DERMATOLOGY

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APPROACH TO THE DERMATOLOGY 2 PATIENT History Physical Exam	DRUG ERUPTIONS
DEFINITIONS	Delayed Hypersensitivity Syndrome Photosensitivity Eruptions Serum Sickness - Like Reaction
ACNEIFORM ERUPTIONS	COMMON SKIN LESIONS
DERMATITIS/ECZEMA	Miscellaneous MALIGNANT SKIN TUMOURS
Dyshydrotic Dermatitis Diaper Dermatitis INFECTIONS	HERITABLE DISORDERS
Bacterial Superficial Skin (Epidermal) Deeper Skin (Dermal) Hair Follicles Periungual Region Others Viral Dermatophytes	SKIN MANIFESTATIONS OF INTERNAL 31 CONDITIONS Autoimmune Disorders Endocrine Disorders HIV Malignancy Others Pruritus
Yeast Parasitic	ALOPECIA (HAIR LOSS)
PAPULOSQUAMOUS DISEASES	Scarring (Cicatricial) Alopecia WOUNDS AND ULCERS
Pityriasis Rosea VESICULOBULLOUS DISEASES	COSMETIC DERMATOLOGY
Pemphigus Vulgaris Bullous Pemphigoid Dermatitis Herpetiformis Porphyria Cutanea Tarda	USEFUL DIFFERENTIAL DIAGNOSES 35 Differential Diagnosis by Morphology Differential Diagnosis by Location
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STEVENS-JOHNSON SYNDROME AND TOXIC EPIDERMAL NECROLYSIS ERYTHEMA NODOSUM	Dry Skin Therapy SUNSCREENS AND PERVENTATIVE 38 THERAPY

HISTORY
☐ age, race, occupation, hobbies
details of skin eruption
• location
• onset
 persistent/intermittent
 factors affecting eruption (aggravating/relieving factors)
 factors affecting eruption (aggravating/relieving factors) associated skin symptoms (itchiness/burning/pain/dry/discharge) past history of skin eruptions associated systemic symptoms environmental and psychological factors
□ past history of skin eruptions
☐ associated systemic symptoms
 environmental and psychological factors
☐ allergies, medications
past medical history in family history of skin and internal disease
☐ family history of skin and internal disease
PHYSICAL EXAM
□ distribution
□ colour
□ type of lesion (see morphological definitions)
☐ arrangement (annular, linear, etc)
 type of lesion (see morphological definitions) arrangement (annular, linear, etc) remember to examine hair, mucous membranes and nails

DEFINITIONS

LICTODY

PRIMARY MORPHOLOGICAL LESIONS

TRIVITATI MORE MOLOGICAL ELSIONS		
Table 1. Types of Lesions		
	< 1 cm diameter	≥ 1 cm diameter
raised superficial lesion	papule (e.g. wart)	plaque (e.g. psoriasis)
palpable deep (dermal) lesion (not necessarily raised)	nodule (e.g. dermatofibroma)	tumour (e.g. lipoma)
flat lesion	macule (e.g. freckle)	patch (e.g. vitiligo)
elevated fluid filled lesions	vesicle (e.g. HSV)	bulla (e.g. bullous pemphigoid)
□ pustule: a vesicle that contains purulent exudate (white, yellow, green) (e.g. pustular acne) □ erosion: a disruption of the skin involving the epidermis alone □ ulcer: a disruption of the skin that extends into the dermis or deeper		

 □ wheal: a special form of papule or plaque that is blanchable and transient, formed by edema in the dermis (e.g. urticaria)
 □ scar: replacement fibrosis of dermis and subcutaneous tissue SECONDARY MORPHOLOGICAL LESIONS ☐ crust: dried serum, blood, or purulent exudate originating from a lesion (e.g. impetigo) scale: excess keratin (e.g. seborrheic dermatitis) fissure: a linear slit-like cleavage of the skin excoriation: a scratch mark lichenification: thickening of the skin and accentuation of normal skin markings (e.g. chronic atopic dermatitis)

xerosis: dryness of skin, eyes and mouth ☐ atrophy: histological decrease in size and number of cells or tissues

OTHER MORPHOLOGICAL LESIONS comedones: collection of sebum and keratin

- open comedone (blackhead)closed comedone (whitehead)
- petechiae: hemorrhagic punctate spot, 1-2 mm in diameter, non-blanchable
 purpura: extravasation of blood resulting in red discolouration
 ecchymosis: macular red or purple hemorrhage > 2 mm diameter

ACNE VULGARIS/COMMON ACNE

(see Colour Atlas A5)

☐ a common inflammatory pilosebaceous disease characterized by comedones, papules, pustules, inflamed nodules and cysts, with occasional scarring

predilection sites: face, neck, upper chest, back

more severe in males than females

pathogenesis

androgens stimulate increased sebum production
sebum is comedogenic, an irritant, and is converted to free fatty acids by microbial lipases made by anaerobic diphtheroid Propionibacterium acnes

free fatty acids + bacteria = inflammation plus delayed hypersensitivity reaction causing hyperkeratinization of follicle lining with resultant plugging

exacerbating factors

menstruation

oral contraceptive pill (OCP) (low estrogen formulations)
 corticosteroids

- lithium, iodides, bromides
- comedogenic topical agents some cosmetics, sunscreens, moisturizers, greases, tars

 • NB: foods are NOT a major aggravating factor

 □ treatment based on severity of acne

4 types of acne severity
I - Comedonal. Few lesions. No scarring
II - Papular. Moderate number of lesions. Little scarring
III - Pustular. Lesions > 25. Moderate scarring
IV - Nodulocystic. Severe scarring

Acne Type	Treatment
Type I – Non-inflammatory	Benzoyl Peroxide (2.5%, 5%, 10%) – bactericidal
Type 1 from minimizery	Adapalene gel/cream
	• not irritating, no interaction with sun
	• expensive
	+/- Tretinoin (Retin-A)
	comedolytic more sun-sensitive
	• start with 0.01% and increase to 0.025% after one month
Type I – Inflammatory	Benzoyl Peroxide
	Tretinoin/ Adapalene gel/cream
	Topical Antibiotic (clindamycin, erythromycin)
	bacteriostatic and anti-inflammatory
Type II	Topical Antibiotic
	Benzoyl Peroxide
	Tretinoin/ Adapalene gel/cream
Type III	Topical Antibiotic
	Benzoyl Peroxide
	Tretinoin
	Oral Antibiotic (tetracycline, minocycline, erythromycin)
Type IV	Isotretinoin (Accutane)
	• 0.5 to 1.0 mg/kg/day for 3-4 months
	 baseline CBC, pregnancy tests, LFT, TG, and cholesterol prior to start of therapy repeat tests at 2/6/10/14 weeks
	 S/E: teratogenic, skin and mucous membrane dryness, hyperlipidemi reversible alopecia, abnormal LFT

other treatments

- cryotherapy (for cysts)intralesional steroids (for cysts)
- dermabrasion
- spironolactone antiandrogen

	 Diane-35 OCP (cyproterone acetate + ethinyl estradiol) high-estrogen OCP differential diagnosis rosacea folliculitis perioral dermatitis
	OSACEA (see Colour Atlas A6) a chronic and recurrent inflammatory disorder of the pilosebaceous units and vasculature of the face characterized by telangiectases, flushing (due to capillary vasodilation), papules, and pustules differentiated from acne by its absence of comedones • F>M, 30-50 years old • symmetrical; forehead, cheeks, nose, chin, eyes • may get conjunctivitis, blepharitis, episcleritis, or keratitis • may develop rhinophyma (nose enlargement) prolonged course common, recurrences common, may disappear spontaneously unknown pathogenesis exacerbating factors • heat, cold, wind, sun, stress, drinking hot liquids, alcohol, caffeine, spices treatment
_	 topical antibiotics (metronidazole 0.75% gel or cream, clindamycin or erythromycin have anti-inflammatory mechanisms) systemic tetracycline or erythromycin 250 mg qid then as needed alternatives: minocycline
۵	 others lasers for telangiectases plastic surgery or laser for rhinophyma camouflage makeup for erythema differential diagnosis SLE carcinoid syndrome acne vulgaris perioral dermatitis
	ERIORAL DERMATITIS discrete erythematous micropapules that often become confluent forming inflammatory plaques on perioral and periorbital skin subset of acneiform conditions • initial lesions usually in nasolabial folds, symmetry common, rim of sparing around vermilion border of lips • 15 to 40 year old • females predominantly • can be aggravated by potent topical (fluorinated) corticosteroids
	treatment • topical • metronidazole 0.75% gel or cream to area bid • systemic • tetracycline 500 mg bid until clear, then 500 mg daily for 1 month, then 250 mg daily for 1 additional month

DERMATITIS (ECZEMA)

 superficial inflammation of the sk papulovesicles, redness, crusting to scratching 	rin, characterized by pruritic g, scaling, and lichenification secondary
hypersensitivity reaction clinical suspicion by discrete area susceptibility to allergen is acqui persists indefinitely allergens include poison ivy, rubbinickel diagnosis by patch testing treatment avoid allergen and its cros wet compresses soaked in change q3h, betamethason	cion caused by cell-mediated delayed a of skin involvement ired and specific sensitivity usually ber/latex, neomycin, dyes, lanolin, s reactants Burow's solution (a drying agent),
IRRITANT CONTACT DERMATITIS (see Colour Atlas A2) □ eczema is ill-marginated □ physical/chemical damage: damage to water and lipid-holding barrier, denaturing keratin and other proteins □ irritants include soaps, weak alkali, detergents, organic solvents, alcohol, oils □ irritant and allergic dermatitis accounts for 30% of industrial-related medical disability □ treatment • avoidance, compresses, topical and oral steroids	
ATOPIC DERMATITIS (see Colour Atlas A3) □ subacute and chronic eczematous reaction caused by Type I (IgE-mediated) hypersensitivity reaction (release of histamine) producing prolonged severe pruritus □ increased personal or family history of atopy (asthma, eczema, hay fever) • 3% of infants – 50% clear by age 13, few persist > 30 years of age • polygenic inheritance: one parent > 60% chance for child; two parents > 80% chance for child □ associated findings • keratosis pilaris (hyperkeratosis of hair follicles, "chicken skin") • xerosis • atopic palms: prominent palmar creases • inflammation, lichenification, excoriations are 2° to relentless scratching	
Table 3. Phases of Atopic Derr	matitis
Phase	Distribution
Infant (onset at 2-6 months old)	Face, scalp, extensor surfaces

Table 3. Phases of Atopic Dermatitis	
Phase Distribution	
Infant (onset at 2-6 months old) Childhood (>18 months) Adult	Face, scalp, extensor surfaces Flexural surfaces Hands, feet, flexures, neck, eyelids, forehead, face, wrists

- treatment
 bath additive (Aveeno oatmeal) followed by application of unscented emollients, or menthol (cooling agent)
 topical corticosteroids with oral antihistamines

 avoid prolonged potent dose; hydrocortisone cream for maintenance
 alternate with lubricants or tar solution

 antibiotic therapy if 2° infection by *S. aureus* avoid systemic corticosteroids

SEBORRHEIC DERMATITIS (see Colour Atlas A4) ☐ greasy, erythematous, yellow, non-pruritic scaling papules and plaques occurs in areas rich in sebaceous glands • sites: scalp, eyebrows, eyelashes, beard, face (flush areas,

behind ears, forehead), trunk, body folds, genitalia

possible etiologic association with the yeast Pityrosporum ovale

increased incidence in AIDS and Parkinson's patients

□ treatment

- face: non-fluorinated hydrocortisone cream
 scalp: salicylic acid in olive oil (to remove scale), 2% ketoconazole shampoo (Nizarole), low potency steroid lotion

STASIS DERMATITIS (see Colour Atlas A1)

- persistent skin inflammation of the lower legs with a tendency toward brown pigmentation, erythema, and scaling
 - commonly associated with venous insufficiency
 - complications: secondary bacterial infections, ulceration

□ treatment

- support stocking
- rest and elevate legs
- moisturizer to treat xerosis
- mild topical corticosteroids to control inflammation
- surgical vein stripping for cosmetic reasons only

NUMMULAR DERMATITIS

- □ annular coin-shaped pruritic plaques
 dry, scaly, lichenified

 - · often associated with atopy and dyshydrotic eczema
- □ treatment
 - potent corticosteroid ointment bid or intalesional triamcinolone injection if severe

DYSHYDROTIC DERMATITIS

- papulovesicular dermatitis of hand and foot; may become lichenified with scaly plaques
- ☐ misnomer pathophysiology is NOT related to sweating
- ☐ treatment
 - topical
 - · high potency corticosteroid with saran wrap occlusion to increase penetration
 - intralesional triamicinolone
 - systemic
 - prednisone in severe cases
 - antibiotics for 2° S. aureus infection

DIAPER DERMATITIS

(see Pediatrics Notes)

BACTERIAL

often involve the epidermis, dermis, hair follicles or periungual region.

☐ may also be systemic

SUPERFICIAL (EPIDERMAL)

Impetigo Vulgaris (see Colour Atlas F5)

☐ acute purulent infection which appears vesicular and progresses to crusting (crust is golden yellow and appears stuck on)
• agent: GABHS, S. aureus, or both

- sites: commonly involves the face, arms, legs and buttocks
- affected: preschool and young adults living in crowded conditions, poor hygiene, neglected minor trauma

complication: post-strep. glomerulonephritis

□ treatment

- · remove crusts and use saline compresses, plus topical antiseptic soaks bid
- topical antibacterials such as mupirocin or fucidin, continued for 7-10 days after resolution
- systemic antibiotics such as cloxacillin or cephalexin

☐ differential diagnosis

infected eczema, herpes simplex, varicella

Bullous Impetigo

- scattered, thin walled bullae arising in normal skin and containing clear yellow or slightly turbid fluid with no surrounding erythema
 - agent: S. aureus group II elaborating exfoliating toxin
 sites: trunk, intertriginous areas, face

- affected: neonates and older children, epidemic especially in day care
- complication: high levels of toxin in immunocompromised or young children may lead to generalized skin peeling or staphylococcal scalded skin syndrome (SSSS)

□ treatment

- cloxacillin
- · topical antibacterials such as fucidin and mupirocin, continued for 7-10 days

Erythrasma

- infection of the stratum corneum that manifests as a sharply demarcated, irregularly shaped brown, scaling patch
 - agent: Corynebacterium minutissimum
 - sites: intertriginous areas of groin, axillae, intergluteal folds, submammary, toes
 - affected: obese, middle-aged, blacks, diabetics, living in warm humid climate
 - diagnosis: "coral-red" fluorescence under Wood's light (365 nm) because of a water-soluble porphyrin

□ treatment

- econazole cream applied bid and showers with povidone-iodine
- erythromycin (250 mg qid for 14 days) for refractory cases or recurrences
- ☐ differential diagnosis
 - tinea cruris (positive scraping for hyphae)
 - seborrheic dermatitis (no fluorescence)

DEEPER (DERMAL)

Table 4. Comparison of Erysipelas and Cellulitis	
Erysipelas (see Colour Atlas F8)	Cellulitis
upper dermis	lower dermis/subcutaneous fat
may be confluent, but well demarcated and raised, often with vesicles	poorly demarcated, not uniformly raised
Group A streptococcus	GAS (most common), S. <i>aureus</i> (usually in significantly sized wounds, doesn't spread as much), H. <i>flu</i> (especially periorbital in kids < 5 years old, may be blue), <i>Pasteurella multocida</i> (dog/cat scratch/bite)
spreads through lymphatics; long term recurrent erysipelas can cause elephantiasis	
PAINFUL (once called St. Anthony's fire)	
systemic symptoms: fever, chills, headache, weakness	systemic symptoms less likely (but may have fever, leukocytosis and lymphadenopathy)
more serious	
complications include scarlet fever, streptococcal gangrene, fat necrosis, coagulopathy	
face and legs	commonly legs
first line: Penicillin, Cloxacillin or Ancef second line: Clindamycin or Keflex If penicillin allergic, can use Erythromycin	first line: Cloxacillin or Ancef/Keflex second line: Erythromycin or Clindamycin Cefuroxime in young kids; TMP/SMX + Metronidazole in diabetic foot infections
 check for history of trauma, bites, saphenous vein graft, etc, but often no inciting cause identified rarely culture bacteria by skin/blood culture; clinical diagnosis. If suspecting necrotizing fasciitis, do immediate biopsy and frozen section histopathology DDx: DVT (less red, less hot, smoother), superficial phlebitis, RSD 	

HAIR FOLLICLES

Superficial Folliculitis □ superficial infection of the hair follicle □ pseudofolliculitis: inflammation of follicle due to friction, irritation or occlusion □ acute lesion consists of a superficial pustule surrounding the hair • can occur on face (Staphylococcus most common), beard area, scalp or legs, trunk (Pseudomonas), or back (Candida) • common in AIDS □ treatment • topical antibacterial (fucidin, mupirocin or erythromycin), • oral cloxacillin for 7-10d • mupirocin for S. aureus in nostril and on involved hairy area
Furuncles (Boils) □ red, hot, tender, inflammatory nodules involving subcutaneous tissue that evolves from a <i>Staphylococcus folliculitis</i> • occurs where there are hair follicles and in areas of friction and sweat (nose, neck, face, axillae, buttocks) □ if recurrent, rule out diabetes or hidradenitis suppurativa (if in groin or axillae)

Carbuncles

- deep seated conglomerate of multiple coalescing furuncles
- ☐ treatment
 - incise and drain large carbuncles to relieve pressure and pain

 - if afebrile: hot wet packs, topical antibiotic
 if febrile/cellulitis: culture blood and aspirate pustules (Gram stain and C&S)
 - cloxacillin for 1 to 2 weeks

PERIUNGUAL REGION

Paronychia

- ☐ inflammation around nail
 - can cause nail dystrophy
 - acute: S. aureus, Streptococcuschronic: C. albicans

□ treatment

- · avoid exposure to moisture
- topical fucidin or clotrimazole

OTHERS

Syphilis sexual

- sexually transmitted infection caused by Treponema pallidum
- characterized by a painless ulcer (chancre)

 ordering inoculation becomes a systemic infection with secondary
- and tertiary stages

 primary syphilis (see Colour Atlas F11)

 single red, indurated, PAINLESS, round/oval, indolent, chancre (buttonlike papule) that develops into
 - painless ulcer with raised border and scanty serous exudate chancre develops at site of inoculation after 3 weeks of incubation and heals in 4-6 weeks
 - regional non-tender lymphadenopathy appears < 1 week after onset of chancre VDRL negative

 - darkfield examination (for primary) spirochete in tissue fluid from chancre or lymph node aspirate
 - M:F = 2:1
 - treatment: benzathine penicillin G 2.4 million units IM
 differențial diagnosis
- chancroid: painful
 HSV: multiple lesions
 secondary syphilis (see Colour Atlas F13)
 appears 2-10 weeks after initial chancre, and 2-6 months after primary infection
 - general exam: generalized lymphadenopathy, splenomegaly, +/- fever
 - lesions heal in 1-5 weeks, and may recur for 1 year
 - types of lesions

 - 1. macules and papules, round to oval, flat top, scaling, non-pruritic, sharply defined, circular (annular) rash

 trunk, head, neck, palms, soles, mucous membranes

 differential diagnoses: pityriasis rosea, tinea corporis, drug eruptions, lichen planus
 - 2. condyloma lata: moist papules around genital/perianal regionexudate teeming with spirochetes

 - differential diagnosis includes condyloma acuminata
 - 3. mucous patches: macerated patches mainly found in oral mucosa
 - associated findings: pharyngitis, iritis, periostosis, "acute illness" syndrome headache, chills, fever, arthralgia, myalgia, malaise, photophobia

 - VDRL positive
 FTA-ABS +ve; -ve after 1 year following appearance of chancre
 TPI +ve; darkfield +ve in all secondary syphilis except macular
 - serologic test may be -ve if undiluted serum, or if HIV-infected
 - treatment as for primary syphilis

 tertiary syphilis extremely rare 3-7 years after secondary main skin lesion: 'Gumma' - a granulomatous nodule independent of other tertiary syphilis manifestations VDRL: blood positive, CSF negative
Gonococcemia (Disseminated Gonococcal Infection) □ pustules on a purpuric erythematous base and hemorrhagic, tender, necrotic pustules (aka "arthritis-dermatitis syndrome") • Gram negative diplococcus <i>Neisseria gonorrheae</i> • skin manifestations develop in gonococcemia with vasculitis • distal aspects of extremities • associated with fever, asymmetric oligoarticular arthritis, and tenosynovitis • conjunctivitis if infected via birth canal • examine contacts and notify authorities • look for syphilis and other STDs • avoid intercourse until negative cultures • do not confuse with skin lesion of meningococcemia: petechiae which may evolve into purpura and ecchymosis (see Colour Atlas F1) □ treatment: ceftriaxone (drug of choice)
VIRAL
Herpes Simplex (see Colour Atlas F12) □ grouped vesicles (herpetiform arrangement) on an erythematous base on skin or mucous membranes • transmitted via contact with erupted vesicles • primary • children and young adults • usually asymptomatic • may have high fever, regional lymphadenopathy, malaise • secondary • recurrent form seen in adults • prodrome of tingling, pruritus, pain • much more commonly diagnosed than primary
HSV I □ recurrent on face, lips □ rarely on mucous membranes (rule out aphthous ulcer) □ virus in posterior root ganglion (Gasserian ganglion of trigeminal nerve, sacral ganglion) □ reactivated by: sunlight, fever, menstruation, stress, upper respiratory infection, physical trauma □ differential diagnosis • impetigo • eczema
HSV II incubation 2-20 days gingivostomatitis (entire buccal mucosa involved with erythema and edema of gingiva) vulvovaginitis (edematous, erythematous, extremely tender, profuse vaginal discharge) urethritis (watery discharge in males) recurrent on vulva, vagina, penis, lasting 5-7 days 8% risk of transmission to neonate via birth canal if mother is asymptomatic diagnosis confirmed with −ve darkfield, −ve serology for syphilis, −ve bacterial cultures • Tzanck smear shows multinucleated giant epithelial cells with Giemsa stain • tissue culture and EM on vesicular fluid • skin biopsy (intraepidermal, ballooning degeneration, giant cells)
antibody titres increase 1 week after primary infection, however, increase in titres are not diagnostic of recurrence

 treatment rupture vesicle with sterile needle tepid wet dressing with aluminum subacetate solution, Burow's compression, or betadine solution acyclovir: 200 mg PO, 5 times a day for 10 days for 1st episode topical therapy is generally not as effective famciclovir and valacyclovir may be substituted complications dendritic corneal ulcers stromal keratitis erythema multiforme herpes simplex encephalitis HSV infection on atopic dermatitis causing Kaposi's varicelliform eruption (eczema herpeticum)
Differential Diagnosis of Genital Ulcerations HSV II multiple syphilitic chancres chancroid Candida balanitis lymphogranuloma inguinale
Herpes Zoster (shingles) (see Colour Atlas F9) a localized infection caused by varicella zoster virus characterized by unilateral pain and vesicular/bullous eruption limited to a dermatome risk factors: old age, immunosuppression, occasionally associated with hematologic malignancy occurs when decreased cellular and humoral immunity to VZV thoracic (50%), trigeminal (10-20%), cervical (10-20%), disseminated in HIV patients eruption begins day 3-5 after pain and paresthesia of a dermatome lesions usually last days-weeks pain: pre-herpetic, synchronous with rash, or post-herpetic and may persist for months and years severe post-herpetic neuralgia often occurs in elderly if tip of nose involved = eye involvement (conjunctivitis, keratitis, scleritis, iritis) treatment compresses with normal saline, Burow's, or betadine solution analgesics NSAID, amitriptyline for patients over 50 years old or with severe acute pain or ophthalmic involvement famciclovir 500 mg tid for 7 days or valacyclovir 1000 mg tid for 7 days or acyclovir 800 mg 5x day for 7 days (if immunocompromised)
Clinical Pearl ☐ In Herpes Zoster, antiviral treatment must be started within 72 hours of the onset of rash unless ophthalmic involvement
 differential diagnosis MI, pleural disease, acute abdomen, vertebral disease contact dermatitis localized bacterial infection zosteriform herpes simplex virus (more pathogenic for the eyes than varicella zoster)
Hand-Foot-and-Mouth Disease ☐ grey vesicles in parallel alignment to palmar and plantar creases of hands, feet and diaper area with a painful ulcerative exanthem over buccal mucosa and palate • young children often presenting with refusal to eat • Coxsackie A16, highly contagious • 3-6 day incubation, resolves in 7-10 days ☐ treatment • xylocaine gel as analgesic

Molluscum Contagiosum (see Colour Atlas F3) ☐ discrete dome-shaped and umbilicated pearly white papules caused by DNA pox virus • afflicts both children and adults • pock avilles trunk peringum evolids (may cause conjunctivitis)
 neck, axillae, trunk, perineum, eyelids (may cause conjunctivitis) M > F, HIV patients (common on face in AIDS patients) transmission: direct contact, auto-inoculation, sexual
 treatment topical cantharidin (painless application, blisters within days) liquid nitrogen cryotherapy (10-15 seconds) curettage
 differential diagnosis fibromata, nevi, keratoacanthoma, basal cell carcinoma
Verruca Vulgaris (Common Warts) (see Colour Atlas F4) ☐ hyperkeratotic, elevated discrete epithelial growths with papillated surface • human papilloma virus (HPV) • trauma site: fingers, hands, knees of children and teens
• paring of surface reveals punctate red-brown specks (dilated capillaries) ☐ treatment
 65-90% resolve spontaneously over several years salicylic acid paste (keratolytic) cryotherapy with liquid nitrogen (10-30 seconds); no scar but
hypopigmentation light electrodesiccation, curettage with local anesthesia
 differential diagnosis seborrheic keratosis, molluscum contagiosum
Verruca Plantaris (Plantar Warts) ☐ hyperkeratotic, shiny, sharply marginated papule/plaque • pressure sites: heads of metatarsal, heels, toes • paring of surface reveals red-brown specks (capillaries),
interruption of epidermal ridges treatment
 none if asymptomatic, disappears in 6 months if tender on lateral pressure, 40% salicylic acid plaster for 1 week then cryotherapy
 differential diagnosis need to scrape ("pare") lesions to differentiate wart from callus and corn callus: paring reveals uniformly smooth surface with no interruption of epidermal ridges
 corn (caused by underlying bony protuberance): paring reveals shiny keratinous core, painful to vertical pressure
Verruca Plana (Flat Wart) ☐ numerous discrete, skin coloured, flat topped papules occurring in linear configuration
• face, dorsa of hands, shins □ treatment
electrodesiccationcryotherapy
Condylomata Acuminata (Genital Warts) (see Colour Atlas D7) skin coloured pinhead papules to soft cauliflower like masses in clusters voung adults, infants, children asymptomatic, last months to years genitalia and perianal areas F: from cervix to labia and perineum
M: from meatus to scrotum
 highly contagious, transmitted sexually and non-sexually can spread without clinically apparent lesions
 this HPV is immunologically distinct from HPV of verruca vulgaris types 6 and 11 are the most common causes types 16, 18, 31, 33 cause cervical dysplasia, squamous cell
cancer and invasive cancer of vagina and penis children vaginally delivered to infected mothers at risk
for anogenital condylomata and respiratory papillomatosis acetowhitening: subclinical lesions seen with 5%
acetic acid x 5 minutes and hand lens (tiny white papules) • false positives due to psoriasis, lichen planus

 treatment podophyllin (contraindicated in pregnancy) liquid nitrogen, electrocautery trichloroacetic acid (80-90%), intralesional interferon surgery only needed for giant lesions differential diagnosis condylomata lata (secondary syphilitic lesion, darkfield strongly + ve) molluscum contagiosum lichen planus pearly penile papules
DERMATOPHYTES (SUPERFICIAL FUNGAL INFECTION OF SKIN) □ caused by Trichophyton, Microsporum, Epidermophyton □ live on dead superficial skin by digesting keratin therefore result in scaly skin, broken hairs and crumbling nails □ diagnose using skin scrapings, hair, and nail clippings analyzed with KOH prep (since these fungi live as molds, look for hyphae, and mycelia) □ general principles of treatment • topicals are not first line therapy for all dermatophytes • topicals may be used as first line agents for tinea corporis/cruris and tinea pedis (interdigital type) • main topicals are clotrimazole or terbinafine • otherwise treat orally with terbinafine (Lamisil) or itraconazole (Sporanox) • itraconazole is a P-450 inhibitor. It alters metabolism of non-sedating antihistamines, cisapride, digoxin, and HMG CoA reductase inhibitors
Tinea Capitis (see Colour Atlas A10) □ Non-scarring alopecia with scale, caused by Trichophyton tonsurans and Microsporum species • affects children (mainly black), immunocompromised adults • may see black-dot broken off hairs, kerion (boggy, elevated, purulent, inflamed nodules or plaques), or yellow crust depending on organism • very contagious and may be transmitted from barber, hats, theatre seats, pets • Wood's light examination of hair: green fluorescence only for microsporum infection □ differential diagnosis • psoriasis, seborrheic dermatitis, alopecia areata, trichotillomania
Tinea Corporis/Tinea Cruris (Ringworm) (see Colour Atlas F14) scaling plaques with papular, sharp margins, occurring in an annular arrangement (with peripheral enlargement and central clearing) T. corporis trunk, limbs, face T. rubrum, E. floccosum, M. canis (kids in contact with puppies or kittens) T. cruris
 intertriginous areas, upper thigh, buttock T. rubrum, E. floccosum often concurrent tinea pedis note: take scraping from advancing border differential diagnosis candidiasis (involvement of scrotum, satellite pustules, no sharp border) erythrasma (coral-red fluorescence with Wood's lamp, rods and filaments, axilla and webs of toes) contact dermatitis (often superimposed on tinea due to home remedy)
Tinea Pedis (Athlete's Foot) acute: red/white, scales, maceration, vesicles, bullae interdigital heat, humidity, occlusive footwear may present as flare-up of chronic tinea pedis frequently become secondarily infected by bacteria

 chronic non-pruritic, pink, scaling keratosis on soles, and sides of foot, often in a "moccasin" distribution differential diagnosis hyperkeratosis allergic contact dermatitis (dorsum/heel) erythrasma, intertrigo (interdigital) psoriasis (soles or interdigital) 	
Tinea Manuum □ acute: blisters at edge of red areas □ chronic: single dry scaly patch • primary fungal infection of the hand is actually quite rare; usua associated with tinea pedis with one hand and two feet affects = "1 hand 2 feet" syndrome □ differential diagnosis • contact dermatitis, atopic dermatitis, psoriasis (all three commonly mistaken for fungal infections) • granuloma annulare (annular)	al eo
Tinea Unguium (Onychomycosis) (see Colour Atlas A9) □ crumbling, distally dystrophic nails • treat with Terbinafine (fingernails 6 weeks, toenails 12 weeks) or with Itraconazole (fingernails 2 pulses, toenails 3 pulses) • a pulse = 1 week per month of 200 mg bid □ differential diagnosis • psoriasis (pitting, may have psoriasis elsewhere) • candidiasis (hands in water) • hyperthyroidism	
YEAST	
Pityriasis (tinea) Versicolour (see Colour Atlas F7) □ chronic asymptomatic superficial fungal infection with brown/white scaling macules • etiology: Malassezia furfur (Pityrosporum orbiculare) • young adults • affected skin darker than surrounding skin in winter, lighter in summer (doesn't tan) • sites: upper trunk most common seen on face in dark skinned individuals • predisposing factors: summer, temperate climates, Cushing's syndrome, prolonged corticosteroid use • diagnosis: direct microscopic exam of scales for hyphae and spores prepared in KOH, Wood's lamp (faint yellow-green fluorescence) □ treatment • scrub off scales with soap and water • selenium sulfide	
 selenium suinde ketoconazole cream or 200mg PO daily for 10 days 	
Candidiasis (see Colour Atlas F10) ☐ Candidal paronychia: painful red swellings of periungual skin ☐ Candidal intertrigo: red patches with pustular borders in areas of skin folds ☐ often under breast, groin, interdigital ☐ predisposing factors - obesity, diabetes, systemic antibiotics	
 Întertrigo starts as non-infectious maceration from heat, moisture and friction; evidence that it has been infected by intertrigo is a pustular border treat by keeping area dry, miconazole 	

PARASITIC

Scabies (see Colour Atlas F2) a transmissible parasitic skin infection (Sarcoptes scabiei, a mite), characterized by superficial burrows, intense pruritus and secondary infection secondary lesions: small urticarial crusted papules, eczematous plaques, excoriations sites: axillae, cubitus, wrist, side of palm, web spaces, groin, buttocks, back of ankle, toes, penis sexual promiscuity, crowding, poverty, nosocomial intractable pruritus worse at night (mite more active; pruritus is also worse at night) also worse at night)
adults: scalp, face, upper back spared
infants: scalp, face, palms/soles involved
immunocompromised: Norwegian Scabies = Crusted Scabies; all over body
scabies mite remain alive 2-3 days on clothing/sheets
incubation = 1 month, then begin to itch
re infection followed by bypersensitivity in 24 hours re-infection followed by hypersensitivity in 24 hours microscopic examination of root and content of burrow with KOH for mite, eggs, feces □ treatment bathe then apply Permethrin 5% cream (i.e. Nix) or Kwellada from head (not neck) down to soles of feet (must be left on for Nix is preferred in children (seizures reported with Kwellada) • may require second treatment 7 days after first treatment change underwear and linens
+/- antihistamine treat family and contacts
 pruritus may persist for 2-3 weeks due to prolonged hypersensitivity reaction ☐ differential diagnosis dermatitis herpetiformis: see vesicles, urticaria, eosinophilia, no burrows asteatotic eczema ("winter itch") · neurotic excoriation Lice (Pediculosis) intensely pruritic red excoriations, morbilliform rash, *Pediculus humanus* scalp lice: nits on hairs · red excoriated skin with secondary bacterial infection, lymphadenopathy pubic lice: nits on hairs excoriations rarely in chronic cases: "maculae ceruleae" = bluish grey, pea-sized macules body lice: nits and lice in seams of clothing
 excoriations and secondary infection mainly on shoulders, belt-line and buttocks □ treatment Permethrin 1% (Nix) cream rinse (ovicidal)
Kwellada shampoo (kills newly hatched nits) · comb hair with fine-toothed comb using dilute vinegar solution toremove nits repeat in 7 days
change and clean bedding, clothing and towels
for body lice, washing clothes is essential ☐ differential diagnosis bacterial infection of scalp: responds rapidly to antibiotic
seborrheic dermatitis: flakes of dandruff readily detached
hair casts: pulled more easily than nits, no eggs on microscopy

PSORIASIS	(see	Colour	Atlas	A7)

types

- plaque psoriasis
- guttate psoriasiserythrodermic psoriasis
- pustular psoriasispsoriatic arthritis

☐ differential

- seborrheic dermatitis
- · chronic dermatitis
- mycosis Fungoides (cutaneous T-cell lymphoma)

PLAQUE PSORIASIS

- a common chronic and recurrent disease characterized by dry, well-circumscribed, silver scales over erythematous papules/plaques, mostly at sites of repeated trauma
 - sites: scalp, extensor surfaces of elbows and knees, trunk, nails, pressure areas

worse in winter (lack of sun and humidity)
multifactorial inheritance: 30% with family history and HLA markers pathogenesis: decrease epidermal transit time from basal to horny layers and shortened cell cycle of psoriatic and normal skin Koebner phenomenon (isomorphic response): induction of new lesion

☐ Auspitz's sign: bleeds from minute points when scale is removed ☐ exacerbating factors: drugs (lithium, ethanol, chloroquine, beta-blockers), sunlight, stress, obesity

topical and systemic

Table 5. Topical Treatment of Psoriasis			
Treatment	Mechanism	Comments	
lubricants	reduce fissure formation		
salicylic acid 1-12%	remove scales		
anthralin .1%, .2%, .4%	increase cell turnover	stains and irritates normal skin	
tar (Liquor carbonis detergent)	inhibits DNA synthesis, increase cell turnover	poor longterm compliance	
calcipotriol (vit. D derivative; Dovenex)	binds to skin 1, 25-dihydroxyvitamin D3 to inhibit keratinocyte proliferation	not to be used on face or skin folds	
corticosteroid ointment	reduce scaling and thickness	use appropriate potency steroid in different areas and degree of psoriasis	
tazarotene	retinoid derivative		
Goeckermann regimen: UVB + tar		UVB 290-320 nm	

Table 6. Systemic Treatment of Psoriasis		
Treatment	Adverse Effects	
methotrexate	bone marrow toxicity, hepatic cirrhosis	
steroids	rebound effect when withdrawn	
PUVA (8 methoxy-psoralen and UVA 360-440 nm)	pruritus, burning, cataracts, skin cancer	
acetretin	alopecia, cheilitis, teratogenicity, epistaxis, xerosis, hypertriglyceridemia	
cyclosporine	renal toxicity, hypertension, immunosuppression	

GUTTATE PSORIASIS ("drop-like")

□ discrete, scattered salmon-pink scaling papules
• sites: generalized (mainly trunk and proximal extremities), sparing palms and soles

often antecedent streptococcal pharyngitis

□ treatment

UVB phototherapy, sunlight, lubricants
penicillin V or erythromycin if Group A beta-hemolytic Streptococcus on throat culture

ERYTHRODERMIC PSORIASIS

- generalized erythema with fine desquamative scale on surface, with islands of spared skin
- may present in patient with previous mild plaque psoriasis
 aggravating factors: lithium, beta-blockers, NSAIDs, antimalarials,
 - phototoxic reaction, infection
 - associated symptoms: worse arthralgia, severe pruritus
- □ treatment
 - hospitalization, bed rest, IV fluids, monitor fluid and lytes
 treat underlying aggravating condition

 - methotrexate
 - PUVA and retinoids

PUSTULAR PSORIASIS

- □ sudden onset of erythematous macules and papules which evolve into pustules rapidly; can be generalized (von Zumbusch type) or localized (acropustulosis or pustulosis of palms and soles)
- ☐ uncommon
 - patient may have no history of psoriasis, or was recently inappropriately withdrawn from steroid therapy. It also may occur in the 3rd trimester of pregnancy (impetigo herpatiformis)
 associated symptoms: fever, arthralgias, diarrhea, \(^{\(\)}\) WBCs
- □ treatment
 - · bed rest, withdraw exacerbating medications, monitor lytes
 - methotrexate and etretinate (start with low dose)
 localized PUVA for pustulosis of palms and soles

PSORIATIC ARTHRITIS

- 5 categories

 - asymmetric oligoarthropathy
 DIP joint involvement is predominant
 rheumatoid pattern symmetric polyarthropathy
 psoriatric arthritis mutilans

 - predominant spondylitis or sacroilitis

Table 7. Psoriasis by distribution			
Location	Signs and Symptoms	Treatment	
scalp	dry, scaling, well demarcated, reddish, lichenified plaques (no hair loss), mild to severe itching, sunlight does not cause remission	tar shampoo followed by betamethasone valerate 0.1% lotion biweekly If severe (thick plaques) remove plaque with 10% salicylic acid in mineral oil and cover with plastic cap overnight (1-3 treatments) fluocinolone cream/lotion with cap overnight maintenance with scalp lotion (clobetasol propionate 0.05%)	
nails	onycholysis, pitting, subungal hyperkeratosis, oil spots	intradermal triamcinolone acetonide 5 mg/mL PUVA methotrexate	
palms and soles	sharply demarcated dusky-red plaques with thick scales on pressure points; can be pustular	PUVA retinoids methotrexate	

LICHEN PLANUS

- acute or chronic inflammation of mucous membranes or skin characterized by violaceous, shiny, pruritic papules topped with Wickham's striae (fine white lines); milky white papules in mouth

 sites: flexor surface of wrists, lumbar region, shins, eyelids, scalp,
 - buccal mucosa, tongue, lips, nails

 scalp lesions associated with alopecia

- spntaneously resolves in weeks or lasts for years (mouth and shin
- mnemonic "6 P's: Purple, Pruritic, Polygonal, Peripheral, Papules, Penis
- precipitating factor: severe emotional stress
- associated with hepatitis C

□ treatment

- topical corticosteroids with occlusion or intradermal steroid injections
- short courses of oral prednisone (rarely)
 PUVA for generalized or resistant cases
- oral retinoids for erosive lichen planus in mouth

differential

- skin
 - drug eruption (chloroquine or gold salts)
 - lichenoid graft vs. host disease
 lupus erythematosus

- contact with colour film development chemicals
 mucous membranes
 - - leukoplakia
 - thrush
 - HIV associated hairy leukoplakialupus erythematosus

PITYRIASIS ROSEA (see Colour Atlas F6)

- acute self-limiting erythematous eruption characterized by red, oral patches and papules with marginal collarette of scale

 sites: trunk, proximal aspects of arms and legs

 etiology: human herpes virus 7

 long axis of lesions follow lines of cleavage producing

 "Chairman trees" next arm on back

- "Christmas tree" pattern on back varied degree of pruritus most start with a "herald" patch which precedes other lesions by 1-2 weeks
- clears spontaneously in 6-12 weeks

□ treatment

- no treatment needed unless itchy
- UVB in first week of eruption (5 exposures) may help pruritis

PEMPHIGUS VULGARIS □ autoimmune blistering disease characterized by flaccid, non-pruritic bullae/vesicles on an erythematous base □ etiology
 IgG produced against epidermal desmoglein 3 leading to acantholysis (epidermal cells separated from each other) producing intraepidermal bullae associated with thymoma, myasthenia gravis, malignancy,
D-penicillamine history
• 40-60 years old, patients are often Jewish or Mediterranean physical
 may present with erosions and secondary bacterial infection sites: mouth (90%), scalp, face, chest, axillae, groin, umbilicus Nikolsky's sign: bulla extends with finger pressure
immunofluorescence shows IgG and C3 deposited in epidermal intercellular spaces
□ course • mouth lesions, months later skin lesions; first
 localized (6-12 months) then generalized lesions heal with hyperpigmentation but no scar may be fatal unless treated with immunosuppressive agents
• prednisone 2.0-3.0 mg/kg until no new blisters, then
 1.0-1.5 mg/kg until clear, then taper steroid sparing agents - azathioprine, plasmapheresis, methotrexate, gold, cyclophosphamide
BULLOUS PEMPHIGOID chronic autoimmune bullous eruption characterized by pruritic, tense,
subepidermal bullae □ etiology
 TgG produced against basement membrane associated with malignancy in some
history 60-80 years old
 physical sites: flexor aspect of forearms, axillae, medial thighs, groin, abdomen, mouth (33%)
 diagnosis direct immunofluorescence shows deposition of IgG and C3 at basement membrane anti-basement membrane antibody (IgG)
coursehealing without scars if no infection
 treatment prednisone 50-100 mg (to clear) +/- steroid sparing agents
such as azathioprine tetracycline 500-1 000 mg/day +/- nicotinamide is effective for some cases dapsone 100-150 mg/day for milder cases
DERMATITIS HERPETIFORMIS intensely pruritic grouped papules/vesicles/urticarial wheals
 etiology 90% associated with gluten sensitive enteropathy (80% are asymptomatic), 30% have thyroid disease, and some have intestinal lymphoma iron or folate deficiency
□ history • 20-60 years old, M:F = 2:1
 90% håve HLA B8, DR3, DQW2 physical sites: extensor surfaces of elbows/knees, sacrum, buttocks, scalp
diagnosis • immunofluorescence: granular IgA and complement deposition
in dermis
• lesions last days - weeks □ treatment
 dapsone for pruritus but multiple side effects gluten free diet

Table 8. Vesiculobullous Diseases			
	Pemphigus Vulgaris	Bullous Pemphigoid	Dermatitis Herpetiformis
antibody	IgG	IgG	IgA
site	intercellular space	basement membrane	dermal
infiltrate	eosinophils and neutrophils	eosinophils	neutrophils
treatment	high dose steroids cyclophosphamide	moderate dose steroid cyclophosphamide	gluten-free diet/dapsone
association			gluten enteropathy

PORPHYRIA CUTANEA TARDA □ autosomal dominant or sporadic skin disorder associated with the presence of excess heme characterized by tense vesicles/ bullae in photoexposed areas
 □ etiology • associated with Hepatitis C, alcohol abuse, DM, estrogen therapy, HIV, ↑ iron
history30-40 years old, M>F
 physical facial hypertrichosis, brown hypermelanosis, "heliotrope" around eyes, bullae on extensor surfaces of hands and feet sites: light-exposed areas subjected to trauma: dorsum of hands and feet, nose, upper trunk may complain of fragile skin on dorsum of hands diagnosis Wood's lamp of urine + 5% HCl shows orange-red fluorescence immunofluorescence shows IgE at dermal-epidermal junctions treatment
 discontinue aggravating substances (alcohol, estrogen therapy) phlebotomy to decrease body iron load hydroxychloroquine if phlebotomy contraindicated
DIFFERENTIAL OF PRIMARY BULLOUS DISORDERS Drug eruptions EM and related disorders Infections - bullous impetigo Infestations - scabies (dermatitis herpetiformis) Inflammation - acute eczema

ERYTHEMA MULTIFORME (EM) / STEVENS-JOHNSON SYNDROME (SJS) / TOXIC EPIDERMAL NECROLYSIS (TEN)

Notes

 $\hfill \square$ spectrum of disorders with varying presence of characteristic skin lesions, blistering, and mucous membrane involvement

EM (minor) EM (major) SJS TEN

	Erythema Multiforme (EM) (see Colour Atlas A10)	Stevens-Johnson Syndrome (SJS)	Toxic Epidermal Necolysis (TEN) (see Colour Atlas A14)
Lesion	macules/papules with central vesicles classic bull's-eye pattern of concentric light and dark rings (target lesions) bilateral and symmetric EM minor - no mucosal involvement, bullae, or systemic symptoms EM major - mucosal involvement, bullae, systemic symptoms, usually drug induced Nikolsky sign (see pemphigus vulgaris)	EM with more mucous membrane involvement, and blistering "atypical lesions" - red circular patch with dark purple center more "sick" (high fever) sheet-like epidermal detachment in <10% Nikolsky sign	 severe mucous membrane involvement "atypical lesions" – 50% have no target lesions diffuse erythema then necrosis and sheet-like epidermal detachment in >30°
Sites	 mucous membrane involvement (oral, genital, conjunctival) extremities with face > trunk involvement of palms and soles 	generalized with prominent face and trunk involvement palms and soles may be spared	generalized nails may also shed
Other organs/ complications	corneal ulcers, keratitis, anterior uveitis, stomatitis, vulvitis, balanitis lesions in trachea, pharynx, larynx	complications: scarring, eruptive nevomelanocytic nevi, corneal scarring, blindness, phymosis and vaginal synechiae	tubular necrosis and acute renal failure, epithelial erosions of trachea, bronchi, G tract
Constitutional symptoms	fever, weakness, malaise	prodrome 1-3 days prior to eruption with fever and flu-like illness	• high fever > 38°C
Etiology	drugs – sulfonamides, NSAIDs, anticonvulsants, penicillin, allopurinol infection – herpes, mycoplasma idiopathic - >50%	50% are drug related occurs up to 1-3 weeks after drug exposure with more rapid onset upon rechallenge	80% are definitely drug related < 5% are due to viral infection, immunization
Pathology/ Pathophysiology	perivascular PMN infiltrate, edema of upper dermis	cytotoxic cell-mediated attack on epidermal cells no dermal infiltrate epidermal necrosis and detatchment above basement membrane	• same as Stevens-Johnson Syndrome
Differential diagnosis	EM minor – urticaria, viral exanthems EM major – SSSS, pemphigus vulgaris, bullous pemhigoid	scarlet fever, phototoxic eruption, GVHD, SSSS, exfoliative dermatitis	scarlet fever, phototoxic eruption, GVHD, SSSS, exfoliative dermatitis
Course and Prognosis	• lesions last 2 weeks	• < 5% mortality • regrowth of epidermis by 3 weeks	• 30% mortality due to fluid loss, secondary infection
Treatment	 prevention – drug avoidance symptomatic treatment corticosteroids in severely ill but controversial 	withdraw suspect drug intravenous fluids corticosteroids – controversial infection prophylaxis	• admit to burn unit

(SSSS = Staphylococcal Scalded Skin Syndrome)

(see Colour Atlas A15)
 acute or chronic inflammation of venules in the subcutaneous fat characterized by round, red, tender, poorly demarcated nodules 15-30 years old, F:M = 3:1 sites: asymmetrically arranged on lower legs, knees, arms lesions last for days and spontaneously resolve in 6 weeks associated with arthralgia, fever, malaise
• sites: asymmetrically arranged on lower legs, knees, arms
• lesions last for days and spontaneously resolve in 6 weeks
 associated with arthralgia, fever, malaise
□ associations
 infections: Group A Streptococcus, primary TB, coccidioidomycosis, histoplasmosis, Yersinia drugs: sulfonamides, oral contraceptives (also pregnancy) inflammation: sarcoidosis, Crohn's > ulcerative colitis
coccidioidomycosis, histoplasmosis, Yersinia
 drugs: sulfonamides, oral contraceptives (also pregnancy)
 inflammation: sarcoidosis, Crohn's > ulcerative colitis
 malignancy: acute leukemia, Hodgkin's lymphoma 40% are idiopathic
• 40% are idiopathic
 investigations: chest x-ray (to rule out chest infection and sarcoidosis throat culture, ASO titre, PPD skin test
☐ treatment
• NSAIDs
 • treat underlying cause □ differential diagnosis
u differential diagnosis
 superficial thrombophlebitis, panniculitis, erysipelas

DRUG ERUPTIONS

EXANTHEMATOUS ERUPTIONS (MACULOPAPULAR ERUPTIONS/MORBILLIFORM)

- symmetrical, widespread, erythematous patches or plaques with or without scales
 - the "classic" adverse drug reaction
 - often starts on trunk or on areas of sun exposure
 - may progress to generalized exfoliative dermatitis especially if the drug is continued
 - penicillin, sulfonamides, phenytoin (in order of decreasing probability)
 - incidence of ampicillin eruption is greater than 50% in patients with mononucleosis, gout or chronic lymphocytic leukemia

URTICARIA (also known as "Hives")

- ☐ transient, red, pruritic well-demarcated wheals
 - second most common type of drug reaction
 - due to release of histamine from mast cells in dermis
 - lasts less than 24 hours
- ANGIOEDEMA

 □ deeper swelling of the skin involving subcutaneous tissues often with swelling of the eyes, lips, and tongue
 □ may or may not accompany urticaria
 □ hereditary Angioedema does not occur with urticaria
 onset in childhood; 80% have positive family history
 recurrent attacks; 25% die from layngeal edema
 triggers: minor trauma, emotional upset, temperature changes
 diagnosis: reduced C₁ esterase inhibitor level (in 85%) or function (in 15%), diminished C4 level
 □ acquired angioedema
 autoantibodies to C₁ esterase inhibitor
 consumption of complement in lymphoproliferative disorder
 diagnosis: C₁ esterase inhibitor deficiency, decreased C₁
 - (unique to acquired form), diminished C4 level
- treatment: prophylaxIS with danazol or stanozolol
 Eprinephrine pen to temporize until patient re
 - Eprinephrine pen to temporize until patient reaches hospital in acute attack

Table 10. Classification of Urticaria			
Туре	Provocative agents/tests	Comments	
acute urticaria	Foods (nuts, shellfish, eggs, fruits) Insect stings Drugs (especially aspirin, NSAID's) Contacts – cosmetics, work exposures Infection – viral (hepatitis, upper respiratory), bacterial, parasitic Systemic diseases – SLE, endocrinopathy (TSH), neoplasm Stress Idiopathic	Attack lasts <6 weeks Each lesion lasts <24 hrs Occurs with or without angioedem	
chronic urticaria	most commonly idiopathic aggravating and causative factors may be similar to those in acute urticaria	• Attack lasts >6 weeks • Each lesion lasts <24 hrs	
Cholinergic urticaria	Increased core body temperature hot shower, exercise	Tiny flesh coloured wheals with surrounding red flare	
Contact urticaria	latex rubber – patch test, allergy test		
Phyiscal urticarias • Aquagenic urticaria	exposure to water		
Adrenergic urticaria	• Stress		
Cold urticaria	• ice cube, swimming pool	Can be life threatening	
Dermographism	Friction, rubbing skin	Immediate and possible delayed types	
Heat urticaria	• local heat		
Pressure urticaria	Located over pressure areas of body (shoulder strap, buttocks)	Immediate and delayed types	
Solar urticaria	Caused by a specific wavelength of UV radiation		
Vibratory urticaria	• Vibration		
Vasculitic urticaria	Infections – hepatitis Autoimmune diseases – SLE Drug hypersensitivity	Painful non-pruritic lesions Lesions last > 24 hrs Must biopsy these lesion	

FIXED DRUG ERUPTION

- sharply demarcated erythematous oval patches on the skin or mucous membranes
 - sites: face, genitalia
 - with each exposure to the drug, the patient develops erythema at the same location as before (fixed location)
 tetracycline, sulfonamides, barbituates, phenolphthalein

- DELAYED HYPERSENSITIVITY SYNDROME

 □ initial fever, followed by symmetrical bright red exanthematous eruption and may lead to internal organitis including hepatitis, arthralgia, lymphadenopathy, and/or hematologic abnormalities

 classically the patient has a first exposure to a drug and develops the syndrome 10 days later

 siblings at risk
 sulfonamides, anticopyulsants, etc.

 - sulfonamides, anticonvulsants, etc...

PHOTOSENSITIVITY ERUPTIONS

- phototoxic reaction: "an exaggerated sunburn" confined to light exposed areas
- photoallergic reaction: an eczematous eruption that may spread to
- areas not exposed to light

 chlorpromazine, doxycycline, thiazide diuretics, procainamide

- SERUM SICKNESS LIKE REACTION

 □ a symmetric drug eruption resulting in fever, arthralgia, lymphadenopathy, and skin rash
 usually appears 5-10 days after drug
 skin manifestations: usually urticaria; can be morbilliform
 cefaclor

COMMON SKIN LESIONS

HYPERKERATOTIC

Seborrheic Keratosis (Senile Keratosis) (see Colour Atlas A8) round/oval, well demarcated waxy papule/plaque, +/- pigment, warty surface, "stuck on" appearance sites: face, trunk, upper extremities benign neoplasm of epidermal cells usually asymptomatic more common with increasing age treatment no treatment usually needed liquid nitrogen for cosmetic reasons differential solar lentigo spreading pigmented actinic keratosis pigmented basal cell carcinoma malignant melanoma (lentigo maligna, nodular melanoma)
Actinic Keratosis (Solar Keratosis) (see Colour Atlas A19) discrete yellow-brown, scaly patches on a background of sun damaged skin
 sites: (areas of sun exposure) - face (forehead, nose, cheeks, temples), ears, neck, forearms, hands, legs middle age and elderly (except in sunny climates), more common in males and fair-skinned people
• 5-FU cream
 liquid nitrogen differential discoid lupus erthematosus Bowen's Disease
Keratoacanthoma (see Colour Atlas A18) □ red/skin coloured, firm, dome-shaped nodule with central keratotic plug • sites: sun-exposed skin of persons over age 50 • benign epithelial neoplasm with atypical keratinocytes • asymptomatic, attains full size in < 4 months, regress in < 10 months • rapidly grow to ~2.5 cm in 6 weeks • spontaneously resolve with disfiguring scar □ treatment
 surgical excision curettage and electrocautery if on lip treat as squamous cell carcinoma differential
• squamous cell carcinoma (grows slower – months)
FIBROUS
Dermatofibroma ☐ firm, red-brown, solitary, well demarcated intra-dermal papules or nodules with central dimpling ☐ site: legs
□ unknown etiology, often with antecedent trauma or insect bites □ dimple sign on pressure □ treatment
 no treatment usually needed (excise if bothersome) differential malignant melanoma, nevus

Skin Type tags (papilloma, acrochordon, fibroepithelial polyp) small, soft, pedunculated, skin-coloured tag sites: neck, axillae, and tunk middle-aged and elderly treatment clipping, cautery
CYSTS
Epidermal Cysts (see Colour Atlas A11) □ round, firm yellow/flesh coloured, slow growing, mobile, epidermally lines cyst filled with keratin • sites: scalp, face, upper trunk, buttocks • may rupture and produce inflammatory reaction • excise completely before becomes infected
Pilar Cysts ☐ hard, pea to grape-sized nodules under scalp • idiopathic, post-trauma (e.g. EEG)
Dermoid Cysts ☐ rare, congenital hamartomas ☐ arise from inclusion of epidermis along embryonal cleft closure lines ☐ most common at lateral third of eyebrow and midline under nose ☐ treatment: excision
Ganglion ☐ cystic lesion originating from joint or tendon sheath ☐ treatment • drainage +/- steroid injection if painful • excise if bothersome
VASCULAR
Hemangiomas ☐ benign proliferation of vessels in the dermis ☐ treatment options: argon laser, tattooing, cosmetics, excision with skin expansion
Nevus Flammeus (Port-Wine Stain) permanently dilated capillaries in dermis, present at birth dermatomal distribution, rarely crosses midline most common site: nape of neck papules/nodules may develop in adulthood, no involution seen in Sturge Weber syndrome treatment: laser or make-up
Cavernous Hemangioma ☐ can ulcerate ☐ 80% without scarring or discoloration
Angiomatous Nevus (Strawberry Nevus) □ congenital □ appears by age 9 months and resolves spontaneously by age 6 years □ can excise if not gone by school age
Spider Angioma ☐ central arteriole with slender branches resembling legs of a spider ☐ faintly pulsatile, blanchable, red macule ☐ associated with hepatic cirrhosis, pregnancy, oral contraceptives
Cherry Hemangioma (Senile Hemangioma, Campbell Demorgan Spot) ☐ bright red, dome-shaped papules, 1-5 mm ☐ site: trunk ☐ more common with increasing age
Melanocytic Nevi (Moles) □ be suspicious of new pigmented lesions in individuals over age 40 □ average number of moles per person:18-40

lanocytic	Table 11. Melanocytic Nevi Classification			
	Age of Onset	Description	Histology	Treatment
	birth	 sharply demarcated pigmented with regular/irregular contours +/- coarse hairs >1.5 cm R/O leptomeninges involvement if on head/neck 		• excuse if suspicious, due to increased risk of developing plaque melanoma
	• early childhood to age 40 • involute by age 60	• benign neoplasm of pigment forming nevus cells • well circumscribed, round, uniformly pigmented macules/papules • <1.5 cm • can be classified according to site of nevus cells		excisional biopsy required if on scalp, soles, mucous membranes, anogenital area, or has variegated colours, irregular borders, pruritic, bleeding, exposed to trauma
		 flat, irregularly bordered, uniformly tan-dark brown, sharply demarcated macule 	melanocytes at dermal-epidermal junction above basement membrane	• same as above
		elevated, regularly bordered, uniformly tan-dark brown papule NOT found on palms or soles	• melanocytes at dermal-epidermal junction; migration into dermis	• same as above
		• soft, dome-shaped, skin-coloured to tan/brown papules	 melanocytes exclusively in dermis 	• same as above
		• variegated macule/papule with irregular indistinct borders and focal elevation • >6 mm • RFs: postive family history 100% lifetime risk with 2 blood relatives with melanoma (0.7% risk for general population)		• follow q 2-6 months with colour photographs • excisional biopsy if lesion changing or highly atypical
	2-3	 dermal/compound nevus surrounded by hypomelanosis 		• none required
	1.5-40	 uniformly blue to blue-black macule/papule with smooth border 6 mm 	 pigmented melanocytes and melanophages in dermis 	• remove if suddenly appears or has changed

MISCELLANEOUS

Keloid excessive proliferation of collagen following trauma to skin, may continue to expand in size for years sites: earlobes, shoulders, sternum, scapular areapredilection for Blacks and Orientals ☐ treatment intralesional steroid injections • silicone compression different from a hypertrophic scar Pyogenic Granuloma ight red pedunculated nodule characterized by proliferation of capillaries develops rapidly on fingers, lips, mouth, trunk, toes ☐ treatment electrocautery laser cryotherapy Solar Lentigo (Aging Spots, Liver Spots) ☐ well demarcated brown/black macules with an irregular outline sites: sun-exposed skin espcially dorsum of hands and face > 40 years old, most common in Caucasians increased number of melanocytes in epidermis □ treatment liquid nitrogen ☐ differential · lentigo maligna seborrheic keratosis pigmented solar keratosis

MALIGNANT SKIN TUMOURS

BASAL CELL CARCINOMA (see Colour Atlas A21) usually a centrally ulcerated, translucent / pearly papule or nodule with a rolled border and fine telangiectasia • 75% of all malignant skin tumours with increased prevalence in the elderly usually due to UV light, therefore > 80% on face may also be caused by scar formation, trauma or arsenic exposure malignant proliferation of basal cells of the epidermis · variants include superficial multicentric, sclerosing, fibroepithelium, and pigmented (brown and often mistaken 95% cure rate if lesion is less then 2 cm in diameter • slow growing and rarely metastatic (< 0.1%) □ treatment • surgical excision +/- MOHS radiotherapy cryotherapy · electrodessication and curettage carbon dioxide laser differential diagnosis nodular malignant melanoma (biopsy) sebaceous hyperplasia eczema tinea corporis SQUAMOUS CELL CARCINOMA (see Colour Atlas A17) ☐ a malignant neoplasm of keratinocytes characterized by erythematous, indurated, scaly/ulcerated papules primarily on sun exposed skin in the elderly

 predisposing factors include UV radiation, ionizing radiation exposure, HPV in the immunosuppressed, PUVA, atrophic skin lesions and chemical carcinogens such as arsenic, coal tar and topical nitrogen mustards, Marjolin's ulcers in burn scars prognostic factors include: immediate treatment, negative margins, and small lesions
 overall control is 75% over 5 years, 5-10% metastasize □ treatment as for basal cell carcinoma lifelong follow-up
Bowen's Disease (like a Squamous Cell Carcinoma in situ) ☐ erythematous plaque with a sharply demarcated red and scaly border • biopsy required for diagnosis • often 1-3 cm in diameter and found on the skin and mucous membranes • evolves to SCC in 10-20% of cutaneous lesions and > 20% of mucosal lesions ☐ treatment • as for basal cell carcinoma
 topical 5-fluorouracil (Efudex) used if extensive and as a tool to identify margins of poorly defined tumours
MALIGNANT MELANOMA (see Colour Atlas A23) □ malignant neoplasm of pigment forming cells (melanocytes and nevus cells)
 sites: skin, mucous membranes, eyes, CNS malignant characteristics of a mole include (ABCD) A - Asymmetry B - Border (irregular) C - Colour (varied) D - Diameter (increasing or > 6 mm) risk factors: numerous moles, fair skin, red hair, positive family history, people who burn but do not tan, large congenital nevi, familial dysplastic nevus syndrome (100%) most common sites: back (M), calves (F) worse prognosis if: male, on scalp, hands, feet, late lesion better prognosis if: pre-existing nevus present classification of invasion - see Plastic Surgery Notes Breslow's Thickness of Invasion 1. <0.76 mm - mets in 0% 2. 0.76-1.5 mm - mets in 25% 3. 1.5-3.99 mm - mets in 50% 4. >4 mm - mets in 66% Clark's Levels of Cutaneous Invasion Level I - above basement membrane - rare mets Level II - to junction of papillary and reticular dermis mets in up to 20% Level IV - into reticular dermis - mets in 40% Level V - into subcutaneous tissue - mets in 70%
Superficial Spreading Melanoma □ atypical melanocytes initially spread laterally in the epidermis then invade the dermis □ irregular, indurated, enlarging plaques with red/white/blue discoloration, focal papules and nodules □ ulcerate and bleed with growth □ 60-70% of all melanomas
Nodular Melanoma □ atypical melanocytes that initially grow vertically with little lateral spread □ uniform, grey-black, and sharply delineated □ rapidly fatal □ 30% of melanomas

Lentigo Maligna (Premalignant Lesion) ☐ malignant melanoma in situ (normal and malignant melanocytes confined to the epidermis) ☐ 2-6 cm, tan/brown/black patch with irregular borders ☐ lesion grows radially and produces complex colours ☐ sites: face, sun exposed areas ☐ 1/3 evolves into lentigo maligna melanoma
Lentigo Maligna Melanoma ☐ malignant melanocytes invading into the dermis ☐ similar to lentigo maligna, but with raised focal papules within the lesion ☐ found on all skin surfaces ☐ 15% of all melanomas
Acrolentiginous Melanoma palmar, plantar, subungual skin histologic picture as lentigo-maligna melanoma metastasize via lymphatics and blood vessels melanomas on mucous membranes have poor prognosis 5% of melanomas
Treatment ☐ excisional biopsy preferable, otherwise incisional biopsy ☐ remove full depth of dermis and extend beyond edges of lesion only after histologic diagnosis ☐ lymph node dissection shows survival advantage if nodes uninvolved ☐ chemotherapy (cis-platinum, BCG) for stage II (regional) and stage III (distant) disease ☐ radiotherapy curative for uveal melanomas, palliative bone and brain metastases
OTHERS
Leukoplakia □ white patch/plaque on lower lip, floor of mouth, buccal mucosa, tongue border or retromolarly • 40-70 years old, M > F, fair-skinned • premalignant lesion arising from chronic irritation or inflammation □ treatment • excision • cryotherapy □ differential diagnosis • lichen planus • oral hairy leukoplakia
Cutaneous T-Cell Lymphoma (Mycosis Fungoides) □ characterized by erythematous, patches/plaques/nodules • > 50 years old • etiology: HTLV • eventually invades internal organs • Sezary's syndrome - erythroderma, lymphadenopathy, WBC > 20 000 with Sezary cells, hair loss, pruritus
 treatment PUVA topical nitrogen mustard radiotherapy> total skin election beam radiation differential diagnosis psoriasis
nummular dermatitis"large plaque" parapsoriasis

ICHTHYOSIS VULGARIS

a generalized disorder of hyperkeratosis leading to dry skin, associated with atopy and keratosis pilaris
 "fish-scale" appearance especially on extremities with sparing of

flexural creases, palms and soles

• "2 A.D.": atopic dermatitis and autosomal dominant

☐ treatment

- immersion in bath and oils
- · emollient or humectant creams and ointments containing urea

NEUROFIBROMATOSIS (NF; VON RECKLINGHAUSEN'S DISEASE)

characterized by cafe-au-lait macules and neurofibromas

· diagnostic criteria include

1) more than 6 cafe-au-lait spots > 1.5 cm in an adult, and more than 5 cafe-au-lait spots > 0.5 cm in a child under age 5

2) axillary freckling

3) iris hamartomas (Lisch nodules)

4) optic gliomas

5) neurofibromas, and others

- autosomal dominant disorder with excessive and abnormal proliferation of neural crest elements
- associated with pheochromocytoma, astrocytoma, bilateral acoustic neuromas, bone cysts, scoliosis, precocious puberty

follow closely for malignancy

VITILIGO (see Colour Atlas A13)

- □ acquired loss of melanocytes characterized by sharply marginated off white macules or patches
 - sites: extensor surfaces and periorificial areas (mouth, eyes, anus, genitalia)
 - associated with streaks of depigmented hair, chorioretinitis

• 30% with +ve family history

- associated with autoimmune disease especially thyroid
- do blood work to rule out thyroid dysfunction, pernicious anemia, Addison's disease, diabetes
- Wood's lamp to detect lesions in fair-skinned patients

management

- camouflage makeup (self-tanning preparations)
- PUVA (psoralens and UVA)

- minigrafting"bleaching" normal pigmented areas (total white colour)
 - done in widespread loss of pigmentation
- sun protection

Disease	Related Dermatoses
AUTOIMMUNE DISORDERS	
systemic lupus erythematosus	malar erythema, erythematous papules or plaques on face, hands, and arms, hemorrhagic bullae, palpable purpura, urticarial purpura, patchy/diffuse alopecia, mucosal ulcers (see Colour Atlas L1)
cutaneous lupus erythematosus	sharply marginated bright red papules and plaques with adherent scales, telangiectasia, marked scarring scarring alopecia $$
scleroderma	Raynaud's, nonpitting edema, waxy/shiny/tense atrophic skin (morphea), ulcers, cutaneous calcification, periungal telangiectasia (see Colour Atlas L8)
dermatomyositis	periorbital heliotrope with edema, violaceous erythema, Gottron's papules (violaceous flat-topped papules with atrophy), periungal erythema, telangiectasia, ulcers (see Colour Atlas L2 and L4)
polyarteritis nodosa	polyarteritic nodules, purpura, erythema, gangrene
ulcerative colitis	pyoderma gangrenosum
rheumatic fever	petechiae, urticaria, erythema nodosum, erythema multiforme, rheumatic nodules
Buerger's disease	superficial migraine thrombophlebitis, pallor, cyanosis, gangrene, ulcerations
ENDOCRINE DISORDERS Cushing's syndrome	purple atrophic striae, hyperpigmentation, hypertrichosis (see Colour Atlas B1)
hyperthyroid	moist, warm skin with evanescent erythema, seborrhea, acne, nail atrophy, hyperpigmentation, toxic alopecia, localized myxedema of pretibial area (see Colour Atlas B2 and B3)
hypothyroid	cool, dry, scaly, thickened, hyperpigmented skin; toxic alopecia with dull, dry, coarse hair
Addison's disease	hyperpigmentation on areas of friction and pressure
diabetes mellitus	increased incidence of skin infections: boils, carbuncles, ulcers, gangrene, candidiasis, tinea pedis and cruris, infectious eczematoid dermatitis; other: pruritus, xanthoma diabeticorum, necrobiosis lipoidica diabeticorum (multicoloured papules on anterior shins) (see Colour Atlas B5)
HIV viral	HSV, HZV, HPV, molluscum contagiosum, oral hairy leukoplakia
bacterial	impetigo, acneiform folliculitis, dental caries, cellulitis, bacillary epithelioid angiomatosis, syphilis
inflammatory dermatoses	seborrhea, psoriasis, pityriasis rosea, vasculitis
malignancies	Kaposi's Sarcoma (see Colour Atlas A20), lymphoma, basal cell carcinoma, squamous cell carcinoma malignant melanoma
MALIGNANCY	
Adenocarcinoma GI cervix/anus/rectum	Peutz-Jeghers: pigmented macules on lips/oral mucosa Paget's Disease: eroding scaling plaques of perineum
Carcinoma breast GI thyroid breast/GU/lung/ovary	Paget's Disease: exzematous and crusting lesions of breast Palmoplantar keratoderma: thickened skin of palms/soles Sipple's Syndrome: multiple mucosal neuromas Dermatomyositis: heliotrope erythema of eyelids and purplish plaques over knuckles
Lymphoma/Leukemia Hodgkin's Acute Leukemia Multiple Myeloma	Ataxia Telegectasia: telengectasia on pinna, bulbar conjunctiva Ichthyosis: generalized scaling especially on extremities Bloom's Syndrome: butterfly erythema on face, associated with short stature Amyloidosis: large, smooth tongue with waxy papules on eyelids, nasolabial folds and lips, as well as facial petechiae
OTHERS pruritic urticaria papules and plaques of pregnancy (PUPPP)	erythematous papules or urticarial plaques in distribution of striae distensae, buttocks, thighs, upper inner arms and lower backs
cryoglobulinemia	palpable purpura in cold-exposed areas, Raynaud's, cold urticaria, acral hemorrhagic necrosis, bleeding disorders; related to hepatitis C infection

Notes

PRURITUS

- a careful history is important, because medical workup may be indicated in 20% of cases
- causes
 - dermatologic generalized
 - winter itch (=xerotic eczema, dry and cracked mainly legs,
 - senile pruritus (may not have dry skin, any time of year)

 - infestations scabies, lice
 drug eruptions ASA, antidepressants, opiates
 - psychogenic statesdermatologic local
 - atopic and contact dermatitis, lichen planus, urticaria, insect bites, dermatitis herpetiformis
 infection varicella, candidiasis
 neurodermatitis (lichen simplex chronicus, vicious

 - cycle of itching & scratching leads to excoriated lichenified plaques)

 - medical usually generalized
 some types of cholestasis (e.g. PBC, chlorpromazine induced)
 chronic renal failure, cholestatic liver disease of pregnancy
 hematologic Hodgkin's lymphoma, multiple myeloma,
 - polycythemia vera, mycosis fungoides, hemachromatosis, Fe deficiency

 - carcinoma lung, breast, gastric
 endocrine carcinoid, diabetes, hypothyroid/thyrotoxicosis • infectious - HIV, onchocerciasis, trichinosis, echinococcosis
- □ treatment
 - treat underlying cause and itch (minimize irritation and scratching)
 - topical corticosteroid and antipuritics such as menthol, camphor or phenol
 - systemic antihistamines H1 blockers are most effective
 - avoid topical anaesthetics which may sensitize the skin

ALOPECIA (HAIR LOSS)

NON-SCARRING (NON-CICATRICIAL) ALOPECIA

Mnemonic telogen effluvium out of Fe, zinc
physical - trichotillomania, "corn-row" braiding
hormonal - hypothyroidism, androgenic
autoimmune - SLE, alopecia areata Ō P Η toxins - heavy metals, anticoagulants, chemotherapy, Vit. A

Physiological

- ☐ male-pattern alopecia
 - temporal areas progressing to vertex, entire scalp may be bald
 action of testosterone on hair follicles
 early 20's-30's (female androgenetic alopecia is

 - diffuse and occurs in 40's and 50's)
- □ treatment
 - minoxidil lotion to reduce rate of loss/partial restoration
 - spironolactone in women

 - hair transplantfinasteride 1 mg/d in men

- ☐ frichotillomania: impulse-control disorder characterized by compulsive
- hair pulling with resultant noticeable hair loss traumatic (e.g. tight "corn-row" braiding of hair)

Telogen Effluvium

- 15% of hair normally in resting phase, about to shed (telogen)
 post-partum, post-birth control pill, severe physical/mental stress can all increase the number of hairs in telogen
 - hair may shed up to 3 months after stimuli
 - will regrow

Alopecia Areata (see Colour Atlas A12) autoimmune disorder characterized by patches of complete hair loss localized to scalp, eyelids, cheek alopecia totalis - loss of all scalp hair and eyebrows alopecia universalis - all body hair associated with dystrophic nail changes - fine stippling exclamation mark pattern (hairs fractured and have tapered shafts, i.e!) may be associated with other autoimmune disease i.e. vitiligo, thyroid disease spontaneously regrow (but worse prognosis if young age of onset and extensive loss) frequent recurrence often precipitated by emotional distress treatment generally unsatisfactory intralesional triamcinolone acetonide can be used for isolated patches (eyebrows, beards) wigs
Metabolic Alopecia □ Drugs: e.g. chemotherapy, Danazol, Vitamin A, anticoagulants □ Toxins: e.g. heavy metals □ Endocrine: e.g. hypothyroidism
SCARRING (CICATRICIAL) ALOPECIA
Physical □ x-ray, burns
Infections ☐ fungal, bacterial, TB, leprosy, viral
Collagen-Vascular discoid lupus erythematosus (treatment with topical/intralesional steroid or antimalarial); note that SLE can cause an alopecia unrelated to discoid lupus lesions which are non-scarring scleroderma - "coup de sabre" when involves center of scalp

Clinical Pearl
☐ Scarring alopecia needs to be biopsied vs. nonscarring which does not

WOUNDS AND ULCERS

Table 9. Different types of ulcers and management				
ulcer type	Symptoms and signs Management			
arterial	wound at tip of toes, cold feet with claudication, gangrene, distal hyperemia, decreased pedal pulses	 Doppler study if ankle: brachial ratio < 0.4, may consider amputation if gangrenous, paint with betadine otherwise promote moist interactive wound healing 		
venous	wound at malleolus, stasis change, edema, previous venous injury	 local wound dressing: moist interactive healing compression: preferably 4 layer after wound heals, support stocking for life 		
neurotropic	wound at pressure point or secondary to unknown trauma	 pressure downloading by using proper shoes or seats promote moist interactive wound healing 		
vasculitic	livedo reticularis, petechiae, extreme tendemess, delayed healing	 biopsy to determine vasculitis serum screening for vasculitis treat vasculitis local moist interactive wound healing 		

CHEMICAL PEELING

(Chemexfoliation, Chemical Resurfacing)

- application of caustic agent(s) to skin to produce a controlled destruction of epidermis or dermis with subsequent re-epitheliazation
- ☐ topical keratolytics are applied 2-3 weeks preoperatively
- ☐ 3 different categories of chemical peeling agents used, depending on their depth of cutaneous penetration required:

Table 13.					
Penetration Type	Peeling Agents	Indications			
Superficial	AHA (glycolic acid) 10-30% trichloroacetic acid Jessner's solution	Fine wrinkling Acute actinic damage Postinflammatory pigment changes Acne vulgaris/rosacea			
Medium	CO2 ice + 35% TCA Jessner's + 35% TCA Glycolic acid + 35% TCA	Moderate wrinkling Chronic photodamage Pigment changes Epidermal/premalignant lesions			
Deep	Baker-Gordon formula	Severe wrinkling Chronic photodamage Superficial neoplasms Pigment changes Epidermal lesions			

complications

 erythema, infection, postinflammatory hyper/hypopigmentation, hypertrophic scars

LASER THERAPY

- □ wavelength is inversely proportional to absorption and directly proportional to penetration depth
 □ purpose: to remove/lessen unwanted pigmentation or vascular lesions
 □ hemoglobin, water, and melanin are the main targets of lasers

- lasers destroy unwanted skin abnormalities based on 3 mechanisms
 - heat energy absorption of heat with 2° spread to adjacent
 - mechanical energy rapid thermoelastic expansion destroys
 - selective photothermolysis wavelength that is maximally absorbed by target only and does not spread to adjacent tissues
- complications
 - erythema, hyper/hypopigmentation, scars, infection

Red Scaling Lesions	Psoriasis (elbows/knees/scalp, nail pits, Koebner's)
Red Scaling Lesions (epidermal cells produced from excessive and abnormal keratinization and shedding)	Psoriasis (elbows/knees/scalp, nail pits, Koebner's) Atopic Dermatitis (flexural folds) Contact Dermatitis (history) Discoid Lupus (don't see hair follicles) Drug reaction (e.g. gold, phenolphthalein in Ex-Lax) Lichen Planus (flat surface, lacy lines on surface) Mycosis Fungoides (girdle area, leonine facies) Nümmular Eczema (coin-like, isolated) Pityriasis Rosea (Christmas-tree distribution) Seborrheic Dermatitis (scalp/nasolabial folds/chest) Secondary Synyllis (nalms + solas conper coloured)
	Seborrheic Dermatitis (scalp/nasolabial folds/chest) Secondary Syphilis (palms + soles, copper coloured) Tinea (well demarcated, raised border)
Discrete Red Papules (elevated/solid lésion < 1 cm)	Acne (teenager, face/chest/back) Bites/Stings (history of outdoors, central punctum) Dermatofibroma ("dimple sign") Folliculitis (in hair follicle) Furuncle (very painful, central plug) Hemangioma (blanching) Hives (whitish border, pruritic) Inflamed Epidermal Cyst (mobile under skin) Inflamed Epidermal Cyst (mobile under skin) Inflamed Seborrheic Keratosis (stuck-on appearance) Lichen Planus (flat surface, lacy lines on surface) Miliaria Rubra (heat/overbundling of child) Psoriasis Pyogenic Granuloma (bleeds easily) Scabies (burrow, interdigital/groin, family members) Urticaria
Flat Brown Macule (circumsrcibed flat and discoloured area)	Actinic/Solar Lentigo (sun-damaged area) Congenital Nevus (contain hair) Cafe-au-Lait (present in childhood, very light brown) Hyper/hypopigmentation (e.g. posttraumatic, Addison's) Freckle (sun-exposed areas, disappears in winter) Junctional Nevus (regular shape) Lentigines associated with underlying disorders (LEOPARD, LAMB, Peutz-Jegher's) Lentigo Maligna (irregular, varied pigmentation) Malignant Melanoma (characteristic atypia) Pigmented Basal Cell Carcinoma Simple Lentigo (non-sun exposed area, irregular) Stasis Dermatitis
Vesicles (circumscribed collection of free fluid > 1 cm)	Viral • HSV (mouth, genitals) • Zoster (dermatomal,painful) • Varicella (generalized, itchy) • Molluscum (umbilicated) • Coxsackie (painful, hand-foot-mouth, summer) Acute Contact Dermatitis (e.g. poison ivy) (exposure history) Cat-Scratch Disease Dyshydrotic Eczema (sides of fingers/palms/soles) Dermatitis Herpetiformis (VERY itchy, gluten Hx) Impetigo Porphyria Cutanea Tarda (hypertrichosis, heliotrope lesion around eyes, alcohol ingestion) Scabies
Bullae (circumscribed collection of free fluid > 1 cm)	Bullous Impetigo (children, other family members) Bullous Pemphigoid (tense, lower limb) Drug eruption EM/SJS/TEN (target lesions) Lupus Erythematosus Pemphigus Vulgaris (flaccid, easy bleeding)
Pustules (elevated, contains purvient fluid, varying in size)	Acne (teenager, face/chest/back) Acne Rosacea (forties, telangiectatic, no comedones) Candida (satellite pustules, areas of skin folds) Dermatophyte infection Dyshidrotic Eczema (sides of fingers/palms/soles) Fölliculitis (in hair follicle) Hidradenitis suppurativa Impetigo (honey-crust) Sepsis (e.g. staph, gonococcal) Pustular Psoriasis (psoriasis) Rosacea Varicella
Ulcer (break in the skin that extends to the dermis, or deeper)	Common: Arterial, Venous, Neurotrophic, Pressure Uncommon: "CHIP IN" mnemonic Cancer (e.g. SCC), Chromosomal (e.g. XXY) Hemoglobinopathy (e.g. Sickle Cell) Inflammatory (e.g. RA, SLE, Vasculitis, Raynaud's) Pyoderma Gangrenosum (e.g. ulcerative colitis, RA) Infectious (syphilis, TB, tularemia, plague) Necrobiosis Lipoidica Diabeticorum (DM)
Oral Ulcers	Aphthous Cancer (Squamous /Basal Cell Ca) Dermatologic Diseases (Lichen Planus, Bullours, Pemphigoid) latrogenic (Chemo, Radiation) Infectious (HSV/HZ, Coxsackie, HIV, CMV, TB, Syphilis, Aspergillosis, Cryptococcosis) Inflammatory (SLE, Seronegatives, EM/SJS/TEN, allergic stomatitis) Traumatic

USEFUL DIFFERENTIAL DIAGNOSES ... CONT.

Location	Common	Less Common and Rare
Scalp	seborrheic dermatitis, contact dermatitis, psoriasis, folliculitis, pediculosis, tinea	pemphigus, DH
Ears	seborrheic dermatitis, psoriasis, infectious eczematoid dermatitis, actinic keratoses	fungal infection
Face	acne, rosacea, impetigo, contact dermatitis, seborrheic dermatitis, folliculitis, herpes simplex, BCC, SCC, actinic keratoses, sebaceous hyperplasia	lupus, actinic dermatitis, dermatomyositis, lentigo maligna melanoma
Eyelids	Contact dermatitis (fingernail polish, hairspray), seborrheic dermatitis, atopic eczema	
Posterior Neck	neurodermatitis (LSC), seborrheic dermatitis, psoriasis, contact dermatitis	acne keloidalis in black patients
Mouth	Aphthae, herpes simplex, geographic tongue, contact dermatitis	syphilis, lichen planus, pemphigus
Axillae	Contact dermatitis, seborrheic dermatitis, hidradenitis suppurtiva	erythrasma, acanthosis nigricans, inverse psoriasis, Fox-Fordyce disease
Chest and Back	Tinea versicolour, pityriasis rosea, acne, seborrheic dermatitis, psoriasis, Herpes Zoster	secondary syphilis, Grover's disease, inverse psoriasis
Groin and Crural Areas	Tinea, Candida, bacterial intertrigo, scabies, pediculosis, granuloma inguinale	
Penis	Contact dermatitis, fusospirochetal and candidal balanitis, chancroid, herpes simplex, Condylomata (HPV), scabies	primary and secondary syphilis, balanitis xerotica obliterans, lichen planus
Hands	Contact dermatitis, dyshydrotic eczema, reaction to fungal infection of the feet (one-hand two feet), warts, atopic eczema, psoriasis	pustular psoriasis, granuloma annulare, erythema multiforme, secondary syphilis(palms) and fungal infection
Cubital Fossae and Popliteal Fossae	Atopic eczema, contact dermatitis and prickly heat	
Elbows and Knees	Psoriasis, xanthomas	atopic ezcema, DH
Legs	Contact dermatitis, stasis dermatitis, ulcers, nummular eczema	pyoderma gangrenosum, erythema nodosum, leukocytoclastic vasculitis, HSP and other vasculitidie
Feet	Fungal infection, primary or secondary bacterial infection, contact dermatitis, atopic eczema, warts	psoriasis, erythema multiforme, secondary syphilis (soles), acral lentiginous melanoma (soles)

VEHICLES

- ☐ for acute inflammation (edema, vesiculation, oozing, crusting, infection)
- use aqueous drying preparation

 or chronic inflammation (scaling, lichenification, fissuring) use a greasier, more lubricating compound

Powders

promote drying, increase skin surface area (i.e. cooling)

- used in intertriginous areas to reduce moisture and friction
- inert or contain medication

Lotions

- suspensions of powder in water
 cool and dry as they evaporate
 leave a uniform film of powder on skin
 easily applied to hirsute areas

Cream

semisolid emulsions of oil in water

- water-soluble, contain emulsifiers and preservatives
- cosmetically pleasing

Gel

☐ transparent, colourless, semisolid emulsion

- liquifies on contact with skin
 dries as a thin, greaseless, nonocclusive, nonstaining film
 aqueous, acetone, alcohol or propylene glycol base

☐ semisolid water in oil emulsions (more viscous than cream)

- inert bases petrolatum
- most effective to transport medications into skin
- retain heat, impede water loss, increase hydration
 occlusive, not to be used in oozing or infected areas

TOPICAL STEROIDS

Table 16. Potency Ranking of Topical Steroids				
Relative Potency	Relative Strength	Generic Names	Trade Names	Usage
weak	x1	hydrocortisone	Emo Cort	intertriginous areas, children, face, thin skin
moderate	х3	hydrocortisone 17-valerate desonide mometasone furorate	Westcort Tridesilon Elocom	arm, leg, trunk
potent	х6	betamethasone 17-valerate amicinonide	Betnovate Celestoderm Cyclocort	body
very potent	х9	betamethasone dipropionate clucinonide	Propaderm Lidex, Topsyn gel	palms and soles
extremely potent	x12	clobetasol propionate	Dermovate Diprolene	palms and soles

Body site: Relative Percutaneous Absorption

forearm 1.0 0.14 plantar foot palm 0.83 1.7 3.7 back scalp forehead 6.0 cheeks 13.0 scrotum 42.0

calculation of strength of steroid compared to hydrocortisone on forearm:

relative strength of steroid x relative percutaneous absorption

DRY SKIN THERAPY

encourage humidifier

decrease excess exposure to water or soap
use mild soaps such as Dove and bath oils
lubricating lotions and creams
are occlusive and soften the skin

humectant agents such as uremol (urea), LacHydrin (lactic acid) and Neostrata (glycolic acid) hold water to skin or affect desquamation of stratum corneum

topical steroid ointment for symptomatic dryness with eczema

SUNSCREENS AND PREVENTATIVE THERAPY

UV Radiation

□ UVA (320-400nm)

- penetrates skin more effectively then UVB or UVC
 responsible for tanning, burning, wrinkling and
- premature skin aging

 penetrates clouds, glass and is reflected off water, snow and cement

 UVB (290-320nm)

 absorbed by the outer dermis

is mainly responsible for burning and premature skin aging
 primarily responsible for BCC, SCC and melanomas

does not penetrate glass and is substantially absorbed by ozone
 UVC (200-290nm)
 is filtered by ozone layer

Sunburn Prevention

Sunburn

- erythema 2-6 hours post UV exposure often associated with edema, pain and blistering with subsequent desquamation of the dermis
- UV index measures the time to burn for a fair skinned individual
 - < 15 minutes = UV index > 9
 - ~ 20 minutes = UV index 7-9
 ~ 30 minutes = UV index 4-7

Sunscreens

- ☐ SPF: under ideal conditions a sun protection factor of 10 means that a person who normally burns in 20 minutes will burn in 200 minutes following the application of the sunscreen, no matter how often the sunscreen is subsequently applied

 Topical Chemical: requires application, at least 15-60 minutes prior to
- exposure
 - UVB absorbers: PABA, Salicylates, Cinnamates, Benzylidene camphor derivatives

- campnor derivatives
 UVA absorbers: Benzophenones, Anthranilates,
 Dibenzoylmethanes, Benzylidene camphor derivatives
 Topical Physical: reflects and scatters UV light
 Titanium dioxide, Zinc oxide, Kaolin, Talc, Ferric chloride and Melanin all are effective against the UVA and UVB spectrum
- less risk of sensitization then chemical sunscreens and waterproof, but may cause folliculitis or miliaria

Sunburn Treatment

☐ if significant blistering present, consider treatment in hospital

• apply cool and wet compresses

- use moisturizers for dryness and peeling
 oral anti-inflammatory: 400 mg ibuprofen q6h to minimize erythema and edema
- topical corticosteroids: soothes and decreases erythema, does not reduce damage
- oral steroids and antihistamines have no role