give separate Rx, the more chronic the condition the longer the duration of steroids the weaker the steroid you should use
  - Face/Intertriginous (more sensitive = use milder agents)
    - Mild: Cortaid (hydrocortisone 0.05%)
    - Mod: Cutivate (fluticasone 0.05%)
    - Strong: Dermatop (prednicarbate 0.1%)
  - Body (less sensitive = can use stronger agents)
- Mild: Kenalog (triamcinalone 0.5%)
- Mod: Diprolene AF (betamethasone 0.05%)
- Strong: Ultravate (halobetasol 0.05%)

- Other
  - Topical immunomodulators (tacrolimus and pimecrolimus) where introduced for a variety of conditions
  - Acid Peels: Melanage Bleaching Cream, Salicylic/Glycolic/Trichloroacetic Acid
  - Biotin (Appearex) for nail growth/health

**Hair**

- Excessive Hair Growth
  - **Hirsutism** (occurs in women at sites where hair is under androgen control (face, chest, areola, line alba, lower back, buttock, abdomen, genitalia, inner thigh) 2/2 presence of androgens (androgen secreting tumor, functional androgen excess, medication induced) which convert vellus hairs to terminal hairs, Tx: androgen suppression w/ Aldactone/OCPs/H2B esp Cimetidine/Stereoids)
  - **Hypertrichosis** (occurs in anyone at any sites where there is an increase in hair density and/or length beyond accepted limits of normal for age/race/sex, 2/2 malignancy***, drugs (phenytoin/cyclosporine/stereoids etc), porphyria, POEMS syndrome, dermatomyositis, hypoTH, acromyria, malabsorption syndromes, various CNS diseases)

- Alopecia (due to shedding of hair aka **effluvium/deffluvium**, (always check RPR, ANA, KOH Prep, TSH, Iron, med list, recent PMHx)
  - Non-Cicatricial Alopecia (no underlying scarring/inflammation/atrophy)
    - **Diffuse**
      - Abnormality w/ Follicle Production
      - Abnormality w/ Hair Shaft
      - Abnormality w/ Cycling
        - **Telogen Effluvium** (Metabolic: post-partum, hypo/hyperthyroidism, perimenopausal, Nutritional: Biotin/zinc/iron/EFA deficiency, caloric/protein deprivation, Drugs: chemo, Enalapril, BB, AC, IFN, Li, Valproic Acid, OCPs, Retinoids, Cimetidine, Physical/Emotional Stress: surgery, systemic illness, not permanent, you can pull out hair easily, inciting event can occur up to 3mo before hairloss, usually not full alopecia but just thinning, hair changes [transverse grooves], Tx: reverse underlying cause and when you do hair should regrow should return to normal in 12mo)
        - **Anagen Effluvium** (kind of a more severe form of TE above, hair usually breaks off rather than falling out, 2/2 radiation therapy, chemo, severe protein deficiency, various poisons, Tx as above)
    - **Alopecia Areata** (refer below)
  - **Localized**
    - Abnormality w/ Follicle Production
    - **Androgenic Alopecia aka Male Pattern Baldness or Post-Menopausal Thinning** (2/2 DHT and genetics from both parents, Tx Men: oral finasteride which inhibits Salph-Reductase which converts testosterone to DHT or topical minoxidil or hair transplant, Tx Women: antiandrogens like Aldactone, etc)
    - Abnormality w/ Hair Shaft
      - **Trichotillomania** (psychological pulling of hair)
      - **Traction Alopecia** (from ponytails or chemicals)
      - **Tinea Capitis** (refer)
    - Abnormality w/ Cycling
      - **Alopecia Areata** (round/oval well demarcated area of hair loss w/ normal scalp over weeks w/ regrowth in 80% of pts w/ 1/3 getting complete recover in 1yr but recurrence is common, seen in young adults, 1% of population will have at least one episode in their lifetime, T-cell autoimmune condition therefore associated w/ other autoimmune conditions, atopy and Down Syndrome, accompanied by nail changes (pitting, ridging, separation from matrix, etc), called AA-Totalis/Universalis if entire scalp/body, Tx: no Tx but topical immunomodulators are emerging)
      - **Syphilis**
    - Cicatricial Alopecia (underlying scarring/inflammation/atrophy, can determine type based on pattern of inflammatory cells on scalp biopsy, Tx: topical steroids) **Chronic Cutaneous Discoid Lupus Erythematosus (CCLE), Lichen Planopilaris (LPP), Classic Psuedopelade, Central Centrifugal Scarring Aloppecia (CCSA), Alopecia Mucinosa, Keratosis Folicularis Spinulosa Decalvans (KFSD), Folliculitis Decalvans, Dissecting Folliculitis**
Nails

- Paronychia (infection of skin fold over distal nail, Tx: abx) leading to Felon (infection of pulp around distal phalanx, Tx: surgery) = Staph
- Onychomycoses aka Tinea Ungium (refer below but also discoloration, cracking, etc, Dx: nail sample via clipping or scraping then KOH prep then Cx, Tx: refer to ID) = Dermatophytes
- Clubbing (hypertrophy of digital pulp) = congenital, familial, CV/lung/GI dz
- Pitting (small pits) = psoriasis, alopecia areata, eczema
- Splinter Hemorrhage (tiny red streaks) = endocarditis vs Subungual Hemorrhage (big bruise) = trauma
- Onycholysis (separation of nail plate from bed w/ collection of debris that then can become secondarily infected) = onychomycosis, trauma, psoriasis
- Beau’s Lines (transverse grooves) = systemic disease, psoriasis (NB longitudinal grooves usually 2/2 OCD picking)
- Koilonychia (spooning) = physiologic in young, trauma in old, Plummer Vinson Syndrome
- Melanonychia (hyperpigmentation) = trauma, nevus, melanoma, ziduvidine
- Leukonychia (hypopigmentation) = inherited, psoriasis, trauma, onychomycosis, cirrhosis = Terry’s Nails, uremia = Lindsay’s Nails, hypoalbuminemia = Muehrcke’s Nails
- Onychauxis (thickening w/o hooking sometimes w/ subungual hyperkeratosis) = psoriasis, onychomycosis
- Onychia (inflammation of matrix resulting in nail shedding) = ?
- Onycholysis (breaking) = ?
- Onychocryptosis (ingrowing) = trauma
- Onychophyrosis (thickening w/ hooking) = trauma
- Onychomalacia (softening)
- Onychorrhexis (longitudinal ridging) = age, psoriasis, lichen planus
- Onychoschizia (splitting or lamination) = lichen planus
- Onychotillomania (compulsive picking/tearing)
- Elkonysis (hole) = psoriasis
- Trachonychia (roughness) = alopecia areata
- Other: Yellow Nail Syndrome

Palms/Soles

- Tylosis aka Palmar/Plantar Keratoderma (esophageal cancer)
- contact dermatitis ESP if dorsal side
- psoriasis
- pityriasis rubra pilaris w/ plantar erythema
- lichen planus
- keratoderma plantaris w/ white thickened sole
- callus over PIP joint
- chilblains which is kind of like Raynaud’s
- black heel aka talon noir which is a black mark on back of heel 2/2 trauma from shoe

Pigmentation Problem

<table>
<thead>
<tr>
<th>Melanocytes</th>
<th>Lentigo</th>
<th>Vitiligo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melanin</td>
<td>Melasma</td>
<td>Albinism</td>
</tr>
</tbody>
</table>

- Hypopigmentation
  - Vitiligo (autoimmune destruction of melanocytes by anti-melanocyte antibodies resulting in loss of pigmentation, associated w/ Addison’s/SLE/T1DM/HypoTH/Pernicious-Anemia and Chororetinitis/Iritis/Uveitis, apigmented macules as on face/axilla/hands/perineum/knees/feet, mainly seen in dark skinned people, can begin at any age but most at 20yo and usually after trauma/stress/illness w/ rapid onset and then it stabilizes, Tx: sunscreen to prevent tanning of non-vitiligo areas and to prevent cancer of vitiligo areas, cosmetic, partial repigmentation w/ topical steroids/psoralens)
  - Albinism (loss of ability of melanocytes to make melanin affecting skin/hair/iris (translucent not blue) 2/2 mutation in enzymes, tyrosinase, present at birth, pts also have strabismus/nystagmus b/c the iris lacks pigmented and thus light is not focused well onto fovea resulting in impaired CN-III development, Tx: sunscreen, see an ophthalmologist for the vision problems, consider to systemic beta-carotene to add color)
  - Post Inflammatory Hypopigmentation
- **Tinea/Pityriasis Versicolor** (refer to fungal infection below)
- **Other:** Tuberculoid Leprosy, Ash-Leaf Spots of Tuberous Sclerosis, Morphea of Systemic Sclerosis

### Hyperpigmentation
- **Lentigo/Nevi/Melanoma** (refer below, varying degrees of increase in melanocyte number)
- **Melasma aka Chloasma aka “Mask of Pregnancy”** (increase melanin production after exposure to sunlight when pt is pregnant or taking OCPs but many cases are idiopathic, symmetric macular hyperpigmentation on face, Tx: topical hydroquinone/azelaic-acid/flucinolone, sunblock)
- **Post Inflammatory Hyperpigmentation**
- **Acanthosis Nigricans** (S/S: Dirty appearing velvety thickened hyperpigmentation in flexural areas (neck, axillae, breasts, groin) w/ accentuated skin lines, Etiology: endocrine (DM, Cushings/Addisons, obesity, hyper-androgen state, hypothyroidism), drugs (niacin, stilbestrol, glucocorticoids, OCPs), malignancy (GI, lung, ovarian adenocarcinoma consider in pts w/ severe AN, tripe hands aka accentuation of palmar ridges, B-Sx, oral mucosa AN w/ furrows), hereditary, Tx: treat underlying cause)
- **Other:** Metabolic (Hemochromatosis, Renal Failure, Addison’s), Drugs (minocycline, amio, 5-FU), Genetic (Mucosal Hyper esp of Lips in Peutz-Jegher’s Syndrome, Café au Lait Spots in Von Recklinghausen’s aka Neurofibromatosis)

<table>
<thead>
<tr>
<th>Number</th>
<th>Benign</th>
<th>Pre-Malignant</th>
<th>Malignant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Childhood</td>
<td>Adolescence</td>
<td>Adulthood</td>
</tr>
<tr>
<td>ABCDEs</td>
<td>None</td>
<td>Some</td>
<td>Many</td>
</tr>
<tr>
<td>Evolution</td>
<td>Involution by 60yo</td>
<td>No Involution</td>
<td>No Involution</td>
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</tbody>
</table>

### Benign
- **Lentigo aka “Solar/Liver/Age Spots”** (tan/brown macule w/ irregular borders in sun-exposed areas, increase transfer of melanin to keratocytes but not increase in melanocytes like nevi, not round/oval but more irregular, never raised, lighter in color vs nevi)

- **Common/Acquired Nevus aka Mole** (sun exposed areas, increase in number of melanocytes, develop during childhood then grow (followed by involution/fibrosis w/ most disappearing by 60yo, classified according to state of evolution (below), as the melanocytes penetrate they lose their ability to make melanin, <1cm, benign ABCDEs, no Tx)
  - **Junctional** (early, at DE jxn, macules, very pigmented)
  - **Compound** (later, invade papillary dermis, papules, mildly pigmented)
  - **Dermal** (late, invade ? dermis, nodules, non pigmented)

- **Other:** Halo Nevus (mole surrounded by a halo of depigmentation 2/2 lymphocytic infiltration that results in death of melanocytes), Blue Nevus (scary looking, firm dark-blue papule/nodule, emerge during childhood, ectopic accumulation of melanocytes in dermis not from DE jxn but from melanoblasts that became arrested during migration from neural crests into skin, often on dorsum of hand/feet, buttock, lower back, often 8x b/c looks like melanoma), Spitz Nevus (during childhood, pink/tan dome-shaped smooth papule on head/neck, often have a history of recent rapid growth), Nevus Spilus (collection of multiple small dark-brown macules speckled across a mildly pigmented light brown patch on trunk), Mongolian Spot (present at birth, seen in Asians, very large blue patch in lumbosacral region, often confused for bruise), Nevus of Ota/Ito (present at birth, Asians, large mottled dusky blue/brown patch in V1-2 region)

### Pre-Malignant
- **Atypical/Dysplastic/Clark’s Nevus** (arise later in life and do not involute like acquired NMN, central brown (usually not as dark as moles) >6mm (larger than normal nevi) macule surrounded by an irregular pink rim (more irregular pigmentation/borders) mainly on trunk, usually fewer in number, excision, precursor AND marker (general 1.2%, if you have atypical nevi 18%) for melanoma)
- **Other:** Congenital (vary in size from small to very large, usually distort skin surface, single lesions, 5% risk of melanoma w/ most developing by 8yo, worst ones are the giant hairy nevi on head/pelvis, Tx: excision)
• Malignant Melanoma
  ▪ Cell: melanocytes
  ▪ Epidemiology: 8th most common cancer, increasing faster than any other cancer, usually ~40yo
  ▪ RFs: Fitzpatrick I/IV skin (fair skin, light red hair, freckles, light eye color), exposure to sunlight (UVA/B) especially infrequent sunburns not constant tanning, high altitude and higher latitude, males (higher risk and worse prognosis), prior melanoma (4%), AA (obviously lower risk but worse prognosis), gene mutation (CDKN2a), Fxh, genetic syndromes (Dysplastic Nevus Syndrome, Xeroderma Pigmentosa)
  ▪ NB even acquired new become a concern when multiple (>50) and large (>5mm)
  ▪ Can be de novo (70%) or arising from pre-malignant lesions (above) (30%)
  ▪ ABCDE
    ▪ A (asymmetry)
    ▪ B (border is irregular and blurred with pigment appearing as if it is spreading thru skin)
    ▪ C (color is not uniform throughout and has weird colors like blue and various blacks/browns)
    ▪ D (diameter is >6mm or a pencil eraser head)
    ▪ E (elevation and evolves over time)
    ▪ Other: (bleeds, itches, etc)
  ▪ Mets: lung, brain, GI viscera, bone
    ▪ NB melanoma of unknown primary represents 3% of melanomas
  ▪ Types (Fe = back/LE vs M = back/UE)
    ▪ Superficial Spreading (65%) often develops from a pre-malignant lesion, grows radially along skin followed by vertical growth therefore less aggressive, usually a more flat lesion w/ color variegation and irregular borders
    ▪ Nodular (20%) does not develop from a pre-malignant lesion, grows vertically therefore much more aggressive, grows fast, no association w/ UV exposure, usually a elevated nodular lesion w/ single color and round border
    ▪ Lentigo Maligna (10%) seen in elderly, least aggressive type, slow growing, only found in sun exposed areas, F<70yo
    ▪ Acral-Lentiginous (5%) found on palms/soles/nailbeds, seen in dark skinned people esp AA/Asians/Hispanics where it comprises 50% of melanomas, NB melanonychia is a benign subungual linear black line, seen in elderly, can be confused for subungual hematoma (in hematoma the entire lesion moves forward unlike melanoma), often large in diameter
    ▪ Ocular/Mucosal (rare) similar to Acral-Lentiginous
  ▪ IHC Markers: S-100 (more sensitive less specific) and HMB-45 (less sensitive more specific)
  ▪ Serum Markers: LDH, S-CysteinylDopa, Melanoma Inhibiting Activity (MIA)
  ▪ Dx: Excisional Bi (best) vs Incisional Bi (if low suspicion, large lesion (>1.5cm), located on disfiguring area of body, etc) NB don’t do Shave Bi Freeze Cauterize
  ▪ Staging (all have initial radial growth followed by vertical growth)
    ▪ TNM Stage w/ Breslow Thickness (mm) and Presence of Ulceration (present worse than not-present) NB if <0.76mm then 8yr survival is >93%  
    ▪ Other: Clark’s Level (histologic layer), Anatomic Location (trunk worse than extremities), Sex (male worse than female)
      ▪ Clark’s Level
        ▪ Level 1: intraepidermal
        ▪ Level 2: papillary dermis
        ▪ Level 3: upper epidermis
        ▪ Level 4: reticular dermis
        ▪ Level 5: subcutaneous fat
  ▪ Screening: no evidence by the USPSTF
  ▪ Prevention: sun-screen and protective eye wear
  ▪ Surveillance: H&P Q3mo x2yrs then Q6mo x3yrs then Qyr, check LFTs/CXR, check for incomplete excision w/ local recurrence

<table>
<thead>
<tr>
<th>Stage % metastasis</th>
<th>Ulceration</th>
<th>T (Breslow Deption)</th>
<th>N</th>
<th>M</th>
<th>Syr Survival</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>1 (&lt;1mm)</td>
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<td>0</td>
<td>95%</td>
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<tr>
<td>15%</td>
<td>B</td>
<td>+</td>
<td>2 (1-2mm)</td>
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<td>0</td>
<td>90%</td>
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<tr>
<td>II</td>
<td>A</td>
<td>+/-</td>
<td>2/3 (2-4mm)</td>
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<td>78%</td>
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<td>B</td>
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<td>3/4 (&gt;4mm)</td>
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<td>0</td>
<td>65%</td>
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<tr>
<td></td>
<td>C</td>
<td>+/-</td>
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<td>0</td>
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<tr>
<td>III</td>
<td>A</td>
<td>-</td>
<td>#</td>
<td>1-2 (1-3 LNs)</td>
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<td>67%</td>
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<tr>
<td></td>
<td>B</td>
<td>+/-</td>
<td>#</td>
<td>1-2 / Satellitosis</td>
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<td>53%</td>
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<tr>
<td></td>
<td>C</td>
<td>-</td>
<td>#</td>
<td>3 (&gt;4 LNs) or Satellitosis</td>
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<td>26%</td>
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### IV

<table>
<thead>
<tr>
<th>1a</th>
<th>#</th>
<th>#</th>
<th>#</th>
<th>Skin Subcut Nodules Distant LNs</th>
<th>19% (usually mean survival is 6mo)</th>
<th>Resection of distant mets only for palliation or with curative intent if there is limited number of foci</th>
</tr>
</thead>
<tbody>
<tr>
<td>1b</td>
<td>#</td>
<td>#</td>
<td>#</td>
<td>Liver SI</td>
<td>Lung</td>
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<tr>
<td>1c</td>
<td>#</td>
<td>#</td>
<td>#</td>
<td>Liver SI Brain Bone</td>
<td>9%</td>
<td></td>
</tr>
</tbody>
</table>

### Vascular Lesions

- **Tumors**
  - Benign
  - Hemangioma
    - Pathophysiology: benign proliferation of endothelial cells
    - Epidemiology: 2% of newborns and 10% at 1 year olds, F>M, White>Black, Preterm
    - 5/5/0: 3mo not formed yet (solitary, soft, compressible but non-blanchable raised nodule/plaque (red, superficial) or nodule (blue, deep) on head/neck 50%, trunk 25%, other 25%) → 3mo-1yr rapid growth phase (plaque rises, darkens in color, and increases in size > growth of child) → 1-10yr slow involution phase (flattens, lightens in color central to peripheral even turning white_gray centrally, decreases in size)
      - 100% resolution at 10yr, 90% resolution at 9yr..., 80% of kids will have perfectly normal skin most of the rest will have minimal signs such as discoloration, scarring, laxity, telangiectasia, etc therefore don’t say that the lesion will “go away”
    - Prognostic Indicators (NB not size of lesion)
      - Good: Involution b/f 4yo, located anywhere except below
      - Bad: involution after 4yo, parotid, lip, nose, anogenital
    - Complications
      - Ulcerations (most common)
      - Infection
      - Hemorrhage
      - GIB
      - Obstruct organs during proliferative phase (periocular = seeing problems, airway = breathing problems, auditory canal = hearing problem, mouth = eating problems, spine = tethered cord, genitourinary = imperforate anus)

- Hemangioma
  - Pathophysiology: benign proliferation of endothelial cells
  - Epidemiology: 2% of newborns and 10% at 1 year olds, F>M, White>Black, Preterm
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- “Cyrano Nose” (big bulb-like lesion on nose tip, hard to surgically correct when big therefore remove early on)
- “Hemangiomatosis” (multiple hemangiomas over skin, in liver and GI tract, can cause high output CHF, anemia, HSM, mortality 50%)
- “PHACE” Syndrome (Posterior fossa brain malformations, Hemangiomas of the face, Arterial anomalies in the brain, Coarctation of aorta, Eye problems, Other: sternal clefting)
- “Von Hippel-Lindau Syndrome” (Hemangiomas of the brain and eye, Cysts of the kidney, pancreas, adrenals, liver, Renal Cell Carcinoma (primary cause of death)
- “Kasabach-Merritt Syndrome” (thrombocytopenia + hemangiomas)

- GI: found in SI/LI but ones in rectum are often large called “cavernous hemangioma of the rectum”, sometimes they can occur diffusely across GI tract called “hemangiomatosis”, single or multiple red/purple/blue 2-20mm nodular lesions, usually a slow GIB, Tx: endoscopic Tx but most require surgery b/c large and multiple

- Dx: MRA (determine size/depth of vascular anomaly)
- Tx
  - NO Complications: no treatment therefore just reassure parents that the lesion will most likely involute using the statistics above
  - YES Complications: 1st: Systemic/Intralesional Corticosteroids, 2nd: Interferon alpha-2a, Other: XRT, Embolization, Laser, Excision, Poor Results: Cryotherapy, Sclerosing Agents

- Pyogenic Granuloma (solitary red/brown nodule on hands/feet/mouth w/ collar of thickened stratum at base, develops after mild trauma, bleeds easily, Tx w/ surgical excision)

- Glomus Tumor (tumor of the glomus body which is made of specialized smooth muscle cells (glomus cells) that surround the Saccuett-Hoyer Canal that connects arteriole and venule and when the glomus cells relax an AV shunt is created, glomus bodies are found in finger pads, nails beds, volar aspect of hand, ear skin, center of face, it is an exquisitely tender nodule w/ paroxysms of painful attacks during cold exposure, Tx w/ excision)

- Malignant
  - Angiosarcoma (malignant vascular tumor, variable in presentation, usually on scalp/forehead or in areas of lymphedema esp post-mastectomy/radiation, Tx is surgery, 10% survival at 5yrs)

- Vascular Malformations
  - Classification: Fast-Flow (arterial, Pulsating, warm, thrill, Steal phenomena may result) vs Slow-Flow (venous)
  - Dx: U/S to determine flow then Angiography, MRI, MRA to map out
  - Tx
- **Capillary Malformations**
  - **Port Wine Stain** (irregularly shaped violaceous plaque that follows dermatomes and is unilateral usually along the trigeminal nerve distribution, present at birth and never disappears, it is usually confined to skin but can involve other structures [Sturge-Weber w/ CNS Problems: ipsilateral leptomeningeal vascular malformations, seizures, MR and Eye Problems: visual field defects, glaucoma], called “Angle Kiss” if on front of head or “Stork Bite” if on back of head)

- **Spider Angioma aka Telangiectasia**
  - Focal network of blanching dilated capillaries that radiate from a central arteriole that may pulsate usually on face/forearms/hands
  - Capillary angiogenesis 2/1 vasculactive factors produced during anoxia, estrogen hormones (pregnant females or cirrhotics), chemicals, infection or in syndromes (below)
    - **Ataxia-Telangiectasia**
    - **Osler-Weber-Rendu Syndrome aka Hereditary Hemorrhagic Telangiectasia**
  - AD mutation (+/FHx in 80% of pts) of various proteins (esp endoglin, activin receptor-like kinase, etc) involved in angiogenesis resulting in telangiectasias of skin (esp hand/fingers/nails/nose/lips), mucosa (conjunctiva and pharynx resulting in epistaxis - usually the presenting sx during first few years of life) and AVMs in GI organs (mainly LGI esp stomach/SI but sometimes LGI w/ GIB and also liver (8-30%) w/ arterioportal shunts, arteriosystemic shunts, telangiectasias, vascular masses, in general screening is not needed b/c most remain asymptomatic and do not need Tx (embolization to transplantation) but if symptomatic then they p/w high CHF and cholestasis from ischemia to biliary tree from arteriovenous shunt, portal HTN from arterioportal shunt, HE from portosystemic shunt,), other organs (brain, lung, etc)
  - Tx: if a discrete lesion is found direct Tx is done but in general there are so many lesions that you can’t therefore some Tx w/ hormones, do regular endoscopy b/t episodes to Tx non bleeding lesions that are larger and likely to bleed
    - Tx: usually nothing but if bothersome then microsclerotherapy with polidocanol, EtOH, hypertonic saline or photothermolysis with laser

- **Cherry Angioma** (common, flat round cherry red macules mainly on trunk, begin to emerge at 30yo and increase in number as you get older such that every elderly person has some, not treated)
Venous Malformations

- Blubber Rubber Bleb Nevus Syndrome (venous malformations on skin/eyes/lungs/liver/spleen/heart/muscle and in GI tract (mainly SI/LI), accompanied by orthopedic abnormalities w/ consumptive coagulopathy, single to multiple raised blue 0.1-5cm lesions w/ wrinkly surface, endoscopic coagulation is dangerous b/c they are often full thickness therefore consider sclerotherapy)
- Klippel-Trenaunay Parkes Weber Syndrome (lower limb nevi + unilateral varicose veins + hypertrophy of all tissues in a limb)
- Venous Lake (blue spongy blebs w/ phleboliths (calcified thrombus) and skeletal abnormalities, may affect growth of neighboring structures, thrombosis can lead to pain, changes in size with change in hormonal status, usually on face/lips/ears, occurs in elderly, etiology unknown but maybe 2/2 sun exposure, sometimes confused for melanoma, Tx w/ electrosurgery/laser)

Arteriovenous Malformations

- AVMs (congenital communication b/t arteries and veins, occur in any internal organ not so much on skin, in GI tract they occur in distal LI, variable in appearance from small single lesions to large lesions occupying a segment of the bowel, Tx is surgery if symptomatic)

Lymphatic Malformations

- Lymphangioma or Cystic Hygroma (group of clear vesicles that ooze clear liquid, super infection is common, involute or grow rapidly, common in neck/axilla/chest, hypertrophy of surrounding tissue (soft tissue and bone), MRI: multiple loculated cystic structure with enhanced fibrous septa, Tx w/ surgery/sclerotherapy)

Erythematous Lesions

- The “Erythemas”
  - E. Nodosum
    - Mech: inflammation of subcutaneous fat aka panniculitis
    - Causes
      - Autoimmune: Sarcoid (most common adult), IBD, Behcet’s
      - Infection: Strep (most common child), Yersinia, TB, Mycoses, Mono, HBV, Parasites
      - Meds: OCPs, Sulfas
      - Other: Lymphoma/Leukemia, Pregnancy
- self-limited (wks) painful dusky red indurated poorly demarcated round subcutaneous nodules (hence “nodular erythema”) over anterior aspect of tibia/arms/abdomen w/ systemic Sx and arthralgia
- Tx: bedrest, leg elevation, heat, NSAIDs/steroids

- E. Multiforme (drug reaction) refer below
- E. Chronic Migrans (Lyme Disease) refer
- E. Marginatum (Rheumatic Fever) refer
- E. Gyratum Repens (malignancy esp breast cancer) refer
- Necrotizing Migratory E. (glucagonoma) refer
- Erythroderma (Sezary Syndrome) refer

### Dermatitis

- **Atopic Dermatitis aka Eczema**
  - Atopic Triad (allergy rhinitis aka Hay Fever, asthma, Atopic Dermatitis aka Eczema)
  - 90% occurs b/f 1st yr of life with 1/3 continue into adulthood
  - Cause: inhalants seen in asthma, proteins found in egg/milk/nuts/soybeans/fish/wheat
  - Exacerbating Factors: skin barrier disruption, skin infection, winter, certain clothes, stress
  - Mech: Type I IgE Mediated Hypersensitivity
  - S/S: lesions vary depending on age but remember “it is the itch that rashes”, NB dermatographism: stroking of skin will lead to blanching (not redness)
    - Early/Young: erythematous, exudative, crusty, lesions affecting face, extensor surfaces, hands, etc,
    - Secondary infections
    - Late/Adult: non-erythematous, dry w/ lichenification, lesions affecting flexor surfaces esp antecubital/popliteal fossa
      - NB Lichen Simplex Chronicus (lichenification 2/2 chronic frequent rubbing usually from atopic dermatitis, the more lichenified the more pruritic creating a vicious cycle, LSC can actually become an erogenous zone where it becomes pleasurable to scratch creating a vicious cycle, Tx: topical steroids w/ occlusive dressing, Unna Boot, intralesional steroids, anti-pruritics)
      - NB Increased r/o cutaneous infections (dermatophytes, staph, warts, molluscum, HSV)
  - Dx: allergy testing
  - Tx: Topical steroids, Wet to Dry Dressings w/ “Dombero” solution, hydration, petroleum jelly, NO soap, Avoid Scratching, Antipruritics, UV therapy, Avoid wool/fragrances/harsh-cleansers, H1B, Asthma Meds, Topical/Systemic Abx if secondary infection, Take short warm showers using gentle soaps and after bathing before dry moisturize using gentle creams/emollients (Eucerin) no lotions/oils, VitC, EFAs, Quercitin (bioflavenoid), Atopica, Pro-topico, Eldel, topical Tacrolimus/Pimecrolimus, Topical Doxepin, Refined Coal Tar

- **Contact Dermatitis** (inflammatory reaction from contact w/ an external agent)
<table>
<thead>
<tr>
<th>Irritant CD</th>
<th>Allergic CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mech</td>
<td>just direct irritation, no sensitization required therefore immediate response minutes after one exposure, everyone develops a reaction it's just certain people develop more easily at type IV hypersensitivity reaction, sensitization for days to even years is required until a response is elicited which takes a few days following subsequent exposures</td>
</tr>
<tr>
<td>Unique S/S</td>
<td>Pain before itching</td>
</tr>
<tr>
<td>------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Types</td>
<td>Dependent on [ ] of agent and state of skin barrier therefore occurs in everyone</td>
</tr>
</tbody>
</table>

**Seborrheic Dermatitis**

- S/S (geometric, localized, linear patterns)
  - Acute: sharp, wet, pruritus, erythema, edema, vesiculation
  - Sub-acute: in between
  - Chronic: ill defined, xerosis/dryness, lichenification, hyperkeratosis, fissuring
- **Dx:** **Patch Test** (various substances are applied to skin in shallow “Finn Chamber” cups x24-48hrs) vs Prick Test (various substances are applied to skin and a needle is passed thru substance into skin)
- Tx: Remove and avoid offending agent, Topical corticosteroids, Wet dressings soaked in Burow’s solution

- **Seborrheic Dermatitis**
  - Mech: hyperactive sebaceous glands 2/2 Malassezia furfur???
  - Epidemiology: 2% of population (very common), RFs (men, immunocompromised, etc), Age (bimodal: <20yo and >50yo), Exacerbations (winter, sunlight, stress, fatigue, hormones, HIV, etc)
  - S/S (chronic benign)
    - Children: same as adult but just on scalp (“cradle cap”)
      - If generalized, diarrhea, FTT then consider Leiner’s Disease which is 2/2 various immunodeficiencies
- Adults: flaking, greasy, yellow scales on erythematous base over scalp ("dandruff" aka "pityriasis sicca"), behind ears, eyelids/lashes ("blepharitis"), face, nasolabial fold, central sternal chest, body folds, groin, sometimes pruritic (NB dandruff could be a severe form of seborrheic dermatitis)
  - Tx
  - Scalp: remove crusts w/ olive oil, OTC antidandruff shampoo containing selenium sulfide (Selsun Blue) / zinc pyrithione, Rx antidandruff shampoo containing ketoconazole
  - Face/Trunk: remove crusts w/ olive oil, Rx topical creams containing ketoconazole/steroids
  - NB increase incidence of Parkinson’s

- Other: Dyshidrotic Eczematous Dermatitis (pruritic vesicles on hands/feet), Nummular Eczema (coin shaped plaque of grouped small papules and vesicles on an erythematous base on lower leg of old males in winter)

- Hypersensitivity Reaction (EM-SJS-TEN)
  - Causes (50% idiopathic)
    - Drugs: beta-lactams, sulfas (Bactrim, HCTZ), NSAIDs (Allopurinol), AEDs (Phenytoin/Tegretol)
    - Infection: HSV, HAV/HBV, Mycoplasma, Strept
    - Other: malignancy, radiation, pregnancy, vaccination
  - S/S: “bull’s eye target lesion” (painful/pruritic maculopapular target lesions) → bullae → diffuse erythema w/ skin sloughing) primarily affecting palms/soles, hands/feet, face, elbows/knees, genitals
  - Complications: super infection, necrotizing tracheobronchitis, meningitis, pre-renal AKI, permanent skin changes
  - Tx: supportive, remove causative agent, fluid/electrolyte replacement, if TEN consider IVIG/steroids, abx if super infection

<table>
<thead>
<tr>
<th>Skin Change</th>
<th>Erythema Multiforme (EM) Minor</th>
<th>Erythema Multiforme (EM) Major aka Steven Johnson Syndrome (SJS)</th>
<th>Toxic Epidermal Necrolysis (TEN)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic Sx</td>
<td>Target Lesion</td>
<td>Bullae</td>
<td>Skin Sloughing</td>
</tr>
<tr>
<td>Location</td>
<td>10-30%</td>
<td>&gt;30%</td>
<td></td>
</tr>
<tr>
<td>BSA</td>
<td>Skin</td>
<td>Skin + Mucosa</td>
<td>Skin + Mucosa</td>
</tr>
<tr>
<td>Mortality</td>
<td>0%</td>
<td>5%</td>
<td>30%</td>
</tr>
<tr>
<td>Causes</td>
<td>Infection</td>
<td>Drug</td>
<td></td>
</tr>
</tbody>
</table>

- Acne Vulgaris
  - Mechanism
    - hyperandrogen state (male, PCOS, etc), drugs (Li, steroids, certain hormones, isoniazide, phenytoin), stress, occlusion/pressure on skin, et al (NB NO link w/ foods like chocolate or fatty foods, dirt, etc) → stimulation of sebaceous gland → increased density of keratin within sebaceous glands results in plugging →
Rosacea

- sebum collects behind obstruction →
- non-inflammatory comedones →
  - Blackhead = open follicle that is plugged w/ keratin
  - Whitehead aka pimple = closed follicle that is plugged w/ keratin
- proliferation of Propionibacterium acnes which converts sebum to FFA and pro-inflammatory mediators →
- inflammatory papulopustules →
- inflammatory nodulocystic →
- scars

- Location: face, trunk, buttocks
- Treatment (when you move to next step cont prior steps)
  - 1st Topical Cleansers (?)
    - benzoyl peroxide (Benzac)
    - azelaic acid (Azelex)
  - 2nd Topical Retinoids (VitA derivatives that normalize desquamation of follicular epithelium thus removes plugs, apply at evening)
    - tretinoin (Retin-A)
    - adapalene (Differin)
    - tazarotene (Avage)
    - Combo: benzoyl peroxide / adapalene (Episud)
  - 3rd Topical Antibiotics (decrease bacterial colonization, apply at morning)
    - clindamycin (Cleocin T)
    - erythromycin (Akne-Mycin)
    - Combo: benzoyl peroxide / clindamycin (BenzaClin, Duac)
  - 4th Systemic Antibiotics (low dose, consider circulating abx, reduce Hz as much as tolerated to reduce long-term SEs)
    - minocycline (Minocin) NB resistant species are emerging
    - azithromycin (Zithromax)
    - amoxicillin (Amoxil)
  - 5th Systemic Retinoids
    - Isotretinoin (Accutane) NB teratogenic therefore concurrently give OCPs, cheilitis, dry skin, arthralgia/myalgia, hepatotoxic, dyslipidemia, VitA deficiency w/ night time blindness therefore should only be prescribed by dermatologist, labs Qmo, only given for 6mo, never use together w/ tetracycline
  - Other
    - Hormones or females: OCPs esp Yasmin and Ortho-Tri-Cyclen to decrease androgens (reduce sebum production) spironolactone for lower ½ face acne
    - Intradermal Steroid Injection w/0.1mL of 2 mg/mL Kenalog w/ 30g needle
    - NB dermabrasion for scars
    - NB if brown scarring then use glycolic acid peel or bleaching cream (hydroquinone 7% + retinA + desonide 30g topical QM/W/F)

- Rosacea
  - Chronic relapsing/remitting benign
  - Stages/Types
    - Stage I: Erythematotulengectatic w/ flushing 2/2 capillary leaking and vasomotor lability
    - Stage II: Papulopustular similar to acne vulgaris on cheeks/chin/forehead w/ thick greasy skin but there are no h/o comedones
    - Stage III: Disfiguring Phymatous 2/2 sebaceous hyperplasia, fibrosis, lymphedema
      - Rhinophyma (enlarged nose)
      - Metophyma (enlarged forehead)
      - Blepharophyma (enlarged eyelid)
      - Otophyma (enlarged ear)
      - Gnathophyma (enlarged chin)
    - NB Ocular w/ burning eyes
  - chronic disorder of pilosebaceous unit resulting in inflammation AND increased reactivity of capillaries to heat
  - typically occurs in ~40yo white F/M pts w/ low Fitzpatrick skin and +FHx, 10% of fair-skinned people
  - worse w/ sunlight/heat/spicy-foods/alcohol/caffeine
  - Tx
    - avoid precipitants and sunscreen
    - topical metronidazole, azelaic acid, sulfacetamide
- Oral T/M/Doxycycline, Isoretinoin
- NO topical steroids (will make worse)

- Hidradenitis Suppurativa
  - Mech: occlusion of the apocrine sweat gland by a keratinous plug → chronic, recurrent
    axillary/anogenital/inframammary/scalp infection of the gland w/ S. aureus → infection ruptures into surrounding tissue
    → tender fluctuant erythematous nodules w/ purulent malodorous drainage → sinus tracts/ulceration/fibrosis of subcutaneous tissue/fascia after repeated episodes
  - Tx: I&D, intralesional steroids, topical clinda, oral isoretinoin, extensive resection w/ graft closure as this is a recurrent problem

- Pityriasis Rosea
  - Self-limited (6-12wks), 2/2 HHV-7, spring/fall, not that common, average age (30yo)
  - S/S: single 2-5cm oval erythematous patch w/ scale at periphery (“Herald Patch”) followed by a generalized rash
    characterized by multiple pink oval scaly patches over trunk and proximal extremities in a Christmas tree distribution
    following Langerhan’s cleavage lines w/ pruritus
  - Dx: clinical but r/o Syphilis
  - Tx: symptomatic for pruritus (Tx w/ steroids and antifungals makes it worse)

- Exfoliative Erythroderma Syndrome
  - Serious life threatening skin reaction characterized by generalized/uniform erythema and scaling involving 100% of skin
    including hair/nail/palm/sole involvement, associated w/ systemic toxicity and generalized LAD
  - ½ of pts have a pre-existing dermatosis (in descending order): psoriasis, atopic dermatitis, drug reaction (CCB, Tegretol, Cimetidine, Gold, Li, Dilantin, Quinidine) leukemia/lymphoma, contact dermatitis, pityriasis rubra pilaris
  - Complication: hypothermia, super infection, high out CHF, electrolyte imbalance
  - Tx: bland emollients, systemic corticosteroids, supportive care

Bullous Lesions
- Acquired: Contact Dermatitis, Drug Eruptions, Insect Bite, 2nd Degree Burn, HSV (more common than genetic/autoimmune causes below)
- **Bullous "Benign" Pemphigoid** (benign, IgG against BM proteins, no mucosal lesions, tense bullae on extremities esp hands and groin that then rupture leaving an erosion then crust, - Nikolsky Sign, ~70yo, subepidermal, Tx: mild immunosuppressants)
- **Pemphigus Vulgaris "Vital"** (life threatening, chronic, IgG against cell surface proteins that hold cells together in epidermis, ~50yo, follows a viral infection, begins with oral blisters then flaccid skin bullae across body that then rupture leaving an erosion then crust, + Nikolsky Sign (direct pressure applied extends lesion laterally), Bx (acantholysis aka keratinocyte separation and + immunofluorescence), you can also monitor serum IgG levels, Tx: strong immunosuppressants and plasmapheresis b/c fatal if not Tx)
- **Dermatitis Herpetiformis** (refer)
- **Hereditary Epidermolysis Bullosa**
- **Herpes Gestationalis**
- **Porphyria Cutanea Tarda**
- **Familial Benign Pemphigus**
- **Pemphigoid Gestations**
- **Epidermolysis Bullosa Acquisita**
- **Linear IgA Dermatosis**

**Few Distinct Lesions**

- **Warts** (last weeks to forever, 2/2 HPV, easily transmitted, contains a central black dot, common (flesh colored on palmar of hand), flat (pink/brown on forehead, mouth, dorsum of hand), filiform/digitate (face), plantar (feet), Tx: topical salicylic acid (Dursal), cidofovir injection is best, also cryotherapy, topical imiquimod, intraleional injected mumps/candidal antigens)
- **Seborrhic Keratosis aka SK** (develop during late adulthood, no known cause, sharply demarcated waxy “stuck on” brown warty papule on trunk, NB can look similar to melanoma and wart, Tx: reassurance but if bothersome cryotherapy or shave if larger, NB “Sign of Lesser Trelat” sudden multiple SKs should raise suspicion of visceral malignancy esp stomach cancer)

**Basal Cell Carcinoma (BCC)**
- RFs: sun exposure, age, low Fitzpatrick category, PUVA Tx for psoriasis, immunosuppression, arsenic, nevus sebaceous of Jadassohn (irregular, raised, yellow-pink waxy smooth plaque on head/neck that is present at birth)
- Cell: basal cells esp where pilosebaceous follicles are concentrated esp head/neck (upper lip and lower eye lid)
- Types
  - Nodular: Early lesions are flesh-colored dome shaped nodules w/ adjacent telangiectasias and pearly translucent surface w/ pruritus/bleeding, late lesions have central ulcerations w/ rolled up edges (most common)
  - Superficial: multiple lesions on trunk that look like scaly eczema
  - Morpheaform/Sclerosing/Fibrosing: looks like a scar
  - Other: Pigmented, Adnexal, Infiltrative, Micro-Nodular
- Syndromes
  - Basal Cell Nevus Syndrome (Gorlin’s Syndrome) AD disorder, pits on palms/soles, jaw cysts, rib abnormalities, mental retardation
  - Xeroderma Pigmentosa
  - Albinism
- Mets: never metastasizes but can cause extensive local destruction esp of sinsuses/brain/orbit if left unTx
- Dx: shave Bx b/c tumor depth is not important but if keratotic/sclerotic then you will need full thickness excision
- Tx: excision w/ 3-7mm margins depending on aggressiveness of histology + radiation if invasive
  - NB Moh’s Surgery if recurrent, high RFs, aesthetically sensitive areas like nose, eyes, etc
  - NB curettage or electrodeexcision for small BCC that is not recurrent, no high RFs, non aesthetically sensitive area
  - NB cryotherapy for small BCC that is over bone/cartilage, tip of nose, around eye, etc

**Squamous Cell Carcinoma (SCC)**
- **RFs:** Immunosuppressed, UV (sunlight, tanning booths, PUVA Tx for psoriasis), chemical exposure (arsenic, pesticides, organic hydrocarbons), virus (HSV, HPV), ionizing radiation, Marjolin’s Ulcer (any chronic wound, burn, pressure, etc), 2/2 genetic changes 2/2 inflammation
- **Cell:** keratinocytes
- **Precursor**
  - **Actinic Keratosis aka AK** (erythematous macules/papules w/ coarse adherent scale, 5% progression, 2/2 sun exposure, Tx: if small then cryotherapy but if large then topical 5-FU, imiquimod cream, topical retinoin, blue light)
  - **Bowen’s Dz aka SCC In Situ** (full thickness cytologic atypia, called Erythroplasia of Queyrat if on penis)
  - **Leukoplakia** (white mucosal patch)
- **Types**
  - Verrucous (slow growing exophytic hence rarely metastasizes)
  - Ulcerative (fast growing invasive hence often metastasizes)
- **S/S:** scalp, hand dorsum, superior pinna, lower lip, upper eye lid with hyperkeratosis w/ variable scaling/crusting (any persistent nodule/plaque/ulcer should be examined for SCC)
  - **NB Keratoacanthoma** (looks just like SCC that grows fast over 6wks followed by involution over 6mo, no Tx)
- **Mets:** 4% mets esp if >2cm, fast growing, ill defined borders, deeper, less differentiated, lesions on face/ears/scalp-vertex
- **Dx:** punch Bx not shave Bx
- **Tx:** same as BCC just wider margins (3-7mm)
- **Psoriasis**
  - **Epidemiology:** 2% of population, ~30yo (the younger the more severe), M=F
  - **Genetic (+FHx, 10/40% risk if one/two parents) chronic (exacerbations w/ remissions worse in winter/trauma/stress) auto-immune inflammatory (T-cell & Neutrophils, red base) hyperproliferation of epidermal cells 2/2 shortened cell cycle w/ defective keratinization resulting in scaling
  - **Flares** 2/2 stress, URTI, skin damage (Koebner’s Phenomenon), EtOH, drugs (Li, BB, steroids, IFN), withdrawal of meds
  - **S/S:**
    - Auspice’s Signs (removing a scale results in pinpoint bleeding)
    - Muhro Microabscess (neutrophils in stratum corneum)
  - **Other**
    - Joint: DIP, 1st MCP, SI seronegative spondyloarthritis w/ enthesitis (25%)
- Nail: pitting, oily spots, onycholysis, subungual hyperkeratosis, elkonyxis, transverse grooves, longitudinal ridging, leukonychia (25%)

- Types
  - Vulgaris
    - Chronic/Stable: well demarcated thick adherent appearing salmon pink based Plaques w/ easily removable silvery scale (non-pruritic/painful except on scalp/anogenital) on extensor surfaces, scalp, and/or sacral but can be Inverse (at flexural sites, intergluteal cleft, intertriginous sites), Palmo-Plantar (on palms/soles), most common representing 80% of psoriasis

- Acute/Eruptive: multiple small 1-2cm pink papules aka Guttate lesions across trunk that appear rapidly and then spontaneously resolve but often evolves into chronic/stable (above), 2% of psoriasis, follows Strept URTI
• Pustularis
  - Palmoplantar Pustulosis: chronic burning/pruritic pustules on palms/soles, more common in females
  - Generalized Pustular Psoriasis of von Zumbusch: abrupt erythema and pustules that spreads w/in hours, pts are toxic appearing w/ leukocytosis and constitutional Sx, high r/o infection resulting in a life threatening condition
• Erythroderma
  - Erythrodermic: generalized lesions w/ significant inflammation affecting nearly 100% of BSA, high r/o infection, often occurs after stopping meds
    - Dx: clinical
    - Tx (NB no cure) Tx below is for Chronic/Stable Psoriasises Vulgaris
      - Localized/Mild Plaque/Inverse/Palmo-Plantar
        - Trunk/Extremities: remove scales by soaking in water or w/ Baker’s P&S scale remover then topical corticosteroids specifically betamethasone valerate / fluocinolone acetonide / betamethasone propionate / clobetasol propionate (w/ occlusion overnight, take M to F w/ weekends off)
          - Steroids can be accentuated w/ topical coal tar (Anthralin), topical VitD analogues (Calcipotriol, Tacalcitol), topical tazarotene (?)
          - when lesions are >10% then give phototherapy light box (Sunlight, Tanning Bed, Broadband UVB, Narrowband UVB, oral Psoralen followed by UVA aka PUVA)
        - Scalp: tar/ketoconazole shampoos (Clonil, Neutrogena T-gel, Nigoral) followed by topical corticosteroids specifically betamethasone valerate
        - Palms/Soles: occlusive dressing w/ topical corticosteroids in petrolatum and phototherapy
        - Nails: topical do not work, injections are painful, some evidence that phototherapy and retinoids are helpful
          - Generalized/Severe: systemic retinoids (Acitretin), systemic immunosuppressants (Methotrexate, Cyclosporine), immunomodulators (Alefacept anti-CD4, Efalizumab anti-CD11, Etanercept/Infliximab anti-TNF)
  - Palms/Soles: occlusive dressing w/ topical corticosteroids in petrolatum and phototherapy
• Granuloma Annulare
- Self-limited asymptomatic chronic dermatosis
- Young females
- Firm shiny papules in an annular arrangement w/ central depression on dorsum of hand, feet, elbows, knees
- Lasting months to years w/ most remitting after 2yrs but 40% recurrence
- Tx: no treatment but cosmetically displeasing then topical glucocorticoids

- **Cylindroma** (firm rubbery bluish-pink nodules on scalp from eccrine glands)

- **Eccrine Poroma** (soft red pedunculated nodules on sole of foot from eccrine glands)

- **Syringoma** (periocular small clear papules from eccrine glands, seen in Down Syndrome)

- **Sebaceous Hyperplasia** (small, shiny, umbilicated yellow-white papules on face, often confused w/ BCC but they are soft and with lateral compression you can feel a small globule of sebum, Tx w/ electrocautery)
- **Epidermoid Cyst** (cystic enclosure of epithelium into dermis containing malodorous cheesy keratinous debris, develop during adulthood, mildly fluctuant flesh colored well-circumscribed nodule w/ punctum on back, often called a sebaceous cyst but a misnomer b/c it is epidermal in origin)

- **Epidermal Inclusion Cyst** (traumatic implantation of epidermis into dermis on hand/fingers but unlike epidermoid cysts they don’t burst easily and must be removed surgically)

- **Milium** (multiple epidermal cysts on eyelids/cheeks/forehead at sites of trauma)
- **Dermoid Cyst** (present at birth, similar to epidermal cyst but w/o punctum, supraorbital ridge)

- **Trichilemmal Pilar Cyst** (similar to epidermal cyst but on scalp)

- **Dermatofibroma** (develop during adulthood, brown-red indurated papule/nodule, + dimple sign where lateral compression with thumb produces a depression, on lower extremities, Tx w/ cryotherapy)
- **Lipoma** (soft moveable flesh colored nodule on trunk/extremities, composed of fat cells, Tx w/ excision)

- **Neurofibroma** (any age, soft compressible pink/flesh colored nodule on trunk/extremities, w/ button hole sign, consists of Schwann cells and endoneurial fibroblasts, Neurofibromatosis also has pigmented hamartomas of the iris (Lisch nodules), multiple sharp light brown 5cm macules (Café-au-Lait spots), and axillary freckling)

- **Pilomatrixoma aka Calcifying Epithelioma of Malherbe** (firm flesh colored nodule associated w/ hair follicle in head/neck/arms)

- **Trichoepithelioma** (multiple pink/flesh colored shiny papules from hair follicles that coalesce into plaques on face looking just like BCC, associated w/ Rasmussen Syndrome (AD disorder w/ triad of trichoepitheliomas + cylindromas + milia)

- **Molluscum Contagiosum** (self-limited, very contagious w/ auto-inoculation very common, 2/2 Pox virus, affects young children, sexually active, immunocompromised adults, multiple, 1-5mm discrete solid skin colored or pearly white waxy papules w/ central umbilication found on face, eyelids, axillae, anogenital, Dx clinical but smear of plug will show inclusion bodies, Px: minimize shaving area as it opens lesions spreading virus, Tx: if immunocompetent then usually regress spontaneously curettage, cryotherapy, electrodessication, laser, imiquimod cream, cantharidin drops)

- **Pyoderma Gangrenosum (PG)**
  - Etiology: 50% Idiopathic or 50% Systemic Disease (IBD, Diverticulosis, Paraproteinemia, Leukemia, Hepatitis, Behcet’s)
  - S/S: initially a single painful hemorrhagic pustule/nodule on leg/buttock/ab/face/stoma that then rapidly develops into an irregular ulcer w/ undermined borders w/ a violaceous rim surrounding a purulent/necrotic base w/ crater like holes, NB pathergy (lesions develop after mild trauma)
  - Tx: treat underlying dz, steroids, no surgery b/c pathergy makes it worse, can last up to years!!!
• Sweet’s Syndrome
  - Mech: usually idiopathic but sometimes associated w/ Yersinia infection, Leukemia, Paraproteinemia
  - S/S: infectious prodrome (URI/Colitis) followed by painful inflammatory plaques on face/neck/arms w/ constitutional Sx
  - Lab: Leukocytosis w/ neutrophilia
  - Bx: neutrophilic infiltrate
  - Tx: steroids ± abx if 2/2 Yersinia

• Lichen Planus (LP)
  - Epidemiology: middle-aged, F>M, prevalence 1%, higher prevalence in Caucasians/South-Asians
  - Pathogenesis: not entirely understood, T-cells specifically target basal keratinocytes b/c of increased abnormal intercellular adhesions molecules (ICAM) → apoptosis
  - Associations
    - HCV
      - General: relationship is uncertain and routine HCV screening is controversial but often performed
      - Mech: HCV changes the cytokine profile leading to inappropriate activation of T-cells
        - NB this mechanism may underlie the case reports of LP emerging after vaccination w/ HBV/Influenza and tattoo w/ certain pigments
      - S/S: disease is usually more generalized and prolonged w/ more mucosal lesions w/ exacerbation occurring during IFN Tx
        - Drug Induced: BB, NSAIDs, ACE-Is, Aldactone, Anti-Malarials, Gold, Penicillamine, et al
        - Autoimmune States: PBC, PSC, UC
  - Dx: clinical but sometimes a Bx is necessary
    - Bx: dense band like infiltration of lymphohistiocytes w/ shaggy fibrinogen at the dermal epidermal junction w/ degeneration of the basal keratinocytes (Civatte Bodies) (NB IF w/ subepidermal Ig deposits)
  - Prognosis: most cases are self-limited remitting anywhere b/t 1mo-7yrs except for mucosal LP
  - S/S
    - Skin
      - “4Ps” (Pruritic, Polyonal, Purple, flat topped Papule/Plaques) grouped together on flexor surface of extremities esp wrists/ankles w/ “Wickham Striae” (fine lacy white lines found on top of lesion) looking like “Lichen”
      - “Koebner Phenomenon” (lesions erupt after trauma/injury)
      - Lesions usually heals w/ post-inflammatory hyperpigmentation
- DDx: Chronic GVHD and Secondary Syphilis
- Tx: few well designed randomized controlled trials, notoriously very difficult to Tx, 1° topical/intralobular corticosteroids, 2° retinoids, phototherapy w/ UVB, 3° griseofulvin, metronidazole, sulfasalazine, azathioprine
  - NB topical calcineurin inhibitors (eg. pimecrolimus) was very effective but FDA placed a "black box" warning on its topical use b/c of the increased r/o lymphoma and skin cancer
- Hair: 10%, scarring alopecia
- Nail: 10%, wedge shaped deformity, ridging, splitting, thinning
- Mucosal: 30-70%

  - Morphology: (1) asymptomatic white reticulated patches w/ Wickham Stria or (2) painful erosions or ulcers or rarely popular/atrophic lesions
  - Location: mouth (lateral buccal mucosa and tongue), genitals (vulva and glans penis), esophagus
  - Complication: marginally (0.8-1.2%) increased r/o oral/genital/esophageal SCC
  - Esophagus
    - Historical Perspective: first reported case in 1982 by Al-Shihabi, et al since then the number of reported cases is low (<100) however this is likely an underestimation b/c of subtle findings
    - Epidemiology: much more common in female otherwise similar demographics to cutaneous dz, much more rare (<1% of LP)
    - S/S: dysphagia/odynophagia, chest pain, weight loss
    - Complications: SCC, to date 4 cases of squamous cell carcinoma have been reported in pts w/ LP but no other RFs including tobacco or alcohol
    - RF: associated w/ the rare variant of vulvovaginal gingival syndrome while cutaneous dz seems to be less common, very few cases series mainly case reports, the largest case series was a Mayo Clinic Series of 27 patients published in CGH in 2010, 95% have pre-existing oral dz hence a thorough oral exam very helpful. b/c the esophageal changes can be non-specific a thorough H&P is emphasized
    - Dx
      - EGD: friable, tissue paper like (b/c of basal cell layer degeneration) mucosa w/ underlying inflammation, often mistaken for erosive reflux esophagitis however dz is usually localized to the proximal esophagus (but distal dz has been reported), if chronic discrete single to multiple strictures/rings or small caliber esophagus, EUS (echofree space b/t mucosa/submucosa)
      - Bx: histopathology is many times it not diagnostic w/ only 40% of cases showing classic unique histologic findings similar to cutaneous dz while 60% demonstrate only non-specific inflammation
      - NB often there is a delay in Dx of LP as the cause of their esophageal dz but one should consider the dx in a middle aged women w/ cutaneous/oral/genital LP w/ no reflux and proximal disease
      - DDx: eosinophilic esophagitis, bullous skin disease, pill esophagitis, fungal/viral infections, Behcet’s, SJS, GVHD
    - Tx
      - There is no clear standardization of Tx or guidelines w/ regimens varying in the literature
- Most successful Tx is with systemic steroids (prednisone 40-60mg PO QD x4-6wks w/ subsequent 4-6wk taper is most successful at 75% but still has an 85% relapse rate and steroid SEs)
  - Case Reports: intralvesional/topical steroids, immunomodulators (oral cyclosporine, intralvesional tacrolimus, etc), retinoids
- If strictures are present then dilation is necessary
  - NB Koebner phenomenon (trauma triggers the formation of lesions of uninvolved skin/mucosa) is important to know when dilating or if undergoing intralvesional injection therefore dilate only involved areas however there has is no documented info showing aggravation of strictures, dz is usually resistant to immunosuppressive therapy requiring frequent dilation
- NB PPIs do not seem to be helpful except if concurrent GERD
- NB spontaneous clearance of dz is unlikely as seen in mucocutaneous dz

***Photosensitivity***
- Sunburn
- Skin Diseases That Exacerbate from UV Exposure (Acne, SLE, Rosacea, Seborrheic Dermatitis)
- Phototoxic/allergy (Drug Induced: FQ, Chlorpromazine, Amio, Lasix, etc vs Plants: lime, cosmetics, etc)
- DNA (Xeroderma Pigmentosa)
- Chronic (Dermatoheliosis aka Photo-Aging, Solar Lentigo, Actinic Keratosis, Skin Cancer)
- Metabolic (Porphyria) (refer to metabolic liver)

***Burns***
- Parkland Formula: 2-4cc/kg/%BSA of LR with 1/2 volume in first 8hrs and other 1/2 volume in next 16hrs (only for 2nd/3rd degree burns >10%)
- After 24hrs use D5W with 5% albumin at 0.55cc/kg/%BSA in/c the massive sodium load in first 24hrs of LR infusion and in/c of massive evaporation of water
  - Inhalation/carbon monoxide injury (soot in sputum/nose/mouth, nasal/facial hair burns, carboxyHgb: throat/mouth erythema, dyspnea, low O2 sat, H, confusion, coma) Tx: intubation, mechanical ventilation, 100% O2
  - Alkal. burns more dangerous b/c body cannot buffer them
  - Electric burns very bad b/c electricity follows route of least resistance aka nerves, vessels, fascia also cardiac dysrhythmias and myoglobinuria w/ RF are common
  - First Degree: epiderm only (painful, dry, red, no blisters)
    - Tx: clean w/ nonionic detergent, topical antibiotics like Neosporin, analgesics, sterile dressing (not IV antibiotics b/c bacteria live in eschar which is avascular)
    - Topical Antibiotics
      - Neosporin
      - Silver Sulfadiazine (Silvadene) for small burns: painless, NO electrolyte disturbance, little eschar penetration, broad – Psuedomonas, neutropenia
    - Mafenide Acetate (Sulfamylon) for contaminated burns: painful, acid-base disturbance, good eschar penetration, broad – Staph, allergic reaction
    - Polymyxin B (Polysporin) for facial burns, Painless, NO electrolyte disturbance, little eschar penetration, narrow spectrum
  - Second Degree: some dermis (very painful, weeping, red, blisters/swollen) Tx: same + remove blisters
  - Third Degree: all dermis (painLESS, dry, white, swollen/charred) Tx: early (w/in 1st wk) excision of eschar and STSG (12-15/1000in), if circumferential around extremity there is a risk of compartment syndrome therefore do longitudinal escharotomy to healthy fat
  - Fourth Degree: bone/muscle
  - Rule of Nines: Head/Neck: 9%, UE: 9%, LE: 18%, Chest: 18%, Back: 18%, Genitals/Perineum: 1% where 1% = palm of hand
  - Referral Criteria: 2nd & >20%, >10% in children/elderly, 3rd & >5%, any burns involving face, hands, feet, perineum, inhalation, other trauma, electric
  - Burn Shock: loss of fluid from intravascular space 2/2 burn injury
  - NGT decompression is necessary b/c most burn pts develop ileus
  - Curling Stress Ulcer Prophylaxis: H2B
  - Burn Wound Infection: change in color of eschar***, surrounding unburned skin affected, 2nd degree that turns into 3rd degree, green pigment (Staph, Psuedomonas, Strep, Candida), send tissue to lab for bacterial count
  - Other infections beside wound infection: pneumonia and central line (in fact so common that central lines are replaced q3-4d)
  - Sodium is the most common electrolyte imbalance

***Infections***
- **NB Intertrigo** (nonspecific inflammation of opposed skin, 2/2 infections: Bacteria (Strep-Impetigo, Corynebacterium-Erythrasma, Psuedomonas) or Fungi (Candida, Malassezia furfur) and dermatosis: inverse psoriasis, seborrheic dermatitis, atopic dermatitis, Tx underlying cause along w/ antibiotic/antifungal powders w/ zinc oxide)
- **Bacterial**
<table>
<thead>
<tr>
<th>Pyoderma Skin Infection</th>
<th>Erythrasma</th>
<th>Location/Epidemiology/Organism</th>
<th>Signs/Symptoms/Complications</th>
<th>Diagnosis/Treatment/Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Location</td>
<td>Chronic infection affecting intertriginous areas mimicking tinea</td>
<td>- KOH Prep and Coral Red Fluorescence under Wood’s Lamp 2/2 Coproporphyrin III followed by Cx</td>
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<tr>
<td>Epidemiology</td>
<td>Epidemiology</td>
<td>Sharply demarcated macerated macule</td>
<td>Treatment: Benzoyl Peroxide 2.5% Gel after showering x7d and Systemic Tetracycline x14d</td>
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<tr>
<td>Organism</td>
<td>Organism</td>
<td>Impetigo/Ecthyma</td>
<td>- NB then Impetigo (S. aureus, NB MRSA is on the rise) vs. NON-Bullous /Contagiosa Impetigo (GABHS)</td>
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<td></td>
<td></td>
<td>Signs</td>
<td>Diagnosis</td>
<td>Prevention</td>
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<tr>
<td></td>
<td></td>
<td>sharp demarcated inflamed lesion anywhere skin has been traumatized</td>
<td>Impetigo (small sharply demarcated inflamed papule → vesicle → pustule → ruptures → honey colored crust) typically on face (nose/mouth)</td>
<td>highly contagious therefore wash hands</td>
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<td>(1) Bullous Impetigo (large macule → large sharply demarcated inflamed bulla → ruptures → honey colored crust) typically on trunk/extremities</td>
<td>NON Bullous / Contagiosa Impetigo (small sharply demarcated inflamed papule → vesicle → pustule → ruptures → honey colored crust) typically on face (nose/mouth)</td>
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<td></td>
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<td>Symptoms</td>
<td>Complications</td>
<td></td>
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<td></td>
<td></td>
<td>Constitutional (usually none)</td>
<td>dehydration if widespread skin loss</td>
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<td></td>
<td></td>
<td>Local (pruritus, burning, pain)</td>
<td>other skin infections, osteomyelitis, septic arthritis, sepsis, meningitis, pneumonia (more common in neonates)</td>
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<td></td>
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<td>poststrep GN (do not protect)</td>
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<td>Complications</td>
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<tr>
<td>Folliculitis</td>
<td>Location</td>
<td>Signs</td>
<td>Diagnosis</td>
<td>Prevention</td>
</tr>
<tr>
<td></td>
<td>Location</td>
<td>sharply demarcated inflamed lesion surrounding hair follicle after it has been cut off from shaving, rubbed by tight clothing, exposed to extreme perspiration/water small pustule typically on neck/legs/arm pits after shaving or groin unlike acne pimple there is a hair in the middle of it</td>
<td>- &quot; &quot;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Follicle (Epidermis + Papillary Dermis)</td>
<td>Symptoms</td>
<td>Treatment</td>
<td>monthly nasal mupirocin BID x5d Qmo</td>
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<tr>
<td></td>
<td></td>
<td>Constitutional (usually none)</td>
<td>hot moist compresses</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Local (pruritus)</td>
<td>typically self-limited therefore no Tx needed</td>
<td></td>
</tr>
<tr>
<td></td>
<td>S. aureus “barber’s itch”</td>
<td>Complications</td>
<td>topical abx or benzoyl peroxide</td>
<td></td>
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<tr>
<td></td>
<td>Fungus “tinea barbae”</td>
<td></td>
<td>keep area clean</td>
<td></td>
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<tr>
<td></td>
<td>Psuedomonas “hot tub folliculitis” b/c low chlorine levels</td>
<td></td>
<td>shave with new razor</td>
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<tr>
<td></td>
<td>Hair Grows Back Into Skin “Psuedofolliculitis barbae”</td>
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<td>minimize friction</td>
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<td>HIV “ eosinophilic folliculitis”</td>
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<tr>
<td>Location</td>
<td>Signs</td>
<td>Diagnosis</td>
<td>Treatment</td>
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<tr>
<td>Follicle (Epidermis +</td>
<td>poorly marred inflamed lesion, NON-elevated</td>
<td>Blood Ct only 5% + (rarely done)</td>
<td>Debride</td>
<td></td>
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<tr>
<td>Reticular Dermis)</td>
<td>central extending red streak w/ LAD/adenitis</td>
<td>Aspiration 8x Ct only 20% + (inject saline at leading edge and then aspirate) (rarely done)</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
<td></td>
</tr>
<tr>
<td>S. aureus</td>
<td>NB erysipelas is well marred inflamed lesion, elevated/palpable plaque, found in elderly: face vs child: extremities</td>
<td>Puncture 8x Ct only 25% + (rarely done)</td>
<td>Debride</td>
<td></td>
</tr>
<tr>
<td>Organism</td>
<td>constitutional (MILD: F, malaise, chills)</td>
<td>The above three are rarely done unless severe and immunocompromised</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
<td></td>
</tr>
<tr>
<td>Polymicrobial rarely</td>
<td>Constitutional (MILD: F, malaise, chills)</td>
<td>Blood Ct only 5% + (rarely done)</td>
<td>Debride</td>
<td></td>
</tr>
<tr>
<td>Staph only (25%)</td>
<td>Local (MILD: pain)</td>
<td>Aspiration 8x Ct only 20% + (inject saline at leading edge and then aspirate) (rarely done)</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
<td></td>
</tr>
<tr>
<td>Cellulitis (Bites,</td>
<td>Sepsis</td>
<td>Blood Ct only 5% + (rarely done)</td>
<td>Debride</td>
<td></td>
</tr>
<tr>
<td>Cuts, &quot;Diabetic Foot&quot;,</td>
<td>Abcess</td>
<td>Puncture 8x Ct only 25% + (rarely done)</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
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<tr>
<td>etc)</td>
<td></td>
<td>The above three are rarely done unless severe and immunocompromised</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
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</tbody>
</table>

**Soft Tissue Infection**

<table>
<thead>
<tr>
<th>Location</th>
<th>Signs</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermis + Superficial</td>
<td>poorly marred inflamed lesion, NON-elevated</td>
<td>Blood Ct only 5% + (rarely done)</td>
<td>Debride</td>
</tr>
<tr>
<td>Fascia + Subcutaneous</td>
<td>central extending red streak w/ LAD/adenitis</td>
<td>Aspiration 8x Ct only 20% + (inject saline at leading edge and then aspirate) (rarely done)</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
</tr>
<tr>
<td>Tissue</td>
<td>NB erysipelas is well marred inflamed lesion, elevated/palpable plaque, found in elderly: face vs child: extremities</td>
<td>Puncture 8x Ct only 25% + (rarely done)</td>
<td>Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF</td>
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<tr>
<td>Polymicrobial rarely</td>
<td>Sepsis</td>
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<tr>
<td>Staph only (25%)</td>
<td>Abcess</td>
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</tr>
</tbody>
</table>

**Diagnosis**

- " 
- " 

**Treatment**

- Debride
- Mark border to assess progression to r/o NF and check every hour until convinced that it is not NF

**Cellulitis (Bites, Cuts, "Diabetic Foot", etc)**

**Signs**

- poorly demarcated inflamed lesion, NON-elevated
- central extending red streak w/ LAD/adenitis
- NB erysipelas is well demarcated inflamed lesion, elevated/palpable plaque, found in elderly: face vs child: extremities
- Constitutional (MILD: F, malaise, chills)
- Local (MILD: pain)
- Sepsis
- Abcess

**Complications**

- superficial/deep thrombophlebitis
- dermatitis
- drug rxx
- cancer
- insect bite
- Sweet’s Syndrome
- Eosinophilic cellulitis

**Mechanism**

- not so much 2/2 bacteria itself but rather the toxin made by the bacteria therefore use bacteriostatic (clindamycin) NOT bacteriocidal agents (beta-lactams) b/c they will actually make the inflammation temporally worse

**Prophylaxis**

- Abx if >2 episodes/yr
- Topical Benzoyl Peroxide
- Warm Compresses
- Elevation
- TEDS
- Diuresis
- Selenium
- Emollients
Clostridium thru a warm, sweaty shoe hence Psuedomonas)
- *Clostridium sordellii* (black tar heroin use)
- High r/o resistance b/c these infections are often chronic

<table>
<thead>
<tr>
<th>Organism</th>
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<tbody>
<tr>
<td>Polymicrobial</td>
<td>Superficial Fascia + Subcutaneous Fascia + Deep Fascia</td>
<td>poorly demarcated inflamed lesion, non-elevated that progresses to blisters, blebs, bullae</td>
<td>MRI (imperative)</td>
<td>Urgent debridement b/c 30% mortality w/ surgical reexaminatin in &lt;24hrs (pts usually require three debridements) many times amputation is required</td>
</tr>
<tr>
<td></td>
<td>usually lower extremities 2/2 trauma, ab wall 2/2 surgery or hernias, or perineum 2/2 untx cysts or hemorrhoids</td>
<td>deep infection resulting in tenderness beyond border of inflammation b/c superficial skin not affected yet</td>
<td></td>
<td>Hyperbaric Oxygen</td>
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<tr>
<td></td>
<td></td>
<td>anaerobic growth resulting in crepitus</td>
<td></td>
<td>Broad Spectrum Parenteral Abx same as above add anaerobic coverage with Clinda</td>
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<tr>
<td></td>
<td></td>
<td>arterial thrombosis resulting in cyanosis, necrosis, loss of overlying skin</td>
<td></td>
<td>IVIG (esp if 2/2 strept)</td>
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</tbody>
</table>

- Polymicrobial (aerobes and anaerobes) otherwise similar to cellulitis
- Location
- Superficial Fascia + Subcutaneous Fascia + Deep Fascia
- usually lower extremities 2/2 trauma, ab wall 2/2 surgery or hernias, or perineum 2/2 untx cysts or hemorrhoids

- Signs
  - poorly demarcated inflamed lesion, non-elevated that progresses to blisters, blebs, bullae
  - deep infection resulting in tenderness beyond border of inflammation b/c superficial skin not affected yet
  - anaerobic growth resulting in crepitus
  - arterial thrombosis resulting in cyanosis, necrosis, loss of overlying skin
  - nerve damage resulting in hypoesthesia
  - progresses fast sometimes before your eyes b/c below fascia
  - Constitutional (SEVERE: F, tachy, hypoTN, AMS)
  - Local (SEVERE: pain that is out of proportion to physical findings especially early on)

- Symptoms
- Constitutional (SEVERE: F, tachy, hypoTN, AMS)
- Local (SEVERE: pain that is out of proportion to physical findings especially early on)

- Complications
  - Dehydration
  - Sepsis
  - Amputation
  - Death

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<table>
<thead>
<tr>
<th>Panniculitis</th>
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<tbody>
<tr>
<td>Pyomyositis aka Gas Gangrene</td>
</tr>
<tr>
<td>- Viral: Influenza, Coxsackie, HIV</td>
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<tr>
<td>- Bacterial: Staph aureus, Lyme</td>
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<tr>
<td>- Fungal: Trichinosis, Toxo</td>
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<tr>
<td>- Common in the tropics but suspect in a pt in the US when they are HIV+</td>
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<td>- Usually the quadriceps muscle is the involved muscle</td>
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<tr>
<td>- Muscle inflammation but no weakness and enzyme levels are typically normal</td>
</tr>
<tr>
<td>- Blood cultures are rarely positive but muscle aspirate cultures could be helpful</td>
</tr>
<tr>
<td>- Tx: iv abx x4wks</td>
</tr>
</tbody>
</table>

- **Fungal** (these infections can extend deeper in immunocompromised pts, other rare types of infections include Mycetomas, Chromomycosis, Sporotrichosis and pulmonary infections disseminated to skin like Crypt, Histo, Coccidio, etc
  - **Tinea/Pytiriasis Versicolor**
    - 2/2 dimorphic fungus Malassezia furfur w/ spherical yeast and short hyphae mold, infects Statum Corneum, various hypo/hyperpigmented macules on upper chest/back/shoulders, asymptomatic, worse in summer b/c the area fails to tan
    - Dx: “spaghetti and meatballs” on KOH prep, + Wood’s Lamp fluorescence,
    - Tx: topical keratolytics like selenium sulfide (Selsun Lotion) or topical/oral –azole
  - **Candidiasis**
    - S/S: creamy white patches on mucosa/skin that can be scraped off
    - Epidemiology: normal in young children, normal in women on abx, immunocompromised esp HIV/DM
Atypical HIV

STDs

Viral

- Tinea capitis (scalp, children, mild scaling w/ broken off hairs (“black dot”) to a kerion that looks like an abscess resulting in scarring alopecia, very contagious)
- Tinea barbae (bearded face) vs facei (non-bearded face)
- Tinea corporis (trunk/limbs), this has the classic ring appearance w/ raised red ring representing active inflammation/infection w/ central clearing
- Tinea cruris (“jock itch”, at crural folds)
- Tinea pedis (“athlete’s foot” maceration b/t toes) 
- Tinea manum (erythema and scaling at palmar creases) 
- Tinea unguium (thickened discolored nail w/ subungual debris) 
  - Onychomycosis refers to any fungal infection of the nail bed including dermatophytes, candida, etc
- Dx (scapel blade brushing, recent cream use makes dx impossible so always ask)
  - Wood’s Lamp: greenish fluorescence (for hair)
  - Heated KOH Prep: branched septated hyphae (Candida has no branching)
- Sabouraud’s Culture: Mycrosorum canis, Trichophyton spp., Epidermophyton floccosum

- Tx (refer to ID Fungal)

Viral

- Shingles aka Herpes Zoster
  - Chicken Pox (primary VZV infection, disseminated pruritic vesicles) vs Shingles (secondary/reactivation VZV infection when immunity to VZV declines
  - Varicella reactivation from dorsal root ganglion to single cutaneous nerve
  - 2/2 emotional/physical stress, lymphoma, radiation, immunosuppressants
  - 15% lifetime risk if patient had chickenpox
  - Pain and constitutional Sx often prior to rash
  - Some pts can have viremia w/ vesicles distant from dermatome
  - Complications: hemorrhage, CN disorders (Ophthalmicus, Ramsay Hunt, etc), cutaneous dissemination
  - Contagious several days before to until last vesicle crusts over
  - Serious if involves CN-V involving eye/eye-lid/forehead/nose-tip (Hutchinson’s Sign)
  - Multiple CNs esp if auditory canal is affected (Ramsay-Hunt Syndrome)
  - Bell’s Palsy
  - Dx: Direct Fluorescent Antibody (DFA) of Ac in Vesicle Scrapings, Viral Cx; Tzanck Smear
  - Tx: moist dressings, -cyclovir, immunize later on, treat pain/insomnia w/ doxepin, steroids to relieve constitutional Sx
  - Post-Herpetic Neuralgia: pain after rash begins to heal, ¾ of pts, increases w/ age, usually spont remission after 6mo, steroids have no effect on preventing PHN, consider TCAs/gabapentin/Capsaicin

- Atypical
- Insect
- STDs
- HIV