

DERMATOLOGY

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HISTORY

- age, race, occupation, hobbies
- details of skin eruption
 - location
 - onset
 - persistent/intermittent
 - 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
- allergies, medications
- past medical history
- family history of skin and internal disease

PHYSICAL EXAM

- distribution
- colour
- type of lesion (see morphological definitions)
- arrangement (annular, linear, etc...)
- remember to examine hair, mucous membranes and nails

DEFINITIONS

PRIMARY MORPHOLOGICAL LESIONS

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

Table 2. Acne Types and Treatments

Acne Type	Treatment
Type I – Non-inflammatory	Benzoyl Peroxide (2.5%, 5%, 10%) – bactericidal Adapalene gel/cream <ul style="list-style-type: none"> • not irritating, no interaction with sun • expensive +/- Tretinoin (Retin-A) <ul style="list-style-type: none"> • 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) <ul style="list-style-type: none"> • 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) <ul style="list-style-type: none"> • 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 <ul style="list-style-type: none"> • S/E: teratogenic, skin and mucous membrane dryness, hyperlipidemia, 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

ROSACEA (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
 - others
 - lasers for telangiectases
 - plastic surgery or laser for rhinophyma
 - camouflage makeup for erythema
- ❑ differential diagnosis
 - SLE
 - carcinoid syndrome
 - acne vulgaris
 - perioral dermatitis

PERIORAL 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

- ❑ superficial inflammation of the skin, characterized by pruritic papulovesicles, redness, crusting, scaling, and lichenification secondary to scratching

ALLERGIC CONTACT DERMATITIS

- ❑ epidermal and dermal inflammation caused by cell-mediated delayed hypersensitivity reaction
- ❑ clinical suspicion by discrete area of skin involvement
- ❑ susceptibility to allergen is acquired and specific sensitivity usually persists indefinitely
- ❑ allergens include poison ivy, rubber/latex, neomycin, dyes, lanolin, nickel
- ❑ diagnosis by patch testing
- ❑ treatment
 - avoid allergen and its cross reactants
 - wet compresses soaked in Burow's solution (a drying agent), change q3h, betamethasone cream
 - systemic corticosteroids for extensive cases (prednisone 1mg/kg and reduce over 2 weeks)

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 Dermatitis

Phase	Distribution
Infant (onset at 2-6 months old)	Face, scalp, extensor surfaces
Childhood (>18 months)	Flexural surfaces
Adult	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 (Nizazole), 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 intralesional 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 triamcinolone
 - 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 soap
 - 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)

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), <i>S. aureus</i> (usually in significantly sized wounds, doesn't spread as much), <i>H. 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
<ul style="list-style-type: none"> ■ 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 *Staphylococcus folliculitis*
 - 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*, *Streptococcus*
 - chronic: *C. albicans*
- ❑ treatment
 - avoid exposure to moisture
 - topical fucidin or clotrimazole

OTHERS

Syphilis

- ❑ sexually transmitted infection caused by *Treponema pallidum* characterized by a painless ulcer (chancre)
- ❑ following 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
 - differential 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 region
 - exudate 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 exanthem
 - 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

Gonococemia (Disseminated Gonococcal Infection)

- ❑ pustules on a purpuric erythematous base and hemorrhagic, tender, necrotic pustules (aka "arthritis-dermatitis syndrome")
 - Gram negative diplococcus *Neisseria gonorrhoeae*
 - skin manifestations develop in gonococemia 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 meningococemia: 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
 - 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
 - electrodesiccation
 - cryotherapy

Condylomata Acuminata (Genital Warts) (see Colour Atlas D7)

- ❑ skin coloured pinhead papules to soft cauliflower like masses in clusters
 - young 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; usually associated with tinea pedis with one hand and two feet affected = “1 hand 2 feet” syndrome
- differential diagnosis
 - contact dermatitis, atopic dermatitis, psoriasis (all three commonly mistaken for fungal infections)
 - granuloma annulare (annular)

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
 - 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
 - intertrigo 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)
 - 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 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 8 hours)
 - 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 to remove 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 psoriasis
 - erythrodermic psoriasis
 - pustular psoriasis
 - psoriatic 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 by injury
- ❑ Auspitz's sign: bleeds from minute points when scale is removed
- ❑ exacerbating factors: drugs (lithium, ethanol, chloroquine, beta-blockers), sunlight, stress, obesity
- ❑ treatment
 - 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; Dovonex)	binds to skin 1, 25-dihydroxyvitamin D ₃ 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

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
acetrelin	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
 - psoriatic arthritis mutilans
 - predominant spondylitis or sacroilitis

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	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> intra-dermal triamcinolone acetonide 5 mg/mL PUVA methotrexate
palms and soles	sharply demarcated dusky-red plaques with thick scales on pressure points; can be pustular	<ul style="list-style-type: none"> 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
 - spontaneously resolves in weeks or lasts for years (mouth and shin lesions)
 - 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 intra-dermal 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 leukoplakia
 - lupus 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 “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
- ❑ diagnosis
 - 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
- ❑ treatment
 - 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 PEMPHEGOID

- ❑ chronic autoimmune bullous eruption characterized by pruritic, tense, subepidermal bullae
- ❑ etiology
 - IgG 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)
- ❑ course
 - healing 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% have HLA B8, DR3, DQW2
- ❑ physical
 - sites: extensor surfaces of elbows/knees, sacrum, buttocks, scalp
- ❑ diagnosis
 - immunofluorescence: granular IgA and complement deposition in dermis
- ❑ course
 - lesions last days - weeks
- ❑ treatment
 - dapsone for pruritus but multiple side effects
 - gluten free diet

	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
- history
 - 30-40 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

- spectrum of disorders with varying presence of characteristic skin lesions, blistering, and mucous membrane involvement

EM (minor) EM (major) SJS → TEN

Table 9. Comparison of Erythema Multiforme, Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis

	Erythema Multiforme (EM) (see Colour Atlas A10)	Stevens-Johnson Syndrome (SJS)	Toxic Epidermal Necrolysis (TEN) (see Colour Atlas A14)
Lesion	<ul style="list-style-type: none"> • 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) 	<ul style="list-style-type: none"> • 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 	<ul style="list-style-type: none"> • severe mucous membrane involvement • "atypical lesions" - 50% have no target lesions • diffuse erythema then necrosis and sheet-like epidermal detachment in >30%
Sites	<ul style="list-style-type: none"> • mucous membrane involvement (oral, genital, conjunctival) • extremities with face > trunk • involvement of palms and soles 	<ul style="list-style-type: none"> • generalized with prominent face and trunk involvement • palms and soles may be spared 	<ul style="list-style-type: none"> • generalized • nails may also shed
Other organs/ complications	<ul style="list-style-type: none"> • corneal ulcers, keratitis, anterior uveitis, stomatitis, vulvitis, balanitis • lesions in trachea, pharynx, larynx 	<ul style="list-style-type: none"> • complications: scarring, eruptive nevocmelanocytic nevi, corneal scarring, blindness, phimosis and vaginal synechiae 	<ul style="list-style-type: none"> • tubular necrosis and acute renal failure, epithelial erosions of trachea, bronchi, GI tract
Constitutional symptoms	<ul style="list-style-type: none"> • fever, weakness, malaise 	<ul style="list-style-type: none"> • prodrome 1-3 days prior to eruption with fever and flu-like illness 	<ul style="list-style-type: none"> • high fever > 38°C
Etiology	<ul style="list-style-type: none"> • drugs - sulfonamides, NSAIDs, anticonvulsants, penicillin, allopurinol • infection - herpes, mycoplasma • idiopathic - >50% 	<ul style="list-style-type: none"> • 50% are drug related • occurs up to 1-3 weeks after drug exposure with more rapid onset upon rechallenge 	<ul style="list-style-type: none"> • 80% are definitely drug related • < 5% are due to viral infection, immunization
Pathology/ Pathophysiology	<ul style="list-style-type: none"> • perivascular PMN infiltrate, edema of upper dermis 	<ul style="list-style-type: none"> • cytotoxic cell-mediated attack on epidermal cells • no dermal infiltrate • epidermal necrosis and detachment above basement membrane 	<ul style="list-style-type: none"> • same as Stevens-Johnson Syndrome
Differential diagnosis	<ul style="list-style-type: none"> • EM minor - urticaria, viral exanthems • EM major - SSSS, pemphigus vulgaris, bullous pemphigoid 	<ul style="list-style-type: none"> • scarlet fever, phototoxic eruption, GVHD, SSSS, exfoliative dermatitis 	<ul style="list-style-type: none"> • scarlet fever, phototoxic eruption, GVHD, SSSS, exfoliative dermatitis
Course and Prognosis	<ul style="list-style-type: none"> • lesions last 2 weeks 	<ul style="list-style-type: none"> • < 5% mortality • regrowth of epidermis by 3 weeks 	<ul style="list-style-type: none"> • 30% mortality due to fluid loss, secondary infection
Treatment	<ul style="list-style-type: none"> • prevention - drug avoidance • symptomatic treatment • corticosteroids in severely ill but controversial 	<ul style="list-style-type: none"> • withdraw suspect drug • intravenous fluids • corticosteroids - controversial • infection prophylaxis 	<ul style="list-style-type: none"> • 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
- ❑ associations
 - infections: Group A Streptococcus, primary TB, coccidioidomycosis, histoplasmosis, Yersinia
 - drugs: sulfonamides, oral contraceptives (also pregnancy)
 - inflammation: sarcoidosis, Crohn's > ulcerative colitis
 - malignancy: acute leukemia, Hodgkin's lymphoma
 - 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
 - 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 laryngeal 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 reaches hospital in acute attack

Type	Provocative agents/tests	Comments
acute urticaria	<ul style="list-style-type: none"> • 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 	<ul style="list-style-type: none"> • Attack lasts <6 weeks • Each lesion lasts <24 hrs • Occurs with or without angioedema
chronic urticaria	<ul style="list-style-type: none"> • most commonly idiopathic • aggravating and causative factors may be similar to those in acute urticaria 	<ul style="list-style-type: none"> • Attack lasts >6 weeks • Each lesion lasts <24 hrs
Cholinergic urticaria	<ul style="list-style-type: none"> • Increased core body temperature • hot shower, exercise 	<ul style="list-style-type: none"> • Tiny flesh coloured wheals with surrounding red flare
Contact urticaria	<ul style="list-style-type: none"> • latex rubber – patch test, allergy test 	
Physical urticarias		
• Aquagenic urticaria	<ul style="list-style-type: none"> • exposure to water 	
• Adrenergic urticaria	<ul style="list-style-type: none"> • Stress 	
• Cold urticaria	<ul style="list-style-type: none"> • ice cube, swimming pool 	<ul style="list-style-type: none"> • Can be life threatening
• Dermographism	<ul style="list-style-type: none"> • Friction, rubbing skin 	<ul style="list-style-type: none"> • Immediate and possible delayed types
• Heat urticaria	<ul style="list-style-type: none"> • local heat 	
• Pressure urticaria	<ul style="list-style-type: none"> • Located over pressure areas of body (shoulder strap, buttocks) 	<ul style="list-style-type: none"> • Immediate and delayed types
• Solar urticaria	<ul style="list-style-type: none"> • Caused by a specific wavelength of UV radiation 	
• Vibratory urticaria	<ul style="list-style-type: none"> • Vibration 	
Vasculitic urticaria	<ul style="list-style-type: none"> • Infections – hepatitis • Autoimmune diseases – SLE • Drug hypersensitivity 	<ul style="list-style-type: none"> • 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, 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
- ❑ treatment
 - 5-FU cream
 - liquid nitrogen
- ❑ differential
 - discoid lupus erythematosus
 - 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 trunk
- 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

Table 11. Melanocytic Nevi Classification				
Nevus Type	Age of Onset	Description	Histology	Treatment
Congenital	birth	<ul style="list-style-type: none"> sharply demarcated pigmented with regular/irregular contours +/- coarse hairs >1.5 cm R/O leptomeninges involvement if on head/neck 		<ul style="list-style-type: none"> excise if suspicious, due to increased risk of developing plaque melanoma
Acquired Melanocytic Nevo Cellular Nevi (MNCN)	<ul style="list-style-type: none"> early childhood to age 40 involute by age 60 	<ul style="list-style-type: none"> 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 	<ul style="list-style-type: none"> melanocytes at dermal-epidermal junction above basement membrane 	<ul style="list-style-type: none"> excisional biopsy required if on scalp, soles, mucous membranes, anogenital area, or has variegated colours, irregular borders, pruritic, bleeding, exposed to trauma
- Junctional NCN		<ul style="list-style-type: none"> flat, irregularly bordered, uniformly tan-dark brown, sharply demarcated macule 	<ul style="list-style-type: none"> melanocytes at dermal-epidermal junction above basement membrane 	<ul style="list-style-type: none"> same as above
- Compound NCN (see Colour Atlas A22)		<ul style="list-style-type: none"> elevated, regularly bordered, uniformly tan-dark brown papule NOT found on palms or soles 	<ul style="list-style-type: none"> melanocytes at dermal-epidermal junction; migration into dermis 	<ul style="list-style-type: none"> same as above
- Dermal NCN		<ul style="list-style-type: none"> soft, dome-shaped, skin-coloured to tan/brown papules sites: face, neck 	<ul style="list-style-type: none"> melanocytes exclusively in dermis 	<ul style="list-style-type: none"> same as above
Clark's Melanocytic Nevus (Dysplastic Nevus)		<ul style="list-style-type: none"> variegated macule/papule with irregular indistinct borders and focal elevation >6 mm RFs: positive family history 100% lifetime risk with 2 blood relatives with melanoma (0.7% risk for general population) 		<ul style="list-style-type: none"> follow q 2-6 months with colour photographs excisional biopsy if lesion changing or highly atypical
Halo	2-3	<ul style="list-style-type: none"> dermal/compound nevus surrounded by hypomelanosis 		<ul style="list-style-type: none"> none required
Blue	1.5-40	<ul style="list-style-type: none"> uniformly blue to blue-black macule/papule with smooth border < 6 mm 	<ul style="list-style-type: none"> pigmented melanocytes and melanophages in dermis 	<ul style="list-style-type: none"> 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 area
 - predilection for Blacks and Orientals
- ❑ treatment
 - intralesional steroid injections
 - silicone compression
- ❑ different from a hypertrophic scar

Pyogenic Granuloma

- ❑ bright 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 especially 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 for nevi)
 - 95% cure rate if lesion is less than 2 cm in diameter
 - slow growing and rarely metastatic (< 0.1%)
- ❑ treatment
 - surgical excision +/- MOHS
 - radiotherapy
 - cryotherapy
 - electrodesiccation 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 - in papillary dermis - mets in 2-5%
 - Level III - 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 margined 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, periungual telangiectasia (see Colour Atlas L8)
dermatomyositis	periorbital heliotrope with edema, violaceous erythema, Gottron's papules (violaceous flat-topped papules with atrophy), periungual 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 diabetorum, necrobiosis lipidica diabetorum (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	Peutz-Jeghers: pigmented macules on lips/oral mucosa
cervix/anus/rectum	Paget's Disease: eroding scaling plaques of perineum
Carcinoma	
breast	Paget's Disease: exzematous and crusting lesions of breast
GI	Palmoplantar keratoderma: thickened skin of palms/soles
thyroid	Sipple's Syndrome: multiple mucosal neuromas
breast/GU/lung/ovary	Dermatomyositis: heliotrope erythema of eyelids and purplish plaques over knuckles
Lymphoma/Leukemia	
Hodgkin's	Ataxia Telegectasia: telengectasia on pinna, bulbar conjunctiva
Acute Leukemia	Ichthyosis: generalized scaling especially on extremities
	Bloom's Syndrome: butterfly erythema on face, associated with short stature
Multiple Myeloma	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

PRURITUS

- 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, skin)
 - senile pruritus (may not have dry skin, any time of year)
 - infestations - scabies, lice
 - drug eruptions - ASA, antidepressants, opiates
 - psychogenic states
 - dermatologic - 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, hemochromatosis, 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 antipruritics 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-CICATRICAL) ALOPECIA

Mnemonic	
T	telogen effluvium
O	out of Fe, zinc
P	physical - trichotillomania, "corn-row" braiding
H	hormonal - hypothyroidism, androgenic
A	autoimmune - SLE, alopecia areata
T	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 transplant
 - finasteride 1 mg/d in men

Physical

- trichotillomania: 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 (CICATRICAL) 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	<ol style="list-style-type: none"> 1. Doppler study 2. if ankle: brachial ratio < 0.4, may consider amputation 3. if gangrenous, paint with betadine 4. otherwise promote moist interactive wound healing
venous	wound at malleolus, stasis change, edema, previous venous injury	<ol style="list-style-type: none"> 1. local wound dressing: moist interactive healing 2. compression: preferably 4 layer 3. after wound heals, support stocking for life
neurotropic	wound at pressure point or secondary to unknown trauma	<ol style="list-style-type: none"> 1. pressure downloading by using proper shoes or seats 2. promote moist interactive wound healing
vasculitic	livedo reticularis, petechiae, extreme tenderness, delayed healing	<ol style="list-style-type: none"> 1. biopsy to determine vasculitis 2. serum screening for vasculitis 3. treat vasculitis 4. 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-epithelialization
- ❑ 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	CO ₂ 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 tissues
 - mechanical energy – rapid thermoelastic expansion destroys target
 - 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 (epidermal cells produced from excessive and abnormal keratinization and shedding)	<p>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) Nummular Eczema (coin-like, isolated) Pityriasis Rosea (Christmas-tree distribution) Seborrheic Dermatitis (scalp/nasolabial folds/chest) Secondary Syphilis (palms + soles, copper coloured) Tinea (well demarcated, raised border)</p>
Discrete Red Papules (elevated/solid lesion < 1 cm)	<p>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 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</p>
Flat Brown Macule (circumscribed flat and discoloured area)	<p>Actinic/Solar Lentigo (sun-damaged area) Congenital Nevus (contain hair) Café-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</p>
Vesicles (circumscribed collection of free fluid > 1 cm)	<p>Viral</p> <ul style="list-style-type: none"> • HSV (mouth, genitals) • Zoster (dermatomal, painful) • Varicella (generalized, itchy) • Molluscum (umbilicated) • Coxsackie (painful, hand-foot-mouth, summer) <p>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</p>
Bullae (circumscribed collection of free fluid > 1 cm)	<p>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)</p>
Pustules (elevated, contains purulent fluid, varying in size)	<p>Acne (teenager, face/chest/back) Acne Rosacea (forties, telangiectatic, no comedones) Candida (satellite pustules, areas of skin folds) Dermatophyte infection Dyshydrotic Eczema (sides of fingers/palms/soles) Folliculitis (in hair follicle) Hidradenitis suppurativa Impetigo (honey-crust) Sepsis (e.g. staph, gonococcal) Pustular Psoriasis (psoriasis) Rosacea Varicella</p>
Ulcer (break in the skin that extends to the dermis, or deeper)	<p>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)</p>
Oral Ulcers	<p>Aphthous Cancer (Squamous /Basal Cell Ca) Dermatologic Diseases (Lichen Planus, Bullours, Pemphigoid) Iatrogenic (Chemo, Radiation) Infectious (HSV/HZ, Coxsackie, HIV, CMV, TB, Syphilis, Aspergillosis, Cryptococcosis) Inflammatory (SLE, Seronegatives, EM/SJS/TEN, allergic stomatitis) Traumatic</p>

Table 15. Differential Diagnosis by Location

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 suppurativa	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 eczema, DH
Legs	Contact dermatitis, stasis dermatitis, ulcers, nummular eczema	pyoderma gangrenosum, erythema nodosum, leukocytoclastic vasculitis, HSP and other vasculitides
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
- ❑ for 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

Ointment

- ❑ 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

Relative Potency	Relative Strength	Generic Names	Trade Names	Usage
weak	x1	hydrocortisone	Emo Cort	intertriginous areas, children, face, thin skin
moderate	x3	hydrocortisone 17-valerate desonide mometasone furorate	Westcort Tridesilon Elocom	arm, leg, trunk
potent	x6	betamethasone 17-valerate amicinonide	Betnovate Celestoderm Cyclocort	body
very potent	x9	betamethasone dipropionate clucinsonide	Propaderm Lidex, Topsyng gel	palms and soles
extremely potent	x12	clobetasol propionate	Dermovate Diprolene	palms and soles

Body site: Relative Percutaneous Absorption

forearm	1.0
plantar foot	0.14
palm	0.83
back	1.7
scalp	3.7
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 than 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
 - 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 than 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