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Derm Notes

Clinical Dermatology Pocket Guide

Benjamin Barankin
Anatoli Freiman

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Includes...

- ✓ **Diagnosis & Treatment**
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- ✓ **Effects of Aging and Pregnancy on Skin**



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DERM Notes

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A Davis's Notes Book



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History Taking in Dermatology

ID: Age, race, sex, occupation, hobbies

HPI:

- When did the rash/lesion start or was first noticed?
- Where did it start?
- Symptoms: E.g., does it itch, hurt, burn?
- Evolution: How has it spread or developed over time?
- What makes the rash or symptoms worse? E.g., heat, cold, sun, exercise, season
- What therapy has been tried?—dose, duration, frequency of actual use

Contacts (where appropriate): Pets or farm animal contact, travel, motel stays

PMHx: Diabetes, hypertension, atopy (eczema, asthma, hay fever), previous skin cancers or other skin problems; STDs, HIV, blood transfusions

Medications: Dosage listed for any derm drugs; specific topical steroid names

Allergies (& specific reaction)

Habits: Smoking, alcohol, drug abuse

FamHx: Psoriasis, melanoma, atopy, genetic conditions (e.g., neurofibromatosis)

Constitutional symptoms (if relevant: infection, previous malignancy): Headache, fever, chills, sweats, fatigue, weakness, anorexia, weight loss

Review of systems: Based on clinical scenario. E.g., If autoimmune connective tissue disease is in the differential, ask about arthralgias, myalgias, aphthous ulcers, keratoconjunctivitis sicca, Raynaud's phenomenon, neurologic or renal problems.

Approach to Describing Skin Lesions

Mnemonic: LEST. CABS

Descriptors

Location & Distribution

Symmetrical, asymmetrical, sun-exposed, flexures/extensors, acral (hands/feet)

Erythema

Erythematous or nonerythematous lesions &/or underlying skin

Surface Features

Crusting, rough, smooth, scaly, warty

Type

Cyst, macule, papule, pustule, ulcer, vesicle

Color

Blue, brown, pink, purple, red, white

Arrangement

Single or multiple, discrete, unilateral, generalized, disseminated, grouped, annular, gyrate, dermatomal, linear, serpiginous

Border & Shape

Well- or poorly defined, active edge, round, oval, irregular, pedunculated

Special Sites/Systemic

Scalp, mouth, nails, genitalia, systemic disease, constitutional symptoms

Terminology of Dermatology

Lesion	Definition	Examples
Abscess	Accumulation of pus in the dermis or subcutaneous tissue	Perianal abscess, <i>Staphylococcus aureus</i> infection
Annular	Ring-shaped	Granuloma annulare, tinea corporis
Atrophy	Depressed surface due to a thinned epidermis and/or dermis and/or subcutis	Lichen sclerosus, necrobiosis lipoidica, prolonged potent topical steroid use
Bulla (blister)	A large, circumscribed, fluid-containing elevation (>5 mm)	Bullous impetigo, bullous pemphigoid, pemphigus vulgaris
Burrow	Linear S-shaped elevated channel in the epidermis	Scabies
Carbuncle	Inflammatory nodules or abscess of numerous contiguous hair follicles	Hidradenitis suppurativa
Comedo	Plugged pilosebaceous follicle containing serum & keratin. Closed = whitehead; open = blackhead	Acne
Crust (scab)	Dried serum, pus, or blood (usually preceded by vesicle, bulla, or pustule)	Herpes simplex or VZV, impetigo
Cyst	Nodule containing fluid, cells or keratin	Epidermoid cyst, mucous cyst, pilar cyst

(Continued text on following page)

Terminology of Dermatology (Continued)

Lesion	Definition	Examples
Ecchymosis ("bruise")	Large, confluent area of purpura	Postsurgery or trauma
Erosion	Partial loss of epidermis. Heals without scarring.	Eczema, lichen simplex, pemphigus vulgaris
Erythema	Redness that blanches on pressure	Cellulitis, erysipelas, erythroderma, sunburn
Excoriation	Shallow abrasion due to scratching. Linear or pinpoint erosions or crusts.	Dermatitis
Exudate	Serum, blood, or pus accumulated on the skin surface	Acute eczema
Fissure	Linear split in epidermis &/or crack in (skin) dermis	Eczema
Folliculitis	Pustule involving a hair follicle	Folliculitis due to <i>S. aureus</i>
Furuncle	Deep necrotizing type of folliculitis	Furuncle due to <i>S. aureus</i> , hidradenitis suppurativa
Keratin/horn	Rough, uneven surface due to accumulation of keratin. Hard to pick off, unlike crust.	Actinic keratosis, corn, squamous cell carcinoma, wart
Lichenification	Focal area of thickened skin with accentuation of skin lines due to rubbing/scratching	Atopic dermatitis, lichen simplex chronicus

(Continued text on following page)


Terminology of Dermatology (Continued)

Lesion	Definition	Examples
Linear	Straight line	Allergic contact dermatitis (poison ivy), Koebner reaction to lichen planus or psoriasis
Livedo	Criss-cross pattern	Cutis marmorata, erythema ab igne, livedo reticularis
Macule	Flat, circumscribed area of altered skin color <1 cm	Freckle, junctional nevus, lentigo, pityriasis versicolor, vitiligo
Nodule	Solid, round circumscribed elevation >5 mm	Erythema nodosum, keratoacanthoma, prurigo nodularis, rheumatoid nodule
Papule	Raised, solid lesion <5 mm	Acne, basal cell carcinoma, lichens planus, molluscum contagiosum, rosacea, skin tag, syringoma
Patch	Flat, pigmented lesion >5 mm	Café au lait patch, melasma, Mongolian spot, nevus of Ota, port-wine stain, vitiligo
Petechia	Small, nonblanching, red-brown macules	Capillaritis, meningococcemia
Plaque	A >5-mm, flat-topped raised lesion (diameter > thickness)	Giant hairy nevus, mycosis fungoides, psoriasis, tinea corporis
Purpura	Visible collection of free red blood cells (red, purple, brown); nonblanchable	Drug-eruption, Henoch-Schönlein purpura
Pustule	A small papule containing pus	Acne, folliculitis, pustular psoriasis rosacea
Reticulate	Fine lace pattern	Oral lichen planus

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Terminology of Dermatology (Continued)

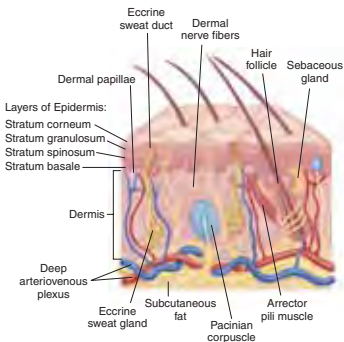
Lesion	Definition	Examples
Scale	Thick stratum corneum due to increased proliferation or cohesion of keratinocytes	Ichthyosis, psoriasis, seborrheic dermatitis, tinea capitis/corporis
Scar (cicatrix)	Healed dermal (or deeper) lesion following trauma/inflammation	Acne scars, hypertrophic scars, keloids
Sclerosis	Skin induration/hardening	Morphea
Serpiginous	Snakelike appearance	Cutaneous larva migrans, elastosis perforans serpiginosa
Stellate	Star-shaped	Stellate pseudoscars on forearms after years of sun damage
Target lesions	Three zones: dark or blistered center surrounded by a second pale zone and a third zone of a rim of erythema	Erythema multiforme
Telangiectasia	Small, dilated superficial blood vessels	Rosacea, steroid side effect
Ulcer	Loss of tissue affecting the entire epidermis and part of the dermis. Surface exudate &/or crusting often present. Heals with scarring.	Decubitus ulcer, pyoderma gangrenosum, venous (stasis) ulcer
Umbilicated	Round depression in center of papule	Molluscum contagiosum, umbilicated vesicles of herpes

(Continued text on following page)

Terminology of Dermatology (Continued)

Lesion	Definition	Examples
Vesicle	A small papule containing clear fluid	Dermatitis herpetiformis, dyshidrotic eczema, hand-foot-and-mouth disease, herpes simplex, pemphigus vulgaris, VZV
Warty/papillomatous	Finger-like projections on the surface	Nevus sebaceous, seborrheic keratosis, warts
Wheal (hive)	Transient swelling due to dermal edema; pruritic	Dermographism, urticaria, urticaria pigmentosa
Whorled	Swirling pattern	Epidermal nevi, incontinentia pigmenti (late)

Structure and Function of Skin



Three Layers of Skin

1. Epidermis: Keratinocytes, melanocytes, Langerhans cells, Merkel cells
2. Dermis: Collagen (types I & III), elastin, proteoglycans, hair follicles, glands (apocrine, eccrine, sebaceous), blood vessels, lymphatics, nerves
3. Subcutis (subcutaneous fat): Adipose tissue, large blood vessels, nerves

Epidermis: 4 Layers

1. Basal cell layer (stratum basalis)
2. Spiny cell layer (stratum spinosum)
3. Granular cell layer (stratum granulosum)
4. Cornified layer (stratum corneum)

Dermis: 2 Layers

1. Papillary dermis (thin zone beneath epidermis)
2. Reticular dermis (bulk of dermis)

Special Tests and Procedures

Special Tests & Procedures in Dermatology

BIOPSIES OF SKIN Choice of technique depends on the pathology suspected

1. Shave biopsy: A superficial skin biopsy with a #10 or #15 blade. Useful for elevated lesions (e.g., benign compound nevi). No sutures; bleeding stopped by Monsel's solution, aluminum chloride, or electrodesiccation.



2. **Punch biopsy:** A round “cookie-cutter” knife that obtains tissue specimen. Usually 3 to 5 mm is taken. Usually 1 to 2 sutures are required.

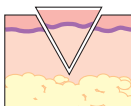
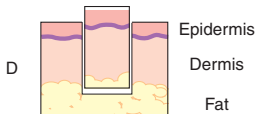
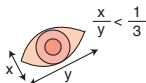
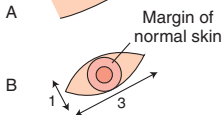
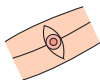
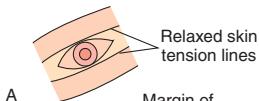


3. **Incisional biopsy (“wedge”):** An elliptical partial removal of a lesion down to fat. Useful, for example, when a malignancy is too large to remove by simple surgery, in scleroderma, panniculitis, and pyoderma gangrenosum. Requires layered closure with sutures.
4. **Excisional biopsy:** An elliptical complete removal of lesion down to fat. Useful in completely removing skin cancers. Requires layered closure with sutures.
5. **Curettage:** For hyperkeratotic lesions (e.g., SK, warts, BCC); often accompanied by cautery.
6. **Snip (scissors):** Skin tags, filiform warts



RIGHT

WRONG



Examples of excision ellipses on facial crease lines

A biopsy is warranted in:

1. Skin neoplasms.
2. Bullous disorders (with immunofluorescence simultaneously).
3. Autoimmune connective tissue diseases—lupus, dermatomyositis.
4. Skin disorders in which clinical exam is insufficient for diagnosis.

DERMOSCOPY A hand lens with built-in lighting and magnification (10–30X). It allows for the noninvasive inspection of deeper layers of the skin (dermal-epidermal junction and beyond). This is especially useful in distinguishing benign and malignant growth patterns in pigmented lesions.



DIASCOPY A microscope slide or magnifying glass is pressed against lesion.

- **Blanching:** Dilated capillaries (erythema)
- **Nonblanching:** Extravasated blood (purpura)
- **“Apple-jelly” color (yellow-brown):** Granulomas as seen in sarcoidosis, necrobiosis lipoidica, tuberculosis of skin, and granuloma annulare

IMMUNOFLUORESCENCE Tissue is transported in liquid nitrogen or Michel’s media (most commonly).

1. **Direct immunofluorescence (DIF):** A histologic stain for antibodies or other tissue proteins. Useful in bullous pemphigoid, cicatricial pemphigoid, dermatitis herpetiformis, epidermolysis bullosa acquisita, Henoch-Schönlein purpura, herpes gestationis, linear IgA bullous disease, lupus erythematosus, and pemphigus

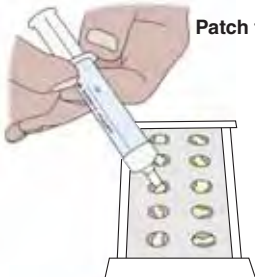
2. Indirect immunofluorescence (IIF): Examines the presence of circulating autoantibodies in the serum. Can give titers that are useful in pemphigus and other diseases to determine disease activity.

KOH (POTASSIUM HYDROXIDE) Scrape presumed dermatophyte infection with an alcohol pad or water and scrape with a #15 blade onto a slide. Then apply 2 drops of 10% to 20% KOH on slide to dissolve keratin for fungal elements. Heating accelerates the process. May have to wait several minutes to visualize.

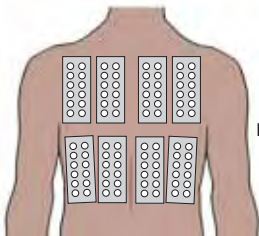
MAGNIFYING LENS Handheld magnifier or dermatoscope, or loupes, provide enhanced morphologic detail; should be carried around at all times.

PATCH TESTING: The test of choice for diagnosing allergic contact dermatitis. Patch test allergens are usually purchased prepared, but can be prepared individually. Allergens are placed in special wells (e.g., Finn chambers®) and applied to the back for 48 hours using Scanpor tape® and then removed; they are interpreted at 48 hours after first application, and then again at 96 hours. Contact hypersensitivity will show as a papular-vesicular reaction.

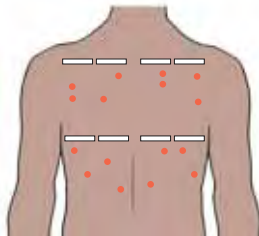
Patch testing methodology



A. Patch tests are prepared



B. Patches are affixed to upper back



C. Hypersensitivity is determined

+/-	= Macular erythema only
+	= Weak reaction: erythema, infiltration, papules (no vesicles)
++	= Strong reaction: edema, vesiculation
+++	= Extreme reaction: spreading, bullous, ulcerative
IRR	= Irritant morphologic appearance
-	= Negative reaction
NT	= Not tested

TZANCK SMEAR Quickly diagnoses HSV and VZV (doesn't distinguish between them). Scrape the base of an early blister, air dry or fix with ethanol, stain with Giemsa or Wright's stain. Look for multinucleated giant cells or atypical keratinocytes with large nuclei.

WOOD'S LAMP A 360-nm light source useful in localizing the site of melanin in Caucasian skin, diagnosing infections and porphyria. Specifically, it is useful in diagnosing disorders of pigmentation such as vitiligo; erythrasma; porphyria cutanea tarda; pseudomonas infection of the skin; tinea capitis; and tinea versicolor.

- Dull yellow: Tinea versicolor
- Yellow-green: Some Dermatophytes (esp. *Microsporum* species; *audouinii*, *canis*, *distortum*, *ferrugineum*, *gypseum*, & *T. schonleinii*)
- Coral red: Erythrasma
- Pale blue-green: Pseudomonas
- Dark pink urine: Porphyria cutanea tarda
- Completely white: Vitiligo

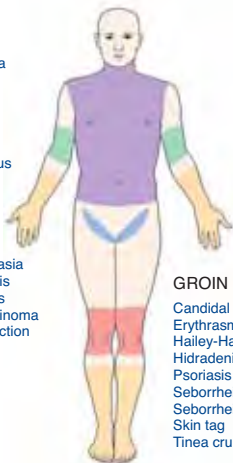
Dermatoses by Location

FACE

Acne
Actinic keratosis
Basal cell carcinoma
Contact dermatitis
Dermatomyositis
Herpes simplex
Impetigo
Keratoacanthoma
Lupus erythematosus
Melasma
Nevus
Perioral dermatitis
Rosacea
Sarcoidosis
Sebaceous hyperplasia
Seborrheic dermatitis
Seborrheic keratosis
Squamous cell carcinoma
Varicella-zoster infection
Vitiligo

LIMBS

Atopic eczema
Bullous pemphigoid
Cellulitis
Dermatofibroma
Erythema multiforme
Granuloma annulare
Henoch-Schönlein purpura
Keratosis pilaris
Lichen planus
Melanoma
Nevus
Psoriasis
Pyoderma gangrenosum
Seborrheic keratosis
Stasis dermatitis
Ulcer
Vasculitis



GROIN

Candidal intertrigo
Erythrasma
Hailey-Hailey disease
Hidradenitis suppurativa
Psoriasis
Seborrheic dermatitis
Seborrheic keratosis
Skin tag
Tinea cruris

GENITALIA

Herpes simplex
Lichen planus
Lichen sclerosus
Molluscum contagiosum
Psoriasis
Scabies
Syphilis (chancre)
Wart
Zoon's balanitis

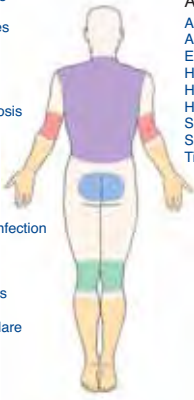
Dermatoses by Location (Continued)

TRUNK

Acne
 Basal cell carcinoma
 Cherry angioma
 Darier disease
 Drug eruption
 Epidermal cyst
 Folliculitis
 Grover disease
 Keloid
 Lipoma
 Melanoma
 Molluscum contagiosum
 Morphea
 Mycosis fungoides
 Neurofibroma
 Nevus
 Pityriasis rosea
 Psoriasis
 Seborrhic keratosis
 Skin tag
 Striae
 Syphilis
 Tinea corporis
 Tinea versicolor
 Urticaria
 Varicella-zoster infection

SCALP

Actinic keratosis
 Alopecia areata
 Androgenetic alopecia
 Dermatitis
 Epidermal or pilar cyst
 Nevus
 Pediculosis (lice)
 Psoriasis
 Seborrhic dermatitis
 Squamous cell carcinoma
 Tinea capitis



AXILLA

Acanthosis nigricans
 Allergic contact dermatitis
 Erythrasma
 Hailey-Hailey disease
 Hidradenitis suppurativa
 Hyperhidrosis
 Seborrhic dermatitis
 Skin tag
 Tinea corporis

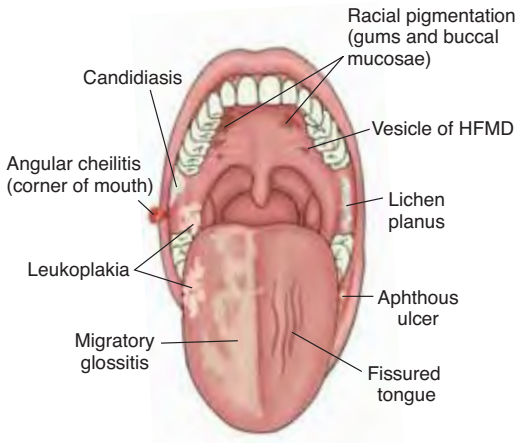
FEET

Contact dermatitis
 Corn
 Granuloma annulare
 HFMD
 Keratoderma
 Lichen planus
 Nevus
 Onychomycosis
 Plantar wart
 Psoriasis
 Tinea pedis

HANDS

Actinic keratosis
 Atopic eczema
 Contact dermatitis
 Erythema multiforme
 Granuloma annulare
 HFMD
 Hyperhidrosis
 Keratoacanthoma
 Lichen planus
 Psoriasis
 Scabies
 Syphilis
 Warts

Dermatoses by Location (Continued)



Diagnostic Hints: What to Do When You're Uncertain

Did you consider:

1. Drug eruption?
2. Factitial?
3. Lupus erythematosus?
4. Mycosis fungoides?
5. Occupational or hobbies?
6. Sarcoidosis?

7. Scabies?
8. Syphilis?
9. Systemic illness?

Did you think of these broad categories?

1. Infectious: E.g., bacterial, fungal, parasitic, spirochetal, viral
2. Neoplastic
3. Inflammatory/autoimmune
4. Allergic: E.g., contact dermatitis
5. Drug reaction
6. Metabolic
7. Genetic

Did you examine for or enquire about:

1. Focus of infection?
2. Foreign travel?
3. Mucosal clues?
4. Nail clues?
5. Over-the-counter, herbal, or natural medicines?
6. Pets?
7. Scalp clues?
8. Tinea pedis?
9. Tumor?

Did you:

1. Biopsy?
2. Culture?
3. Envision the lesion at another site?
4. Order blood studies?
5. Order imaging studies?
6. Patch test?
7. Perform a KOH?
8. Really listen to the patient?

Pruritus/Itch

Causes of Localized Pruritus

Body Region	Examples
Scalp	Pediculosis capitis (lice), psoriasis, seborrheic dermatitis
Trunk	Contact dermatitis (axillae, waistline), erythrasma (axillae), pediculosis corporis, psoriasis, scabies, seborrheic dermatitis (chest), seborrheic keratoses, urticaria
Groin	Candida, contact dermatitis, erythrasma, pediculosis pubis, scabies, tinea cruris
Anal region	Candidiasis, contact dermatitis, gonorrhea, hemorrhoids, pinworms, psoriasis, tinea cruris
Hands	Contact dermatitis, eczema (atopic, dyshidrotic), scabies
Legs	Atopic dermatitis, dermatitis herpetiformis (knees), lichen simplex chronicus (malleoli), nummular eczema, stasis dermatitis
Feet	Contact dermatitis, dyshidrotic eczema, pitted keratolysis, tinea pedis

Causes of Generalized Pruritus

Body Region	Examples
Skin diseases	Contact dermatitis; dermatitis herpetiformis; eczema—atopic, nummular, bullous pemphigoid, mycosis fungoides, psoriasis, PUPPP, urticaria, xerosis
Endocrine disorders	Carcinoid syndrome, diabetes, hypo- & hyperthyroidism

(Continued text on following page)

Causes of Generalized Pruritus (Continued)

Body Region	Examples
Infectious diseases	Hepatitis C, HIV, pediculosis, scabies
Psychiatric disease	Anxiety, delusions of parasitosis, depression
Systemic disease	Chronic renal failure, drugs (medicine, drugs of addiction/abuse), hemochromatosis, hepatic cholestasis, pregnancy (e.g., cholestasis of pregnancy), primary biliary cirrhosis
Hematopoietic disorders	Hodgkin's disease, iron deficiency, anemia, lymphoma, polycythemia rubra vera

Skin Manifestations of Systemic Disease

Skin Manifestations of Chronic Renal Failure

Changes in skin pigmentation: Bruising, hyperpigmented palms & soles, pallor, photodistributed or diffuse hyperpigmentation, uremic frost, yellow tinge

Infections: Onychomycosis, tinea pedis

Nail changes: Half-and-half nails, pale nails, splinter hemorrhages

Pruritus

Xerosis

Keratotic palmoplantar pits

Perforating disorders: Kyrle disease, reactive perforating collagenosis

Calcinosis cutis

Skin Manifestations of Endocrine Diseases

Acromegaly: Acanthosis nigricans, soft tissue hypertrophy, seborrheic dermatitis

Addison's disease: Increased skin pigmentation

Cushing's syndrome: Acanthosis nigricans, acne, fat maldistribution (e.g., "Buffalo hump"), hirsutism, ruddy skin with telangiectasia, striae, thin & atrophic skin

Diabetes: Acanthosis nigricans, bullous diabeticorum, diabetic dermopathy, foot ulcers, fungal & bacterial skin infections, granuloma annulare (controversial), necrobiosis lipoidica (diabeticorum), pruritus, scleredema, skin tags, yellow skin & nails

Hyperthyroidism: Fine hair, hair loss, hyperhidrosis, onycholysis, pretibial myxedema, pruritus, warm smooth skin

Hypothyroidism: brittle nails, broken hair, coarse hair, diffuse hair loss, loss of outer third of eyebrow, pruritus, thickened yellow skin, xeroderma

Skin Manifestations of Gastrointestinal Diseases

Acrodermatitis enteropathica: Abnormality in zinc absorption

Dermatitis herpetiformis: Gluten-sensitive enteropathy

Eczema (flexural): Malabsorption, mild zinc deficiency

Gardner's syndrome: Large intestinal polyps

Liver disease, chronic: Dilated abdominal wall veins, gynecomastia, jaundice, loss of body hair, palmar erythema, peripheral edema, purpura, spider angiomas,

Perineal ulceration, sinus tracts: Crohn's disease

Peutz-Jeghers syndrome: Small intestine polyps

Pyoderma gangrenosum: Crohn's disease, rheumatoid arthritis, ulcerative colitis

Skin Manifestations of Other Systemic Diseases

Hyperlipidemia: Xanthomas—tendinous, tuberous, eruptive

Neurofibromatosis: Café au lait macules, freckling (axillary, inguinal; "Crowe's sign"), multiple cutaneous neurofibromas

Pellagra (nicotinic acid deficiency): 3Ds—dementia, dermatitis, diarrhea; erythema following sun exposure

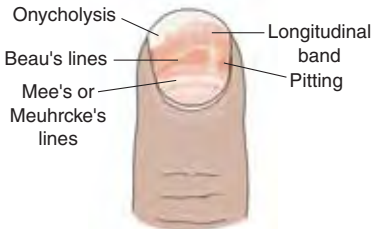
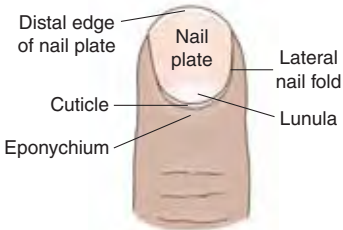
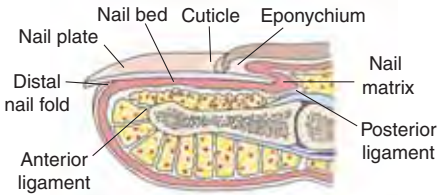
Porphyria: Blister formation, hypertrichosis, milia, photosensitivity, pigmentation, skin fragility

Sarcoidosis: Blue-red subcutaneous nodules, lupus pernio, sarcoid in scar

Scurvy: Bleeding gums, coiled hairs, perifollicular purpura, poor wound healing

Tuberous sclerosis: Confetti macules, enamel pits, facial angiofibromas, forehead plaque, gingival fibromas, hypomelanotic macules, periungual fibromas, Shagreen patch

Nails



Nail Findings and Associated Systemic Conditions

Nail Finding	Associated Systemic Condition
Shape or Growth Change	
Beau's lines	Any severe systemic illness that disrupts nail growth, pemphigus, Raynaud's disease, trauma
Clubbing	Asbestosis, atrioventricular malformations, chronic bronchitis, cirrhosis, congenital heart disease, COPD, endocarditis, fistulas, inflammatory bowel disease, pulmonary malignancy
Koilonychia	Hemochromatosis, iron deficiency anemia, nail-patella syndrome, Raynaud's disease, SLE, trauma
Onycholysis	Amyloidosis, connective tissue disorders, hyperthyroidism, infection, psoriasis, sarcoidosis, trauma
Pitting	Alopecia areata, incontinentia pigmenti, psoriasis, Reiter's syndrome
Yellow nail	Bronchiectasis, immunodeficiency, lymphedema, nephrotic syndrome, pleural effusion, Raynaud's disease, rheumatoid arthritis, sinusitis, thyroiditis, tuberculosis
Color Change	
Azure lunula	Hepatolenticular degeneration (Wilson's disease), quinacrine therapy, silver poisoning
Dark longitudinal streaks	Benign nevus, chemical staining, melanoma, normal variant in darkly pigmented people
Half-and-half nails	Specific for renal failure
Longitudinal striations	Alopecia areata, atopic dermatitis, vitiligo, psoriasis
<i>(Continued text on following page)</i>	

Nail Findings and Associated Systemic Conditions (Continued)

Nail Finding	Associated Systemic Condition
Mees' lines	Arsenic poisoning, carbon monoxide poisoning, chemotherapy, CHF, Hodgkin's disease, leprosy, malaria, other systemic insults
Muehrcke's lines	Specific for hypoalbuminemia
Splinter hemorrhage	Antiphospholipid syndrome, malignancies, oral contraceptive use, pregnancy, psoriasis, rheumatoid arthritis, SLE, subacute bacterial endocarditis, trauma
Telangiectasia	Dermatomyositis, rheumatoid arthritis, scleroderma, SLE
Terry's (white) nails	Cirrhosis, CHF, diabetes mellitus, hepatic failure, hyperthyroidism, malnutrition

Skin Disorders With Nail Changes

Disease	Incidence	Findings
Alopecia areata	10%	Fine pits, rough nail surface
Darier's disease	High	Longitudinal ridging, red & white longitudinal bands, V-neck, hyperkeratosis
Eczema	Common	Coarse pitting, shiny nails, transverse ridging
Lichen planus	10%	Coarse pitting, shiny nails, transverse ridging
Pityriasis rubra pilaris	Majority	Hyperkeratotic, yellow-brown
Psoriasis	10%–50%	"Oil spots," onycholysis, pits, splinter hemorrhages, subungual hyperkeratosis
Scleroderma	Frequent	Pterygium inversus unguium

Drug Eruptions in Dermatology

Typical presentations (in descending order of frequency):

Exanthema (maculopapular, morbilliform)

Urticaria and angioedema

Fixed drug eruptions

Erythema multiforme

Stevens-Johnson syndrome

Exfoliative dermatitis

Photosensitivity reactions

Anaphylaxis

Toxic epidermal necrolysis

Drugs most commonly resulting in an adverse skin reaction:

Amoxicillin

Trimethoprim-sulfamethoxazole

Ampicillin

Whole blood

Cephalosporins

Allopurinol

Carbamazepine

Drugs Causing Skin Pigmentation Changes

Color	Drug
Blue-gray	Gold
Brown	ACTH, bleomycin, minocycline, oral contraceptives, zidovudine
Gray	Amiodarone, antimalarials, minocycline, phenothiazines
Red	Clofazimine
Yellow	Beta-carotene, quinacrine

Drug Eruptions & Likely Patterns

Drug	Pattern of Eruption
Antimalarials	Lichen planus-like eruption
Barbiturates	Blistering of lower limbs
Beta-blockers	Psoriasiform
Iodides and bromides	Acneiform eruption
Minocycline	Hyperpigmentation
Penicillamine	Pemphigus-like eruption
Penicillins, sulfonamides	Morbilliform, erythema multiforme, SLE
Phenothiazines	Photosensitive dermatosis
Salicylates	Angioedema, urticaria

Skin Reaction Patterns & Drugs That Cause Them

Reaction Pattern	Likely Drugs
Acne	Corticosteroids, lithium
Alopecia	Anti-thyroid drugs, coumarin anticoagulants, cytotoxics (e.g., chemo)
Erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis	Allopurinol, antibiotics, anticonvulsants, NSAIDs, sulfonamides
Erythema nodosum	Oral contraceptives, sulfonamides
Erythroderma	Antibiotics, anti-rheumatic drugs
Exfoliative dermatitis	Gold, isoniazid, phenylbutazone
Photosensitivity	Phenothiazines, tetracyclines
Psoriasiform eruptions	Antimalarials, beta-blockers, lithium
SLE-like syndrome	Hydralazine, minocycline, penicillin, sulfonamides

(Continued text on following page)

Skin Reaction Patterns & Drugs That Cause Them (Cont'd)

Reaction Pattern	Likely Drugs
Toxic erythema	Antibiotics (e.g., ampicillin), anti-rheumatics, barbiturates, sulfonamides
Vasculitic and purpuric eruptions	Indomethacin, phenytoin

Effects of Aging on Skin

Intrinsic

HISTOPATHOLOGIC *Decreased:* corneocyte adhesion, dermal collagen, dermal elastic fibers, dermal ground substance, eccrine sweat glands, epidermal thickness, number of hair follicles, subcutaneous fat (site-dependent)

CLINICAL Gray hair, thinning & ridging of nails

Extrinsic (Primarily UV Light)

Comedones (e.g., Favre-Racouchot syndrome)

Dry skin (xerosis)

Elastosis (yellow skin)

Freckles

Idiopathic guttate hypomelanosis

Senile/solar purpura

Solar lentigo

Telangiectases

Venous lakes

Wrinkling/rhytides

Also: Actinic keratoses, Bowen's disease, rosacea, roughness, sallow skin tone, seborrheic dermatitis, seborrheic keratoses, skin cancers (BCC, SCC), stasis dermatitis & chronic venous insufficiency, stucco keratoses

Aging Effects by Disease Process

Autoimmune: Bullous pemphigoid

Benign tumors: Cherry angiomas, chondrodermatitis nodularis helices, seborrheic keratosis, skin tags

Eczemas: Asteatotic/dry skin, contact, seborrheic
 Infections: Candidiasis, herpes zoster, onychomycosis, scabies
 Photodamage: Actinic elastosis, colloid milium, Favre-Racouchot syndrome, freckling, photoaging (wrinkling, solar lentigo), poikiloderma of Civatte
 Premalignant: Actinic keratosis, Bowen disease
 Malignancies: BCC, lentigo maligna melanoma, MF, SCC
 Ulcerations: leg, pressure/decubitus
 Other: Cutaneous horn, pruritus

Effects of Pregnancy on Skin

Dermatoses of Pregnancy

1. Pemphigoid gestationis/herpes gestationis
 2. Cholestasis of pregnancy
 3. Pruritic urticarial papules and plaques of pregnancy (PUPPP)/polymorphic eruption of pregnancy (PEP)
 4. Prurigo of pregnancy—may include pruritic folliculitis
- #1,2: Fetal risks; #3,4: no fetal risks.

Pigmentary Changes of Pregnancy

1. Hyperpigmentation: anogenital region, areola-nipple complex, axillae, inner thighs
2. Linea nigra on abdomen
3. Melasma
4. Nevi and freckles darken

Vascular Changes of Pregnancy

1. Gingival hyperemia or hyperplasia
2. Hemorrhoids
3. Nonpitting edema
4. Palmar erythema
5. Pyogenic granulomas
6. Spider angiomas
7. Varicose veins
8. Vasomotor instability

Note: Impetigo herpetiformis (acute form of pustular psoriasis in pregnancy) no longer considered a specific dermatosis of pregnancy.

Hair Changes of Pregnancy

1. Hirsutism
2. Postpartum telogen effluvium
3. Postpartum androgenetic alopecia

Nail Changes of Pregnancy (None are Pathognomonic)

1. Subungual hyperkeratosis
2. Onycholysis
3. Transverse grooving
4. Brittleness

Connective Tissue Changes of Pregnancy

Striae gravidarum/stretch marks (in up to 90%)

Glandular Changes of Pregnancy

1. Increased eccrine & sebaceous function
2. Decreased apocrine function

Diseases Potentially Improved During Pregnancy

1. Acne
2. Atopic dermatitis
3. Fox-Fordyce disease
4. Hidradenitis suppurativa
5. Psoriasis
6. Rheumatoid arthritis
7. Sarcoidosis

Diseases Potentially Worsened During Pregnancy

1. **Infections:** Candida vaginitis, condyloma acuminata, herpes simplex, leprosy, pityrosporum folliculitis, trichomoniasis, VZV
2. **Immune-mediated diseases:** Dermatomyositis/polymyositis, lupus erythematosus, pemphigus vulgaris, systemic sclerosis
3. **Metabolic diseases:** Acrodermatitis enteropathica, porphyria cutanea tarda
4. **Connective tissue disorders:** Ehlers-Danlos syndrome, pseudoxanthoma elasticum
5. **Miscellaneous:** AIDS, cutaneous T-cell lymphoma, erythrokeratoderma variabilis, hereditary hemorrhagic telangiectasia, neurofibromatosis

Dermatologic Emergencies

Vesiculobullous Disorders

Stevens-Johnson syndrome, toxic epidermal necrolysis, pemphigus vulgaris, other generalized blistering disorders (e.g., bullous pemphigoid)

Infections

Cysticercosis cutis, hemorrhagic fevers, leprosy (reversal reaction upon treatment) mucormycosis, necrotizing fasciitis & other gangrene (e.g., gas gangrene, progressive bacterial synergistic gangrene), neonatal herpes simplex, Rocky Mountain spotted fever, Still's disease, trichinosis, tularaemia

Autoimmune Disorders

Acute eruption, systemic and neonatal lupus erythematosus & dermatomyositis

Inflammatory Cutaneous Disorders

Acne fulminans, acute drug eruptions (e.g., red man syndrome), acute pustular psoriasis, capillary hemangiomas (affecting respiratory tract or oral cavity, periorbital), exfoliative erythroderma, Kawasaki syndrome, pyoderma gangrenosum, scarring alopecias ("hair emergency")

Environmental

Burns, child abuse, cholesterol emboli, sclerema neonatorum

Presentation and Worrisome Differential

1. Generalized red rash with fever
 - a. Bacterial infections with toxin production
 - b. Drug eruptions
 - c. Rickettsial exanthems
 - d. Viral exanthems
2. Generalized red rash with blisters and prominent mouth lesions
 - a. Bullous pemphigoid
 - b. Drug eruptions
 - c. Erythema multiforme (major)
 - d. Toxic epidermal necrolysis

3. Generalized rash without redness with blisters, erosions, and mouth lesions
 - a. Pemphigus
4. Generalized red rash with pustules
 - a. Drug eruptions
 - b. Pustular psoriasis
5. Generalized rash with vesicles
 - a. Disseminated herpes simplex
 - b. Drug eruptions
 - c. Generalized herpes zoster
 - d. Varicella
6. Generalized red rash with scaling over whole body
 - a. Exfoliative erythroderma
7. Generalized wheals and soft tissue swelling
 - a. Urticaria and angioedema
8. Generalized purpura
 - a. Drug eruptions
 - b. Purpura fulminans
 - c. Thrombocytopenia
9. Generalized purpura that can be palpated
 - a. Bacterial endocarditis
 - b. Vasculitis
10. Multiple skin infarcts
 - a. Disseminated intravascular coagulopathy
 - b. Gonococcemia
 - c. Meningococcemia
11. Localized skin infarcts
 - a. Antiphospholipid antibody syndrome
 - b. Atheroembolization
 - c. Atherosclerosis obliterans
 - d. Calciphylaxis
 - e. Warfarin necrosis
12. Facial inflammatory edema with fever
 - a. Erysipelas
 - b. Lupus erythematosus

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Topical Medication Vehicles

Ointment

- Emulsion of *water in oil*
- Greasy vehicle that provides enhanced penetration; occlusive, therefore don't use on infected or oozing skin
- #1 (most lubricating) for dry skin, hyperkeratotic, and lichenified lesions (e.g., psoriasis, lichen simplex chronicus, chronic eczema)

Cream

- Emulsion of *oil in water*
- Less moisturizing than ointments, but more cosmetically appealing
- Useful in acute and subacute dermatoses

Gel

- Usually alcohol-based, transparent and colorless
- Liquefies on contact with skin
- Nongreasy, non-occlusive, nonstaining
- Use in *hair-bearing* areas or when *drying* is desired

Solution

- A liquid vehicle: propylene glycol, water or alcohol
- Useful under cosmetics, in *hair-bearing* regions, and when drying is desired

Lotion

- Suspension of a *powder in water*; leaves a film of uniform powder on skin
- Less moisturizing than a cream
- Good for *hair-bearing* areas

Foams

- Any area, but especially *scalp*
- Easy application, with less grease/residue

Powders

- Promote drying
- Used in *intertriginous* areas to reduce moisture, friction, maceration

Paste

- Mixture of powder and ointment
- Ihle's paste used for *diaper and intertriginous dermatitis*

Topical Steroids

Memorize and become comfortable prescribing 1 to 2 **topical steroids** within each steroid potency group.

Potency*

1 (Most Potent)

Cormax—Clobetasol propionate	0.05% (cream, ointment, solution)
Diprolene—Betamethasone dipropionate	0.05% (optimized vehicle; ointment)
Olux—Clobetasol propionate	0.05% (foam)
Psorcon—Diflorasone diacetate	0.05% (optimized vehicle; ointment)
Temovate—Clobetasol propionate	0.05% (optimized vehicle; cream, ointment)
Ultravate—Halobetasol propionate	0.05% (cream, ointment)

2

Cyclocort—Amcinonide	0.1% (ointment)
Diprolene AF—Betamethasone dipropionate	0.05% (cream)
Diprosone—Betamethasone dipropionate	0.05% (ointment)
Elocon—Mometasone furoate	0.1% (ointment)
Florone—Diflorasone diacetate	0.05% (ointment)
Halog—Halcinonide	0.1% (cream)
Kenalog—Triamcinolone acetonide	0.5% (ointment)
Lidex—Fluocinonide	0.05% (cream, gel, ointment)
Maxiflor—Diflorasone diacetate	0.05% (ointment)
Topicort—Desoximetasone	0.25% (cream, ointment), 0.05% (gel)

3

Aristocort A—Triamcinolone acetate	0.1% (ointment)
Aristocort HP—Triamcinolone acetate	0.5% (cream)
Benisone—Betamethasone benzoate	0.025% (gel)
Cutivate—Fluticasone propionate	0.005% (ointment)

*N.B.: Occlusion (e.g., under saran wrap) can increase potency 10 to 100 times.

Cyclocort—Amcinonide	0.1% (cream, lotion)
Diprosone—Betamethasone dipropionate	0.05% (cream)
Florone—Diflorasone diacetate	0.05% (cream)
Halog—Halcinonide	0.1% (ointment)
Lidex E cream—Fluocinonide	0.05% (cream)
Luxiq—Betamethasone valerate	1.2 mg/g (foam)
Maxiflor—Diflorasone diacetate	0.05% (cream)
Topicort LP emollient cream—Betamethasone	0.05% (cream)
Uticort—Betamethasone benzoate	0.025% (gel)
Valisone—Betamethasone valerate	0.1% (ointment)

4

Aristocort—Triamcinolone acetonide	0.1% (ointment)
Benisone—Betamethasone benzoate	0.025% (ointment)
Cordran—Flurandrenolide	0.05% (ointment)
Elocon—Mometasone furoate	0.1% (cream)
Halog—Halcinonide	0.025% (cream)
Kenalog—Triamcinolone acetonide	0.1% (ointment)
Synalar HP—Fluocinolone acetonide	0.2% (cream)
Synalar—Fluocinolone acetonide	0.025% (ointment)
Topicort LP—Desoximetasone	0.05% (cream)
Uticort—Betamethasone benzoate	0.025% (ointment)
Valisone—Betamethasone valerate	0.1% (lotion)
Westcort—Hydrocortisone valerate	0.2% (ointment)

5

Aristocort—Triamcinolone acetonide	0.25% (cream)
Benisone—Betamethasone benzoate	0.025% (cream)
Betatrex—Betamethasone valerate	0.1% (cream)
Cloderm—Clocortalone	0.1% (cream)
Cordran—Flurandrenolide	0.05% (cream)
Cutivate—Fluticasone propionate	0.05% (cream)
Dermasmoothe/FS—Fluocinolone acetonide	0.01% (oil)
Dermatop—Prednicarbate	0.1% (cream)
Diprosone—Betamethasone dipropionate	0.02% (lotion)
Fluonid—Fluocinolone acetonide	0.025% (cream)
Locoid—Hydrocortisone butyrate	0.1% (cream)
Synalar—Fluocinolone acetonide	0.025% (cream)
Uticort—Betamethasone benzoate	0.025% (cream)
Valisone—Betamethasone valerate	0.1% (cream)
Westcort—Hydrocortisone valerate	0.2% (cream)

6		
Aclovate—Aclometasone dipropionate	0.05% (cream, ointment)	
Desowen—Desonide	0.05% (cream)	
Kenalog—Triamcinolone acetonide	0.1% (cream, lotion)	
Synalar—Fluocinolone acetonide	0.01% (cream, solution)	
Tridesilon—Desonide	0.05% (cream)	
Valisone—Betamethasone valerate	0.1% (lotion)	

7 (Least Potent)

Cortaid—Hydrocortisone	0.5%, 1%, 2.5%
Decadron phosphate—Dexamethasone	0.1% (cream)
Generic—Hydrocortisone	0.5%, 1%, 2.5%
Hytone—Hydrocortisone	0.5%, 1%, 2.5%
Medrol—Methylprednisolone	1%

General Rules of Use Based on Location

Potency	Sites
Low	Babies' skin, face, genital skin, skin folds
Intermediate	Similar to "Low," but where skin is more thickened/chronic
High	Scalp; thick or chronic skin lesions
Ultra-high	Elbows, hyperkeratotic dermatoses, knees, palms, soles

Amounts Required

Area Treated	1 Application (g)	Bid for 1 Wk (g)	Bid for 1 Mo (g)
Anogenital, face, hands, head	2	28	120
1 arm, posterior or anterior trunk	3	42	180
1 leg	4	56	240
Entire body	30–60	420–840	1.8–3.6 kg

Use the adult finger-tip unit as your guide



Finger-tip measurements
 1 finger-tip length = 0.5 g
 2 finger-tip lengths = 1 g
 1 pump unit = 1 g

Topical steroids: approximate single application requirements

Child

0.5 g
Face and neck

2 g
Trunk (front and back)

0.5 g
One arm

1 g
Hands and feet

1 g
One leg



Adult

1.5 g
Face and neck

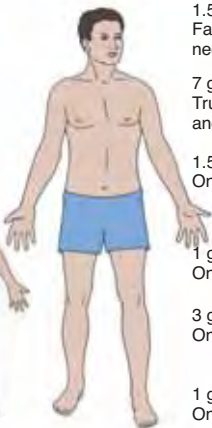
7 g
Trunk (front and back)

1.5 g
One arm

1 g
One hand

3 g
One leg

1 g
One foot



Regional Differences in Penetration: Most to Least

1. Mucous membrane
2. Scrotum
3. Eyelids
4. Face & scalp
5. Chest & back
6. Upper arms & legs
7. Lower arms & legs
8. Dorsa of hands & feet
9. Palmar & plantar skin
10. Nails

Local Skin Side Effects*

Acne	Rosacea
Atrophy—epidermal, dermal	Skin infections—worsened
Folliculitis	Steroid addiction syndrome
Hirsutism	Striae
Hypopigmentation	Telangiectasia
Perioral dermatitis	Ulceration
Purpura	Wound healing – delayed

Surgical Procedures Wisdom

- **Elderly** patients should slowly sit up and stay seated for a minute following a procedure, as their blood pressure needs time to adjust.
- Prior to and immediately after surgery, **avoid the Gs**: ginkgo, garlic, green tea, ginseng, & ginger (can cause bleeding after surgery). Also avoid Vitamin E and feverfew.
- Minor **bleeding** can often be stopped by Monsel's solution (most effective) or 20% aluminum chloride.
- To **protect yourself medicolegally**, ask patients "Do you not like the cosmetic appearance of this?" before cosmetic removal of SKs, moles, etc.

*N.B.: These are uncommon if the correct potency is used in the correct location, for the appropriate duration.

- To protect yourself from **needle sticks**, make a rule in your office that whomever uses a sharp object, disposes of that object. Never recap a needle.
- For small lesions, hand patients a **mirror** to point them out. You don't want to treat the wrong lesion.
- If using liquid nitrogen cryotherapy for **warts/molluscum** in kids, apply Maxilene 5 (or EMLA) by swab immediately after treatment to reduce pain.
- Dark-skinned patients and those having procedures on the upper back, shoulders or chest should always be warned of the risk of **keloids**.
- Consider having **consent forms** for every surgical, and certainly any cosmetic procedure.

Pregnancy Categories of Medication

FDA Pregnancy Drug Risk Categories

Drug Risk Category	Definition
X	Contraindicated in pregnancy No reason to risk use of drug in pregnancy.
D	Evidence for risk to human fetus However, benefits may outweigh risks of drug.
C	Risk cannot be ruled out—human studies lacking. Animal studies may or may not show risk. Potential benefits may justify potential risk.
B	No risk to human fetus despite possible animal risk. <i>Or</i> no risk in animal studies, and human studies have not been performed.
A	Controlled studies show no fetal risk.
Unrated	No pregnancy category assigned.

Dermatologic Drugs in Pregnancy

Topical drugs are in blue.

U	A	B	C	D	X
Cantharidine Nystatin Resorcin Salicylic acid* Ivermectin		Azelaic acid Bactroban Ciclopirox Clindamycin Clotrimazole EMLA Erythromycin Imiquimod Lindane Malathion Metronidazole Mupirocin Nafifine Penlac Permethrin Silver sulfa* Terbinafine Zonalon Acetaminophen	Adapalene Anthralin Bacitracin Benzoyl peroxide Calcipotriene Coal tar Crotaimton Hydroquinone Ketoconazole Levulan Kerastick Minoxidil Pimecrolimus Podophyllum Sodium sulfacetamide Steroids Sulfur Tacrolimus	Mechlorethamine Nitrogen mustard Aldactone/ Spironolactone Azathioprine Bleomycin Carmustine Colchicine Cyclophosphamide Danazole Flutamide Hydroxyurea Kanamycin Potassium iodide Progestogens Streptomycin Tetracyclines	Fluorouracil Tazarotene Acitretin Bexarotene Estrogens Finasteride Isotretinoin Leflunomide Methotrexate Thalidomide

(Continued text on following page)

Dermatologic Drugs in Pregnancy (Continued)

U	A	B	C	D	X
		Alefacept Azithromycin Cephalosporins Cetirizine Chlorpheniramine Cimetidine Cyproheptadine Diphenhydramine Etanercept Erythromycin Famciclovir Ibuprofen Infliximab Loratadine Metronidazole Nystatin	Tretinoin Vaniqua Acyclovir Aspirin Bactrim/Septa Beta carotene Chloroquine Ciprofloxacin Clotrimazole Codeine Cyclosporine Dapsone Denileukin Difitox Efalizumab Fexofenadine Fluconazole Fluoroquinolones Gold Griseofulvin		

(Continued text on following page)

Dermatologic Drugs in Pregnancy (Continued)

U	A	B	C	D	X
		Penicillins Prednisone Sulfasalazine Tacrolimus Terbinafine Valacyclovir	Hydroxychloroquine Infliximab Interferons Itraconazole Ivermectin IVIG Kenalog— intralesional Ketoconazole Methoxsalen Mycophenolate mofetil Nicotinomide Psoralens Rifampin Steroids Sulfonamides Thiabendazole Trimethoprim		

*Should be avoided in late pregnancy.

Cryotherapy



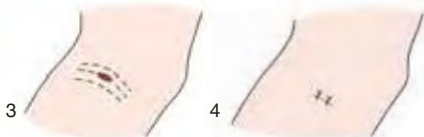
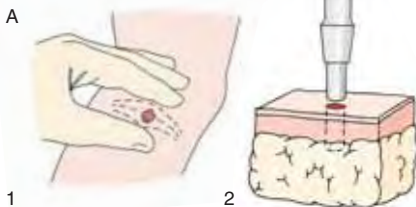
Liquid nitrogen—direct from spray canister ($-195.8\text{ }^{\circ}\text{C}$) or a dipped swab (much less cold)

Liquid nitrogen cryotherapy is most commonly used for: actinic keratoses, Bowen disease (SCC in-situ), molluscum contagiosum, myxoid cysts, pyogenic granulomas, skin tags, solar lentigines, and warts.

(See Image B on page 43.)

Punch Biopsy

A



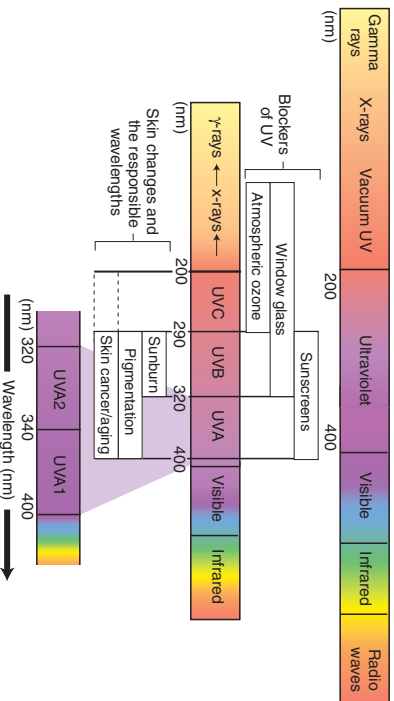
B



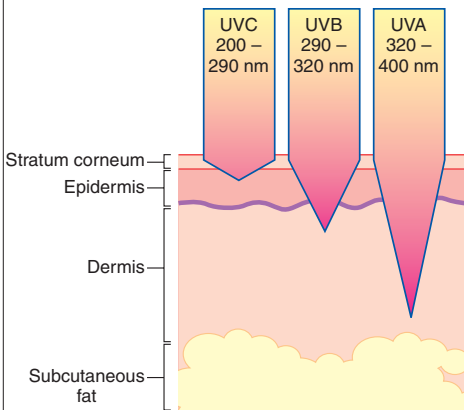
Photobiology & Phototherapy

UVA: 320–400nm
 (UVA-1 = 340–400nm; UVA-2 = 320–340nm) UVB: 290–320nm
 UVC: 100–290nm

Electromagnetic Spectrum with Expanded UV Region



Penetration of Different UV Wavelengths

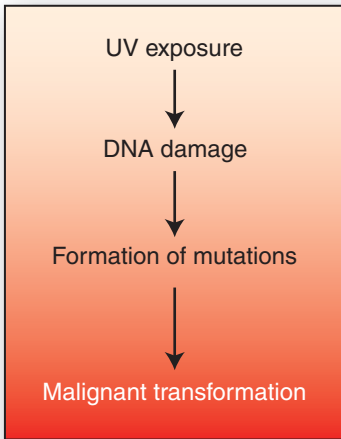


Human Skin Phototypes (SPT)

Skin Phototype	Unexposed Skin Color	Sun Response History
I	White	Always burns, never tans
II	White	Always burns, minimally tans
III	White	Burns minimally, tans gradually and uniformly
IV	Light brown	Burns minimally, always tans well
V	Brown	Rarely burns, tans darkly
VI	Dark brown	Never burns, tans darkly

*Those in SPT groups I & II are at highest risk for skin cancer.

Photocarcinogenic Cascade



Phototherapy is particularly helpful for:

- Eczema/atopic dermatitis
- Mycosis fungoides/cutaneous T-cell lymphoma
- Palmoplantar pustulosis
- Parapsoriasis
- Pityriasis rosea
- Polymorphous light eruption (prevention)
- Pruritus (esp. in renal failure)
- Psoriasis
- Vitiligo

- Other diseases may be helped: acne, alopecia areata, graft-versus-host disease, granuloma annulare, Langerhans cell histiocytosis, lichen planus, localized scleroderma, lymphomatoid papulosis, photodermatoses, pityriasis lichenoides, and urticaria pigmentosa

Photo/sun-aggravated dermatoses:

- Acne vulgaris (small %; most improve)
- Atopic dermatitis (small %; most improve)
- Bullous pemphigoid
- Darier disease
- Dermatomyositis
- Disseminated superficial actinic porokeratosis
- Erythema multiforme
- Grover disease
- Hailey-Hailey disease
- Lichen planus
- Lupus erythematosus
- Mycosis fungoides/cutaneous T-cell lymphoma (small %; most improve)
- Pellagra
- Pemphigus
- Pityriasis rubra pilaris
- Porphyria
- Psoriasis (small %; most improve)
- Rosacea
- Seborrheic dermatitis
- Viral infections—e.g., HSV

Drugs causing photosensitivity:

- ACE inhibitors
- Amiodarone
- Ciprofloxacin
- Nifedipine
- NSAIDs
- Phenothiazines
- Sulfonamides
- Tetracyclines (esp. doxycycline)
- Thiazides

Lasers

Laser Wavelengths

Lasers	Wavelength	Target	Uses
Resurfacing Lasers			
Carbon dioxide (CO ₂) laser	10,600 nm	Water	Resurfacing
Erbium:YAG laser	2940 nm	Water	Resurfacing; more superficial
Vascular Lesion Lasers			
Argon*	488, 514 nm	Oxyhemo- globin	Telangiectases, thick port-wine stains, venous lesions; high risk of scarring
Argon-pumped tunable dye	585–690 nm	Oxyhemo- globin	Telangiectases, thick port-wine stains
Copper vapor, yellow*	578 nm	Oxyhemo- globin	Telangiectases, thick port-wine stains
Flashlamp pulsed dye	585, 595 nm	Oxyhemo- globin	Telangiectases, thin port-wine stains, cherry angiomas, childhood hemangiomas, warts, scars, striae, poikiloderma of Civatte, leg veins

(Continued text on following page)

Laser Wavelengths (Continued)

Laser	Wavelength	Target	Uses
Krypton, yellow*	1568 nm	Oxyhemo- globin	Telangiectases, thin port-wine stains
Long-pulsed KTP	532 nm	Oxyhemo- globin	Facial telangiec- tases, leg veins
Long-pulsed Nd:YAG (safest in dark skin types)	1064 nm	Oxyhemo- globin	Leg veins
Pigmented Lesion Lasers			
Copper vapor, green*	511 nm	Melanin	Epidermal pig- mented lesions
Frequency- doubled Nd: YAG	532 nm	Melanin	Epidermal pigmented lesions, some red tattoos
Krypton, green*	521, 531 nm	Melanin	Epidermal pigmented lesions
Hair Removal Lasers**			
Alexandrite	755 nm	Melanin in hair	Hair removal
Diode	810 nm	Melanin in hair	Hair removal
Nd:YAG	1064 nm	Melanin in hair	Hair removal
Ruby	694 nm	Melanin in hair	Hair removal

(Continued text on following page)

Laser Wavelengths (Continued)

Laser	Wavelength	Target	Uses
Tattoo Lasers			
Frequency-doubled Nd:YAG	532 nm	Tattoo pigment	Red tattoos
Q-switched alexandrite	755 nm	Tattoo pigment	Black, blue, and green tattoos
Q-switched Nd:YAG	1064 nm	Tattoo pigment	Black and blue tattoos
Q-switched ruby	694 nm	Tattoo pigment	Black, blue, and green tattoos; Nevus of Ota

*These lasers are uncommonly used

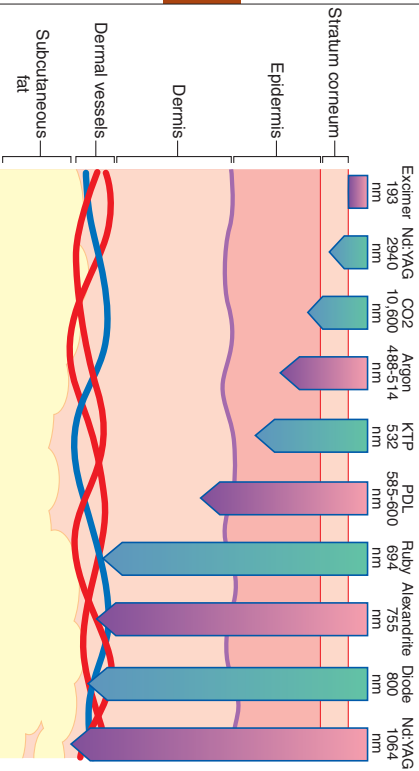
**Only effective on darkly pigmented hair; blond or gray hair resists treatment

N.B. #1: Q-switched technology allows higher energy & shorter pulses.

N.B. #2: New laser technology:

1. Excimer laser (308 nm): psoriasis, striae & vitiligo
2. 1450 diode or 1320 Nd:YAG laser: dermal collagen remodeling for wrinkles and atrophic scars.

Depth of Penetration by Various Lasers



Acanthosis Nigricans (AN)



Etiology: May be due to:

- a. Heredity
- b. Endocrine disorder—most commonly associated with insulin resistance (i.e., diabetes mellitus), hyperandrogenic state, hypothyroidism
- c. Obesity
- d. Drugs—e.g., nicotinic acid, niacinamide, oral contraceptives, steroids
- e. Malignancy, usually adenocarcinoma—e.g., gastrointestinal (60% stomach), lung, breast

History: Rule out aforementioned causes.

Physical: Hyperpigmented velvety, typically symmetrical plaques predominantly on the neck, axillae, and groin.

Physical location & morphology may be similar for AN of different etiologies; hence, Hx and Px should aim to include/exclude these associated conditions.

DDx: Eczema (e.g., lichen simplex chronicus), hyperpigmented nevus (e.g., Becker, epidermal), confluent and reticulated papillomatosis of Gougerot and Carteaud

Investigations: Screen for diabetes (glycosylated Hgb) and insulin resistance (plasma insulin); consider searching for underlying malignancy in adult onset.

Management

- Treat underlying disorder (e.g., remove tumor).
- Cases associated with obesity and/or insulin resistance may improve with weight loss and/or metformin.
- Management is difficult and mostly for cosmetic purposes.
- **Tx options:** Topical tretinoin, ammonium lactate, laser therapy, dermabrasion.

Acne Vulgaris



Inflammatory disorder of pilosebaceous follicles with a 90% prevalence in adolescence and young adulthood.



Etiology: Abnormal follicular keratinization, increased sebum 2° to androgens, *Propionibacterium acnes* (bacteria), inflammation. Genetic factors; occlusive cosmetic agents; medications: steroids, ACTH, androgens, danazol, iodides, lithium, antiepileptics, oral contraceptives; diseases: congenital adrenal hyperplasia (CAH), polycystic ovarian syndrome (PCOS); worse with emotional stress.

History: Often asymptomatic lesions (especially comedones), although can be tender (nodules).

Physical: Two types of lesions, predominantly affect face, neck, chest and back.

- Noninflammatory—open (“black heads”) and closed (“white heads”) comedones.
- Inflammatory—papules, pustules, cysts, nodules; deep lesions leave scars, inflammatory papules, pustules & nodules on back and chest with early scarring.

DDx: Folliculitis, perioral dermatitis, rosacea

Investigations: If irregular periods, hirsutism, virilization, or not responding to conventional therapy, work-up to rule out virilizing tumor or PCOS.

Management

Treatment Options Based on Acne Type

Treatment	Comedonal	Inflammatory	Nodulocystic
Topical Therapy			
Salicylic acid	X		
Retinoids	X	X	
Azelaic acid	X	X	
Benzoyl peroxide	X	X	
Antibiotics	X	X	
Systemic Therapy			
Oral contraceptives	X	X	X
Antibiotics		X	X
Isotretinoin	X	X	X

Selected Topical Medications for the Treatment of Acne

Medication	Dosage	Side Effects
Retinoids*		
Adapalene (Differin)	Qhs–bid	Same as tretinoin but less severe
Tazarotene (Tazorac)	Qhs	Same as tretinoin but more severe
Tretinoin (Retin-A)	Qhs	Dryness, scaling, erythema, burning, irritation, and photosensitivity
Tretinoin microsphere (Retin-A Micro)	Qhs	Same as tretinoin but less severe
Antibiotics		
Clindamycin	Qd–bid	Local irritation, dryness
Erythromycin	Qd–bid	Local irritation, dryness
Other		
Azelaic acid (Azelex)	Qd–bid	Dryness, scaling, erythema, burning, irritation, pruritus; rarely, hypopigmentation
Benzoyl peroxide (2.5%–10%)	Qd–bid	Erythema, peeling, contact dermatitis, dryness, bleaches bedding/clothing
Salicylic acid (Stri-Dex, Clearasil)	Qd–bid	Dryness, irritation
Sulfacetamide (Klaron)	Bid	Itching, redness, irritation; contraindicated if sulfa allergy
Sulfacetamide/sulfur (Sulfacet-R)	Bid	Same as sulfacetamide
Combination products (Duac, Benzamycin)	Qd–bid	Erythema, peeling, contact dermatitis, dryness, bleaches bedding/clothing

*Due to irritation, retinoids can be used every second day or as tolerated.

Selected Systemic Medications Acne Treatment

Medication	Dosage	Side Effects
Erythromycin	250 mg bid–qid; with meals	N/V/D, abdominal pain and cramps, rash, stomatitis, elevated liver transaminase levels, jaundice
Doxycycline	50–100 mg qd–bid (or 20 mg bid subantimicrobial dose); with meals	N/V/D, photosensitivity, stomatitis, discolored teeth (if <8 yr), esophagitis, lightheadedness, dizziness, vertigo, headache, pseudotumor cerebri
Tetracycline	500 mg bid; take on empty stomach	Same as doxycycline
Minocycline	50–100 mg qd–bid; with meals	Same as doxycycline (except less photosensitivity), plus rare lupus-like syndrome, or rare hypersensitivity reaction, and skin and mucous membrane hyperpigmentation
Isotretinoin	Start at 0.5–1 mg/kg/day. After 1 mo, full dose of 1 mg/kg/day to cumulative dose of 120–150 mg/kg (usually 15–20 wk) Lab tests: CBC, TG, ALT, B-hCG—baseline & monthly	<i>Common:</i> cheilitis, dry skin and mucous membranes, pruritus, epistaxis, conjunctivitis, photosensitivity, arthralgia, hypertriglyceridemia, elevated liver transaminase levels, decreased night vision <i>Rare:</i> pseudotumor cerebri, hepatotoxicity, major birth defects (Counsel regarding birth control/abstinence), cataracts, neutropenia, thrombocytopenia, reported cases of depression

N/V/D=5-nausea/vomiting/diarrhea.

- Other options: comedone extraction, intralesional cortisone injection (for papulonodules, cysts), oral contraceptives (esp. anti-androgenic such as cyproterone acetate, Yasmin®), photodynamic therapy, spironolactone (reduces androgen production; 50–200 mg/day); dermabrasion, chemical peels
- Treatment improves cosmesis and psychosocial well-being, and helps prevent further scarring and hyperpigmentation.
- Patients should be forewarned of acne exacerbations in the first month of systemic therapy as deep-seated acne comes to the surface. Also, systemic therapy can require 6 wk before benefits are noted.
- **Isotretinoin** should be taken with a fatty snack or meal that has some fat in it

Actinic Keratosis (AK; Solar Keratosis)



Most common precancerous lesions in humans, and more prevalent in fair-skinned individuals who tan poorly and burn easily.

Etiology: Sun exposure, ionizing radiation, and arsenic exposure.

Physical: Slightly erythematous, rough, scaly papules on sun-exposed areas, i.e., head, neck, backs of hands, and forearms.

May be difficult to see, but often feel rough: Palpation is essential to diagnosis. Occur on sun-exposed and damaged skin, most commonly in the elderly. Course: <1% of all untreated AKs will progress to squamous cell skin cancer.

DDx: BCC, Bowen disease, SCC

Investigations: Biopsy if recurrent, unresponsive to treatment, or pronounced hyperkeratosis and induration.

Management

TX Options

- Liquid nitrogen cryotherapy for limited # or thick lesions.
- Electrodesiccation and curettage, CO₂ laser (esp. for actinic cheilitis).
- Topical 5-fluorouracil (e.g., Efudex®) cream applied bid for 2–4 wk.
- Imiquimod (Aldara™ 5% cream) applied qd three to five times weekly for 12 wk.
- Patients must be warned that the treated areas will become irritated, inflamed, and eroded with both 5-fluorouracil and imiquimod; follow-up visit in 1–2 wk to evaluate severity of side effects.
- Photodynamic therapy, dermabrasion, & chemical peels (for numerous AKs).
- Multiple lesions are a harbinger of sun damage; patients should be checked annually for AKs and other skin cancers. Biopsy thick lesions or those not responding to above treatment as they may represent invasive squamous cell carcinoma.
- Sun-awareness and protection advice.

Alopecia



Alopecia (Continued)



Basics of Hair

3 Phases of Hair Development

1. Anagen (growing): 3–7 yr for scalp; 80%–90% of hairs; 50–100 switch to catagen/d (falling out)
2. Catagen (resting): 3–4 wk; 1% of hairs
3. Telogen (shedding): 3 mo; hairs have short club root; 50–100 hairs shed/day; 10%–15% of hairs in this phase

N.B.: Normal scalp contains 100,000 hairs. Blonds have 120,000 and those with red hair have 80,000 hairs. There are fewer hairs per square cm in Africans & Asians.

Drug Causes of Hair Loss

Drug Class	Drug
Anticoagulants	Coumarins, heparin
Antithyroid drugs	Carbimazole, thiouracil
Cytotoxic drugs	Adriamycin, colchicines, cyclophosphamide, mercaptopurine derivatives, vinca alkaloids

**Diffuse Hair Loss**

1. Alopecia areata (diffuse, although most commonly localized)
2. Drug-induced: cytotoxics, anticoagulants, retinoids
3. Endocrine disorders: hypothyroid, hypopituitarism
4. Hair-shaft defects: pili torti, monilethrix
5. Male-pattern baldness/androgenetic alopecia
6. Nutritional: iron deficiency
7. Syphilis
8. Systemic lupus erythematosus
9. Telogen effluvium

Localized Hair Loss

1. Alopecia areata
2. Discoid lupus erythematosus
3. Fungal infections (e.g., kerion)
4. Lichen planopilaris
5. Nevoid abnormalities
6. Traction (e.g., corn-rows, ponytails)
7. Trichotillomania

Nonscarring Alopecia: Intact Hair Follicles

1. Telogen effluvium (TE)
2. Alopecia areata (AA)
3. Anagen effluvium (following chemotherapy)
4. Androgenetic alopecia (AGA): male or female pattern baldness
5. Trichotillomania
6. Infections: fungal (i.e., tinea capitis, kerion)
7. Hair shaft abnormalities

Scarring alopecia: Loss of Hair Follicles

1. Discoid lupus erythematosus (DLE)
2. Lichen planopilaris (LPP)
3. Infections: fungal, bacterial, TB, leprosy

N.B.: If scalp biopsy performed (e.g., scarring alopecia), ideally a 4-mm punch biopsy is taken of a symptomatic or early active disease for transverse sectioning and H&E; if lupus suspected, take additional 4-mm punch biopsy—bisect this piece vertically, and submit half for vertical sectioning, and half for DIF.

*In general, scarring alopecias should be referred to a dermatologist.

Alopecia Areata



Autoimmune T-cell mediated disease in genetically predisposed persons. Uncommon association with other autoimmune diseases—diabetes, vitiligo, thyroid disease

Physical: Well-circumscribed areas of complete hair loss on any hair bearing surface (scalp = 90%); may see nail pitting; “exclamation point” hairs at periphery of bald patch

■ **Totalis** = entire scalp; **universalis** = all hair-bearing areas; **ophiasis** = hair loss confluent along temporal & occipital scalp

DDx: androgenetic alopecia, tinea capitis, trichotillomania, telogen effluvium

Investigations: ferritin, TSH, ANA, scalp biopsy if unsure

Management

- High-potency topical steroids; intralesional triamcinolone acetonide (5–10 mg/cc) q 4–6 wk in adults has best outcome.
- Some benefit reported with topical anthralin, topical minoxidil.
- Less commonly: Topical immunotherapy (i.e., diphencyprone), oral steroids (use in early rapidly progressing widespread disease), cyclosporine.
- Course: 95% regrow in 1 yr (except totalis or universalis, which have poor prognosis); 30% recurrence.
- Poor prognostic signs: Atopic dermatitis, childhood onset, duration > 5 yr, onychodystrophy, ophiasis, widespread involvement.

Androgenetic Alopecia (Male and Female Pattern Baldness)



Etiology: Heredity (polygenic); androgen stimulation

Physical: Different pattern for men and women; men—temporal areas affected, progressing to vertex; females—diffuse pattern.

DDx: Alopecia areata, anagen/telogen effluvium

Investigations: ferritin, TSH, androgen profile (if suspicious of virilization).

Management

- Topical minoxidil lotion (Rogaine®; 2% or 5%) use daily to stabilize hair loss and regrowth in some cases.
- Finasteride (Propecia®) 1 mg po qd in men (inhibits synthesis of dihydrotestosterone).
- Spironolactone 100–200 mg po daily in women.
- Hair transplantation (from occipital scalp).
- Hair prosthesis (wig).

Telogen Effluvium



Diffuse decrease in hair density secondary to rapid conversion of anagen to telogen hair. Precipitated by stressful events, illness, fever, pregnancy, crash diet, medications; hair can be shed 2–6 mo after precipitant.

Physical: Diffuse hair loss; positive hair pull test (>10% club hairs).

Investigations: CBC, ferritin, TSH; punch biopsy if unsure.

DDx: Alopecia areata, anagen effluvium, androgenetic alopecia.

Management

Reassurance. Wait for regrowth, and treat any underlying cause. Topical minoxidil may be beneficial.

Trichotillomania



Self-induced (neurotic) compulsive hair pulling/plucking.

Physical: Circumscribed area of alopecia with irregular borders and broken hairs of different length. May find scalp excoriations and perifollicular petechiae; usually only one area (frontoparietal or frontotemporal) affected. Eyebrows and eyelashes may also be plucked.

Investigations: Punch biopsy if unsure.

DDx: Alopecia areata, traction alopecia, tinea capitis.

Management

- When in doubt, biopsy confirms diagnosis.
- Ask about traumatic events (death, separation, school troubles)
- Difficult problem; look for precipitant; may need referral to psychiatry (psychotherapy, behavior therapy, SSRIs).
- Most children outgrow condition, but can be difficult to manage in adults.

Angioma (Cherry Angioma)



Most common benign cutaneous vascular growth, which increases in size and number with age.

History: asymptomatic; uncommonly bleeds with trauma

Physical: Cherry-red dome-shaped round papules predominantly on trunk of adults; 0.5–5 mm.

Investigations: Biopsy if diagnosis doubtful.

DDx: Angiokeratoma, insect bite, melanoma.

Management

Treatment for cosmetic purposes

Tx options: shave excision, electrodesiccation, vascular laser, cryotherapy

Angular Cheilitis



Etiology: Mechanical irritation (e.g., intertrigo), infectious (e.g., candidiasis), nutritional (e.g., iron or riboflavin deficiency), contact dermatitis. More common with dentures and in elderly.

History: Often chronic; discomfort and burning at corners of mouth.

Physical: Inflammatory moist fissures radiating down and out from the lip commissures; may observe: atrophy, fissures or maceration, erythema, crusting and scaling of the angles of the mouth.

Investigations: Culture to rule out candidiasis & bacterial infection; KOH prep.

DDx: Lip-licking dermatitis, impetigo.

Management

- Improving dentures and/or dentition
- Topical miconazole or nystatin cream
- Topical mupirocin
- Combination cortisone, antibacterial, anticandidal ointment often beneficial
- Injectable fillers

Aphthous Ulcers (Aphthous Stomatitis; "Canker Sores")



Etiology: Idiopathic; may have genetic basis, infectious and immune mechanisms play a role.

History: Common (10%–20%), often recurrent, painful mouth ulcerations. May have a prodromal stage of burning or pricking sensation of the oral mucosa immediately prior to ulcers appearing. Precipitating factors include local trauma or food hypersensitivity.

Physical: Round to ovoid shallow mouth ulcers (3–8 mm) with circumscribed margins and erythematous borders. Most common on buccal & labial mucosa and edges of tongue.

Investigations: Viral culture to exclude herpes infection.

DDx: Behçet or Crohn's disease, herpes simplex, pemphigus vulgaris, traumatic ulcers, neutropenic ulcers.

Management

Lesions resolve in 1–2 wk w/o Tx; avoid spicy or citrus foods. Choice of therapy dependent upon severity of symptoms and frequency of recurrence.

- symptomatic relief with topical anaesthetics (e.g., lidocaine gel/sol'n).
- NSAIDs.
- Topical or intralesional corticosteroids; 1–3 days of prednisone hastens/aborts attack.
- Antimicrobial mouth rinses (e.g., tetracycline).
- Sucralfate suspension qid to ulcers.
- continuous debilitating episodes: oral steroids, colchicine 0.6 mg po bid to tid, or rarely, thalidomide.

Atopic Dermatitis (Atopic Eczema)



Atopic Dermatitis (Atopic Eczema) (Continued)



Chronic inflammatory dermatosis affecting 10%–20% of children (esp. infants and young children)

Etiology: Cutaneous immune dysfunction; IgE-mediated; genetics. Strong association with personal and family history of atopy (eczema, asthma, hay fever).

History: Pruritus is a hallmark (“the itch that rashes”). Aggravating factors: sweating, contact sensitivity, secondary infection, wool, food allergy, stress/anxiety.

Physical:

- Acute: Erythematous, excoriated, scaling patches and plaques; more lichenification in chronic forms
- Distribution:
 - Infant: Cheeks, forehead, scalp, extensor surfaces of extremities.
 - Child: Flexural surfaces—antecubital and popliteal fossae, wrists, ankles.
 - Adults: Hands.
- Lesions can be secondarily infected with staphylococcal organisms: impetigo.

DDx: Contact dermatitis, psoriasis, seborrheic dermatitis.

Investigations: Allergy or RAST testing of little value.

Atopic Dermatitis U.K. Working Party Criteria (1994)

Basal Cell Carcinoma (BCC)



Most common skin cancer derived from stem cells of epidermis.

Etiology: Chronic UV exposure, radiation, immunosuppression, genetics (e.g., nevoid basal cell carcinoma syndrome).

History: Persistent, nonhealing papule or nodule that ulcerates or bleeds. Common in elderly Caucasians.

Physical: Pearly papule or nodule with telangiectases, rolled border; central crust or ulceration.

Distributed mostly on sun-exposed areas, i.e., head and neck (85%). **Variants:** Cystic, superficial, nodular, sclerosing (morpheaform), pigmented.

DDx: Melanoma, nevus, SCC.

Investigations: Biopsy must be performed to confirm diagnosis and classify subtype.

Management

- Advise that metastases and death are extremely rare. Vast majority cause no major problem, but should be treated.
- **Tx** options depend on histological subtype and location: Liquid nitrogen cryotherapy (aggressive; requires experience), curettage & electrodesiccation (most common Tx), excision, Mohs micrographic surgery, imiquimod (Aldara™) cream for superficial subtype, 5-FU, CO₂ laser, radiation therapy.
- Advise on sun protection, regular TBSE.

Bullous Pemphigoid (BP)



Bullous Pemphigoid (BP) (Continued)



Etiology: IgG produced against antigens in the dermal-epidermal basement membrane leads to subepidermal tense bullae.

History: Autoimmune bullous skin disease observed predominantly in patients > 60 yr; usually self-limited over 5–6-yr period. It may be accompanied by pruritus, especially in urticarial (early) form of BP.

Physical: Large tense bullae and denuded areas on erythematous or normal skin; 20% have oral lesions.

Investigations: Lesional biopsy for histology and perilesional for immunofluorescence.

DDx: Bullous impetigo/tinea, pemphigus (or many other autoimmune blistering conditions), drug-induced bullous disorders.

Management

- Topical strong steroids, e.g., clobetasol cream/ungt bid (for localized disease).
- Can use oral prednisone (0.75 mg–1 mg/kg/d) +/- steroid sparing agents (e.g., azathioprine, mycophenolate mophetil).
- Methotrexate (lower doses than psoriasis; often 7.5 mg–15 mg sufficient).
- Tetracycline (1g–2 g/d) and nicotinamide (1.5 g–2 g/d) effective in some cases.

Candidiasis



Courtesy of Dr. William Gerstein

Etiology: Cutaneous or mucous membrane infection caused by *Candida albicans* yeast. More prevalent in immunosuppressed patients, diabetics, patients who've had prolonged use of antibiotics, corticosteroids, or immunosuppressive agents; heat, humidity, and shear friction promote infection.

History: Skin surfaces in close proximity that provide a warm and moist environment. Pruritic red rash. Paronychia and onychomycosis present as painful and red areas around the nail and can be associated with immersion of hands in water and with diabetes mellitus.

Investigations: KOH microscopy of scrapings from lesions reveals mycelium & spores; confirmatory fungal culture.

DDx: Eczema, psoriasis, seborrheic dermatitis.

Candidal intertrigo is a common cutaneous pattern: E.g., groin and gluteal folds, the inframammary region, axilla and the interdigital spaces of the hands and feet affected.

Physical: Initially vesicles, pustules, or erythematous plaques, progressing to maceration and fissuring. Central erythematous plaque often bordered by discrete pustules in a "satellite pattern."

Oral candidiasis = thrush: Chronic mucocutaneous candidiasis may be associated with autoimmune diseases and endocrinopathies.

Physical: White plaques occur on the mucosal surfaces of the mouth (thrush); lesions may be removed by scraping, yielding an erythematous base.

Candida paronychia (nail infection) is often a result of chronic water exposure and trauma: Painful red swelling of periungual skin.

Management

Prevention: Avoid heat, humidity, and tight-fitting clothing.

Tx

- Identify and control underlying diseases, e.g., diabetes.
- Topical antifungals: E.g., nystatin, ketoconazole, clotrimazole, bid x 2 wk
- Oral antifungals in extensive mucocutaneous infections: E.g., ketoconazole 200 mg po qd x 10 d.
- Vaginal candidiasis: Single dose oral fluconazole 150 mg effective; topical & suppositories can be tried.

Cellulitis



Etiology: Soft tissue infection and inflammation caused by *Streptococcus pyogenes* or *Staphylococcus aureus*. Facial cellulitis in children is most commonly caused by *Haemophilus influenzae*.

History: Local trauma, abrasions, and dermatoses (e.g., stasis dermatitis and tinea pedis) can be predisposing factors; drug use; impaired lymphatic drainage (e.g., lymphedema). Local pain and swelling are common. Fever, chills, and malaise may be noted.

Physical: Area of spreading erythema, warmth, tenderness, pitting; associated with fever and an elevated white blood cell count.

Investigations: If moderate-severe, consider: CBC&D, blood cultures, BUN, Cr, culture fluid from bulla or abscess.

DDx: Erysipelas, impetigo, insect bites, stasis dermatitis.

Management

Appropriate antibiotic therapy, e.g., cephalexin 250 mg po qid or cloxacillin 250 mg—500 mg po qid x 14 d.

Intravenous antibiotics in severe disease, facial cellulitis, patients with underlying medical problems.

Chickenpox (Varicella)





Etiology: Varicella zoster virus (VZV) causes chickenpox; after primary infection, VZV remains dormant in sensory nerve roots for life, reactivation results in herpes zoster (shingles).

History: Often pruritic. Usually mild, self-limited illness in children; more severe in adults; rarely complicated by staph/strep bacterial superinfection, pneumonia, cerebellar ataxia, encephalitis.

Physical: Characteristic exanthem—Crops of erythematous macules progress to edematous papules and finally vesicles over 24–48 hr. Vesicles resemble “dew drops on rose petals.” Lesions are in different stages (polymorphous).

Incubation: 10–21 d. Infectious from 4 d prior to onset of lesions to crusting of final lesion (5 d after onset).

Investigations: Direct fluorescent antibody testing on fluid from vesicle base

DDx: Other viral exanthema, drug eruption, herpes simplex, insect bites.

Management

Prevention: Vaccine now available.

Tx

- Supportive/symptomatic treatment in children.
- VZIG if immunocompromised or exposed neonate within 96 hr.
- Oral antiviral therapy (acyclovir, valacyclovir, famcyclovir) in selected patient populations (≥ 13 yr, diabetes, CF, HIV, inborn errors of metabolism, severe fulminant skin disease, & visceral involvement).

Chondrodermatitis Nodularis Helicis



Etiology: Common, benign condition of the helix or antihelix of the ear in the elderly and due to persistent local pressure, e.g., from sleeping on one side.

History: Very painful; awakens from sleep.

Physical: Firm, tender nodule, occasionally with crust on helix or antihelix of ear.

Investigations: Biopsy if unsure of diagnosis.

DDx: AK, BCC, gouty tophus, KA, SCC.

Management

Rarely resolves on its own.

Tx

- Relieve or eliminate pressure at site of nodule: E.g., with a pressure-relieving ear cushion.
- Potent topical or intralesional steroids.
- Cryotherapy.
- Surgical options: Wedge excision of involved skin and cartilage, curettage and electrodesiccation.

Contact Dermatitis



Can be divided into two main subtypes: Irritant contact dermatitis (~80%) and allergic contact dermatitis (~20%).

Irritant Contact Dermatitis (ICD)

Etiology: Acute or chronic/cumulative direct nonimmunologic inflammatory reaction to toxic injury, e.g., by chemical/irritant agents. Occurs in all individuals exposed to chemicals, given sufficient exposure times and concentrations; no previous exposure necessary.

History: Regular exposure to common irritants: water, soap, detergents, solvents, alcohol. Pain, burning, and stinging exceed pruritus early on. Those with history of atopic dermatitis are more susceptible.

Physical: Ill-defined erythematous, scaling, papulovesicular dermatitis; chronic—scaling lichenified dermatitis.

Investigations: Patch testing to rule out ACD.

DDx: Dermatitis—allergic contact atopic, nummular, seborrheic, stasis; cellulitis; drug reaction.

Management

Prevention: Avoid irritant exposure by use of barriers (gloves, barrier creams) and/or by using less irritating materials as substitutes.

Treatment: Liberal use of nonsensitizing moisturizers (e.g., plain petroleum jelly) +/- topical steroids.

Allergic Contact Dermatitis (ACD)

Etiology: Type IV delayed-type hypersensitivity reaction of the skin. Develops following exposure to chemicals to which the individual has been previously sensitized; prevalence varies with allergen. *Common allergens:* poison ivy/oak/sumac, nickel, neomycin, bacitracin, rubber/latex, fragrances, formaldehyde.

History: Detailed history of exposure to allergens, workplace or hobbies; history of pre-existing skin disease (e.g., atopic or stasis dermatitis); medications.

Physical: Erythematous, scaling, papulovesicular dermatitis at sites of contact with the allergen; chronic—scaling lichenified dermatitis.

Investigations: Skin patch testing (on back) to establish diagnosis (different from prick testing).

DDx (ACD, ICD): Atopic or nummular dermatitis, drug reaction, scabies.

Management

■ Proper counseling on allergen avoidance, including cross-reactants.

■ Acute cases: Oral antihistamines, potent topical steroids or systemic prednisone in severe cases.



- Patients with multiple allergies or sensitivities should be advised to test out new skin care products by applying them to the inner aspect of their arm, twice a day without occlusion, for 1 wk before applying elsewhere on the body.

Misconceptions About Allergic Contact Dermatitis (ACD)

Misconceptions	In fact
Rash should quickly follow contact.	Rash is usually delayed 1–2 d and may not appear for a week after contact.
Allergy develops mainly to new substances.	Allergy can develop at any time, to any product, even after years of contact.
Allergy is dose dependent.	Allergies are not necessarily dose dependent.
If a consumer changes exposure to a product and the rash doesn't clear, the product is not the cause.	Many products contain the same or cross-reacting antigens, and product composition can change without notification.
Contact allergy occurs only at the site of exposure to the antigen.	Although the dermatitis is often most severe at the site of exposure, dermatitis can become generalized or can be spread to distant sites (e.g., nail polish transferred to eyelids).
Negative scratch/prick testing or negative radioallergen sorbent testing (RAST) as carried out by allergists rules out ACD.	Only patch testing is diagnostic.
Expensive products are less likely to be allergenic.	Cost of products does not determine its allergic properties.

Cutaneous Horn



History: A clinical diagnosis of an asymptomatic conical keratotic projection resembling a miniature horn.

Etiology: >50% of lesions have a benign base. Common underlying lesions include: AK, BCC, SCC, SK, and wart, although many others have been reported.

Physical: A hyperkeratotic papule with hornlike projection often on sun-exposed skin.

Investigations: Biopsy down to base of lesion.

DDx: AK, BCC, KA, SCC.

Management

Tx

Depends on underlying etiology, but typically cryotherapy or surgical excision.

Cysts



Epidermoid Cyst

Proliferation of epidermal cells within a circumscribed space of the dermis

History: Slow growing; usually asymptomatic; tender if ruptures.

Physical: Smooth & shiny mobile subcutaneous mass; cyst wall may be thin and can rupture if distended or traumatized; if the cyst contents leak into the adjacent dermis, inflammation leads to erythema and tenderness. Variants: epidermal/sebaceous (odor), pilar/trichilemmal (odorless; scalp), milium.

DDx: Lipoma; a pit or punctum can often be seen on the cutaneous surface overlying the cyst, differentiating it from lipoma.

Investigations: Rarely, ultrasound or fine-needle aspiration can be performed.

Management

Usually for cosmesis. Surgical excision or enucleation is definitive. Drain ruptured cysts. Occasionally intralesional cortisone is beneficial.

Dermatofibroma



Benign slow-growing asymptomatic skin tumor.

Etiology: Sometimes associated with a history of trauma, such as insect bite or ruptured cyst.

Physical: Small (4 mm–10 mm) firm, often raised, dermal red-brown nodule; usually on legs of adults. “Dimple” sign: dermatofibroma dimples into the surrounding skin upon lateral pinching.

Investigations: Biopsy if in doubt.

DDx: BCC, keloid, melanoma, nevus.

Management

- Reassurance; Tx for cosmesis.
- Liquid nitrogen cryotherapy or intralesional cortisone can be attempted.
- Surgical excision to subcutaneous fat.
- Should be removed if rapidly growing.

Dermatomyositis



Autoimmune inflammatory myopathy with systemic manifestations.

History: Fatigue, pruritus, proximal muscle weakness, dysphagia.

Physical: Skin findings often the initial manifestation—pink-violet eyelid rash (heliotrope) and Gottron's papules (violaceous) over dorsal knuckles are specific cutaneous findings; also, poikiloderma in a photosensitive distribution (shawl pattern), violaceous erythema and scaling on extensor surfaces (Gottron sign), periungual and cuticular dilated telangiectases; calcinosis cutis in children. Internal malignancies

associated with dermatomyositis (in adults); screening should be done (esp. ovarian ca; nasopharyngeal ca. in Asians).

Investigations: Muscle enzymes (CK, aldolase) +/- muscle biopsy, skin biopsy, malignancy work-up.

DDx: Lupus, other connective tissue disorders, lichen planus.

Management

- R/O underlying malignancy.
- Muscle disease: Oral prednisone (1 mg/kg/d) for 2–3 yr, steroid-sparing agents: Methotrexate, azathioprine, cyclosporine, mycophenolate mophetil, IVIG.
- Cutaneous disease: Sunscreens, topical steroids, topical pimecrolimus or tacrolimus, hydroxychloroquine (plaquenil; 200 mg–400 mg/d).
- Often co-managed by dermatologist and rheumatologist.

Eczema: Nummular



Eczema: Nummular (*Continued*)



Common, round, and itchy form of dermatitis.

Etiology: Often occurs on background of dry skin, in colder climates; worse in winter.

Physical: Erythematous, coin-shaped (nummular) plaques; mostly on extremities and in elderly.

Investigations: If diagnosis in doubt, scraping and KOH for tinea and skin biopsy to rule out other causes.

DDx: Atopic dermatitis, contact dermatitis, psoriasis, tinea corporis.

Management

- Humidify air.
- Avoid excessive bathing; take brief lukewarm showers; reduce use of soaps.
- Moisturize skin after bath or shower with thick cream or ointment.
- Potent topical steroids to nummular plaques bid x 2–3 wk.
- Antihistamines prn if diffuse and poor sleep (use sedating type at night, e.g., hydroxyzine).

Erysipelas



Etiology: Superficial bacterial skin infection usually caused by *Streptococcus pyogenes*; affects lymphatics.

History: May be accompanied by fever, chills, nausea, vomiting, headache, and arthralgias.

Physical: Warm, swollen, erythematous plaque with sharp border; face & legs.

Investigations: CBC&D, blood and wound cultures, ESR, urinalysis.

DDx: Cellulitis, contact dermatitis, herpes zoster.

Management

- Adults: Systemic antibiotics (e.g., penicillins; erythromycin if pen. allergic) that offer coverage for both staphylococcal and streptococcal organisms for 10 d; cold compresses. Elevate & rest affected limb.
- Children between the ages of 6 and 36 mo: Cover for *H. influenzae*.

Erythema Multiforme



A self-limited (1–4 wk) skin reaction pattern to a variety of stimuli.

Etiology: Herpes simplex virus (cold sore) and mycoplasma are common precipitants, but often idiopathic; controversial whether drugs are triggers.

History: May be accompanied by malaise and arthralgias.

Physical: Classic iris or target lesions (3 rings) in a symmetrical & acral distribution (best observed on palms/soles); centers may be vesicular or dusky; Koebner phenomenon (lesions at sites of injury).

Investigations: Punch biopsy; in severe cases: CBC&D, ESR, LFTs, BUN, Cr, urinalysis, electrolytes, and blood/urine/sputum cultures.

DDx: Drug eruption, lupus, urticaria.

Management

- Supportive: Antipyretics, antihistamines, analgesics, topical steroids prn.
- If recurrent with HSV, consider treating with chronic suppressive oral antivirals & sun block for photoinduced outbreaks.
- Less commonly: Dapsone, hydroxychloroquine, azathioprine.
- Role of prednisone is controversial; consider use if severe pruritus and discomfort.

Erythema Nodosum



Common form of septal panniculitis that is usually a reactive skin response to an associated trigger

Conditions potentially associated with erythema nodosum

- Infections: Bacterial (e.g., streptococcal pharyngitis), mycobacterial (tuberculosis), yersinia, etc.
- Drugs: E.g., oral contraceptives, sulfonamides
- Sarcoidosis
- Inflammatory bowel disease

- Behçet disease
- Malignancies, esp. hematological
- Pregnancy
- Idiopathic

History: Lesions may be associated with fever, malaise, leg edema, and arthralgia. Recurrence not uncommon. Resolution after 2–6 wk.

Physical: Painful, dull, erythematous nodules 1 cm–5 cm in diameter, located on anterior lower legs of young women. No ulceration, discharge or scarring.

Investigations: Thorough review of systems; CBC, urinalysis, chest X-ray, ASOT titer and throat swab (w/ streptococcal pharyngitis).

DDx: Insect bite reaction, erysipelas, erythema induratum, urticaria.

Management

Investigate for and treat underlying cause.

Most patients benefit from bed rest, gentle support hose, applying ice or cool compresses, elevating legs, & NSAIDs.

Less common Tx choices: Potassium iodide 300 mg–900 mg/d x 2–4 wk, oral prednisone, colchicine, dapsone.

Erythroderma



Cutaneous reaction pattern associated with a diverse range of dermatoses.

History: Chills, malaise, and fever may develop due to excessive vasodilation. Pruritus common.

Physical: Generalized erythematous skin eruption involving >90% of the body. May be life threatening due to generalized failure of skin functions, e.g., fluid loss through skin may result in hypotension +/- electrolyte imbalance. Skin infections common.

Investigations: Ascertain history of potential underlying dermatosis, skin biopsies.

DDx: Common cutaneous diseases that may present with or develop into erythroderma

- Idiopathic
- Dermatitis (atopic, contact, or seborrheic)
- Psoriasis (#1 identifiable cause)
- Drug reaction (allopurinol, gold, carbamazepine, phenytoin)
- Cutaneous T-cell lymphoma (Mycosis fungoides & Sézary syndrome)
- Pityriasis Rubra Pilaris (PRP)
- Leukemia
- Paraneoplastic

Management

- **Treatment:** Depends on etiology—stop offending drug; cyclosporine and acitretin in psoriatic erythroderma; acitretin in PRP. Occasionally prednisone, methotrexate and azathioprine used.
- **Supportive management:** Hospital admission, proper hydration, nutrition, electrolyte monitoring, cardiac monitoring, temperature support.
- **Skin care:** Emollients, soaks, & compresses, mild-mod topical steroids.
- Antihistamines for pruritus, sleep.
- Antibiotics if any signs of infection.

Folliculitis



Etiology: Inflammation of hair follicles due to infection, physical or chemical irritation. Often idiopathic. Most commonly bacterial infection, often due to *S. aureus*; *Pseudomonas* ("hot tub folliculitis"); *Pityrosporum folliculitis*; viruses and fungi rarely.

History: Frequently initiated by mild physical injury to follicles, such as friction of tight-fitting garments, or by ingrown hairs in the male beard area. More common in obese & diabetics. Ask about hot tub exposure.

Physical: Superficial pustules and/or papules in the distribution of hair follicles; affects—face, scalp, chest, back, thighs, and buttocks.

Investigations: Swab for culture and sensitivity of opened pustules; KOH prep; biopsy.

DDx: Acne, insect bites, miliaria, scabies.

Management

- Eliminating the offending agent(s): Antibiotics if bacterial, antifungals if *Pityrosporum*, antivirals if herpes, & symptomatic management if *Pseudomonas*.

- If fails therapeutic antibacterial trial, consider more extensive work-up (cultures, KOH, biopsy).
- Shower with antibacterial soaps/washes.
- Topical antibacterials: Fucidin, mupirocin, erythromycin.
- Systemic antibacterials: E.g., cloxacillin x 7–10 d or antifungal preparations (depending on culture). Occasionally long-term tetracycline or its derivatives required.

Granuloma Annulare (GA)



Common benign inflammatory granulomatous dermatosis of unknown etiology.

History: Usually asymptomatic; most pts. <30 yr

Physical: Papules and annular plaques with central depression; commonly on dorsae of hands, feet, elbows (acral).

Variants: Arcuate dermal erythema, localized, generalized, micropapular, patch, perforating, & subcutaneous.

Generalized (15%; older pts.; poorer response to Tx) and perforating GA (5%; small umbilicated papules) may be associated with diabetes mellitus (controversial).

Course: Usually self-limiting; resolves within 2 yr in 75% of patients; high recurrence rate.

Investigations: Biopsy to confirm diagnosis.

DDx: Erythema elevatum diutinum, lichen planus, sarcoidosis, tinea corporis.

Management

- Reassurance & clinical observation since benign & self-limited.
- Mainly for cosmetic purposes.
- Localized GA: intralesional or potent topical steroids, cryotherapy.
- Generalized GA: PUVA, prednisone, pentoxifylline, oral retinoids.

Hand, Foot, and Mouth Disease



Courtesy of Dr. Henry Foong

Self-limited (7–10 d) systemic infection usually caused by coxsackievirus A16 affecting young children. Highly contagious, and spreads by direct contact.

History: Often accompanied by low-grade fever, malaise, and sore mouth; refusal to eat.

Physical: Multiple small vesicles with red halo on fingers and toes, ulcerative oral lesions (90%).

Investigations: Clinical diagnosis; can culture virus from mucosal or cutaneous lesions.

DDx: Aphthous stomatitis, chickenpox, erythema multiforme, herpes simplex.

Management

- Symptomatic: Topical anesthetic gel (lidocaine).
- Antipyretics: E.g., acetaminophen, ibuprofen.
- Ensure adequate fluid intake.

Hemangiomas (Infantile; “Strawberry Hemangioma”)



Benign vascular tumor of endothelial cells.

History: 40% present at birth; remainder develop within 8 wk; 60% on head & neck. Rapid growth phase at 4–8 wk until 6–9 mo; spontaneous regression begins at 18–24 mo; 50% involute by 5 yr, 90% by 9 yr.

Physical: Divided into 3 types—Superficial (red and papular), deep (blue-gray and nodular), and mixed; *residua*: hypopigmentation, telangiectases, scarring (if ulcerated), redundant tissue.

Complications: Obstruction depending on location (e.g., vision, breathing), ulceration, infection.

Investigations: Consider MRI or U/S if in beard, oral or periocular areas involved.

DDx: Congenital hemangioma, vascular or lymphatic malformation.

Management

- Indications for treatment: Kasabach-Merritt syndrome; high output failure, obstruction of vital function (feeding, respiration, passage of urine/stool); nasal, auditory, or ocular obstruction; ulceration; cosmetic disability.
- Observation and reassurance in most cases.

- Referral to subspecialist (e.g., ophthalmology, ENT) if local obstruction.
- Prednisone 2 mg—4 mg/kg/d until 1 yr; occasionally intralesional steroids.
- Laser (pulsed dye), especially if ulcerated.
- Less commonly, excision; rarely interferon alpha.
- Newer treatment options may be considered: potent topical steroids, imiquimoid 5% cream, becaplermin gel 0.01% (for ulcerated hemangiomas).
- When pt. older, may need plastic surgery or laser for redundant tissue and persistent telangiectases, respectively.

Henoch-Schönlein Purpura (HSP)



Courtesy of Dr. Henry Foong

Common small vessel vasculitis of childhood with annual incidence of 20 per 100,000, highest in boys between 4 and 6 yr. Frequently associated with a URTI; peak occurrence in winter & fall.

History: 2—3-wk history of fever, headache, myalgias, arthralgias, and abdominal pain may precede rash.

Physical: Purpuric papules on buttocks, legs, & extensor extremities. Usually benign and self-resolving condition, lasts 6—16 wk.

- Classic tetrad of symptoms: Cutaneous purpura (100%), arthralgias/arthritides (~80%), abdominal pain (~70%), and gastrointestinal bleeding (33%).
- Renal involvement observed in 25%–50% of patients, heralded by asymptomatic microscopic hematuria and proteinuria, but is usually mild and self-limiting; with increasing age at presentation, nephrotic syndrome, hypertension, and acute renal failure may occur.
- Complete recovery in 94% of children and 89% of adults.

Investigations: Skin biopsy; early DIF helpful—reveals IgA deposits in walls of small blood vessels; U/S & x-ray for GI symptoms; occasionally renal biopsy needed; blood work: CBC&D, BUN, Cr, urinalysis, complement, ASOT.

DDx: Erythema nodosum, LCV, lupus

Management

- Mostly supportive, adequate hydration, NSAIDs to treat arthralgias.
- Close follow-up of renal status w/ repeat urinalyses and renal function tests, referral to nephrologist if renal complications occur.
- Corticosteroids (most evidence for abdominal and joint symptoms), dapsone, cytotoxic agents and IVIG might be of benefit.

Herpes Simplex (HSV)



Herpes Simplex (HSV) (Continued)



HSV-1

Common viral infection affecting the oral mucocutaneous surface. HSV-1 is most commonly associated with oral-facial lesions.

History & Physical: Most patients have subclinical primary episode, others experience primary gingivostomatitis, i.e., 3–5 d of fever, malaise, lymphadenopathy, oral mucosal erosions

- Recurrent HSV-1 infection (herpes labialis) after primary infection may be reactivated by a number of triggers: stress, fever, UV light, trauma, menstruation; burning and itching precede appearance of grouped umbilicated vesicles on erythematous base, herpetiform arrangement, commonly on the vermilion border of the lip. face; crust over in 7–10 d.

DDx: aphthous stomatitis, hand-foot-and-mouth disease, impetigo

HSV-2

Common viral infection affecting the genital mucosa, although oral mucocutaneous involvement reported with increasing prevalence.

History & Physical: Most are sexually transmitted; primary episodes more severe, with painful erosive balanitis, vulvitis, or vaginitis; recurrent infections are milder or subclinical.

DDx: Chancroid, syphilis, fixed drug eruption.

Investigations: Direct fluorescent antibody testing of fluid from base of vesicle; viral culture or PCR from lesion swab; Tzanck smear shows multinucleate giant epithelial cells.

Herpetic whitlow = herpes of fingertip; *Herpes gladiatorum* = herpes among wrestlers & rugby players; *Eczema herpeticum* = eczema secondarily infected with herpes.

Management

- Immunocompetent patients, 1st or occasional episode: Tx often supportive; can treat if severe or disseminated.
- Within 72 hr, oral antiviral therapy may be instituted to reduce pain, viral shedding, and time to healing: Acyclovir, famciclovir, valacyclovir can be used with dosage depending on whether 1^o vs 2^o episode.
- If severe, frequent (>6/yr) recurrences, consider chronic suppressive antiviral therapy.
- Consider antiviral prophylaxis prior to skiing or tropical vacations (& sunscreen), extensive dental work, or cosmetic procedures on the face.
- Topical antivirals of no benefit.

Herpes Zoster ("Shingles")





Etiology: Varicella zoster virus (VZV) causes chickenpox; after primary infection, VZV remains dormant in sensory nerve roots for life, and reactivation results in herpes zoster (shingles). Reactivation may be idiopathic, but may occur with immunosuppression or stress. Increased incidence with increasing age; more common in HIV & hematologic malignancy.

History: Pain/tenderness in a unilateral dermatomal distribution often precedes rash.

Physical: W/i 3–4 d of onset of symptoms, clusters of erythematous papules and vesicles develop in a dermatomal distribution (thoracic > cranial > lumbar); new groups of lesions continue to appear over several days, eventually followed by crusting and desquamation over a 2–4-wk period.

Generalized zoster. Lesions involve several nonadjacent dermatomes and cross the midline. **Complications:** Post-herpetic neuralgia (PHN), scarring, secondary bacterial infection. PHN occurs in 50% of pts > 60 yr, & is present at 1 yr in 10%–25%; characterized by shooting or burning pain in the previously involved sites; more common in older patients and when the trigeminal nerve is involved.

Investigations: Diagnosis is mostly clinical; if unsure, same work-up as for chickenpox and biopsy can be helpful.

DDx: VZV, herpes simplex, folliculitis.

Management

- If identified within 72 hr, oral antiviral therapy can speed healing and decrease pain (PHN): Acyclovir 800 mg 5 x d x 7 d, famciclovir 500 mg tid x 7 d, or valacyclovir 1000 mg tid x 7 d.
- If tip of nose involved/V1 dermatome facial involvement: Urgent ophthalmology consult—Ocular involvement can lead to blindness.
- PHN: Difficult to manage, best prevented by immediate antiviral therapy; analgesics, topical lidocaine, capsaicin, narcotics, nerve blocks, gabapentin, tricyclic antidepressants may be helpful; pain clinic referral. Prednisone occasionally used in elderly in addition to antivirals for herpes zoster (controversial).

Hidradenitis Suppurativa



Etiology: Chronic recurrent inflammatory condition wherein hair follicles are occluded and become secondarily infected. Associated with obesity, diabetes, and smoking; genetic and hormonal components.

History: Pain, odor, and drainage affecting axillae and/or groin.

Physical: Comedones, pustules, and nodules, abscesses, sinuses, and scarring. Severe form associated with acne conglobata, pilonidal sinus, & dissecting cellulitis of scalp.

Investigations: Clinical diagnosis; can swab for bacterial cultures.

DDx: Folliculitis, infected cysts, inflammatory bowel disease.

Management

- Distressing condition with no satisfactory treatment.
- Topical and systemic antibiotics (e.g., clindamycin and tetracycline), intralesional corticosteroids, hormonal therapy, retinoids (e.g., isotretinoin, acitretin).
- Daily cleansing with antibacterial soaps.
- Female patients benefit from antiandrogen therapy such as cyproterone acetate or spironolactone.
- Surgery is required for more recalcitrant cases and can be curative.
- Encourage patients to quit smoking and lose weight.

Hyperhidrosis



Etiology: Excessive sweating, either generalized or focal (e.g., palmar, palmoplantar, axillae), and affecting 2%–3% of general population; most common in adolescence and young adults. Generalized hyperhidrosis can be associated with underlying systemic disorder, e.g., infectious (e.g., TB), endocrine, or neurologic; focal hyperhidrosis often idiopathic.

History & Physical:

Diagnostic criteria for primary focal idiopathic hyperhidrosis.

- Focal, visible, excessive sweating of at least 6 mo duration without apparent cause with at least 2 of the following characteristics:

- Bilateral and relatively symmetrical sweating.
- Frequency of at least 1 episode per wk.
- Impairment of daily activities.
- Age at onset < 25 yr.
- Positive family history.
- Cessation of sweating during sleep.

Investigations: Starch iodine test can be used to outline the area of excessive sweating.

DDx: Thyrotoxicosis, medication-induced hyperhidrosis, pheochromocytoma.

Management

Important to rule out systemic causes—infections, malignancy (ask about night sweats).

Topical: Aluminum chloride hexahydrate solution in ethanol (e.g., Drysol®), glycopyrrolate iontophoresis.

Systemic: Anticholinergics (e.g., Robinul® 1 mg bid–tid), diltiazem, clonidine, Botulinum toxin (Botox®) injections—very effective; can last 6–12 mo.

Surgery: Endoscopic thoracic sympathectomy, subcutaneous liposuction.

Impetigo



Etiology: Superficial infection of the skin caused by *Staphylococcus aureus* or group A beta-hemolytic *Streptococcus* (GABHS). Can be primary or secondary, e.g., impetiginization of an underlying dermatosis, as in atopic dermatitis, insect bites, scabies, and viral infections.

History: Spread by direct contact and most common in children. Highly contagious. Usually asymptomatic.

Physical: Superficial pustules covered by moist, honey-colored crusts; lesions may be localized or extensive; face and extremities commonly affected. Pt. nontoxic.

■ May see vesicles and bullae with yellow-brown crust (bullous impetigo), typically caused by phage group II of *S. aureus*.

Course: Generally self-limited; risk of post-strep glomerulonephritis, but not rheumatic fever.

Investigations: Culture and sensitivity.

DDx: Herpes simplex, eczema, contact dermatitis, scabies.

Management

■ **Localized:** Topical antibiotics (mupirocin or fusidic acid tid).

■ **Generalized:** Oral cephalexin, cloxacillin, or erythromycin x 7–10 d.

■ Saline compresses prn to remove crust.

■ Consider intranasal mupirocin (bid x 5–7 d) if recurrent.

Keratoacanthoma (KA)



Skin tumor, possibly an early or abortive phase of SCC; rare association with Muir-Torre syndrome.

History: Rapid growth (over 2–6 wk) and may resolve spontaneously.

Physical: Firm, dome-shaped, crater-like nodule most common on sun-exposed skin of older adults.

DDx: AK, cutaneous horn, SCC.

Investigations: Excisional or deep incisional biopsy.

Management

- Excision, curettage, and electrodesiccation. Uncommonly: Aggressive cryotherapy, radiotherapy, intralesional therapy (e.g., 5-FU, bleomycin).

Keratosis Pilaris (KP)



Common, benign disorder of keratotic follicular papules.

Etiology: Genetic component more common in atopic and xerotic patients, and worse in winter.

History: Usually asymptomatic, with occasional pruritus, improves with age.

Physical: Hyperkeratotic follicular papules (+/- erythema) typically distributed over the outer aspects of upper arms and thighs, less commonly the face, forearms, & buttocks.

Investigations: Clinical diagnosis.

DDx: Atopic dermatitis, folliculitis, lichen spinulosus.

Management

Treatment often disappointing in all but mild cases.

Tx choices: Lactic acid (e.g., Lac-Hydrin lotion®), urea-based emollients, salicylic acid in urea, tretinoin. Mild cortisone can be added if erythema present.

Lichen Planus (LP)



Lichen Planus (LP) (Continued)



Acute or chronic inflammatory disorder affecting the skin, mucous membranes, and nails (5%–10%).

Etiology: Likely an immunologically mediated reaction; oral erosive lichen planus associated with hepatitis C.

History: Pruritus common; oral lesions may or may not be symptomatic. Ask about Hep C risk factors (e.g., transfusions, IV drug use) for oral-erosive LP.

Physical: 7 P's—Planar (flat-topped), pruritic (intense), purple, polygonal, papules, penile (commonly affected), prolonged course (up to 18 mo; longer in some cases); distributed on flexor surfaces, penis (glans), mouth (lacy white patches or erosions—Wickham striae). Many clinical variants reported.

Koebner phenomenon = appearance of lesions at sites of trauma.

Course: May resolve spontaneously or have chronic course (esp. oral LP); increased risk of oral SCC in oral LP.

DDx: Drug reaction, pityriasis rosea, psoriasis

Investigations: Skin biopsy if unsure of diagnosis.

Investigations: Biopsy if SCC suspected.

DDx: Lichen planus, morphea, vitiligo.

Management

Extragenital LS&A: Often no treatment; topical steroids or phototherapy can be tried.

Genital LS&A: Ultrapotent topical corticosteroids, e.g., clobetasol, eventually tapered; can try topical tacrolimus or pimecrolimus as maintenance.

Lipoma



Slow-growing, common, benign tumor of fatty tissue in 1% of population. Several uncommon associated syndromes include lipomas.

History: Usually asymptomatic; slow growth.

Physical: Mobile and compressible soft subcutaneous mass; commonly on trunk. Sacrococcygeal lipoma may be a marker for spinal dysraphism.

Investigations: Clinical diagnosis; uncommonly, imaging studies or biopsy.

DDx: Cyst, DF, leiomyoma

Management

Excision or liposuction for cosmesis; rarely painful.

Lupus Erythematosus



Etiology: Heterogeneous autoimmune disease resulting from the interplay of genetic, environmental, and hormonal factors.
F > M.

History & Physical: Spectrum of disease varies from limited cutaneous involvement to severe systemic disease.

**Systemic Lupus Erythematosus (SLE): 1997 Update
of the 1982 ACR Revised Criteria**

Management

LE-specific skin manifestations are divided into 3 categories (other less common forms exist as well).

	Clinical	Comment
Acute cutaneous LE	Classic "butterfly" malar rash. Often association with anti-dsDNA Ab and lupus nephritis.	Evaluate for evidence of systemic disease. Hydroxychloroquine. Systemic steroids (0.5 mg–1 mg/kg/d) + steroid-sparing agents (azathioprine, methotrexate, mycophenolate mophetil).
Subacute cutaneous LE	Two subtypes: Annular or papulosquamous presentation. Often associated with anti-Ro Ab.	Sun protection. Corticosteroids (topical, intralesional) and hydroxychloroquine.
Chronic cutaneous/discoid LE	Most often in head/neck area: Atrophic inflammatory plaques. Can lead to scarring alopecia.	5%–10% will get systemic disease Tx: Photoprotection, topical or intralesional steroids, hydroxychloroquine

Other important variants of lupus: Drug-induced lupus, lupus profundus, neonatal lupus.

Investigations: Diagnosis requires clinicopathologic correlation; often multiple skin biopsies needed before diagnosis made.

DDx: Eczema, neurodermatitis, parapsoriasis.

Mycosis fungoides (MF)

The most common clinicopathologic subtype of primary cutaneous T-cell lymphoma (CTCL).

Management

Choice of therapy and management setting depends on stage of disease: "stage-directed therapy"

Overview of Mycosis Fungoides Therapies

Skin-directed Therapies

Topical therapies
 Steroids (mid to strong potency)
 Topical chemotherapy
 ■ Nitrogen mustard
 ■ Carmustine (BCNU)
 Bexarotene 1% gel
 Imiquimod 5% cream
 Phototherapy: PUVA, BB/NB-UVB, UVA-1
 Radiation therapy
 Local x-ray therapy
 Electron Beam Therapy

Systemic Therapies

Biological/immune therapies
 Bexarotene, Acitretin Denileukin
 Diftitox
 Interferon
 Extracorporeal photopheresis (ECP)
 Monoclonal antibodies
 Cytokine therapy
 Chemotherapy
 Methotrexate, gemcitabine
 Pentostatin and purine analogues
 Combination chemotherapy
 Bone marrow/stem cell transplantation

Melanoma



Etiology: Melanocyte-derived skin cancer. May arise within a previously existing nevus or dysplastic nevus, but ~70% arise *de novo*.

History:

Risk factors

- Fair complexion: Red/blonde hair, blue/green eyes, tendency to freckle and burn.
- Sun exposure, particularly blistering sunburns during childhood.
- Personal or family history of melanoma; Genes involved in some cases: CDKN2A, BRAF.
- Giant congenital melanocytic nevi or multiple dysplastic nevi.

Physical: Pigmented macule or plaque with some or all of the following features (ABCDE of melanoma): Asymmetry, Borders (irregular), Color variegation, Diameter (>6 mm), Evolution (lesion change by history).

Classically divided into subtypes based on clinical and histopathologic features:

1. Superficial spreading malignant melanoma—60%—70% of melanomas.
2. Nodular melanoma.
3. Acral-lentiginous melanoma—Most common form in blacks, Asians, and Hispanics, mostly on volar skin of the palms or soles and the nailbeds.
4. Lentigo maligna melanoma—Develops from a lentigo maligna, usually on the face of elderly, slow-growing.
5. Amelanotic melanoma—Pink-red.
6. Rare variants.

Most common sites of local and/or regional metastases—Draining lymph node basins and the skin between the primary site and the lymph nodes; most common sites of systemic metastases—Lung, liver, brain (#1 cause of death), bone, and gastrointestinal tract.

Investigations: Dermoscopy (ABCD rule or 7-point checklist; requires expertise); excisional biopsy if melanoma is suspected.

■ Most important prognostic indicator is maximal thickness of tumor invasion on biopsy (Breslow depth in mm).

DDx: BCC, blue or dysplastic nevus, SK.

AJCC 2002 Revised Melanoma Staging

Reprinted with permission: Balch CM, et al. Final version of the American Joint Committee on Cancer staging system for cutaneous melanoma. J Clin Oncol. 2001 Aug 15;19(16):3635-48.



Management

Wide surgical excision, based on Breslow depth: 5 mm for melanoma in-situ; 1 cm for melanoma <1 mm; 2 cm for melanoma >1 mm.

Sentinel lymph node biopsy depending on current guidelines. Adjuvant high-dose interferon & IL-2 may provide benefit for metastatic disease.

Perfusion chemotherapy for extremity melanoma.

Radiation—symptomatic.

Melanoma vaccines being studied; patients with metastases should be referred for clinical trials.

Melasma



Etiology: Acquired brown macular hyperpigmentation, mostly on face, from exposure to sunlight.

Can be associated with pregnancy (“mask of pregnancy”), oral contraceptives, menopause.

History: Mostly females, asymptomatic, often starts in pregnancy, worsens in summer.

Physical: Well-demarcated, hyperpigmented macules, mostly on the malar prominences, forehead and mandible.

Investigations: Wood’s lamp exam accentuates the epidermal pigment.

DDx: Addison disease, ephelides/freckles, postinflammatory hyperpigmentation.

Management

- Daily sun protection is essential with high SPF and broad-brimmed hat; sun blocks preferred.
- Hydroquinone (2% OTC; 4% by Rx) are most commonly used—Azelaic acid; tretinoin.
- Kligman or modified Kligman formula combines a hydroquinone with a mild cortisone and tretinoin.
- Chemical peels.
- Lasers occasionally used.

Molluscum Contagiosum





Etiology: Self-limited viral infection of skin caused by poxvirus (MCV) affecting mainly children.

Immunocompromised patients (e.g., HIV) may develop more widespread and larger lesions.

History: Asymptomatic; occasional perilesional pruritus.

Physical: 2- to 6-mm flesh colored, dome-shaped, umbilicated pearly papules; most common affected sites—trunk and face of children, genitals/inner thighs of sexually active adults.

Course: Lesions typically involute spontaneously within 9-12 mo; may develop adjacent eczema (in 10%).

Investigations: Clinical diagnosis, biopsy if uncertain.

DDx: BCC, milia, other infection, warts.

Management

- Observation.
- Liquid nitrogen cryotherapy.
- Curettage; may be uncomfortable.
- Papule incision with a scalpel blade or at home sharp fingernail and expression of contents.
- Topical cantharidin: Blistering (intentional) will occur; apply with wooden end of cotton swab and cover with tape for 30 min, then wash off with soap and water; ideal in children.
- Imiquimod cream.
- Tretinoin cream.
- In children with widespread involvement, may consider oral cimetidine (40 mg/kg/d x 2 mo).
- Testing for other STDs recommended in adults with lesions in the genital area.

Nevi



Melanocytic Nevi (Moles)

- Benign growths composed of nests of melanocytes.
- More common in light/fair skin.
- First appear in childhood as flat brown macules, evolve during adulthood into dome-shaped and fleshy papules or nodules with loss of pigmentation.
- Divided into various subtypes: Acquired melanocytic nevi, Spitz nevi, blue nevi.
- Many nevi regress in late adulthood.
- Need monitoring, biopsy if melanoma suspected.



Congenital Nevi	<ul style="list-style-type: none"> ■ Present at birth or become apparent during infancy. ■ Often larger than typical melanocytic nevi and have associated hair. ■ Need monitoring, biopsy if changing or if melanoma suspected. ■ Small = <1.5 cm; medium = 1.5 cm—19.9 cm; large = >20 cm. ■ Giant congenital nevi associated with increased melanoma risk.
Dysplastic Nevi (Atypical Nevi, Clark's Nevi)	<ul style="list-style-type: none"> ■ Nevi with irregular outline, variable pigmentation, indistinct borders, and often >6 mm. ■ Multiple dysplastic nevi herald an increased risk for developing melanoma. ■ Need careful monitoring, biopsy if change in size/color.
Ephelides (Freckles)	<ul style="list-style-type: none"> ■ Small brown macules on sun-exposed skin; more common in fair-complexioned. ■ Darken in response to the sun and fade with UV abstinence. ■ No risk of melanoma if diagnosis certain.
Lentigines (Solar Lentigines, Simple Lentigines, "Liver Spots")	<ul style="list-style-type: none"> ■ Occur in response to sunlight and persist even in absence of sunlight. ■ Vary in color from tan to dark brown, and can be up to 1 cm in diameter. ■ Location: Dorsal hands and face.
Spitz Nevus	<ul style="list-style-type: none"> ■ Smooth-surfaced, firm, round, brown to pink-red papule usually developing in childhood. Often on face. ■ Benign, but histologically can be confused with melanoma.
DDx: Melanoma, pigmented BCC, SK.	

Management

Surgical excision if malignancy suspected.

Onychomycosis (Tinea Unguium)



Etiology: Dermatophyte infection of nail plate, often associated with tinea pedis. Less commonly due to yeasts and nondermatophyte molds.

History: Change in nail color and more brittle; usually asymptomatic.

Physical: Yellow discoloration, thickening, nail dystrophy, subungual hyperkeratosis, onycholysis. Toenails > fingernails.

Patterns include the distal subungual form (most common), the proximal white subungual form (may be a sign of HIV disease), and the white superficial form.

Investigations: 20% KOH direct microscopy, nail clipping/scraping for culture.

DDx: Nail psoriasis or trauma, eczema, lichen planus.

Management

- Topical antifungals much less effective. Ciclopirox (Penlac®) nail lacquer may be tried if po Tx not an option.
- Important to culture fungus prior to initiating oral Tx



- Terbinafine (Lamisil) 250 mg po qd x 6 wk for fingernails, x 12 wk for toenails; itraconazole (Sporanox) 200 mg po bid x 7 d, then 3 wk off—2 pulses for fingernails, 3 pulses for toenails.
- Other less common options: Griseofulvin (esp. in kids), fluconazole.
- Hepatotoxicity and leukopenia are rare side effects of terbinafine use, some check baseline and monitor CBC and LFTs during Tx; itraconazole—mostly hepatotoxic S/E.
- Educate patients that due to slow rate of new nail formation, it may take many months after treatment to see clinical improvement.

Paronychia



Etiology: Inflammation around nail due to *Staphylococcus aureus*, *Streptococcus*, or *Candida albicans* (chronic).

History: Often painful and tender skin around nails.

Physical: Periungual swelling and erythema; tenderness to palpation.

Investigations: Usually clinical; Gram staining, KOH prep, or Tzanck smear to confirm infectious etiology.

DDx: Contact dermatitis, cellulitis.

Management

- Avoid exposure to excessive moisture.
- Rule out offending drugs (e.g., retinoids, protease inhibitors).
- Potent topical steroids; less effective are topical or systemic antifungals in chronic paronychia.
- Topical or systemic antimicrobials.
- Incision and drainage if acute and tense subcutaneous collection.

Pediculosis (Lice)



Courtesy of Dr. Art Huntley

Etiology: Infestation by wingless, 6-legged insects (*Pediculus humanus* & *Phthirus pubis*). Spread by direct contact or through fomites (e.g., clothing, bedding).

History: Symptoms vary from none to extreme pruritus.

Physical:

Pediculosis capitis (head louse)

Gray-white nits/eggs or lice firmly adhere to hair shaft; postauricular and occipital regions commonly affected; children > adults.

Pediculosis pubis (crab louse)

Eyelashes and pubic hair may be affected; usually transmitted by sexual contact; nits seen on hair, red excoriated skin.

Body lice

Infest individuals with poor hygiene or those living in crowded conditions; eggs commonly found along seams of clothing.

Investigations: Magnifying glass—assisted observation of nits or mature lice.

DDx: Impetigo, insect bites, scabies.



Management

- Head lice: Wash hair, rinse, and towel dry; then apply permethrin cream (Nix[®], Elimite[®]) rinse for 10 min and rinse out; repeat in 7 d.
- Pyrethrin, malathion, crotamiton, petrolatum may be used; latter rubbed into hair to suffocate lice.
- For pediculosis pubis—affecting eyelashes, apply petrolatum to eyelashes bid—tid x 10 d.
- Nits may be removed with vinegar soaks (vinegar:water 1:1) and a fine-toothed comb.
- Soak combs and brushes in permethrin shampoo for 10 min or boil.
- Bedding, clothing, and head gear should be washed and heat-dried; environment should be vacuumed; unwashable items can be sealed in plastic bags for 2 wk.
- Contacts should be treated similarly.
- Ivermectin (oral) increasingly used in resistant or complicated cases of lice & scabies.

Pemphigus Vulgaris





Etiology: IgG produced against epidermal desmoglein 1 and 3, leading to acantholysis and intraepidermal bullae.

History: Serious and uncommon autoimmune blistering disease with peak prevalence at 50–60 yr of age.

Painful skin and/or oral mucosae; pruritus uncommon.

Physical: Flaccid blisters or bullae, or residual erosions; mouth often involved—Mouth ulcers & odor often initial presentation (60%); healed lesions don't scar, but leave hyperpigmented patches.

Variants: Pemphigus foliaceus, pemphigus vegetans, fogo selvagem (S. America), pemphigus erythematosus, paraneoplastic pemphigus.

Nikolsky sign: Rubbing the skin in unaffected site induces lesion.

Asboe-Hansen sign: Blister extends with lateral finger pressure.

Investigations: Lesional biopsy for histology and perilesional for immunofluorescence.

DDx: Bullous pemphigoid, linear IgA bullous disease, pemphigus foliaceus.

Management

- Requires management by an experienced physician.
- May need hospital admission (often require burn unit care).
- Prednisone high-dose initially (>1 mg/kg), then taper; add bisphosphonates, calcium, Vitamin D to protect bones.
- Steroid-sparing agents started early & help control disease while tapering prednisone: azathioprine, mycophenolate mofetil, methotrexate, cyclophosphamide, IVIG.
- Antibiotics if secondarily infected.
- Symptomatic oral treatment (viscous lidocaine, diphenhydramine elixir).



Perioral (Periorificial) Dermatitis



Etiology: Inflammatory acneiform skin condition exacerbated by potent or chronic steroid application; may be related to irritation or allergy from ingredients (e.g., detergents) in creams.

History:

Affects mainly females, 15–40 yr who complain of a burning sensation and tightness.

Physical: Erythematous macules, papules, and plaques coalescent on perioral, perinasal, and/or periorbital skin.

Investigations: None.

DDx: Acne, contact dermatitis, rosacea.

Management

Avoid steroids on face (discontinue, or wean).

Topical: Metronidazole gel or cream bid; topical tacrolimus or pimecrolimus can be tried to wean off steroid.

Systemic: Tetracycline 500 mg po bid helpful for papulopustular lesions.

Pityriasis Rosea



Acute, self-limited papulosquamous inflammatory skin exanthem in 15–40 yr.

Etiology: Possibly triggered by human herpes virus 7.

History: May be pruritic or asymptomatic; preceding URTI or mild constitutional symptoms; larger “herald” patch with central clearing, followed 5–10 d later by trunk lesions.

Physical: Scaly pink-erythematous plaques; predominantly trunk affected, in “Christmas-tree” pattern.

Course: Self-resolving in 6–8 wk.

Investigations: Clinical; scraping for KOH (R/O tinea) or order RPR (R/O syphilis), or skin biopsy if uncertain.

DDx: Eczema, psoriasis, tinea corporis, 2^o syphilis, seborrheic dermatitis.

Management

- Reassurance is key.
- Symptomatic management of pruritus (e.g., mild topical steroids, antihistamines prn).
- Light therapy (e.g., UVB) may help hasten resolution and relieve pruritus.



Pityriasis Versicolor (Tinea Versicolor)



Etiology: Common superficial cutaneous fungal infection caused by *Malassezia furfur* or *globosa* yeast.

History: Usually asymptomatic, young adults typically affected; commonly recurs; more common in hot and humid environments.

Physical: Round to oval macules and patches on the trunk; different colors: White, orange-brown, dark-brown; very fine scale; postinflammatory hypopigmentation is often noted rather than initial erythema.

Investigations: KOH test reveals diagnostic "spaghetti & meatballs" (hyphae & spores); Wood's lamp shows yellow-brown fluorescence. Skin biopsy if diagnosis uncertain.

DDx: Tinea corporis, vitiligo, psoriasis.

Management

- Topical or systemic treatment.
- Nizoral shampoo (ketoconazole 2%) applied onto dampened trunk skin, lathered for 5 min before rinsing, x 3 d.

- Topical selenium sulfide 2.5% shampoo applied for 15 min x 3 d.
- Topical nizoral cream 2% (ketoconazole) bid x 2 wk.
- Systemic ketoconazole 200 mg po qd x 5–7 d or fluconazole 400 mg x 1. Postinflammatory hypopigmentation may persist for 6–12 mo.
- Even with treatment, tendency to recur in summer.
- Zinc pyrithione soap lather may be applied to the body 1–3 times weekly for prevention.

Psoriasis





Common (1%–2%), chronic, recurrent inflammatory skin disease with unpredictable course.

Etiology: Genetic & environmental factors; abnormal epidermal differentiation and hyperproliferation initiated and maintained primarily by T-cells.

History: Visible itchy red plaques with increased skin scaling and peeling. May have joint pain. May have preceding URTI. Worsened by smoking, alcohol, & stress.

Physical: Characteristic erythematous plaques with silvery-white scale; distributed over extensor surfaces (elbows, knees), scalp, sacrum, and other sites,

Nail psoriasis: Pitting, thickening, “oil-drop,” lifting of nail plate off of distal nail bed (onycholysis).

Koebnerization = new lesions occur at sites of skin trauma (as with lichen planus).

Geographic tongue occurs in 10% of patients with psoriasis (see Fig. 2–1).

Variants: Vulgaris (#1), palmoplantar, guttate (may follow *streptococcal* pharyngitis), erythrodermic, pustular (generalized = von Zumbusch type), inverse (affects folds & flexor surfaces).

Drugs may precipitate or exacerbate psoriasis: Antimalarials, clonidine, indomethacin, iodine, lithium, NSAIDs, quinidine, some beta-blockers, steroid withdrawal, and terfenadine.

In ~10%–20%, may be associated with *psoriatic arthritis*, most commonly of the small joints of the hands and feet; requires systemic Tx.

Investigations: Clinical diagnosis; skin biopsy if uncertain.

DDx: (Plaque psoriasis): Drug reaction, eczema, lichen planus, pityriasis rubra pilaris.



Management

Overview of Psoriasis Therapy

Therapy	Mechanism of Action	Comments
Topical		
Steroids 5%–10% LCD (tar) Anthralin Salicylic acid 3%–10% Calcipotriol (Dovonex) Tazarotene (retinoid) Emollients	Inhibits DNA synthesis, cell turnover. Increases cell turnover. Keratolytic. Vitamin D ₃ analogue. Retinoid (RXR receptor).	Potency & vehicle selected based on body site. Stains and irritates normal skin.
Systemic		
Methotrexate Cyclosporine Acitretin	Folic acid antagonist that inhibits DNA synthesis & causes immunosup- pression. Immunosuppressant that inhibits IL-2 production & thus reduces T-cell proliferation. Inhibits cell replication by modulating cellular differentiation within the epidermis.	Bone marrow and hepatic toxicity. Renal toxicity, hypertension. Preferred for palmoplantar or pustular psori- asis. Use in males & post- menopausal females.

(Continued text on following page)



Overview of Psoriasis Therapy (Continued)

Therapy	Mechanism of Action	Comments
Phototherapy UVB or PUVA	Interferes with DNA synthesis, decreases cellular proliferation, and induces apoptosis of cutaneous lymphocytes leading to localized immunosuppression.	Skin cancer development, photoaging.
Newer Biological Agents	++ Expensive.	
Alefacept	Fusion protein of Fc receptor of human IgG1 and LFA3.	IM; when effective, long remissions; monitoring of CD4 q wk; slow onset.
Efalizumab	Humanized monoclonal antibody to CD11a; blocks LFA-1/ICAM interaction	SC; rapidly effective, but rebound; risk of thrombocytopenia
Etanercept	Fusion protein directed against soluble TNF α .	SC; effective for psoriasis & psoriatic arthritis; rapid onset; no monitoring; use +/- MTX.
Infliximab	Chimeric monoclonal antibody against TNF α .	IV; Fast onset and very effective; must do PPD; infusion reactions.
Adalimumab	Fully humanized anti-TNF monoclonal antibody.	SC; newest of the biologics, still being studied.

Note TNF drugs avoid or use with caution in presence of CHF, demyelination disorders. Increased risk of opportunistic infections.

- Scalp: 10% salicylic acid in mineral oil qhs, tar shampoo qd, steroid lotion, calcipotriol lotion.
- Nails: Intra-dermal triamcinolone acetonide injections (painful), methotrexate, new biologics.
- Generalized psoriasis (>10% BSA) benefits from systemic therapy, best administered by an experienced physician.

Pyoderma Gangrenosum



Uncommon ulcerative skin condition with many potential associations.

Etiology: ~50% are associated with systemic diseases, including: inflammatory bowel disease, polyarthritis, hematologic diseases/disorders (leukemia, myeloma, monoclonal gammopathies), hepatitis, Behçet syndrome.

History: Acute onset with significant pain; arthralgias, malaise, mouth sores (aphthae), and/or abdominal pain may be present; lesions often occur after trauma to skin.



Physical: Initially superficial hemorrhagic pustule, then develops into painful boggy ulceration with dusky-violaceous undermined borders (#1 on lower leg), and heals with cribriform scars; can be single or multiple lesions. Several variants exist (ulcerative, bullous/atypical, pustular, & superficial granulomatous).

Investigations: Biopsy is not specific and is mostly done to rule out other diagnoses. May swab for bacterial culture if signs and symptoms of infection.

DDx: Antiphospholipid antibody syndrome, arterial insufficiency, traumatic ulceration, Sweet syndrome.

Management

Investigate for and treat underlying diseases

- **Medical management:** Surgery should be avoided.
- **Topical care:** Appropriate dressings, topical or intralesional steroids or tacrolimus 0.1% ointment qd to immediate surrounding area.
- Oral prednisone (1 mg/kg/d) initial therapy until healing begins; steroid-sparing agents: azathioprine, cyclosporine, mycophenolate mophetil, dapsone, infliximab
- **Other:** Cyclosporine, cyclophosphamide, clofazimine, sulfapyridine, dapsone, minocycline.

Pyogenic Granuloma



Common benign friable vascular growth (“proud flesh”).

Etiology: Idiopathic or follows trauma to the skin; more common with oral retinoids & indinavir, and during pregnancy.

History: Evolves quickly in children and young adults and bleeds easily; asymptomatic.

Physical: Solitary, red, firm papule; often ulcerated and crusted; often on hands, fingers, face; may be sessile or pedunculated.

Investigations: Clinical diagnosis; biopsy if uncertain.

DDx: BCC, cherry angioma, Kaposi sarcoma, melanoma.

Management

Remove offending drug if implicated; pregnancy-related lesions may self-resolve after delivery.

Tx options: Excision, curettage & electrodesiccation, laser, liquid nitrogen cryotherapy.

Rosacea



Common chronic inflammatory disorder of pilosebaceous units and vasculature of the face. Role of *Demodex* mite controversial.

History: Easy and recurrent flushing, exacerbated by heat (shower, hot drinks), spicy foods, sunlight, cold, alcohol, stress. Sensitive skin. May complain of dry and gritty eyes. Peak incidence 30–50 yr; F > M.

Physical: Erythema, telangiectases, papules, and pustules of central face; no comedones in contrast to acne. Sebaceous hyperplasia, seborrheic dermatitis & facial lymphedema more common.



4 Major Subtypes

Erythematotelangiectatic, papulopustular, ocular, phymatous.

- Chronic inflammation may progress to rhinophyma (enlarged nose; in males). Ocular involvement common (e.g., gritty, conjunctival injection, styes, photophobia).

Investigations: Clinical diagnosis; uncommonly, skin biopsy to rule out lupus or sarcoidosis.

DDx: Acne, lupus erythematosus, perioral dermatitis, sarcoidosis, seborrheic dermatitis.

Management

- Based on severity and subtype.
- **Lifestyle modification:** Avoid triggers; sun protection & avoidance; facial massage for lymphedema.
- **Topical antibiotics**
 - Metronidazole 0.75% gel or 1% cream bid.
 - Sodium sulfacetamide lotion 10% bid.
- **Oral antibiotics** (moderate to severe cases with inflammatory papulopustular component):
 - Tetracycline 500 mg po bid
 - Minocycline 100 mg po od–bid.
 - Doxycycline 20 mg po bid (subantimicrobial dose therapy) or 100 mg po qd–bid.
- Isotretinoin (low dose); less commonly, topical retinoids may be used.
- **Laser therapy** (e.g., PDL, IPL) for telangiectases & ablative laser (e.g., CO₂) for rhinophyma.
- **Camouflage makeup** (e.g., Dermablend™, Covermark®) for erythema.
- Ophthalmologist to assess for ocular involvement (blepharitis, conjunctivitis, episcleritis).

Sarcoidosis



Idiopathic condition characterized by noncaseating epithelioid granulomas affecting any organ system, most commonly the lungs.

History: May have joint pain, fever, fatigue, weight loss, enlarged lymph nodes, anterior uveitis or other ocular findings. Lungs often affected, 1/3 symptomatically. Cutaneous involvement in 25%. Increased incidence in African Americans.

Physical: Owing to potential multisystem involvement, clinical presentation is often variable.

- Cutaneous lesions typically consist of oval red-purple-brown infiltrated plaques; tendency to infiltrate scars. *Lupus pernio* presents with violaceous indurated plaques on the face and is often associated with respiratory tract involvement. *Erythema nodosum*, a hypersensitivity reaction, may occur.

Investigations: Biopsy needed to confirm diagnosis, with staining and cultures to R/O infectious causes of granuloma formation, e.g., mycobacterial and deep fungal infections; investigation for systemic involvement of sarcoidosis.

DDx: Cutaneous tuberculosis, drug reaction, granuloma annulare or faciale, lichen planus, lupus.

Management

Localized cutaneous disease: Topical or intralesional corticosteroids

Widespread systemic disease: Oral prednisone up to 1 mg/kg/d, hydroxychloroquine, steroid-sparing agents (e.g., methotrexate, azathioprine, cyclosporine), thalidomide.

Scabies



Courtesy of Dr. Loretta Fiorillo



Courtesy of Dr. Henry Foong

Etiology: Parasitic infection by *Sarcoptes scabiei var. hominis* mite.

History: Generalized and often debilitating pruritus, worse at night; family members or sexual partners may also itch. Usually transmitted by direct close human contact; incubation period 3–6 wk. More prone: Overcrowding & older patients in nursing homes, young children, & sexually active adults.



Physical: Often nonspecific; papules & excoriations often present in the finger webs, flexor aspects of wrists, elbows, buttocks, genitalia (glans, nipples), with sparing of the head and neck. 4–8-mm linear ridges (burrow) with a gray dot at one end (mite) may be observed in the finger webs. Blisters may be noted (in infants).

- Scabies often misdiagnosed as eczema; should be in differential diagnosis in the setting of persistent generalized pruritus.
- *Crusted “Norwegian” scabies:* More extensive thick nodular infestation by numerous mites; observed in immunocompromised or neurologically impaired (e.g., Down syndrome) patients, & nursing homes. May not be pruritic.

Investigations: If burrow is observed, scabies mites may be visible with a microscope upon examination of a scraping from a lesion prepared with mineral oil or KOH.

Dermatoscope may also be helpful to visualize mite.

DDx: Bug bites, eczema, lice, lichen planus, prurigo nodularis.

Management

- Permethrin 5% cream (i.e., Nix®, Kwellada-P®) is the most reliable topical scabicide.
- Patients should be instructed to apply cream to entire skin surface (neck down in adults, and entire body of infants and young children) with particular attention to finger-web spaces, feet, genitals, and intertriginous sites, and under nails; wash off in 8–14 hr (i.e., overnight application).
- Despite 90% cure rate after a single treatment, repeat treatment 1 wk later; treatment resistance on the rise.
- All household members and sexual partners should be treated simultaneously to prevent “ping-pong” re-infections.
- All clothes worn within 2 d of treatment, towels, and bed sheets should be machine washed in hot water or dry cleaned.
- Patients must be warned that pruritus may persist for weeks owing to delayed hypersensitivity response to the mite; treat with mid-potency steroids or oral prednisone course with taper and antihistamines prn.
- Ivermectin po increasingly being used (esp. crusted scabies) owing to high efficacy, safety, & ease of use.

Scars



Indurated and thickened, reddish plaque that forms at the site of the original injury and may be pruritic & tender. Unlikely to improve without treatment.

- Most commonly found on the neck, shoulders, deltoid, and sternal region.
- More common in Asians and blacks, & 10–30 yr.

Hypertrophic scar remains confined to the site of injury and flattens in time and unlikely to be pruritic.



Other Scars

Acne scars: Due to prolonged duration of inflammatory papules, pustules, and nodules; major reason for treating acne early and aggressively. There are 5 types of acne scars:

1. Icepick scar: Jagged edge, steep sides, narrow, deep, extends into deep dermis or subcutaneous tissue
2. Boxcar scar: Round pits with sharp vertical edges
3. Rolling scar: Distensible, soft-shouldered, with rolling or undulating texture
4. Hypertrophic scar: Thick, raised, usually darker than surrounding skin
5. Train-track scar (shown): Criss-crossed scar resulting from wounds closed under tension and prolonged duration of sutures



Investigations: Clinical diagnosis; biopsy if uncertain.

DDx: None.

Management

- Prevention is key; minimize surgeries in known keloid formers (ask people to tell or show you how they heal); minimize wound tension; avoid where possible surgery to prone areas & treat back and chest acne aggressively to avoid keloids.
- Intralesional steroid injections with triamcinolone acetonide (Kenalog), 10mg–40 mg/ml, every 4–6 wk to flatten the scar and reduce pruritus.
- Liquid nitrogen cryotherapy.
- Laser therapy (CO₂, PDL).
- Occlusive dressings (e.g., silicone gel sheets) and mechanical compression (esp. earlobe keloids).



- Excision followed by intralesional steroids, topical imiquimod (Aldara®) 5% cream or radiation.
- Acne scars: Dermabrasion or microdermabrasion, chemical peels, subcision, punch techniques, and augmentation with fillers.
- "Train-track" scars: Surgical scar revision.

Sebaceous Hyperplasia



Common benign proliferation of sebaceous glands.

History: Asymptomatic, very slow growing; more numerous in transplant pts. on cyclosporine.

Physical: Single or multiple whitish-yellow small soft papules with central umbilication on face.

Investigations: Clinical diagnosis; biopsy if uncertain (i.e., to R/O BCC).

DDx: BCC, milia, molluscum contagiosum.

Management

- Benign, treatment for cosmesis.
- Liquid nitrogen cryotherapy, electrodesiccation & cautery, bichloroacetic acid, CO₂ laser, photodynamic therapy, excision, isotretinoin.

Seborrheic Dermatitis



Etiology: Common chronic inflammatory skin disease of sebum-rich areas linked to *Malassezia furfur*, a commensal yeast.

History: History of burning, itching, or scaling in typical distribution. More prevalent and severe in HIV and neurological diseases (e.g., Parkinson's). Affects 3%–5% of population; two peaks, one in infancy and the other postpuberty; improves in summer.

Physical: Excessive dandruff, orange-erythematous patches with loose dry or greasy scale; distributed in sebaceous gland regions—face (eyebrows, nasolabial folds, and retroauricular areas), scalp, chest, ears; infants: “cradle-cap.”

Investigations: Clinical diagnosis; skin biopsy or fungal culture if uncertain.

DDx: Contact or other forms of eczema, psoriasis, tinea.



Management

Topical Antifungals

- Nizoral shampoo (ketoconazole 2%), qd then taper with improvement.
- Nizoral cream bid to body.
- Oral antifungals in more resistant cases: ketoconazole 200 mg po qd x 10 d.
- Mild topical steroids (often combined with topical antifungals) or steroid-sparing immunomodulators (tacrolimus or pimecrolimus).
- Tar, zinc, sulfur, selenium sulfide, and salicylic acid shampoos regularly used & applied for 5–15 min prior to washing are beneficial for maintenance.

Seborrheic Keratosis (SK)



Common idiopathic benign epidermal growth in middle-age and elderly.

History: Gradual development; occasionally pruritic or sore.

Physical: Variably pigmented, waxy round-oval papules and plaques with verrucous or crusted surface, “stuck-on” appearance; most commonly: trunk, neck, arms, scalp; wide range of size.

Leser-Trélat sign: Sudden, eruptive seborrheic keratoses in elderly individuals may be a sign of an associated internal (stomach cancer & others) malignancy; controversial.

Investigations: Clinical diagnosis; skin biopsy if uncertain.

DDx: AK, BCC, lentigo, nevus, melanoma, skin tag, wart.

Management

- Treatment is for cosmesis or pruritus.
- Liquid nitrogen cryotherapy.
- +/- Curettage.
- Excision.
- Electrodesiccation.
- Laser (CO₂).
- Topical tazarotene cream.

Skin Tag (Achrochordon)



Common benign fibrous growth of skin.

History: Increase in size and number over time; usually asymptomatic, but may become bothersome and tender after trauma or torsion. More common in older people, females (especially during pregnancy), and the obese.

Physical: Soft skin-colored to dark brown pedunculated polyp; commonly occur in axillae, neck, & groin.

Investigations: Clinical diagnosis; shave/snip biopsy if uncertain.

DDx: Nevus, SK, wart.

Management

- Benign and only require management if irritated or for cosmetic purposes.
- Snipping with scissors (use local anesthesia if broad-based stalk).
- Liquid nitrogen cryotherapy.
- Electrodesiccation.

Squamous Cell Carcinoma (SCC)



2nd most common form of skin cancer (after BCC) arising in sun-exposed areas in elderly; due to malignant keratinocytes with metastatic potential.

Etiology: Observed with: Chronic UV-damage, immunosuppression, burns, leg ulcers, chemical carcinogens (e.g., tar), HPV; can occur in discoid lupus, lichen sclerosis, & any scarring processes.

History: Slow-growing, nonhealing scaly papule on sun-exposed area of head, neck, dorsal hands, and forearms; also affects mucous membranes (lower lip: M >> F, smokers, 10%–15% metastatic rate); rare in dark skin. Actinic keratosis is considered a precursor lesion; actinic cheilitis is the precursor on lip.

Physical: Firm indurated papule, plaque, or nodule with adherent rough scale.

Investigations: Biopsy (to mid-dermis) for confirmation.

DDx: AK, BCC, SK, amelanotic melanoma, wart.

Management

- Electrodesiccation & curettage.
- Excision.
- Liquid nitrogen cryotherapy.
- Mohs micrographic surgery; less commonly topical imiquimod or radiation.
- Advice on sun protection, regular skin exams.

Stasis Dermatitis



Etiology: Common inflammatory skin condition of the legs usually associated with venous insufficiency due to varicose veins or other circulatory problems in middle-aged & elderly.

History: Pruritus, leg edema, darkening color of legs.

Physical: Erythema, scaling, eczema, commonly over the medial and anterior aspects of the shin and malleoli; edema & varicosities often noted; lichenification & hyperpigmentation develop in chronic cases.

■ Can be accompanied by a secondary autosensitization "*id*" dermatitis elsewhere on body (esp. if using prolonged topical antibiotics or anesthetics on legs).

Investigations: Vascular studies prior to compression stockings if arterial disease suspected; skin biopsy if associated with chronic nonhealing ulcer.

DDx: Cellulitis, contact dermatitis, pigmented purpuric dermatitis, pretibial myxedema.

Management

- Improve venous return by daily wear of compression support stockings (medical grade, fitted) & leg elevation; surgical intervention if not candidate or fails medical Tx.
- Low-potency topical steroids and oral antihistamines prn.
- Increased incidence of allergic reactions to ingredients in topical medicaments, hence avoid products containing lanolin, fragrances, bacitracin, neomycin, and other common sensitizers for prolonged periods of time.

Stevens-Johnson Syndrome (EM Major) and Toxic Epidermal Necrolysis (TEN)



Etiology: Spectrum of mucocutaneous drug-induced or idiopathic reaction pattern associated with impaired capacity to detoxify intermediate drug metabolites; genetic susceptibility; keratinocyte apoptosis (due to Fas & FasL interaction).

History: Rare, high mortality rate (SJS, 5%; TEN, 30%) characterized by skin tenderness, erythema, desquamation, epidermal necrosis and sloughing affecting skin & mucous membranes, with systemic symptoms:



- **SJS:** lesions generally more macular, 2 mucous membranes involved and <10% epidermal sloughing; typically drug started 1–3 wk prior to rash or feeling unwell with fever cough or sore throat (e.g., URTI with *Mycoplasma*).

- **TEN:** Similar to SJS but > 30% of body surface area sloughed; stronger (95%) association with drugs as cause than SJS; typically drug started 1–3 wk prior to rash.

Physical: Fever, stinging eyes, pain on swallowing, trunk lesions (dusky red macules that become gray in color) & flaccid blisters followed by spread; erosions & erythema of buccal, genital, & ocular mucosa in most cases.

- **Nikolsky sign:** Rubbing unaffected skin induces lesion.

- **Common offending drugs:** Anticonvulsants (esp. carbamazepine, phenytoin), NSAIDs, allopurinol, sulfa drugs; much higher incidence in HIV.

Complications: Potentially life-threatening because of multisystem involvement, e.g., renal failure; severe ophthalmic involvement possible.

Investigations: Skin biopsy; CBC&D, electrolytes, liver enzymes, Cr, urinalysis, INR, PTT, cultures of blood and denuded skin; CXR; consider bronchoscopy or upper GI series.

DDx: Burn, bullous pemphigoid or lupus, linear IgA bullous disease, pemphigus—vulgaris or paraneoplastic.

Management

- Requires Mx by experienced physician—usually dermatologist and/or plastic surgeon.
- Can determine SCORTEN prognostic score (based on age, blood parameters, surface area, malignancy, & heart rate).
- Remove & avoid offending drug (most important!).
- Supportive care, often need ophthalmology assessment and ICU/burn unit admission.
- Proper wound dressings, and antibiotics if any evidence of infection; antihistamines and topical steroids for pruritus; pain control and oral hygiene (e.g., Chlorhexidine rinse) important.
- IVIG therapy is becoming standard of care; controversial.

Striae (Stretch Marks, Striae Distensae)



Etiology: Progressive or rapid stretching of skin can lead to stretch marks, as with exercise, pregnancy (striae gravidarum), weight gain, rapid growth, anabolic steroids, Cushing's disease.

History: Occasionally pruritic. Hx of recent growth spurt, pregnancy, or steroid (topical, oral, anabolic) use.

Physical: Initially pink hue and occasionally bright red-purple, eventually becoming thinned silvery-white depressed bands. Commonly on the abdomen, breasts, buttocks, lumbosacral, and thighs.

Investigations: Clinical diagnosis; rarely need to R/O Cushing's disease.

DDx: Cushing's disease, linear focal elastosis.

Management

Treatment is unsatisfactory.

Wait & see: some improve with time, and pink-red color fades into silvery-white bands.

Topical tretinoin (not during pregnancy) or laser therapy may potentially be effective.

Syphilis



Courtesy of Dr. William Gerstein



Courtesy of Dr. Henry Foong



Courtesy of Dr. Art Huntley



Etiology: A chronic systemic disease due to *Treponema pallidum* spirochete transmitted sexually or through maternal-fetal route.

History & Physical:

It can affect multiple systems, with affinity for neural, cardiac, bone and cutaneous tissue, leading to widespread symptoms.

- Three distinct phases: primary, secondary, then a period of latency (early, late) followed by tertiary phase.
- 1^o: Painless chancre (usually genital) & enlarged regional lymph nodes, incubation period 10–90 d (avg = 3 wk) from inoculation, resolves spontaneously in a few weeks; asymptomatic infections common.
- 2^o: 3–8 wk after primary infection, generalized nonpruritic red-brown macules and papules with palms and soles frequently involved, erosive patches in the mouth, condylomata lata (moist, pink nodules and plaques) on genital areas, “moth-eaten” alopecia; malaise, fever, & generalized lymphadenopathy; develops in all pts. in absence of Tx; resolves spontaneously within 3–8 wk; followed by a period of latency, may be life-long.
- 3^o: 15%–40% of patients with untreated syphilis; multisystem disease which affects cardiac, nervous, and skeletal tissue as well as the skin: Ascending aortic aneurysms, CNS abnormalities, gummas (red-brown granulomatous plaques that erode into underlying cartilaginous and osseous tissue creating deformities). Rare.

Investigations: Darkfield microscopy examination of serous fluid from 1^o and most 2^o lesions; presumptive diagnosis made via positive VDRL/RPR titer, but many false negatives and positives; FTA-ABS & TPPA treponemal assays are more specific for confirmation.

DDx: Drug eruptions, erythema multiforme, herpes simplex, pityriasis rosacea.



Management

- Benzathine penicillin G 2.4 million units IM (in all stages of disease), schedule depends on stage.
- Doxycycline, tetracycline, or erythromycin are second-line Tx choices (if penicillin allergic).
- Patients should be tested for other STDs (esp. HIV).

Tinea (Dermatophyte) Infections



Etiology: Dermatophytes digest and invade keratin and may infect skin, nails, and hair; incubation = 1–3 wk.

Trichophyton, *Microsporum*, *Epidermophyton* species commonly involved. Human-to-human (anthropophilic), animal-to-human (zoophilic; intense inflammation), or soil-to-human (geophilic; moderate inflammation) spread.

Risk Factors: Hot, humid environments, sweating or maceration of the skin, occlusive footwear, diabetes mellitus, immunosuppression (e.g., AIDS).

History: Asymptomatic; occasionally mild pruritus.

Physical: Scalp hair and general body surfaces mostly affected during childhood; hand, foot, or nail infections are more common after puberty.

■ Immunologic response to a dermatophyte infection may result in a dermatophytid or “id” reaction: Vesicular eruption on acral surfaces, especially the palms.

Investigations: Skin scraping analysis with KOH prep—Septate hyphae branching at various angles are seen; fungal culture (~4 wk to ID dermatophyte species); biopsy—PAS or GMS stain can reveal presence of fungal elements.

DDx: Eczema, granuloma annulare, psoriasis.

Tinea Corporis

Affects trunk and extremities: Erythematous annular scaly patches with "active border," central clearing; #1 cause = *T. rubrum*.



Tinea Pedis



1. Interdigital type: Macerated, scaly plaques in toe web spaces, can be portal of entry for cellulitis of the foot, especially in diabetics. (Tip: in recurrent leg cellulitis, look for tinea pedis!)
2. "Moccasin" type: Dryness, scaling and erythema of the plantar and/or lateral foot.

3. Vesicular type: vesicles, pustules, or bullae on the feet.
 ■ "One hand, two feet disease": Common clinical presentation of tinea pedis involving one hand and both feet.



Tinea Cruris



Inner thighs and inguinal folds; tinea faciei: face; tinea manuum: hands; tinea barbae: beard area.

Tinea Capitis

Alopecia with scale, *kerion* (boggy mass), or discrete pustules; very contagious.



Tinea Unguium/Onychomycosis

See onychomycosis section.

Management

Patient education: Avoid factors which predispose to infection, absorbent powders in intertriginous areas, e.g., for tinea pedis—shower-shoes in public facilities.

Topical antifungals for tinea corporis/cruris/pedis (unless lesions are extensive): Terbinafine, ciclopirox, clotrimazole, ketoconazole applied qd or bid x 3 wk, or continue 1 wk until after resolution of lesions.

Systemic antifungals for tinea capitis.

1. Terbinafine (Lamisil) <20 kg = 62.5 mg po qd, 20–40 kg = 125 mg po qd, >40 kg = 250 mg po qd x 2–4 wk.
2. Micronized griseofulvin with food: 20 mg/kg/d po x 8 wk; adults: 500 mg po qd x 1 mo.
3. Patient and household contacts should use an antifungal shampoo, such as selenium sulfide or ketoconazole to reduce spread.

Ulcers, Leg





Etiology: Most common lower extremity ulcers are: venous, arterial or neuropathic; can be mixed.

Other causes: Trauma (+ above), vasculitis, pyoderma gangrenosum, bacterial infections, malignancy, vasospastic (e.g., cryoglobulinemia, sickle cell anemia).

Ulcer Type	Clinical	Management
Venous (most-common; 70%)	Occur in malleolar region; irregular borders; stasis changes, edema, varicose veins, hemosiderosis, painful.	Compression wraps and, once healed, support stockings for life.
Arterial	Occur in pressure sites & toes; necrotic base, punched out w/ shiny atrophic surrounding skin; history of claudication, decreased/absent pulses & prolonged capillary refill.	If possible, surgery to restore arterial blood flow.
Neuropathic	Occur over pressure areas; punched out w/ surrounding callus; decreased sensation; most commonly associated with diabetes.	Relieve (off-load) pressure by special shoes, casts or crutches, better diabetes control.

History: Ask about—onset & course, symptoms, PMHx, social history, & meds. Major morbidity and financial cost.

Physical: Inspect & document—location, size, shape, odor, ulcer edge & base features, surrounding skin (cellulitis, dermatitis), peripheral pulses, & capillary refill.

Investigations: Vascular studies, blood tests, culture & biopsy wound edge; bone scan if you suspect chronic process or osteomyelitis.

DDx: Pyoderma gangrenosum, vasculitis, SCC.

Management

Principles of Wound Care

Treat any underlying cause:

- Venous insufficiency—compression.
- Arterial insufficiency—surgery.
- Neuropathic or decubitus ulcer—pressure relief.
- Infection—antibiotics.
- Neoplasm—surgery, radiation.
- Promote wound healing.
- Débride: Enzymes, surgery/curette. Dressings: Promote moisture, but not oozing/weeping; know your dressings—foams, alginates, hydrogels, hydrocolloids, hydrofibers.
- Infection control: Topical or oral Abx.
- Skin grafting.
- Less commonly: skin substitutes, growth factors.

Allergic contact dermatitis common with long-term topical antibiotic use (esp. bacitracin, neomycin); use petroleum jelly—low allergenicity, safe, & cheap.

Compression stockings required if evidence of pitting/stasis dermatitis; obtain ankle-brachial index prior to initiating treatment; medical grade better than over-the-counter.

Topical metronidazole eliminates odor. Short-contact topical tretinoin appears to be beneficial.

Biopsy ulcer if fails to improve with care.

Ulcers, Pressure (Decubitus)





Etiology: The trigger event is compression of the tissues against an external object: e.g., mattress, wheelchair, or bed rail. Persistent (i.e., > 2 hr) pressure in excess of capillary filling pressure (32–35 mm Hg) leads to tissue ischemia, necrosis, and ulceration. Shear forces and friction aggravate the effects of pressure.

History: Immobility is the key risk factor, with the elderly and neurologically impaired most prone. High morbidity and mortality, particularly because of superinfection of pressure ulcers.

Physical:

Pressure Ulcer Staging

Stage 1	Nonblanchable erythema of intact skin
Stage 2	Partial-thickness skin loss involving the epidermis and/or dermis
Stage 3	Full-thickness skin loss with damage to the subcutaneous tissue that may extend down to, but not through, the underlying fascia
Stage 4	Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures.

Investigations: Culture to identify and quantify bacterial colonization; biopsy in persistent nonhealing ulcers.

DDx: SCC, pyoderma gangrenosum, osteomyelitis.

Management

Prevention is paramount: Turn and reposition the patient often (e.g., q 2 hr).

Specialized pressure-diffusing beds and support cushions.

Surrounding intact skin must be kept clean and dry.

Assess and treat bacterial infection.

Débridement (chemical, physical) of necrotic tissue.

Adequate nutrition necessary for good wound healing.

Consider Wound Vac®.

Multidisciplinary approach is best.

Urticaria (Hives)



Etiology: Common vascular reaction pattern of the skin.

Histamine release from mast cells in the dermis from immunologic or nonimmunologic mechanisms.

Causes of acute urticaria:

Foods: Fish, shellfish, eggs, nuts, strawberries.

Infections #2: viral (e.g., URTI), bacterial (e.g., strep throat, sinusitis, UTI), parasitic.

Drugs: Aspirin, NSAIDs, alcohol, codeine, morphine, penicillin, sulfonamides, thiazides.

Insect stings.

Systemic illness—i.e., lupus, malignancies.

Idiopathic—#1

History: Transient migratory wheals, individual lesions last <24 hr, marked pruritus; up to 15% lifetime incidence; F > M.

May be acute <6 wk or chronic >6 wk.

Special forms of urticaria: cholinergic, contact, physical (aquagenic, adrenergic, cold, dermatographism, heat, pressure, solar, vibratory), urticarial vasculitis (wheals last >24 hr), C1 esterase inhibitor deficiency (rare).

Physical: Annular or arciform erythematous, edematous papules and plaques; may become confluent to form serpiginous or polycyclic patterns.

Investigations: Often not warranted in acute urticaria; order tests to confirm suspected causes, e.g., CBC, ESR, C4, TSH, ASOT titers, sinus films, U/A, stool for O&P; skin biopsy if lesions persist >24 hr; consider work-up if chronic.

DDx: Insect bites, urticarial bullous pemphigoid, mastocytosis, erythema multiforme, urticarial vasculitis, angioedema.

Management

- Ask about facial edema or difficulty breathing, which may be warning signs of systemic anaphylaxis.
- Identify and eliminate etiologic agents.
- Avoid *aggravating factors*: ACE-I, ASA, codeine, morphine, NSAIDs.
- Minimize: Stress, heat, alcohol.

Symptomatic relief:

Mild cases: Low sedating H1 blockers Zyrtec (cetirizine) 10 mg po qd, Allegra (fexofenadine) 60 mg po bid or Claritin (loratidine) 10 mg po qd or Aerius/Clarinet (desloratidine) 5 mg po qd.

Moderate cases: Doxepin 25 mg po qHS.

Severe cases: Prednisone taper x 3 wk for symptomatic relief (avoid in chronic cases); other: cyclosporine, plasmapheresis, IVIG

Epipen on discharge, if severe reaction.

Vasculitis, Leukocytoclastic





Etiology: Often a reaction pattern to internal or external agent; broadly divided into categories: infections, drugs, malignancy, autoimmune connective tissue disease, idiopathic.

History: Symptoms include itching, burning, or pain. Can also be asymptomatic. May have internal involvement and symptoms, e.g., fever, arthritis, abdominal pain, diarrhea, cough, weakness, sinusitis, & hematuria.

Physical: Palpable purpura, most commonly over lower legs (dependent areas).

Investigations: Work-up for potential trigger and assessment of systemic involvement, esp. renal; CBC&D, liver enzymes, urinalysis, CRP, Hep B & C, cryoglobulins, ANA, ENA, ANCA, RF, complement; skin biopsy (reveals small vessel vasculitis); immunofluorescence to rule out HSP.

DDx: Thrombocytopenia or ITP or TTP, meningococemia, other vasculitic syndromes.

Management

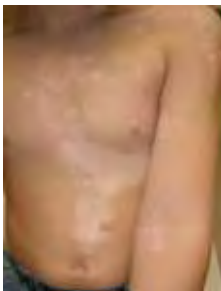
Supportive care and treatment of underlying cause (e.g., infections—Hep C).

Removal of causative agent (e.g., drugs—especially antibiotics, allopurinol, thiazides).

Therapy depends on whether there is evidence of internal vasculitis.

Oral prednisone, colchicine, dapsone, steroid-sparing immunosuppressive agents (e.g., azathioprine).

Vitiligo



Acquired skin disorder characterized by well-defined areas of complete epidermal depigmentation; various pathogenesis theories; likely a large autoimmune component.

History: Asymptomatic; 50% present before age 20; 1%–2% affected; rare in infancy & old age.

■ Associated with immune disorders; therefore work-up for thyroid disease (TSH), diabetes, pernicious anemia, and other autoimmune diseases occasionally indicated (e.g., family history).

Physical: Stark white patches with well-demarcated border; hair in vitiligo patches appears white or gray; several variants: segmental, acrofacial, mucosal, etc.

Course: increased risk of sunburn and skin cancer in amelanotic areas; frustrating to Tx.



Investigations: Wood's lamp accentuates areas of vitiligo & helps determine extent; skin biopsy uncommonly; consider TSH and fasting blood glucose.

DDx: Leprosy, pityriasis alba, tinea versicolor, tuberous sclerosis, nevus anemicus, or depigmentosus.

Management

- Mainly for cosmetic/psychological burden; consider assessment for diabetes, thyroid & Addison disease.
- Topical steroids and new topical immunomodulators (tacrolimus & pimecrolimus), PUVA, NB-UVB phototherapy, camouflage makeup; oral steroid rarely used.
- Note that facial and more proximal lesions respond better than acral lesions.
- Surgical transplants can be considered in stable vitiligo—uncommon
- If extensive, rarely can consider bleaching of nonaffected areas to result in total white color.
- Advise re: importance of sun protection.
- Autologous transplantation or grafting of cultured autologous melanocytes in stable vitiligo (still in research); 308-nm excimer laser (being studied).

Warts (Verrucae)



Etiology: Cutaneous intraepidermal viral infection caused by different subtypes of human papilloma virus (HPV) transmitted directly through broken skin.

History: Affects up to 20% of school-aged children; more common in immunosuppressed and transplant patients; occasionally painful (plantar); can cause psychosocial distress.

Types: Vulgaris (common warts), plantar, flat, genital (condyloma acuminata).



Physical: Flesh-colored hyperkeratotic firm papules, disrupt normal fingerprint lines and display small black dots (thrombosed capillaries).

Course: Genital HPV types 16, 18, and 31 can act to promote development of SCC.

DDx: Molluscum contagiosum, SK, SCC.

Management

- Conservative: Wait & see, 20%–30% clear w/i 6 mo, 50% w/i 1 yr, & 66% w/i 2 yr.
- Patient counseling if anogenital warts to reduce transmission (e.g., condoms) and tests for other STDs. Also at higher risk for anogenital malignancies—females should have annual PAP smears.
- The goal of therapy is superficial destruction of affected skin and elicitation of immune response.
- Plantar warts are thick and should be pared down; can soak foot in water to ease paring.
- Liquid nitrogen—swab or cryospray (avoid in pts. with dark skin), repeat every 3–4 wk.
- Podophyllin 25% for genital warts x 4–6 hr (avoid contact with normal skin) and washed at home; repeat weekly.
- Salicylic acid (up to 40%) OTC—home treatment.
- Duct tape—may be useful in plantar warts, leave on for 1 wk then replace.
- Electrodesiccation +/- curettage.
- Trichloroacetic acid (80%–90%).
- Immunotherapy with diphencyprone (DPC).
- Intralesional bleomycin, candidal antigen for resistant warts.
- Topical retinoids (esp. flat warts).
- Imiquimod 5% cream (esp. genital warts) or topical 5-FU.
- Scissors excision.
- CO₂, PDL laser vaporization.
- In case of extensive perivaginal or perianal warts, referral to gynecologist or gastroenterologist, respectively, to assess internal involvement.
- Vaccines currently in development (for HPV 6,11,16,18).

Acronyms & Abbreviations

Abx	Antibiotics
ACD	Allergic contact dermatitis
ACE-I	Angiotensin converting enzyme inhibitor
AK	Actinic keratosis
ANCA	Antineutrophil cytoplasmic antibodies
ANA	Antinuclear antibody
ASA	Acetyl salicylic acid (aspirin)
BCC	Basal cell carcinoma
BID	<i>Bis in die</i> (twice a day)
BP	Bullous pemphigoid
BSA	Body surface area
BUN	Blood urea nitrogen
Bx	Biopsy
CBC (&D)	Complete blood count (& differential)
CF	Cystic fibrosis
CHF	Congestive heart failure
CHILD syndrome	Congenital <i>hemidysplasia</i> , <i>ichthyosiform erythroderma</i> , and <i>limb defects</i>
CK	Creatine kinase
Cr	Creatinine
CREST	Calcinosis cutis, <i>Raynaud's</i> phenomenon, esophageal dysfunction, sclerodactyly, telangiectasia
CRP	C-reactive protein
C&S	Culture & sensitivity
CTCL	Cutaneous T-cell lymphoma
DEJ	Dermal-epidermal junction
DF	Dermatofibroma
DHEA(S)	Dehydroepiandrosterone (sulfate)
DIF	Direct immunofluorescence
DLE	Discoid lupus erythematosus
EM	Electron microscopy
ENA	Extractable nuclear antigen
ESR	Erythrocyte sedimentation rate
F	Female
FSH	Follicle-stimulating hormone
5-FU	5-Fluorouracil

GVHD	Graft-versus-host disease
Hgb	Hemoglobin
H&E	Hematoxylin & eosin
HPV	Human papillomavirus
HSP	Henoch-Schönlein purpura
IBD	Inflammatory bowel disease
ICAM	Intracellular adhesion molecule
IIF	Indirect immunofluorescence
ILVEN	Inflammatory linear verrucous epidermal nevus
IM	Intramuscular
ITP	Idiopathic thrombocytopenic purpura
IV	Intravenous
IVIG	Intravenous immunoglobulin
KA	Keratoacanthoma
KOH	Potassium hydroxide
KP	Keratosis pilaris
LCV	Leukocytoclastic vasculitis
LH	Luteinizing hormone
LP	Lichen planus
LS & A	Lichen sclerosus et atrophicus
M	Male
MCV	Molluscum contagiosum virus
MF	Mycosis fungoides
MM	Malignant melanoma
MTX	Methotrexate
Mx	Management
NF	Neurofibromatosis
NSAIDs	Nonsteroidal anti-inflammatory drugs
OD	Once a day
OTC	Over the counter
PCOS	Polycystic ovarian syndrome
PCR	Polymerase chain reaction
PCT	Porphyria cutanea tarda
PHN	Postherpetic neuralgia
PMHx	Past medical history
PPD	Paraphenylenediamine
PPD	Purified protein derivative (TB skin test)
PRP	Pityriasis rubra pilaris

PUPPP	Pruritic urticarial papules & plaques of pregnancy
R/O	Rule out
SC	Subcutaneous
SCC	Squamous cell carcinoma
SJS	Stevens-Johnson syndrome
SK	Seborrheic keratosis
SLE	Systemic lupus erythematosus
STDs	Sexually transmitted diseases
TBSE	Total body skin examination
TEN	Toxic epidermal necrolysis
TPPA	<i>Treponema pallidum</i> particle agglutination test
TTP	Thrombotic thrombocytopenic purpura
Tx	Treatment
Ungt	Ointment
URTI	Upper respiratory tract infection
U/S	Ultrasound
UV(L)	Ultraviolet (light)
VZV	Varicella zoster virus
yo	Years old

Spanish-English Vocabulary & Translation

Abscess	El absceso
Acne	El acné
Allergic dermatitis	La dermatitis alérgica
Allergies	Las alergias
Allergy	La alergia
Address	La dirección
Adverse effect	La reacción adversa, el acontecimiento adverso (for FDA and IRBs)
Antiallergenic	El antialérgico
Antibiotic	El antibiótico
Antifungal	El antimicótico, para la infección por hongos

(Continued text on following page)



Spanish-English Vocabulary & Translation *(Cont'd)*

Antihelminthic	Medicamento para las lombrices
Antihistamine	Los antihistamínicos
Arthralgia, joint pain	El dolor en las articulaciones, dolor en las coyunturas, el cuerpo "cortado," la artralgia
Bacterial infection	La infección bacteriana/infección bacterial
Bandage	La venda, el vendaje, el apósito
BID (twice a day)	Bid, dv/d
Birth mark	La mancha de nacimiento
Bite	La mordida, mordedura
Biopsy	La biopsia
Blister	La ampolla
Blistering disease	La enfermedad ampollosa
Blood chemistry	La química sanguínea
Blood culture	El hemocultivo
Blood work	El análisis de sangre
Body hair	El vello
Boils	Los forúnculos
Bruising	Los moretones, cardenales
Burn	La quemadura
Candida/yeast infection	La candidosis/la candidiasis
Cash	El efectivo
Canker sores	El afta/la estomatitis aftosa
Chickenpox	La varicella
Condom	El preservativo/condón
Contraceptive	El anticonceptivo
Contraceptive pill	La píldora anticonceptiva

(Continued text on following page)

Spanish-English Vocabulary & Translation (Cont'd)

Contraindications	Las contraindicaciones
Co-pay	El pago compartido, copago
Cosmetics	Los cosméticos
Cream	La crema
Credit card	La tarjeta de crédito
Cyst	El quiste
Dandruff	La caspa
Dermatitis	La dermatitis, ronchas
Dermatologist	El dermatólogo (la dermatóloga)
Dermatology	La dermatología
Diaphragm	El diafragma
Difficult to treat	Difícil de tratar
Diarrhea	La diarrea
Dosage	La dosificación
Dose	La dosis
Drug	El medicamento, fármaco, la medicina
Drug (illicit, recreational)	La droga
Drug eruption	La erupción provocada por un medicamento/fármaco—la erupción yatrógena/la erupción medicamentosa/el exantema medicamentoso
Drug reaction	La reacción a un medicamento/fármaco—la reacción yatrógena/la reacción medicamentosa
Dry mouth	La boca reseca
Dry skin	La piel reseca
Drug interactions	Las interacciones farmacológicas

(Continued text on following page)

**Spanish-English Vocabulary & Translation (Cont'd)**

Eczema	El eczema
Family physician	El médico familiar
Father	El padre
Fatigue	El agotamiento, la fatiga
Fever	La fiebre, calentura
Fill-out	Llenar, completar
Folliculitis	La foliculitis
Form	El formulario, la forma (México)
Full name	El nombre completo
Fungus infection	La infección micótica, la infección por hongos
Gauze	La gasa
Gel	El gel
Generic drug	Los medicamentos genéricos
Genital area	El área genital
Genitals	Los genitales
Hair	El pelo/el cabello
Hair loss	La alopecia/la pérdida del cabello/ la caída del cabello
Harmless growth	La masa benigna/el tumor benigno/ la neoplasia benigna
Hives/urticaria	Las ronchas/la urticaria
Hospital	El hospital, la clínica, el sanatorio
Hyperpigmentation	La hiperpigmentación
Hypertrichosis	La hipertrichosis/el hirsutismo
Hypopigmentation	La hipopigmentación
Immune globulin	La inmunoglobulina
Impetigo	El impétigo

(Continued text on following page)

Spanish-English Vocabulary & Translation (Cont'd)

Inherited skin disease	La enfermedad hereditaria de la piel/ la dermatosis hereditaria/ la dermatopatía hereditaria
Insect bite	El piquete de insecto
Intravenous (i.v.)	Intravenoso(a), endovenoso(a)
Interpreter	El intérprete (la intérprete)
Irritant dermatitis	La dermatitis irritativa
IUD	El dispositivo intrauterino/el "aparato"
Keloid	La cicatriz queloide/el queloide
Laser(s)	El láser/los láser
Leprosy	La lepra
Lice	Los piojos
Lotion	La loción
Lupus (erythematosus)	El lupus/el lupus eritematoso
Lymph nodes	Los ganglios linfáticos
Malaise	El malestar
Married	Casado
Measles	El sarampión
Medical (adj)	Médico, médica
Medical history	Los antecedentes médicos, el historial médico, la historia médica
Medical insurance	Seguro médico
Melanoma	El melanoma
Mole	El lunar
Mother	Madre (su señora madre, su mamá, mi mamá, mi madre. Do not use "su madre.")
Myalgia	Los dolores musculares, mialgia

(Continued text on following page)



Spanish-English Vocabulary & Translation *(Cont'd)*

Nails	Las uñas
Nausea	Las náuseas, la náusea
NSAIDs	Los medicamentos antiinflamatorios no esteroides
Nurse (n)	La enfermera (el enfermero)
Occupation	Ocupación, empleo, trabajo
Office (medical)	El consultorio
Office hours	Horas hábiles
Ointment	Ungüento
Old age spot (harmless)	Las manchas de la vejez
Orally	Por vía oral
Oral medication	El medicamento oral
Over-the-counter medications	Los medicamentos de venta libre; medicamentos que no requieren receta médica
Pain medication	Los medicamentos para el dolor, los analgésicos
Patient	El paciente (la paciente)
Perfume	El perfume, el agua de colonia
Pharmacy	La farmacia, la botica, el dispensario médico (small towns)
Pill, the	La píldora/la pastilla
Please take a seat	Por favor tome asiento; tenga a bien tomar asiento
Precancer	Precáncer
Pregnancy test	La prueba de embarazo
Prescription drugs	Los medicamentos de venta controlada; medicamentos que requieren receta médica
PRN (as needed)	PRN (según se requiera)

(Continued text on following page)

Spanish-English Vocabulary & Translation (Cont'd)

Problem	El problema
Pruritus	La comezón, el prurito
Psoriasis	La psoriasis
Pus	El pus
QD (every day)	Diario
QD (every day)	Todos los días
Rash	La erupción de la piel, la urticaria
Receptionist	La recepcionista
Redness	El enrojecimiento
Referring physician	El médico remitente
Relatives	Los parientes
Resident	El residente (la residente)
Results	Los resultados
Rheumatoid arthritis	La artritis reumatoide
Rosacea	La rosácea
Roseola	La roséola
Salve	El bálsamo, la pomada
Scar	La cicatriz
Scabies	La sarna
Scalp	El cuero cabelludo
Sexually transmitted disease	Las ETS/las enfermedades de transmisión sexual/las enfermedades venéreas
Shampoo	El champú
Skin cancer	El cáncer de la piel
Skin rash	El exantema/la erupción cutánea
Skin tag	La turgencia cutánea/el acrocordón/ el papiloma cutáneo

(Continued text on following page)

**Spanish-English Vocabulary & Translation (Cont'd)**

Sore	La llaga/la úlcera
Sores	Las lesiones, las úlceras
Steroids	Los esteroides
Strep throat	La infección de la garganta por estreptococos
Subcutaneous	Subcutáneo(a)
Surgeon	El cirujano (la cirujana)
Surgery	La cirugía
Swelling	La hinchazón
Symptom	El síntoma
Syphilis	La sífilis
Tablespoon	La cucharada
Tablet	El comprimido, la tableta, la pastilla
Teaspoon	La cucharadita
Tenderness	Un poco de dolor
Thrush	El algodoncillo
TID (three times a day)	tid, tv/d (tres veces al día)
Toilet/commode	El inodoro, la taza, el water, el WC (escusado, slang)
Topical	Tópico(a)
Topical medication	El medicamento tópico
Ulcer	La úlcera
Viral infection	La infección vírica/infección viral
Vitiligo	El vitiligo/la leucodermia adquirida
Vomiting	El vómito
Warts	Las verrugas
Wife	La esposa

Courtesy of Verónica Albin.

Synonyms Index

Medical Terminology	Synonyms and Layman's Terms
Acrochordon	Skin tag
Actinic keratosis	Solar keratosis
Actinic purpura	Solar or senile purpura
Androgenetic alopecia	Male-pattern baldness
Aphthous ulcers	Canker sores
Asteatotic eczema/ dermatitis	Eczema craquelé
Atopic dermatitis	Atopic eczema; eczema
Bloom syndrome	Congenital telangiectatic erythema
Bowen disease	Squamous cell carcinoma in-situ
Buerger syndrome	Thromboangiitis obliterans
Buschke-Ollendorf syndrome	Dermatofibrosis lenticularis
Campbell de Morgan spot	Cherry (hem)angioma
Candidiasis	Moniliasis
Churg-Strauss syndrome	Allergic granulomatosis
Clark nevus	Dysplastic nevus
Coccidiomycosis	San Joaquin Valley fever
Condylomata acuminata	Genital warts
Crowe sign	Axillary freckling
Cutaneous larva migrans	Creeping eruption
Cutaneous T-cell lymphoma	Mycosis fungoides
Darier disease	Keratosis follicularis
Decubitus ulcer	Pressure ulcer
Degos disease	Malignant atrophic papulosis
Dermatoheliosis	Photoaging

(Continued text on following page)

Synonyms Index (Continued)

Medical Terminology	Synonyms and Layman's Terms
Digital mucous cyst	Digital myxoid cyst; synovial cyst
Discoid lupus erythematosus	Chronic cutaneous lupus erythematosus
Dyshidrotic eczema	Pompholyx
Eczema herpeticum	Kaposi's varicelliform eruption
Ephelides	Freckles
Epidermoid cyst	Sebaceous cyst; wen; epidermal cyst
Epidermolytic hyperkeratosis	Bullous congenital ichthyosiform erythroderma
Erythema induratum (of Bazin)	Nodular vasculitis
Erythema infectiosum	Fifth disease
Fabry syndrome	Angiokeratoma corporis diffusum
Favre-Racouchot syndrome	Nodular elastosis with cysts and comedones
Fish tank granuloma	<i>Mycobacterium marinum</i> infection
Fox-Fordyce disease	Apocrine miliaria
Furunculosis	Boils
Geographic tongue	Migratory glossitis
Gianotti-Crosti syndrome	Papular acrodermatitis of childhood
Glomus tumor	Glomangioma
Glossodynia	Burning mouth syndrome
Goltz syndrome	Focal dermal hypoplasia
Gorlin syndrome	Nevoid basal cell carcinoma syndrome; basal cell nevus syndrome
Gougerot-Carteaud papillomatosis/syndrome	Confluent and reticulated papillomatosis

(Continued text on following page)

Synonyms Index (Continued)

Medical Terminology	Synonyms and Layman's Terms
Granuloma inguinale	Donovanosis
Grover disease	Transient acantholytic dermatosis
Günther disease	Erythropoietic porphyria
Hailey-Hailey disease	Benign familial pemphigus
Hansen disease	Leprosy
Hemangioma of infancy	Strawberry hemangioma; infantile hemangioma
Herpes labialis	Cold sore; fever blister
Herpes zoster	Shingles
Hutchinson freckle	Lentigo maligna
Hyperhidrosis	Excess sweating
Job syndrome	Hyper IgE syndrome
Kawasaki syndrome	Mucocutaneous lymph node syndrome
Koilonychia	Spoon nails
Kyrle disease	Hyperkeratosis follicularis et parafollicularis in cutem penetrans
Leukocytoclastic vasculitis	Hypersensitivity vasculitis
Lichen sclerosus	Lichen sclerosus et atrophicus
Measles	Rubeola
Melasma	Chloasma
Melkersson-Rosenthal syndrome	Cheilitis granulomatosa
Miliaria rubra	Prickly heat
Morphea	Localized scleroderma
Mucha-Habermann disease	Pityriasis lichenoides et varioliformis acuta (PLEVA)

(Continued text on following page)

Synonyms Index *(Continued)*

Medical Terminology	Synonyms and Layman's Terms
Mucocele	Ranula
Mycetoma	Madura foot
Mycosis fungoides	Cutaneous T-cell lymphoma
Myxoid cyst	Digital mucous cyst
Nevus or nevi	Mole(s)
Nevus sebaceus	Organoid nevus
Norwegian scabies	Crusted scabies
Nummular eczema	Discoid eczema
Ofuji's disease	Eosinophilic folliculitis
Onychomycosis	Tinea unguium
Osler-Weber-Rendu disease	Hereditary hemorrhagic telangiectasia
Papular urticaria	(arthropod) bite reaction
Pediculosis	Lice
Pediculosis pubis	Crab lice; pubic lice
Pemphigoid gestationis	Herpes gestationis
Pilomatrixoma	Calcifying epithelioma of Malherbe
Pityriasis versicolor	Tinea versicolor
Port-wine stain	Nevus flammeus
Pseudofolliculitis barbae	Beard bumps
PUPPP	(Pruritic urticarial papules & plaques of pregnancy) Polymorphic eruption of pregnancy (PEP)
Pyogenic granuloma	Lobular capillary hemangioma
Reiter syndrome	Reactive arthritis
Roseola infantum	Exanthem subitum; sixth disease
Rothmund-Thomson syndrome	Poikiloderma congenita

(Continued text on following page)



Synonyms Index (Continued)

Medical Terminology	Synonyms and Layman's Terms
Rubella	German measles
Schamberg disease	Progressive pigmentary purpura
Scleroderma	Progressive systemic sclerosis
Seborrheic dermatitis	"Cradle cap" (infants); dandruff
Sneddon-Wilkinson disease	Subcorneal pustular dermatosis
Spider angioma	Spider nevus; nevus araneus
Spitz nevus	Juvenile melanoma
Stein-Leventhal syndrome	Polycystic ovary syndrome
Striae	Stretch marks
Sweet syndrome	Acute febrile neutrophilic dermatosis
Syphilis	Lues
Temporal arteritis	Giant cell arteritis
Tinea corporis	Ringworm
Tinea cruris	Jock itch
Tinea pedis	Athlete's foot
Trichilemmal cyst	Pilar cyst; wen
Ulerythema ophryogenes	Keratosis pilaris atrophicans faciei
Urticaria	Hives
Varicella	Chickenpox
Verruca vulgaris	Wart (common)
Von Recklinghausen disease	Neurofibromatosis type I
Wells' syndrome	Eosinophilic cellulitis
Werner syndrome	Progeria
Woringer-Kolopp disease	Pagetoid reticulosis
Xerosis	Dry skin
Zoon balanitis	Balanitis circumscripta plasmocellularis

Websites of Interest

For Healthcare Workers

American Academy of Dermatology (AAD): <http://www.aad.org>

Dermanities: <http://www.dermanities.com>

Dermatology Information System (DermIS): <http://dermis.multimedica.de>

Dermatology Nurses Association (DNA): <http://dna.inurse.com>

Dermatology Physician Assistants: <http://home.pacifier.com/~jomonroe>

Dermatology Times: <http://www.dermatologytimes.com/dermatologytimes>

DermMD: www.gizmodoc.com/dermmd

Electronic Textbook of Dermatology: <http://www.telemedicin.org/stamford.htm>

E-medicine dermatology: www.emedicine.com/derm/index.shtml

Evidence-Based Dermatology portal: <http://www.ebderm.org>

Medical Student Core Curriculum from the AAD: <http://www.aad.org/professionals/Residents/MedStudCoreCurr>

For Patients

Alliance for Humane Dermatology: <http://www.ahd.ca>

DermNet: <http://www.dermnetnz.org/index.html>

Healthfinder: <http://www.healthfinder.gov>

Quackwatch: <http://www.quackwatch.org>

Skin Care Physicians: <http://www.skincarephysicians.com>

Support Groups

American Autoimmune Related Diseases Association:
<http://www.aarda.org>

American Behcet's Disease Association: <http://www.behcets.com>

American Hair Loss Council: <http://www.ahlc.org>

American Porphyria Foundation: <http://www.porphyrifoundation.com>

Ataxia-Telangiectasia Children's Project: <http://www.atcp.org>

Camp Discovery: <http://www.aad.org/public/Parentskids/CampDisIntro.htm>

Congenital Nevus Support Group: <http://www.nevusnetwork.org>



- Dystrophic Epidermolysis Bullosa Research Association of America (DeBRA): <http://www.debra.org>
- Ehlers-Danlos National Foundation: <http://www.ednf.org>
- Foundation for Ichthyosis & Related Skin Types (FIRST): <http://www.scalyskin.org>
- Genetic Alliance: <http://www.geneticalliance.org>
- Gluten Intolerance Group of North America—Dermatitis Herpetiformis: <http://www.gluten.net>
- Hereditary Hemorrhagic Telangiectasia Foundation: <http://www.hht.org>
- Histiocytosis Association of America: <http://www.histio.org>
- International Forum for the Study of Itch & Living with Itch: <http://www.itchforum.org>
- International Pemphigus Foundation: <http://www.pemphigus.org>
- Leukemia & Lymphoma Society: <http://www.leukemia.org>
- Lupus Foundation of America: <http://www.lupus.org>
- Med Help: <http://www.medhelp.org>
- Mycosis Fungoides Foundation (MFF): <http://www.mffoundation.org>
- National Alopecia Areata Foundation: <http://www.naaf.org>
- National Association for Pseudoxanthoma Elasticum: <http://www.pxenape.org>
- National Eczema Association for Science & Education (NEASE): <http://www.nationaleczema.org>
- National Foundation for Ectodermal Dysplasias: <http://www.nfed.org>
- National Institutes of Health: <http://www.nih.gov>
- National Organization for Albinism and Hypopigmentation (NOAH): <http://www.albinism.org>
- National Organization for Rare Disorders: <http://www.rarediseases.org>
- National Psoriasis Foundation: <http://www.psoriasis.org>
- National Rosacea Society: <http://www.rosacea.org>
- National Vitiligo Foundation: <http://www.vitiligofoundation.org>
- Nevus Outreach: <http://www.nevus.org>
- Pityriasis Rubra Pilaris Support Group: <http://prp-support.org>
- Proteus Syndrome Foundation: <http://www.proteus-syndrome.org>



Pseudoxanthoma Elasticum, Inc.: <http://www.pxe.org>
Scleroderma Foundation: <http://www.scleroderma.org>
Scleroderma Research Foundation: <http://www.srfcure.org>
Sjögren's Syndrome Foundation: <http://www.sjogrens.com>
Skin Cancer Foundation: <http://www.skincancer.org>
Sturge-Weber Foundation: <http://www.sturge-weber.com>
Tuberous Sclerosis Alliance: <http://www.tsalliance.org>
Vascular Birthmarks Foundation: <http://www.birthmark.org>
Xeroderma Pigmentosa Society: <http://www.xps.org>

Additional Skin Diseases & Disorders



Amyloidosis



Angiofibroma



Atrophie blanche



Becker nevus

Additional Skin Diseases & Disorders (*Cont'd*)



Burn (oven)



Callus



Cuti rhomboidalis nuchae



Dermatoheliosis

Additional Skin Diseases & Disorders *(Cont'd)*



Dyshidrotic eczema



Elephantiasis nostra verrucosum



Erosio interdigitalis blastomycetica



Fixed drug eruption



Additional Skin Diseases & Disorders (Cont'd)



Graft versus Host Disease



Hailey-Hailey Disease



Halo Nevus



Juvenile xanthogranulomas

Additional Skin Diseases & Disorders *(Cont'd)*



Koebnerization – in psoriasis



Leishmaniasis



Leprosy – tuberculoid



Lichen amyloidosis



Additional Skin Diseases & Disorders (*Cont'd*)



Linear epidermal nevus



Lipodermatosclerosis



Palmoplantar Keratoderma



Periungual fibroma

Additional Skin Diseases & Disorders *(Cont'd)*



Perniosis



Phytophotodermatitis
(to lime juice)



Pityriasis alba



Port wine stain

Additional Skin Diseases & Disorders *(Cont'd)*

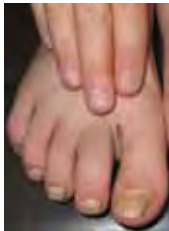
Sporotrichosis



Steroid atrophy



Subungal hemorrhage



Twenty Nail dystrophy

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