



UNIVERSITY of CALIFORNIA, SAN DIEGO
MEDICAL CENTER

Medical Student Guide



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A copy of this file is accessible at: www.dermbridge.com

Welcome to the Division of Dermatology at UCSD. We are excited that you have chosen Dermatology as your rotation and hope that you will find the field as challenging and satisfying as we do. We strive to provide a training program that can prepare our students to recognize and describe standard dermatological disorders, recognize dermatological emergencies, and know when to refer a patient to dermatology throughout his/her medical career. In order to achieve this goal we have created a training program that provides students with extensive experience in clinical dermatology, both adult and pediatric. Students will also be exposed to dermatologic surgery.

This handbook has been developed to provide you with an introduction to the field of dermatology. We have included introductions to our UCSD Dermatology Faculty, information on fundamental descriptive nomenclature, and useful educational websites. Students are required to understand and complete the American Academy of Dermatology Medical Student Curriculum (clinical portions). Information useful for preparation for the USMLE is also included.

We hope you will find this guide useful.

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Lawrence Eichenfield, M.D., Director of Pathophysiology of the Skin Course
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UCSD Dermatology provides the highest quality of care to patients suffering with a variety of diseases of the skin, hair and nails.

Outpatient Dermatology Clinics are located:

1. Ambulatory Care Centers in Hillcrest
2. Perlman Ambulatory Care Center in La Jolla
3. Veterans Administration Hospital in La Jolla
4. Children's Hospital

Residents and attending physicians provide treatment to patients with common, as well as complex dermatologic conditions in all locations.

Services include:

- Dermatologic medicine: treatment of diseases of the skin, hair & nails
- Dermatologic surgery: excisional, including Mohs Micrographic
- Dermatologic Allergy testing: photo & patch
- Cosmetic Dermatology, including Botox, filler injections & lasers
- Dermatopathology
- Pediatric Dermatology

Dermatology Department
UCSD Hillcrest
Mail Code #8420
200 W. Arbor Drive
CTF-C 216
San Diego, CA 92103

Interpreter Services

Phone: 619.543.5730

UCSD Dermatology aims to provide the highest quality of education:

Med 428, Course Description and requirements:

Students will participate in outpatient dermatology clinics at UCSD Hillcrest, the Veterans Administration San Diego, and Children's Hospital, with duties including patient interviews, case presentations to resident and attending dermatologists as well as standard documentation of the patient visit. Students will learn biopsy techniques and assist in surgical procedures. Students attend weekly lectures and conferences at UCSD Hillcrest and the Naval Medical Center. Each student will give two 10-minute power point presentations on a chosen topic at and will be expected to provide oral and/or written materials as requested throughout the rotation.

Pathophysiology of the Skin. *This innovative 2nd year UCSD medical student elective course designed by Drs. Andrew Krakowski and Lawrence Eichenfield is open to 20 to 40 students. The inaugural course starts January 3rd, 2008 and meets every Thursday afternoon from 1-4 pm. This course consists of a lecture series by dermatology faculty. A hands-on interactive "mole check" session will follow the lectures on nevi and melanoma. An interactive session- field trip to the San Diego Zoo is included for the purpose of exploring the descriptions of animal's skin and foliage, using standard dermatologic nomenclature.*

The UCSD Dermatology Interest Group (DIG) has formed to provide students with the opportunity to be exposed to the field of dermatology early on in their education. This group provides education, research, outreach and mentoring opportunities and encourages students to make connections with faculty members in the department. The group is advised by dermatologist Dr. Sharon Jacob and is not limited to students interested in dermatology - as all UCSD medical students are welcome to attend the organizational meetings, educational programs and activities to learn more about our specialty and its applications. UCSD-DIG is a new chapter of the parent organization DIGA (<http://www.derminterest.org/updates.htm>) and provides the UCSD medical students with leadership opportunities both on campus and at regional/national levels.

Rotating Medical Student Dermatology Schedule

	Monday	Tuesday	Wednesday	Thursday	Friday
AM	<p>8 AM (7:50AM on 1st day of rotation) <i>UCSD Clinic ACC, Hillcrest Medical Center 3rd Floor</i></p> <p>One session (after week 1) may be at Pediatric Dermatology Clinic/Children's 8010 Frost Street 6th Floor <i>One session (after week 1) may be at Mohs Surgery UCSD ACC 3rd Floor</i></p>	<p>8 AM (7:50AM on 1st week of rotation)</p> <p><i>VA Clinic Area 1B Drs. Gallo & Romero</i></p>	<p>7:45 – 9:00 AM</p> <p>Lecture – Dr. Nelson VA Area 4</p> <p>9-12 <i>VA Area 4 Clinic Drs. Maier & Jacob</i></p>	<p>8:30 AM</p> <p><i>Grand Rounds UCSD ACC 3rd Floor OR Navy Hospital Bldg 2, 3rd Floor Dermatology</i></p> <p>See schedule at: http://plus.calendar.s.net/ucsdderm</p>	<p>8AM</p> <p><i>UCSD Clinic ACC Hillcrest Medical Center 3rd Floor</i></p>
PM	<p>1:00 PM</p> <p>--Two students with Dr. Jacob at the VA.</p> <p>--One student may be at Children's <i>Pediatric Dermatology Clinic/Children's 8010 Frost Street 6th Floor</i></p> <p>--Rotate locations each week</p>	<p>1:30 PM</p> <p><i>UCSD Clinic ACC 3rd Floor</i></p>	<p>12:30 PM</p> <p><i>VA Surgery Clinic Area 4</i></p>	<p>1 PM</p> <p>One student One student may be at Children's <i>Pediatric Dermatology Clinic/Children's 8010 Frost Street 6th Floor</i></p> <p>---Independent study</p>	<p>1PM</p> <p><i>VA Clinic Area 1B Drs. Nelson & Jacob</i></p>

Web References:

<http://medicine.ucsd.edu/derm/education/medicalstudents.htm>

<http://plus.calendars.net/ucsdderm>

<http://www.childrenspecialists.com/body.cfm?id=21&action=detail&ref=3>

<http://www.aad.org/professionals/Residents/MedStudCoreCurr>

<http://www.emedicine.com/derm/index.shtml>

http://www.doctorfungus.org/imageban/ib_res2.pl

<http://www.buddycom.com/bacteria/gnr/gnreenter.html>

<http://www.dermis.net/doia/mainmenu.asp?zugr=d&lang=d>

<http://dermatlas.med.jhmi.edu/derm/>

www.dermnet.org,

<http://fm.mednet.ucla.edu/derm2/>

<http://www.lumen.luc.edu/lumen/MedEd/medicine/dermatology/melton/atlas.htm>

<http://www.globalrph.com/dermatology.htm>

<http://www.pathmax.com/dermlink.html>

University of California San Diego School of Medicine, Division of Dermatology Faculty Members:

<p>Victoria Barrio, M.D. Clinical Assistant Professor, Departments of Pediatrics and Medicine Rady Children's Hospital</p>
<p>Areas of Interest: pediatric dermatology</p>
<p>Bari B. Cunningham, M.D. Associate Clinical Professor, Departments of Pediatrics and Medicine Director, Dermatologic Surgery and Phototherapy; Rady Children's Hospital San Diego</p>
<p>Areas of Interest: pediatric dermatology</p>
<p>Anna Di Nardo, MD PhD Assistant Professor, Department of Medicine</p>
<p>Areas of Interest: Mast cells and innate skin immunity; High risk pigmented lesion clinic (Moore Cancer Center)</p>
<p>Lawrence Eichenfield, M.D. Professor, Departments of Pediatrics and Medicine Chief, Pediatric and Adolescent Dermatology; Rady Children's Hospital</p>
<p>Areas of Interest: pediatric dermatology</p>
<p>Magdalene Dohil, M.D. Assistant Professor, Departments of Pediatrics and Medicine; Rady Children's Hospital</p>
<p>Areas of Interest: pediatric dermatology</p>
<p>Sheila Fallon Friedlander, M.D. Clinical Professor, Departments of Pediatrics and Medicine; Rady Children's Hospital</p>
<p>Areas of Interest: pediatric dermatology</p>
<p>Richard Gallo, M.D., PhD Professor, Departments of Medicine and Pediatrics; Chief, Division of Dermatology; Chief, Veterans Administration Medical Center Dermatologic Research</p>
<p>Areas of Interest: Innate immune defense systems of the skin</p>
<p>Tissa Hata, M.D. Associate Professor, Department of Medicine Director of Clinics and Clinical Research</p>
<p>Areas of Interest: Clinical dermatology, research</p>
<p>Wendy Havran, PhD Professor, Department of Immunology, The Scripps Research Institute</p>
<p>Skin T cell functions</p>
<p>Chun-Ming (Eric) Huang, PhD Associate Professor, Division of Dermatology</p>
<p>Proteomics; Acne vaccine development</p>

<p>Sharon E. Jacob, M.D. Assistant Professor, Department of Medicine Associate Director, Dermatology Residency Program</p>
<p>Areas of Interest: medical education & T cell mediated inflammatory disorders (atopic & contact dermatitis, psoriasis)</p>
<p>Colin Jamora, PhD Assistant Professor, Department of Biology (Cell and Developmental Biology); Adjunct Assistant Professor, Department of Medicine (Dermatology)</p>
<p>Areas of Interest: epidermal homeostasis and regeneration; hair follicle morphogenesis</p>
<p>S. Brian Jiang, M.D. Associate Clinical Professor of Medicine (Dermatology) Director of Dermatologic Surgery</p>
<p>Areas of Interest: Laser & Mohs surgery</p>
<p>Lisa Maier, M.D. Assistant Clinical Professor Department of Medicine (Dermatology)</p>
<p>Areas of Interest: Acne</p>
<p>Fern Nelson, M.D. Associate Clinical Professor Department of Medicine (Dermatology), VAMC</p>
<p>Areas of Interest: Medical Student Education; Medical Dermatology</p>
<p>Terence O'Grady, M.D. Clinical Professor of Medicine (Dermatology) and Pathology Director, Dermatology Residency Training Program</p>
<p>Areas of Interest: Dermatopathology, Resident Medical Education</p>
<p>Laura Romero, M.D. Assistant Professor and Clinical Director, Veterans Hospital San Diego</p>
<p>Areas of Interest: Dermatopathology</p>
<p>Richard Schweitzer, M.D. Clinical Attending Professor; Department of Medicine (Dermatology), VAMC</p>
<p>Areas of Interest: Cutaneous manifestations of systemic diseases</p>
<p>Daniel W. Shaw, M.D. Associate Clinical Professor, Department of Medicine (Dermatology) Director, Contact Dermatitis Clinic</p>
<p>Areas of Interest: Contact Dermatitis</p>
<p>Benjamin D. Yu, M.D., Ph.D. Assistant Professor, Department of Medicine (Dermatology)</p>
<p>Areas of Interest: Genetic research</p>

Medical Spanish:

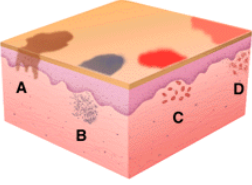
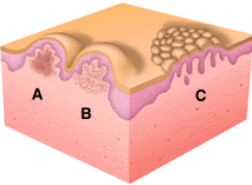
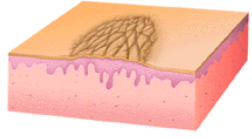
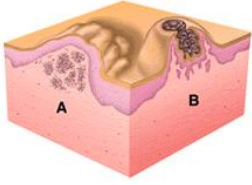
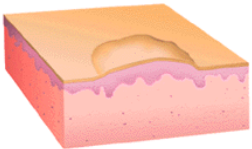

Good morning/ afternoon	Buenos días/tardes.
What is your name?	¿Cómo se llama?
Do you speak English?	¿Habla inglés?
Do you have medical problems?	¿Tiene problemas de medicina?
Does it hurt?	¿Le duele?
Abscess	Absceso
Allergies	Alergias
Benign	Benigno
Big	Grande
Biopsy	Biopsia
Black	Negro
Blister	Ampolla
Blue	Azul
Brown	Café
Bumps	Protuberancia
Burn	Ardor
Cancer	Cáncer
Chicken pox	Varicela
Cream	Crema
Cyst	Quiste
Dermatologist	Dermatólogo
Diagnosis	Diagnóstico
Disease	Enfermedad
Dry	Seco/a
Exam	Examen
Few	Pocos
Green	Verde
Grey	Gris
Gums	Encías
Hair	Cabello
Hives	Urticaria
Infection	Infección
Inflammation	Inflamación
Injection	Inyección
Injury	Herida
Insect bite	Mordedura de
Itching	Picazón
Left	Izquierdo/a
Lesions	Lesiones
Lump	Bulto
Malignant	Maligno
Many	Muchos/as

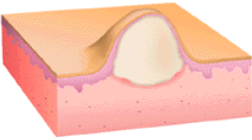
Mild	Moderado
Nail	Uña
New	Neuvo
Nothing	Nada
Old	Viejo
Pain	Dolor
Palm	Palma de mano
Pathologist	Patólogo
Physical exam	Examen físico
Pimple	Grano
Pink	Rosado
Plaque	Plaga
Prescription	Recete médica
Procedure	Procedimiento
Purple	Morado
Pus	Pus
Rashes	Sarpullidos
Red	Rojo
Right	Derecho/a
Scab and scale	Costra
Scar	Cicatriz
Severe	Severo
Skin	Piel
Skin cancer	Cáncer de la piel
Skin disease	Enfermedad de la piel
Skin rash	Erupción en la piel
Small	Chico/a
Soap	Jabón
Sore (wound)	Llaga
Stitches	Puntos
Sun	Sol
Swelling	Hinchazón
Syphilis	Sifilis
Tape	Cinta
Test	Examen
Trauma	Trauma
Ulcer	Úlcera
Varicose veins	Venas varicosas
Wet	Mojado/a
White	Blanco
Yellow	Amarillo

Descriptions of Skin Lesions

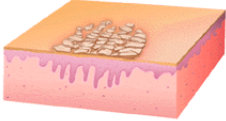
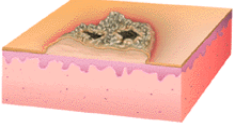

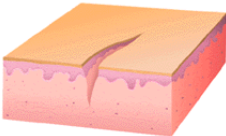
There is an art to describing skin lesions. With a thorough and accurate description a comprehensive differential diagnosis and accurate final diagnosis is within reach. Below is a list of the nine categories used for describing lesions and examples of common terms.

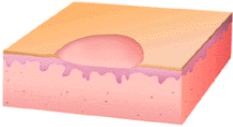
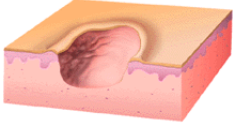
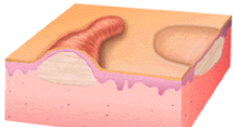
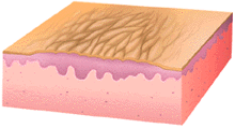
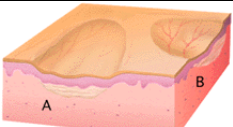
Number of lesions		
Discrete (lesions that are separated)/ confluent (lesions that run together)		
Circumscribed (limited or confined)		
Shape:	Annular (ring shaped)	Arcuate (in the form of an arc)
	Circinate (circular)	Discoid (disc-shaped)
	Guttate (drop-like)	Gyrate (twisted spiral)
	Iris (circle within a circle)	Linear (in lines)
	Moniliform (beaded)	Serpiginous (snake-like)
	Zosteriform (linear arrangement along a nerve)	
Color:	Erythema (red), Violaceous (purple)	
Depth:	superficial, deep	
Surface changes:	Keratosis (horny thickening), Necrotic, Umbilicated, Ulcerated	
Configuration:	Grouped (lesions in clusters), Linear, Arciform, Zosteriform	
Distribution:	Generalized (widespread), Universal (entire skin affected)	

Primary Lesions	Definition	Morphology	Examples
Macule	Flat (nonpalpable) circumscribed skin discoloration		Café au lait Vitiligo Freckle Junctional nevi Ink tattoo Tinea versicolor Melasma
Patch	Large macule, ≥ 1 cm	Same as above	Vitiligo Nevus flammeus
Papule	Solid elevation, < 0.5 cm in diameter		Acrochordan (skin tag) Acne Nevus Melanoma Molluscum contagiosum
Plaques	Broad papule or confluence of papules, > 0.5 cm, no deep component		Psoriasis Eczema Tinea corporis Mycosis fungoides
Nodules	Solid elevation, > 0.5 cm in diameter, larger deeper papule		Rheumatoid nodule Xanthoma Lipoma Metastatic carcinoma Erythema nodosum
Tumors	Large nodule	Same as above	Lipomas, Melanoma, TB
Wheals	Evanescent, pruritic edematous, plaque (a hive)		Urticaria Dermographism Urticaria Pigmentosum
Vesicle	Papule that contains clear fluid (a blister), epidermal, may be unilocular or multilocular		Herpes simplex Herpes Zoster Contact Dermatitis Poison Ivy Dermatitis

Bulla	Large vesicle, >0.5 cm in diameter	Same as above	Pemphigus Vulgaris Bullous Pemphigoid Bullous impetigo
Pustule	Papule that contains purulent fluid		Acne Folliculitis Pustular psoriasis Impetigo
Cyst	Nodule that contains fluid		Acne Epidermal inclusion cyst Pilar cyst

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Secondary Lesions	Definition	Morphology	Examples
Scales (Exfoliation)	Thick stratum corneum resulting from hyper-proliferation or increased cohesion of the keratinocytes		Psoriasis Toxic Epidermal Necrolysis Staphylococcal Scalded Skin Syndrome Eczema Ichthyosis
Crusts (Scabs)	Collection of dry debris, dried sebum, pus or blood		Impetigo Late syphilis Third degree burns
Excoriations & Abrasions (Scratch Marks)	Linear erosions caused by mechanical means		Eczema Scabies
Fissures (Cracks, Clefts)	Linear cleft into the epidermis or dermis		Dry skin from soaps or detergents, chapping

Erosions	Loss of all of the epidermis (heals without a scar)		Herpes zoster Herpes simplex Impetigo
Ulcers	Loss of the epidermis and portions of the dermis (heals with scarring)		Basal Cell Carcinoma Decubitus Dystrophic epidermolysis bullosa
Scars	New connective tissue replacing the lost dermal tissue (dermo-epidermal damage)		Discoid lupus Hypertrophic scars Keloids
Lichenification	Hyperplasia of the epidermis		Caused by chronic scratching or rubbing Atopic Dermatitis
Atrophy	Thinning of the epidermis and/or dermis		Results from topical steroid use or corticosteroid injections

Photos are used with permission from the University of Washington.

Special Lesions	Definition	Examples
Abscess	Collection of purulent material in the dermis or subcutaneous fat	Infections
Burrow	Tunnel in the skin	Parasite Scabies
Comedone	Keratin, sebum, micro-organism and epithelial debris within a dilated follicular opening	Acne
Cyst	Nodule that contains fluid	Acne Epidermal inclusion cyst
Ecchymosis	Deposition of blood in the extravascular tissue, larger than petechia	Bruise Trauma
Fistula	Channel that communicates between two surfaces	Crohn's Disease Surgeries
Hematoma	Extravasations of blood, usually causing a swelling	Trauma Needle Stick
Petechia	Pinpoint, red, lesion, caused by blood in the extravascular tissue	Infections (Endocarditis) Vitamin K Deficiency
Sinus	Elongated tunnel, opening on one surface and having a blind pouch on the other end	Infection Foreign Body
Telangiectasia	Superficial cutaneous small vessels	Basal Cell Carcinoma

***Medical Student Core Curriculum
for Dermatology Rotations***

The following excerpts have been taken from the ***medical student clinical curriculum on the AAD website*** that was sponsored by the Interdisciplinary and Postgraduate Education Task Force of the American Academy of Dermatology and the Association of Professors of Dermatology.

The figures and quizzes found on the AAD website at www.aad.org are located at the back of the guide.

Acne Vulgaris, Folliculitis, and Acne Rosacea

Sonya K. Brown, M.D., and Alan R. Shalita, M.D.

Acne vulgaris, folliculitis and acne rosacea are common disorders of the pilosebaceous units, which consist of sebaceous glands and their associated hair follicles. The most common anatomic sites of involvement are those that have the largest and greatest density of sebaceous glands: the face, neck, upper chest and back and upper arms.

Acne vulgaris is a common disorder that peaks in incidence around the time of puberty. The pathogenesis of acne vulgaris is multifactorial. Abnormal keratinization in the upper canal of the hair follicle causes formation of hyperkeratotic, adherent plugs that are derived from desquamated epithelial cells, resulting in comedones (appearing clinically as whiteheads and blackheads), and the noninflammatory lesions of acne vulgaris. Androgens stimulate the secretion of lipid-rich sebum from the sebaceous glands; sebum, in turn, provides a growth substrate for the commensal *Propionibacterium acnes*, an anaerobic diphtheroid. Proliferation of *P. acnes* is particularly facilitated by the anaerobic environment of the follicles that are plugged by comedones. This leads to the production of proinflammatory mediators that are largely responsible for the appearance of the inflammatory lesions of acne vulgaris: papules, pustules, and nodules.

Treatments for acne vulgaris target one or more of its pathogenetic factors. Topical agents alone may be used for mild cases of acne, whereas systemic agents are generally reserved for patients with moderate to severe involvement. Comedolytic agents act primarily against comedones and include tretinoin and adapalene, both of which are available only as topical preparations. By diminishing the growth of *P. acnes*, antibiotics and antibacterials, available in various topical and systemic preparations are effective against the inflammatory lesions of acne vulgaris. Antiandrogens, administered orally, diminish sebum production, which results in the improvement of inflammatory lesions. Isotretinoin (13-cis-retinoic acid) is a potent systemic agent that affects all of the major pathogenetic factors of acne vulgaris. However, because it can cause a variety of adverse effects, some of which are potentially serious, isotretinoin is generally reserved for the treatment of severe, treatment-resistant acne.

Folliculitis

Folliculitis is a somewhat nonspecific term that refers to inflammation of the hair follicle (in clinical practice, this term does not include acne vulgaris). The most common etiology of folliculitis is bacterial infection, often due to *Staphylococcus aureus*. The usual clinical presentation is superficial pustules and/or papules in the distribution of the hair follicles. The face, chest, back, thighs, and buttocks are often involved. Folliculitis is frequently initiated by mild physical injury to the follicles, such as friction caused by tight-fitting garments, or by ingrown hairs in the beard area in men. Less commonly, folliculitis is caused by infection by fungi, such as dermatophytes or *Pityrosporum*. Folliculitis due to *Pseudomonas aeruginosa* ("hot tub folliculitis") may occur in patients exposed to water sources that are contaminated by that organism. Treatment of folliculitis is aimed at eliminating the offending agent(s), and includes topical and/or systemic antibacterial or antifungal preparations.

Acne Rosacea

Rosacea is an inflammatory disorder of uncertain etiology that most commonly affects adults of northern European ancestry, between 30 and 50 years of age. The earliest manifestation of this disease can be recurrent episodes of flushing and blushing, often triggered by stimuli such as ingestion of hot beverages, spicy foods, and ethanol or exposure to ultraviolet radiation. Clinical findings in the fully developed eruption include papules, pustules, erythema, and telangiectasias. The central face, including the nose, forehead, chin and cheeks, is involved predominantly. Chronic inflammation may lead to permanent enlargement (phyma) of the affected areas due to sebaceous gland and soft tissue hypertrophy; rhinophyma ("W. C. Fields nose") refers to enlargement of the nose. Involvement of the eye may lead to conjunctivitis and/or blepharitis. Rosacea may clinically resemble acne vulgaris; however, in contrast to acne, comedones are absent. Treatments of mild rosacea include topical metronidazole gel, lotion or cream, or a combination of sodium sulfacetamide and sulfur. In moderate to severe cases, oral antibiotics, of which the tetracyclines are the most widely used, may be added. Isotretinoin may be used in severe, recalcitrant cases.

References

1. Rothman KF, Lucky AW. Acne vulgaris. *Adv Dermatol* 1993; 8: 347-74.
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Actinic Keratoses and Non-Melanoma Skin Cancer

Thomas W. McGovern, M.D. and David J. Leffell, M.D.

Actinic Keratoses

The actinic keratosis (AK) is the earliest identifiable lesion that can eventually develop into an invasive squamous cell carcinoma (SCC). These lesions are diagnosed in 14% of all visits to dermatologists, following only acne and dermatitis in frequency. Debate swirls around the nomenclature for these lesions as some consider them "pre-cancerous" and others consider them to be a SCC confined to the lower portion of the epidermis.

Actinic keratoses typically occur in fair-skinned individuals. In various northern hemisphere populations, 11-25% of adults have at least one, compared to 40-60% of adult Australians who live closer to the equator. One prospective study estimates that one AK/1000/year transforms into SCC, while retrospective studies predict that from 5-20% of all untreated AKs will progress to SCC. AK's are typically produced by ultraviolet radiation, but ionizing radiation, arsenic, or polycyclic hydrocarbon exposure may also cause them. At least two prospective studies have demonstrated that sunscreen reduces the likelihood of developing more AKs.

On physical examination, the typical AK is a poorly-demarcated, slightly erythematous papule or plaque found on sun-exposed areas such as the face, balding scalp, posterior neck, and dorsal upper extremity. Characteristically, AKs feel rough or "gritty" and may be difficult to see. Therefore, palpation of high-risk areas under an intense light source is essential to accurate diagnosis. Microscopically, one sees large keratinocytes with atypical nuclei in the lower portion of the epidermis. Liquid nitrogen, 5-fluorouracil cream, trichloroacetic acid, electrodesiccation and curettage, and CO2 laser can all eradicate AKs. Two newer treatment modalities include photodynamic therapy and the topical immunomodulator, imiquimod.

Non-Melanoma Skin Cancer

Non-melanoma skin cancer (NMSC) generally refers to the two most common cancers in the world: basal cell carcinoma (BCC) and cutaneous squamous cell carcinoma (SCC). In 2003, there were an estimated 900,000-1,200,000 cases of NMSC (of which 75-80% were BCCs) in the United States, virtually equal in incidence to the 1,334,100 cases of all other cancers of all types combined. NMSC's account for approximately \$650 million in annual medical care in the United States. The primary risk factors for NMSC include skin phenotype, which determines one's ability to tan, and age, which is a proxy for total ultraviolet exposure. Other risk factors include smoking, immunosuppressed state, outdoor work, and tanning bed use. Organ transplant patients have an increased risk of SCC due to their medical immunosuppression. SCC incidence correlates best with total, lifetime ultraviolet radiation exposure, while BCC occurrence corresponds better with intermittent sunlight exposure and severe sunburns. Rates of NMSC increase with decreasing latitude, as SCC doubles for each 8-10 degree decline. The case-fatality rate for BCC is less than 0.05% and for SCC it is less than 0.7%. These rates have been decreasing for the last 20 years. Approximately 2200 Americans die annually from NMSC, and the vast majority of these die of metastatic SCC. Metastatic BCC is incredibly rare. After developing an initial BCC or SCC, patients have approximately a 50% chance of developing another NMSC within 5 years. For Americans born in 1996,

the lifetime risk of developing NMSC is approximately 20%. Appropriate sun protection including hats, clothing and regular sunscreen are recommended for prevention of actinic keratoses and NMSC.

Superficial spreading BCC appears as a red, scaly, finely wrinkled plaque that may be confused with dermatitis. The typical nodular BCC is a shiny or pearly, translucent papule with overlying telangiectasia and rolled borders. Because the center often outgrows its blood supply, there may be a central, depressed ulcer with or without overlying hemorrhagic crust. Infiltrative or morpheaform BCCs often feel indurated, resemble scars, and possess histologic margins far wider than would be suspected clinically. Microscopically a basal cell carcinoma is characterized by islands of intensely basophilic keratinocytes with peripheral palisading seen extending from the bottom of the epidermis or freely as islands in the dermis. In the more infiltrative types of BCC, thin strands of atypical cells are found within scar-like collagen.

Squamous cell carcinomas are generally erythematous, scaly papules or plaques with ill-defined borders, and they may be confused with large, hypertrophic AKs. It is often difficult to differentiate these AKs from early SCCs without a biopsy. Microscopically squamous cell carcinomas show a proliferation of pleomorphic keratinocytes confined to the epidermis (SCC in-situ) or extending into the dermis (invasive SCC).

Definitive diagnosis of NMSC requires a biopsy, and a shave, or tangential, biopsy is the preferred method. NMSCs may be treated with excision, electrodesiccation and curettage, liquid nitrogen, radiation, or topical imiquimod. Cure rate is 80-95% for BCCs or SCCs treated by these methods. Recurrent or large lesions, those located on high-risk areas or places where maximal normal tissue preservation is essential (such as the nose), and those with aggressive histologic patterns may be referred to a dermatologic surgeon for Mohs micrographic surgery. Mohs surgery achieves a 99% five-year cure rate for primary tumors and a 95% cure rate for recurrent lesions. With this technique, thin layers of tissue are excised and tumors are mapped under microscopic control. Further layers are excised only from areas that have tumor remaining.

All patients with skin cancer and actinic keratoses should protect themselves from sun exposure by the appropriate use of hats, protective clothing, sunscreens and avoidance of peak sunlight exposure. Approximately 60% of the cancer-causing ultraviolet rays reach the Earth's surface during a four-hour period centered on solar noon. The 'shadow-rule' is a simple rule-of-thumb for patients to remember: avoid direct sunlight when your shadow is shorter than you are.

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Pigmented Lesions - Ephelides, Lentigines, Melanocytic Nevi and Melanomas
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Ephelides, simple lentigines, and junctional melanocytic nevi are all small, tan to brown macules. An **ephelis** (*freckle*) is typically found on sun-exposed skin, usually on the face or dorsal forearms and hands of children or young adults with a fair-skinned phenotype. They darken in response to the sun and fade with UV abstinence. Histologically, the only differences that can be detected in lesional skin as compared to normal surrounding skin are larger-sized melanocytes (which are normal in number) with more prominent dendrites and an increased transfer of larger, darker melanosomes to surrounding keratinocytes (see section 3).

Lentigines

are classified into two main varieties. **Solar lentigines** (*liver spots, age spots, senile lentigines*) also occur in response to sunlight, but are more common in middle-aged or older patients, are thought to be caused by years of cumulative UV exposure, and tend to persist even in the absence of sunlight. They favor sites of maximum sun exposure such as the dorsal surface of the hands and the extensor forearms, vary in color from tan to dark brown, and can be up to 1cm in diameter. **Simple lentigines**, in contrast, occur at any age, have no predilection for sun-exposed areas or lighter skin types, and do not darken in response to sunlight. Lentigines have a distinct histologic pattern of elongated, club-shaped rete ridges which often anastomose. Solar lentigines typically have a *normal number* of melanocytes plus increased melanin in the basal layer of the epidermis, overlying a background of solar elastosis (abnormal elastic fibers in the dermis due to cumulative UV exposure). In contrast, simple lentigines typically have an *increased number* of melanocytes, singly arranged along the basal layer of the epidermis. They may be impossible to distinguish clinically from early nevi and are believed by some authors to occasionally evolve into junctional nevi.

Melanocytic nevi

are benign neoplasms, histologically distinguished from lentigines or ephelides by the presence of nevus cells, which are melanocytes that group in well-demarcated nests. Acquired nevi tend to first appear in childhood as flat brown macules or minimally elevated papules, usually less than 6mm in diameter. They evolve during adulthood, becoming more elevated and dome-shaped and then eventually fleshy papules or nodules with loss of pigmentation. Further aging of a nevus can lead to a pedunculated skin tag-like lesion or even complete disappearance of the nevus. It is unusual to see melanocytic nevi in individuals older than 80 years of age. Congenital nevi, in contrast, are present at birth or become clinically apparent during early infancy. They may be small, medium or large in size; the latter is often referred to as a giant or garment nevus. Congenital nevi may have irregular or serrated borders and more frequently involve the hair follicles when compared to common acquired nevi.

Histologically, nevi are classified depending on the location of the melanocytic nests. In junctional nevi, the nevus cells are at the dermo-epidermal junction just above the basement membrane zone of the epidermis; the clinical correlate is a darkly pigmented flat or minimally elevated nevus. As nevi mature, the nests of melanocytes gradually are assimilated into the dermis; they are then classified as either compound when the nests are present at the dermo-epidermal junction *and* within the dermis or as intradermal when the nests are exclusively within the dermis. As the nests descend, they become uniformly smaller and are composed of smaller-sized melanocytes which produce less pigment; the surrounding stroma becomes infiltrated by fibrofatty tissue.

Dysplastic nevi

(*atypical nevi, Clark's nevi, nevi with disordered architecture and cytologic atypia*) are a subgroup of nevi which have an irregular outline, variable pigmentation, indistinct borders, and can be larger than 6mm in diameter. Often described as having a "fried-egg" appearance, they typically have a dome-shaped central brown papular component surrounded by a flatter zone of light brown or tan pigmentation. They show disordered histological architecture, typified by less circumscription of the nevus cell nests and extension of the junctional nests beyond the intradermal component. Dysplastic nevi also show an increased number of single melanocytes in the basal layer of the epidermis; pleomorphism of cells; and nests that vary in size, shape, and spacing. The upper dermis usually shows fibrosis and contains a host response of lymphocytes. When multiple dysplastic nevi are present in a patient with a family history of melanoma, they herald an increased risk for the development of melanoma in that patient. The presence of a single or few dysplastic nevi outside the context of a family history of melanoma may or may not portend an increased risk for that patient.

Cutaneous melanoma

may arise within a previously existing nevus or dysplastic nevus, but approximately 70% of the time, they arise *de novo*. Melanomas are classically divided into subtypes based on their clinical and histopathologic features. Histologically, all are typified by large, pleomorphic, and hyperchromatic melanocytes with loss of orderly architecture and maturation. Most commonly, melanomas begin as minimally elevated, asymmetrical pigmented papules or plaques with irregular, sometimes scalloped, borders and variations in color. At this stage they may be difficult to distinguish from dysplastic nevi.

Superficial spreading melanomas typically show the ABCD's of melanoma (Asymmetry, Border irregularity, Color variegation and Diameter greater than 6mm). If neglected, the depth of tumor invasion can continue to increase; a clinical correlate would be the development of nodularity within the melanoma. Nodular melanoma classically does not have a macular or plaque phase and presents as a blue or black papule or nodule. The superficial spreading and nodular types of melanoma together account for approximately 80% of all melanomas. Both types occur most commonly in patients with lighter skin phenotypes, and may occur anywhere, but have a predilection for the upper back in men and women and the lower legs in women. Risk factors for developing these variants of melanoma include a family history of melanoma (with or without the presence of multiple dysplastic nevi), the presence of numerous common acquired nevi, and a history of blistering sunburns.

The other subtypes of melanoma, lentigo maligna melanoma and acral lentiginous melanoma, are not correlated with intense, intermittent sun exposure. Lentigo maligna melanoma accounts for approximately 5% of all melanomas and is most commonly seen at sites of maximum sun exposure in patients with obvious photodamage, e.g., the face, hairless ("bald") scalp, extensor forearms and upper trunk. Acral lentiginous melanomas

comprise 3-8% of all melanomas and by definition arise on the volar skin of the palms or soles and the nailbeds. Acral lentiginous melanomas have no known correlation with sun exposure and are the most common form of melanoma in African-Americans, Asians, and Hispanics.

The most important indicator of prognosis for all subtypes of melanoma is the Breslow's depth, which is the maximal thickness of tumor invasion as measured by an ocular micrometer, from the top of the granular layer of the epidermis to the base of the neoplasm. Breslow's depth is recorded in millimeters with lesions less than 1.0 mm having an excellent prognosis with infrequent metastases and melanomas thicker than 4 mm having a rather poor prognosis with a 5-year survival of approximately 50%. The most common sites of local and/or regional metastases are the draining lymph node basins and the skin between the primary site and these lymph nodes whereas the most common sites of systemic metastases are the lung, liver, brain, and gastrointestinal tract.

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Common Benign Cutaneous Growths: Seborrheic Keratoses, Cherry Hemangiomas, and Epidermoid Cysts

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Seborrheic Keratoses may be located anywhere, but are most commonly found on the trunk, sides of the neck and the arms. Patients may only have a few lesions, however some have a myriad of these growths. They appear as variably pigmented papules and plaques with sharply defined borders. The surface of a seborrheic keratosis is frequently verrucous and may be crusted, particularly if it has been irritated. Some lesions can attain

a considerable size (over several centimeters in diameter). If a seborrheic keratosis is heavily pigmented, it can simulate a cutaneous melanoma. Eruptive seborrheic keratoses in elderly individuals may be a sign of an associated internal malignancy (sign of Leser-Trélat). Histologically, there is thickening of the epidermis with overlying hyperkeratosis of the stratum corneum.

Cherry hemangiomas are bright red vascular growths that first appear in adolescents and young adults. They slowly increase in size and number over time. The lesions consist of ruby or cherry-red papules, 1 to several mm in size, that are elevated and palpable. Microscopically, these papules contain a benign proliferation of capillaries.

Epidermoid cysts (also called epidermal inclusion cysts) are the most common type of cutaneous cyst. They are primarily found on the face, neck and trunk. These cysts are quite common in adults, particularly those who have had acne vulgaris in the past. These cysts are more commonly found in men and vary in size from less than a centimeter to several centimeters. A pit or punctum can often be seen on the cutaneous surface overlying the cyst. Epidermoid cysts can be a component of genodermatoses such as Gardner syndrome and the basal cell nevus syndrome. Histologically, a cystic space containing laminated keratin is seen. The wall of the cyst is composed of several layers of flattened keratinizing squamous epithelial cells with a granular layer on the luminal surface. The cyst wall may be thin and can rupture if distended or traumatized. If the cyst contents leak into the adjacent dermis inflammation occurs that results in clinically apparent erythema and tenderness.

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Dermatitis, including Atopic, Contact, Seborrheic, and Stasis

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The eczematous dermatitides include a group of diseases which present with a common morphology: erythematous and papulovesicular when acute; erythematous and scaling (with or without fissures and lichenification) when chronic. Included under this rubric are atopic dermatitis, contact dermatitis, nummular dermatitis, seborrheic dermatitis and stasis dermatitis.

Atopic Dermatitis

Atopic dermatitis is a common infantile eczema which affects approximately 10-20% of children in the United States. The disease has a strong association with allergic rhinitis and asthma and occurs in approximately one-third of children with a personal or family

history of these disorders. Although IgE antibodies may be elevated in up to 80% of individuals with atopic dermatitis, the skin manifestations do not seem to be a purely IgE-mediated process. The etiology of atopic dermatitis is unknown. Current hypotheses have concentrated on the possibility that an aberrant T cell response, perhaps to staphylococcal superantigen, results in the activation of T_H2 cells.

Clinically, atopic dermatitis has been called the "itch that rashes". Pruritus is a hallmark of the disease. Infants less than 18 months of age typically present with an acute to subacute dermatitis which may involve the scalp, the face (particularly the cheeks), the posterior neck, the trunk and the extensor aspects of the extremities. After approximately two years of age, most individuals will present with a more chronic, lichenified and scaling form of the disease distributed about the face, neck, trunk and especially the flexural aspects of the extremities (antecubital and popliteal fossae). The reason for the change in distribution of the dermatitis on the extremities is not clear. Individuals with atopic dermatitis are prone to develop secondary infection with staphylococcal organisms, as well as with viruses and fungi. When the disease is flaring, these secondary infections must be excluded. Acute, weeping dermatitis benefits from drying agents whereas chronic lichenified dermatitis requires emollients. Topical glucocorticoids or macrolide immunosuppressants can be helpful in reducing inflammation. Topical antihistamines are best avoided because they may induce a secondary allergy. Phototherapy with ultraviolet B or psoralen plus ultraviolet A (PUVA) can also be beneficial but should be undertaken by trained specialists. Other therapeutic modalities are available for treating more resistant disease but again should be rendered by specialty care.

Contact Dermatitis

Contact dermatitis can be broken down into two main areas: irritant contact dermatitis and allergic contact dermatitis.

Irritant contact dermatitis

is the direct result of injury to the skin caused by chemical exposure. Irritation can be further subdivided into acute corrosion (caused by a single exposure to strong acids and alkalis), acute irritation (caused by a single exposure to chemicals such as strong solvents and non-corrosive acids and bases), cumulative irritation (the most typical and caused by repeated exposures particularly to surfactants and emulsifiers) and phototoxicity (caused by exposure to irritating chemicals which require light for their activation). The clinical presentation of irritation can therefore vary from the acute onset of a third degree chemical burn (corrosion following phenol exposure) to the chronic scaling and xerotic dermatitis of "dishpan hands". Because irritant contact dermatitis is a function of the chemical, it will occur in all individuals exposed to this chemical given sufficient exposure times and concentrations. Nonetheless, it is clear that the skin of some individuals is much more irritable than others. The determinants of hyperirritable skin are numerous and include age, genetics, ambient environment, underlying skin disease(s), and concomitant chemical exposure.

Treatment for irritant contact dermatitis rests upon avoiding the irritants by the use of barriers (gloves or other mechanisms) and/or by using less irritating materials as substitutes. In addition, treatment for corrosion and acute irritation should proceed as that for the appropriate level burn. Treatment for chronic, cumulative irritant contact dermatitis involves avoiding contact with the known irritants and the liberal use of non-sensitizing moisturizers (such as plain petroleum jelly) with or without topical glucocorticosteroids. Phototoxic dermatitis is similarly treated; in addition, broad spectrum, UVA-blocking sunscreens may be beneficial. The pruritus and/or discomfort of irritant dermatitis often require systemic antihistamines and/or non-steroidal anti-inflammatory agents. Topical antihistamines are best avoided for fear of inducing a secondary allergic contact dermatitis.

Allergic contact dermatitis (ACD)

Develops following exposure to chemicals to which the individual has previously become sensitized. It is a type IV or delayed-type hypersensitivity reaction of the skin. There are over 3,000 environmental allergens which have been reported to cause this condition. The prevalence of ACD varies with the allergen. Typically, the patient will develop an erythematous, scaling, papulovesicular dermatitis at the sites of contact with the allergen. Longstanding, low grade allergens can create a more subacute to chronic, scaling lichenified dermatitis. Diagnosis of allergic contact dermatitis is made by patch testing. Treatment is allergen avoidance. The acute disease can be managed with topical glucocorticosteroids, oral antihistamines and other modalities.

Nummular Dermatitis

Nummular dermatitis is characterized by its "coin-shaped" lesions. As with other dermatitides, the acute form is papulovesicular whereas the chronic form is scaly and lichenified. Mild to severe pruritus accompanies the disease which most frequently affects men, typically in the sixth decade or beyond. The prevalence of the disease is unknown, but it would appear to be lower than that for most of the other eczemas. Treatment includes the use of systemic antihistamines and topical emollients. Glucocorticosteroids may also be used as indicated. Ultraviolet B and PUVA phototherapy can be beneficial for resistant disease.

Seborrheic Dermatitis

Seborrheic dermatitis is one of the most common cutaneous diseases and affects from 3 to 5% of the population. One proposed etiology is overgrowth of the *Pityrosporum* yeast that normally inhabits sebaceous skin (e.g., scalp, eyebrows, and central face). The disease has two peaks, one in infancy and the other post-pubertal. Infantile seborrheic dermatitis typically occurs within the first months of life and affects the scalp ("cradle cap") and intertriginous areas with scales and crust. The skin about the ears and the neck may also be involved. In contrast, seborrheic dermatitis in adults typically involves the scalp, face, neck, mid upper chest and intertriginous zones (axillae, groin, submammary,

and in obese patients beneath the pannus). On the face, it particularly concentrates about the eyebrows, nasolabial folds and retroauricular areas. Treatment for seborrheic dermatitis includes keratolytic shampoos and gels, topical antifungals, topical metronidazole and/or topical glucocorticosteroids as indicated. Topical macrolide immunosuppressants can also be very effective for disease involving especially the face and neck, although such use has not yet gained FDA approval. Of note, seborrheic dermatitis can be particularly recalcitrant in individuals who are immunosuppressed (e.g., patients with AIDS) and may require phototherapy when resistant to topical therapies.

Stasis Dermatitis

Stasis dermatitis is an eczematous process of the skin of the lower extremities which results from non-specific inflammation presumably induced by the leakage of serum secondary to venous hypertension. The disease is particularly common over the medial and anterior aspects of the shin and malleolar areas. When significant inflammation occurs, it can be accompanied by a secondary autosensitization dermatitis referred to as an "id". Treatment should be geared towards improving blood return by surgical intervention or by the use of graduated compression support hose. However, it is important that the hose have a proper pressure gradient or the disease can be worsened. In addition, low potency topical glucocorticosteroids and oral antipruritics can be of benefit. Patients with stasis dermatitis seem to have an increased incidence of allergic reactions to ingredients in topical medicaments and, as much as possible, should avoid products containing lanolin, fragrances, neomycin, and other common sensitizers.

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Pruritus and Xerosis

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Pruritus

is the medical term for itching. Itching is an important symptom of many skin diseases, such as psoriasis, atopic dermatitis, and scabies infestation. These conditions can usually be recognized because of the specific features of their clinical appearance, although rubbing and scratching sometimes make diagnosis difficult by obscuring diagnostic features. Pruritus can also be an important and early symptom of certain underlying systemic diseases, such as obstructive liver disease (especially primary biliary cirrhosis), renal failure, hyperthyroidism, Hodgkin's disease, and other lymphomas. In these systemic diseases, the itching can elicit rubbing and scratching which cause a non-specific rash. The cutaneous findings in some early cases of scabies, winter itch from excessively dry skin, and a number of other primarily dermatologic disorders may be very subtle or so altered by rubbing and scratching that their diagnostic features are camouflaged. The schematic diagram in, where the thick arrows represent changes caused by rubbing and scratching, signifies that patients with different causes of itching can end up having similar clinical presentations. It follows that patients with generalized pruritus, with or without a rash, must be carefully evaluated, and that a search for underlying systemic disease must be considered if the diagnosis of a primary dermatologic disorder has not been established.

Itch is generally viewed as a primary sensory modality with certain similarities to pain. Itch is not a form of pain, however. Note, for example, that morphine relieves pain and causes itch. The itch receptor probably resides in free nerve endings in the epidermis and dermis, and the itch sensation is transmitted by slowly-conducting, histamine-responsive unmyelinated C-fibers. These fibers are insensitive to mechanical stimuli. They enter the spinal cord through the dorsal horn and ascend to the thalamus via the lateral spinothalamic tract. Itch is then perceived in the somatosensory cortex with projections to the motor areas, which produce the urge to scratch.

Before itch can be treated it is desirable to make a precise diagnosis of the underlying cause. The underlying skin or systemic disease should then be treated as specifically as possible. Symptomatic treatment of itching is far from satisfactory, as there is no medication even as helpful for itching as aspirin is for pain.

Xerosis

is the medical term for dryness of the skin. This is a common problem in colder climates. When cold, dry air is artificially heated it becomes even dryer, acts almost like a sponge, and "pulls" water from the skin through enhanced surface evaporation. Since water is the main "softener" of the skin, dry skin may become rough, scaly, and eventually red, inflamed, and itchy. In severe cases these changes will have the appearance of dermatitis. The treatment "winter itch" is to 1) increase the relative humidity of the air; 2) decrease factors that may exacerbate the problem, such as excessive bathing and the use of harsh

soaps; and 3) moisturize the skin with emollient creams, lotions, or ointments. When the skin is especially inflamed, a mild topical corticosteroid may also be required.

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Psoriasis

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Psoriasis is an inflammatory disorder of the skin in which activation of T lymphocytes results in release of cytokines that leads to proliferation of keratinocytes. In normal skin, the cells of the epidermis are regenerated every 28 days, while in psoriatic skin epidermis is regenerated every two to four days.

There are four clinical types of psoriasis. The most common type, *plaque psoriasis*, is characterized by sharply demarcated erythematous scaling plaques that most commonly occur on the elbows and knees, and in the scalp and groin. More extensive involvement can occur, with psoriasis affecting most of the cutaneous surface. Nail involvement is common, e.g., yellow discoloration, thickening, surface pitting, and lifting of the nail plate off of the distal nail bed (onycholysis). *Guttate psoriasis* commonly occurs after streptococcal pharyngitis. It is characterized by multiple scaling red papules on the trunk and extremities.

In *erythrodermic psoriasis*, the entire skin surface becomes red, inflamed and scaly. Patients lose many of the protective functions of the skin in this form of psoriasis. They may lose fluids through the skin, resulting in hypotension or electrolyte imbalance. They may lose nutrients, resulting in anemia, or they may lose control of body temperature, resulting in fever or hypothermia. Ultimately, some patients succumb to infection. In *pustular psoriasis*, patients can develop sterile pustules that are either localized to the palms and soles, or are generalized. As in erythrodermic psoriasis, this latter form of psoriasis can be life-threatening. Approximately one out of ten patients with psoriasis develops psoriatic arthritis, most commonly of the small joints of the hands and feet.

Mild or limited psoriasis can be treated with topical therapy, including topical corticosteroids, tars, anthralin, calcipotriene (a vitamin D₃ analog), or tazarotene (a retinoid). In more generalized cases phototherapy with ultraviolet B is effective.

Sun exposure can also be helpful. Treatment with oral psoralens plus ultraviolet A exposure, called PUVA, is effective in most patients, but has been associated with an

increased risk of skin cancers after many treatments over several years. Recently, narrowband UVB has been introduced for the treatment of psoriasis. It uses a narrow portion of the spectrum of ultraviolet B around 311nm, the spectrum which is optimal for the treatment of psoriasis. Narrowband UVB is more effective than traditional broadband UVB, but may be somewhat less effective than PUVA. In the few years since it has been available, it has not been associated with the skin cancer risks seen in patients treated with PUVA.

Oral retinoids, cyclosporine, and methotrexate have also been used for treatment of severe generalized psoriasis, erythrodermic psoriasis, and pustular psoriasis. Most recently, biologic agents have been introduced for the treatment of psoriasis.

Alefacept, which was approved by the USFDA in 2003, works by interfering with T-cell activation and reducing circulating CD45RO+ T-cells. It is a fusion protein consisting of the Fc receptor of human IgG1 and LFA3, a costimulatory ligand, which interacts with CD2 on the surface of T-cells. When it is effective, it offers long remissions. Patients treated with alefacept must have weekly monitoring of CD4 cells during the period of therapy to make sure that CD4 counts don't fall too low.

Efalizumab has been recommended for approval for psoriasis. This humanized antibody to CD11a interferes with T-cell trafficking into inflamed tissues and prevents T-cell activation. It is rapidly effective, but upon discontinuation, some patients experience rebound of their psoriasis.

Three agents that block TNFa have also been used to treat psoriasis. Etanercept, a fusion protein directed against soluble TNFa, has been approved for the treatment of psoriatic arthritis and is also effective for psoriasis of the skin. Infliximab, a chimeric monoclonal antibody against soluble and cell bound TNFa, is dramatically effective against psoriasis. Adlimumab, a human monoclonal antibody to TNFa, has begun trials for psoriasis and initial results are promising.

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Impetigo and Cellulitis

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Impetigo

Impetigo is a superficial infection of the skin caused by either streptococcal or staphylococcal organisms, or, on occasion, both. It is most commonly observed during childhood and in those living in warm, humid climates. Poor personal hygiene may predispose individuals to impetigo. Additionally, impetigo is a common complication of insect bites, scabies, and virus infections. Systemic symptoms are rare, but lymphadenopathy may be present. Nonbullous and bullous impetigo represent the primary clinical subtypes.

Nonbullous impetigo

accounts for the majority of cases of impetigo and begins as subtle vesicles, evolves into superficial pustules and has characteristic overlying honey-colored crusts (**Figure 1**). Sites of predilection include uncovered areas such as the face and less commonly, the extremities, especially following trauma. The lesions may be localized or extensive. *Staphylococcus aureus* and less often group A streptococcus (or the two organisms together) are the predominant etiologic agents in nonbullous impetigo in industrialized nations.

Bullous impetigo

is characterized by localized bullae arising on normal skin (**Figure 2**). The bullae are easily ruptured and form shallow erosions with an adjacent yellow-brown crust. Bullous impetigo is typically caused by phage group II *Staphylococcus aureus*. *Streptococcus pyogenes* is a rare cause of bullous impetigo in the United States and Europe, but may be a common cause in developing countries. The bullous lesion is caused by an exfoliative toxin produced by the bacteria that causes cleavage within the granular cell layer of the epidermis. In children who lack antibodies or in adults with immunodeficiency or renal failure, systemic absorption of this toxin can cause the staphylococcal scalded skin syndrome, a generalized eruption characterized by painful erythema and superficial skin separation. Children, as opposed to adults, usually do not appear seriously ill.

In both forms of impetigo, superficial skin cultures confirm the presence of the responsible bacterial organisms. Neither *S. aureus* nor *S. pyogenes* is part of the normal skin flora. Organisms may be spread from infected to uninfected persons or bacterial

colonization may first occur in the nose and then spread to skin, as *S. aureus* is present in the nares of approximately 20 to 30% of the population. Topical mupirocin is an effective therapy for localized nonbullous impetigo whereas systemic penicillinase-resistant antibiotics are recommended for bullous impetigo and widespread nonbullous impetigo.

Cellulitis

Cellulitis represents an infection of the soft tissue and may be caused by group A beta hemolytic *Streptococcus* or *Staphylococcus aureus*. It is often associated with impaired lymphatic drainage and may be seen in patients who have undergone surgical procedures such as lymph node dissections or saphenous vein harvesting for coronary artery bypass grafts. Local trauma, abrasions and dermatoses such as stasis dermatitis and tinea pedis are other predisposing factors. Cellulitis is a common complication of intravenous drug abuse.

Clinically, cellulitis is characterized by an area of rapidly spreading erythema. The borders are less distinct than in erysipelas (see below) and areas of involvement are warm to touch. Associated symptoms include tenderness, fever, malaise and an elevated white blood cell count. Facial cellulitis in children is most commonly caused by *Haemophilus influenzae*. Usually there is extensive reddish-blue, unilateral swelling involving the cheek or periorbital area. The cutaneous findings often follow an upper respiratory tract infection. However, there has been a significant decrease in the incidence of this form of cellulitis since the introduction of the *H. influenzae* vaccine.

Erysipelas

is a distinctive variant of cellulitis that involves the lymphatic vessels and superficial layers of the skin; it is caused by group A streptococcus and rarely, *S. aureus*. Erysipelas can be distinguished from cellulitis by its sharp margins and its plaque-like elevation of involved skin. The legs and face are the most common sites of involvement and the cutaneous findings are often preceded by fever, chills, nausea, vomiting, headache and arthralgias. Recurrences of both erysipelas and cellulitis are fairly common.

In most patients, the diagnosis of cellulitis or erysipelas is made on the basis of clinical findings. A Gram stain of a tissue aspirate or skin biopsy specimen and blood cultures can sometime provide additional useful information. In immunocompromised hosts, tissue cultures of skin may prove helpful. For adults, treatment with systemic antibiotics that offer coverage for both staphylococcal and streptococcal organisms is recommended. Children between the ages of 6-36 months should be covered for *H. influenzae*.

Necrotizing fasciitis

is an infection of subcutaneous tissue that results in progressive destruction of the fascia and fat. Given the life-threatening nature of this disease, the responsible organisms are commonly known as "flesh-eating bacteria", and typically include group A streptococcus

or a mixture of facultative aerobic and anaerobic bacteria. The extremities are the most common sites of involvement. If this disease is not recognized and treated promptly, it can rapidly progress and result in shock and multi-organ failure. Predisposing factors for necrotizing fasciitis include penetrating injuries, burns, surgical procedures, diabetes mellitus, childbirth, and even minor cuts. The clinical manifestations of this infection include erythema, edema and pain followed by a dusky blue discoloration of the skin or the formation of bullae, which rapidly become hemorrhagic. Fulminant gangrene manifesting as myonecrosis and spread of the disease along fascial planes may then occur, unless there is rapid intervention.

It is often difficult to distinguish necrotizing fasciitis from cellulitis. In its early stages, a frozen section of a biopsy specimen examined for organisms may be useful in making the diagnosis. In addition, a Gram stain and culture of a biopsy specimen may help establish a definitive diagnosis. However, if the diagnosis of necrotizing fasciitis is suspected, these procedures should not delay appropriate treatment and surgical exploration. Preoperatively, magnetic resonance imaging may be useful in determining the depth of involvement. Treatment includes both surgical debridement of necrotic tissue and intravenous antibiotics. Poor prognostic factors include: (1) age greater than fifty years; (2) underlying diabetes mellitus or peripheral vascular disease; (3) delay of diagnosis and surgical debridement by greater than a week; and (4) involvement of the trunk.

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Syphilis

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Syphilis is a disease caused by the spirochete, *Treponema pallidum*. This organism has particular affinity for neural, cardiac, osseous and cutaneous tissue leading to widespread

symptoms. Indeed in the pre-AIDS era, it was said by Sir William Osler "Syphilis simulates all other disease. It is the only disease necessary to know." The disease is most often sexually transmitted, although transfusions and maternal-fetal infections may also occur. It cannot be transmitted via toilet seats, doorknobs, swimming pools or hot tubs.

Clinical

The organism causes three distinct phases: primary, secondary, then a period of latency followed by a tertiary phase. Primary disease follows an incubation period of 10 to 90 days from the time of inoculation. The clinical hallmark of this phase is one to several round 1-2cm, punched-out ulcers or chancres with exudative bases. A chancre is characteristically pain-free, unless secondarily infected. These ulcers are most often seen on mucous membranes, but can also occur on keratinized skin when infection is acquired through a prior skin abrasion or bite. The lesion(s) resolve spontaneously.

Secondary syphilis occurs 6 to 12 weeks after the primary infection and presents as widespread red-brown, round to oval macules and papules; the palms and soles are frequently involved. Erosive patches may be seen in the mouth. On the vulva and around the anus, moist, pink nodules and plaques known as condylomata lata can be found; these are teeming with spirochetes. A patchy alopecia with a "moth-eaten" appearance may also be present. Secondary lesions resolve spontaneously within 4-12 weeks. All primary and secondary syphilis lesions are contagious. In immunocompromised patients, secondary syphilis may also be associated with neurologic symptoms and the organism can be isolated from the cerebrospinal fluid.

The secondary phase is followed by a period of latency in which there are no clinical signs or symptoms of syphilis and the diagnosis is made by serological tests alone. The duration of latency is variable. In normal hosts, it may extend for life, but in HIV-infected and other immunocompromised patients, it may be brief. Only 15-40% of patients with untreated syphilis go on to tertiary disease. The tertiary phase is a multisystem disease which affects cardiac, neural and osseous tissue as well as the skin. Characteristic sequelae include ascending aortic aneurysms and a myriad of CNS abnormalities. Tertiary skin lesions consist of thickened, red-brown granulomatous plaques that can erode into underlying cartilaginous and osseous tissue creating significant deformities. The skin lesions of tertiary syphilis are less infectious than those of primary and secondary syphilis. If left untreated, tertiary syphilis can be a fatal disease.

Diagnostic Testing

A darkfield microscopic examination is the gold standard; the specimen consists of serous fluid obtained by squeezing the lesion gently. Unfortunately, this test is not always available in the outpatient setting. A presumptive diagnosis of syphilis can be made by obtaining a positive VDRL (or RPR) titer. However, in early primary disease, this test may not yet be positive, i.e., it is falsely negative. (False-positive VDRL's can also be seen, particularly in patients who have systemic lupus, other bacterial infections, or are pregnant.) The more specific treponemal FTA assay is often needed for confirmation.

Once infected with syphilis, the FTA remains positive indefinitely in at least 85% of patients. This does not, however, confer immunity to the patient who can become re-infected if exposed to syphilis.

After successful therapy, the VDRL titer should return gradually to zero (non-reactive), with the titer falling four-fold every three months in the normal host. Rarely, HIV-infected patients acquire an infection without becoming VDRL-positive; in contrast, HIV infected patients with a positive VDRL may also remain VDRL-positive after clinical resolution of the disease.

Differential Diagnosis

The differential diagnosis of chancres includes herpes simplex (grouped vesicles which rapidly become eroded), chancroid (tender mucous membrane ulcers), and trauma. The differential diagnosis of secondary syphilis includes pityriasis rosea and viral exanthems.

Treatment

The optimal therapy of syphilis can change rapidly with the introduction of new antimicrobials, and one must keep abreast of new developments on at least a yearly basis. At the present time, the recommended antibiotics are penicillin, doxycycline, tetracycline and erythromycin. Patients with sexually-acquired syphilis have, by definition, experienced unprotected intercourse and should be tested for other sexually transmitted diseases such as gonorrhea and chlamydial infections. Testing for infection with HIV may also be warranted.

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Viral Infections - Verrucae, Herpes Simplex, and Herpes Zoster **David A. Wrone M.D. and George T. Reizner M.D.**

Verrucae

The human papilloma virus (HPV) causes verruca, more commonly referred to as warts. Warts are rare in infants, but the incidence rises during school years. About 10% of children between the ages of 2 and 12 have warts. In general there is an incidence peak between the ages of 12-16. Condylomata acuminata (genital and peri-anal warts), however, are more common in an older population with peak incidence between the ages

of 25 and 45. *Verrucae vulgares* (common warts) are hyperkeratotic dome shaped papules or nodules located most commonly on the hands. Frequently, dark pinpoint spots can be seen representing clotted off capillaries near the surface. HPV types 1, 2 and 4 are most commonly implicated. *Verruca plana* (warts on the bottom of feet) can cause great discomfort. These warts tend to be endophytic with thick callous surrounding a central depression. When several warts coalesce, they are called mosaic warts. HPV types 2, 27 and 57 have been found most consistently in these lesions. Anogenital warts, *condylomata acuminata*, are discrete, sessile, smooth surfaced papillomas that can be flesh colored, brown or whitish. They are most commonly found on the external genitalia, perineum or peri-anal areas. HPV types 6 and 11 are the most commonly implicated, but types 16, 18 and 31 are seen associated with malignancy.

Warts are transmitted by contact. The virus infects the basal cells of the epidermis, thus a breach in the skin barrier is an important predisposing factor. Host immune function is extremely important in the infectious process as well. Once an immunocompetent patient has been infected with a specific HPV type, it is very unlikely that he/she will experience infection with that same type in the future. Patients who are immunosuppressed for any reason, however, can present with numerous, treatment resistant warts as well as recurrent infections with the same HPV type.

Treatment of verrucae is challenging. Common warts and plantar warts may regress spontaneously in 1-2 years. Therapeutic options include cryotherapy with liquid nitrogen, electrodesiccation, laser ablation, intralesional bleomycin, topical cantharone, salicylic acid or imiquimod, or sensitization with a compound such as candida antigen or. Topical irritants like tretinoin gel may be effective on flat warts. No treatment works all the time in all patients, and some treatments may leave scars that are more painful or disfiguring than the original wart. In children especially, conservative treatment such as occlusion with tape followed by gentle debridement with pumus stone can be tried. Treatment of *condylomata acuminata* must be done carefully. Because one cannot clinically differentiate among the various HPV types, patients with anogenital warts need to be followed carefully due to the risk of squamous cell carcinomatous change.

Herpes Simplex

Infections with herpes simplex viruses (HSV-1 and HSV-2) are common. The oral and genital mucocutaneous surfaces are the primary sites of infection. HSV-1 is most commonly associated with oral-facial lesions and HSV-2 is associated with genital lesions. This distinction is becoming more obscure as more and more genital HSV-1 infections are seen. Currently somewhere between 10-30% of genital lesions are attributed to HSV-1.

Most people (80%) with oral-facial HSV infections have a subclinical primary episode. Subsequent to primary infection, the patient can become a carrier for an indefinite period of time. Some patients will experience a primary gingivostomatitis which is characterized by 3-5 days of fever, malaise, tender lymphadenopathy and extensive erosions of the oral mucosa including tongue and palate. Complete recovery may take up to two weeks. It is

estimated that 90% of the world population between 20 and 40 years of age carry antibodies to HSV-1.

Recurrence of oral-facial HSV infections, herpes labialis, is frequently precipitated by some sort of stress. Fever, sun exposure, menstruation are commonly implicated. Burning and itching often precede the development of grouped vesicles. The vermilion border of the lip is the most common location, but lesions can occur on the skin around the mouth or even in the nasal mucosa. The progression from vesicle to crust usually takes 7 to 10 days. Vesicle fluid is infectious and care should be taken with exposure until the lesions are well crusted over.

Primary genital HSV infections tend to be more severe with excruciating painful erosive balanitis, vulvitis or vaginitis. The buttocks and perineum may also be involved. There may be painful inguinal lymphadenopathy or dysuria. Women tend to have more systemic complications. Twenty percent experience extragenital lesions, 10-15% have dysuria and 10% suffer from aseptic meningitis. Recurrent infections are generally less severe or even sub clinical.

HSV infections can be seen outside of the oral-facial or genital regions. In patients with atopic dermatitis, herpetic infections may be widespread (eczema herpeticum). Herpetic whitlow is infection involving the finger. Herpes Gladiatorum is not uncommon in wrestlers and is most commonly seen on the shoulders, upper trunk or upper back. Herpetic infections of the eyes can lead to keratoconjunctivitis (superficial ulcerations and opacities).

Treatment in immunocompetent patients is often symptomatic. If lesions are caught within 72 hrs, oral antiviral therapy may be instituted (acyclovir, famciclovir, valcyclovir). Oral therapy will reduce pain, viral shedding and time to healing. Suppressive therapy may be indicated for patients having > 6 recurrences per year. In healthy patients, the infection is self limited even without treatment. Immunosuppressed patients, however, tend to have chronic disease and require anti-viral therapy.

Herpes Zoster

Herpes zoster (shingles) is the result of reactivation of latent varicella zoster virus (VZV) infection. Primary infection often occurs in childhood in the form of varicella (chickenpox). After the patient recovers, the virus enters latency in the dorsal root ganglion and/or the trigeminal ganglion. Later, when the patient's immunity is suppressed or stressed, the virus reappears in the form of zoster. Zoster affects 20% of healthy adults and 50% of immunocompromised patients. Over 60% of patients are greater than 45 years, and zoster is rarely seen in children. The likelihood of zoster at younger ages is increased if the primary infection occurred before 1 year.

The heralding symptom of herpes zoster is pain/tenderness in a dermatomal distribution. Within 3-4 days, clusters of erythematous papules typically develop within the same dermatomal distribution. These papules rapidly progress to vesicles and pustules. New

groups of lesions continue to reappear over several days. Crusting eventually occurs and patients experience desquamation as the lesions heal. The process usually lasts between 2-4 weeks. In rare cases, the prodrome is not followed by rash. This is termed "zoster sine herpete". Disseminated zoster is defined by more than 20 vesicles outside of the involved dermatome(s) and may be associated with visceral disease. When the lesions involve several non-adjacent dermatomes and cross the midline, it is termed generalized zoster. Zoster in immunocompromised patients can have a variety of atypical presentations and a very high index of suspicion must be maintained when evaluating these patients.

Complications include post herpetic neuralgia (PHN), scarring, secondary bacterial infection, ophthalmic zoster, Ramsay Hunt syndrome, meningoencephalitis, motor paralysis, pneumonitis and hepatitis. PHN occurs in 10-15% and can last for years. It is characterized by shooting or burning pain in the previously involved sites. It is more common in older patients and is more frequent when the trigeminal nerve is involved. Ocular involvement occurs in 7% and carries the risk of severe sequelae including blindness. Ramsay Hunt syndrome is characterized by involvement of the 8th cranial nerve ganglia and the geniculate ganglion of the facial nerve. It consists of severe ear pain, hearing loss, vertigo, facial nerve paralysis. A good exam of the auditory canal and tympanic membrane is essential in patients with periauricular lesions.

If identified within 72hrs, oral anti-viral therapy (acyclovir, famciclovir or valcyclovir) can speed healing and decrease pain. Intravenous treatment is indicated for immunocompromised patients and those with severe involvement. Ocular zoster should be managed by an ophthalmologist. Corticosteroids may be added in Ramsay Hunt. Post herpetic neuralgia can be difficult to manage. Analgesics, EMLA, lidocaine patches, capsaicin, narcotics, nerve blocks, biofeedback, gabapentin and tricyclic antidepressants may be helpful.

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Pityriasis Versicolor and Candidiasis

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Pityriasis (tinea) versicolor

Pityriasis, or tinea, versicolor is a very common superficial fungal disease of the skin caused by the genus of lipophilic yeast known as *Malassezia*. Though commonly referred to as tinea versicolor, pityriasis is a fitting descriptive term used to denote any dermatosis

characterized by branny desquamation. Versicolor refers appropriately to the varied hues of the lesions clinically. Seven species of the genus *Malassezia* are human pathogens, with *M. furfur* and *M. globosa* most commonly indicted as causative agents in pityriasis versicolor. Frequently the older names *Pityrosporum ovale* and *P. orbiculare* are encountered in the literature for the now correct *Malassezia furfur*. In addition to tinea versicolor, *Malassezia* species may cause folliculitis, inverse tinea versicolor, and rarely systemic disease in pediatric patients on parenteral lipid supplementation.

Pityriasis versicolor occurs in either sex, most commonly in post-pubertal age groups. Found in all geographic regions, cases are more common in tropical climates and subtropical areas with high temperatures and increased humidity. Cases found in these regions tend to be more severe. Other factors that contribute to the development of pityriasis versicolor include malnutrition, Cushing's disease, immunosuppression, oral contraceptives and heredity. The lesions of pityriasis versicolor are found on the "seborrheic areas" of the body, most notably the trunk. This is consistent with *Malassezia*'s opportunistic nature and lipid requirement for growth. Lesions exhibit a slightly branny or furfuraceous scale and may be flat, nummular or even confluent. Lesion shades vary from white to red brown. Lesions may be pruritic and erythematous. In addition to the trunk, other body areas affected may include the proximal extremities and face (more common in children). In extreme cases, even the distal extremities may have some involvement. *Malassezia* folliculitis usually presents as pruritic pustules of the trunk.

Malassezia may be found on normal unaffected skin, but biopsy of lesions demonstrates the hyphal forms of the yeast invading the stratum corneum. The yeast may also be visualized within the follicle admixed with debris in *Malassezia* folliculitis. Clinical suspicion can be readily confirmed by microscopically observing these hyphae and budding cells ("spaghetti and meatballs") in KOH (10% potassium hydroxide) preparations of scales scraped from lesions. In a similar fashion, scale may be lifted from lesions with cellophane tape, prepped with methylene blue and examined microscopically for *Malassezia*. Culture is rarely needed, but if performed requires the addition of a lipid source, such as olive oil, to the growth medium.

There are a number of treatments available for pityriasis versicolor, both topically and orally. These treatments have various mechanisms, including fungistatic, fungicidal and nonspecific destruction of the stratum corneum. Fortunately, most of these are very effective; however, there can be a tendency for the disease to recur. Zinc pyrithione shampoo, applied for five to ten minutes and then rinsed off every evening for one to two weeks has therapeutic and prophylactic effects. Topical selenium sulfide 2 ½ % shampoo is an effective treatment when applied for 15 minutes, and washed off well, every day for three days; or for one or two overnight applications. Topical azole, triazole, allylamine, imidazole, benzylamine and hydroxypyridone antifungals have shown efficacy when applied twice daily for 2-3 weeks. Multiple vehicles may be used with preparations like Nizoral shampoo providing significant ease of application. Specific topicals such as ketoconazole and bifonazole may be effective as a single dose. For extensive or recurrent lesions and with patients whom compliance is an issue, oral therapy is sometimes used.

Ketoconazole has been used at a 200 mg dose daily for 5-7 days, or 400 mg once weekly for two weeks. Itraconazole has been very effective at a dose of 200 mg daily for five to seven days. Also single doses of fluconazole 400 mg have been effective. Neither terbinafine nor griseofulvin has been effective when used orally for pityriasis versicolor, although topical terbinafine twice daily for 1 to 2 weeks is effective. To prevent relapse of pityriasis versicolor, particularly in the warmer months, a zinc pyrithione soap lather may be applied to the body at least two or three times weekly.

Candidiasis

Candidiasis is an infection of the skin, mucous membranes, and occasionally internal organs caused by yeast of the genus *Candida*. Most frequently these infections are due to *Candida albicans* but other species with increasing frequency cause human disease. *Candida* species are normal inhabitants of the gastrointestinal tract, but rarely colonize the skin unless there is some break in the integument, for example from dermatitis, cracks, or fissures. *Candida* species are dimorphic fungi that occur as budding yeast or blastoconidia phase, and a mycelial or pseudomycelial phase. Usually the mycelial phase is the form causing superficial disease, while the yeast or blastoconidia is the colonizing or hematogenously disseminating form.

Candida infections of the skin and mucous membranes results from an interplay between a variety of *Candida* virulence factors (for example, hyphae formation, contact sensing, and lytic enzymes), and a variety of host defense mechanisms (including epidermal proliferation, T-cell immunity, phagocytosis, and immunoglobulins). Any factor that adversely affects normal immune function may predispose a person to candidiasis. Examples include: genetic susceptibilities, such as Down's syndrome and chronic mucocutaneous candidiasis; endocrine disorders such as diabetes; malignancies; immunodeficiency states; debility due to chronic or advanced disease; and prolonged use of antibiotics, corticosteroids, or immunosuppressive agents. Heat, humidity, and friction between skin surfaces are environmental factors that may also contribute to infection.

Candidiasis may display a wide clinical spectrum of disease with varied patterns of infection of the skin, mucosa and internal organs. Infections solely limited to the skin and mucous membranes are referred to as superficial candidiasis. The usual sites of the infection include the skin folds; the perioral, vulvovaginal, and anal mucocutaneous junctions; and the nail unit. In immunocompromised or debilitated patients, candidiasis may become systemic and disseminated. This life threatening infection commonly occurs in neutropenic patients via extension from a colonized gastrointestinal tract, aspiration, or by invasion through an impaired mucosa or integument.

The most common cutaneous pattern of *Candida* infection is candidal intertrigo. The genitocrural and gluteal folds, the submammary region, and the interdigital spaces of the hands and feet are usually affected. These often pruritic lesions may begin as vesicles, pustules or erythematous plaques, and eventually lead to maceration and fissuring, leaving behind a denuded, red base. Often the central lesion is bordered by a number of discrete pustules in a "satellite pattern".

Cutaneous candidal infection in the diaper area of infants is one cause of so-called diaper dermatitis. The occlusive nature of the diaper appears to create an environment ripe for candidal colonization. Erythematous or pustular lesions radiate out from the perianal area into the gluteal folds, and subsequently spread to the rest of the perineum, genitalia, buttocks, and thighs. This is not to be confused with congenital cutaneous candidiasis, where a papular or vesiculopustular eruption occurs over the face, neck, trunk, limbs, as well as the palms and soles. Congenital cutaneous candidiasis is most often noted at birth or within twelve hours after delivery, and is presumably caused by ascending infection of the skin by *Candida albicans* through the birth canal. Most of these infants do not demonstrate signs of systemic infection, but the disorder has been fatal in infants with low birth weight or preterm delivery.

Candidal infection of the nail unit may occur, usually caused by *Candida albicans* or *C. parapsilosis*. *Candida* paronychia is usually the result of chronic water exposure and trauma. The proximal or lateral nail folds may be affected, with erythema, edema, scaling, and occasionally a purulent discharge. A resultant onychodystrophy may occur with a greenish-yellow discoloration under the nail. This is usually attributed to secondary invasion by *Pseudomonas aeruginosa*. Actual invasion of the nail plate is rare, but in the inherited disorder chronic mucocutaneous candidiasis nail plate invasion does occur. In this disorder, patients have chronic and recurrent *Candida albicans* infections of the skin, nails, and mucous membranes associated with abnormalities of cell-mediated immunity. Chronic mucocutaneous candidiasis may be associated with other disorders such as autoimmune diseases and endocrinopathies.

The most common oral form of candidiasis is thrush or pseudomembranous candidiasis. In this infection, removable white plaques occur on the mucosal surfaces of the mouth. Characteristically, these lesions may be removed by scraping, yielding an erythematous red base. Several other forms of oral candidiasis have been described and may be associated with thrush. Perleche, or angular cheilitis, may present with burning, tenderness and erythema, cracking, fissuring or maceration of the oral commissures. An erythematous, edematous candidal infection of the palate associated with denture colonization has been described, especially with ill-fitting dentures worn for long periods of time. Erythematous candidiasis may present as atrophic, painful, red patches of the tongue or lips, commonly associated with antibiotic administration.

Another common form of mucosal candidiasis is *Candida* vulvovaginitis and balanitis. In the former, vaginal mucous membranes are inflamed, with associated pruritus, erythema, and a creamy white discharge. *Candida* balanitis usually occurs in uncircumcised males and presents as erythema and pustules, with exudate, on the glans penis or prepuce. Both of these infections may later spread to the perineal areas as intertrigo.

In all of the aforementioned candidal infections, KOH microscopy of scrapings from lesions will usually reveal yeast and hyphae consistent with candidiasis. Material may be sent on Sabouraud's dextrose agar for confirmatory fungal culture.

Treatment involves use of the appropriate topical and/or oral antifungal agent but importantly must address host and environmental factors as well. Heat, humidity and tight fitting clothing should be avoided, and moist or occluded areas must be "dried out". This is especially important in intertrigo, paronychia associated with wet work, and diaper dermatitis where frequent diaper changes and proper skin hygiene are essential. All underlying diseases, such as diabetes, should be identified and controlled. Numerous effective topical agents are currently available. One of the most commonly used agents is nystatin, which is available in a number of vehicles for treating oral and cutaneous candidiasis. The azole family of topical antifungals (e.g. ketoconazole, clotrimazole, econazole, or oxiconazole) is also effective. Ciclopirox, terbinafine, naftifine, Castellani's paint and thymol may also be used. The topical antifungals are usually rubbed in once or twice daily for approximately two weeks, and for a week after the signs of the infection have apparently cleared. In significant cutaneous or mucous membrane candidal infections, the oral azole agents such as ketoconazole, fluconazole, or itraconazole are very effective. A variety of oral treatment regimens are available, usually involving daily therapy for a week or two. There are certain infections, for example vaginal candidiasis, for which single dose oral therapy with fluconazole has been advocated. Disseminated life threatening candidiasis requires treatment with systemic antifungals. Amphotericin B, with or without flucytosine, historically has been first line therapy, but fluconazole, and the newer agents (voriconazole and caspofungin) can be used.

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Tinea (Dermatophyte) Infections

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Superficial fungal infections caused by a dermatophyte (species of fungi belonging to the genera *Trichophyton*, *Microsporum*, or *Epidermophyton*) are referred to as "tinea." Dermatophytes digest and invade keratin and may infect skin, nails, and hair. Infections may be acquired through human-to-human (anthropophilic) spread, animal-to-human (zoophilic) spread, or soil-to-human (geophilic) spread. The latter two categories tend to produce a more severe cutaneous inflammatory response in immunocompetent hosts. Infections of scalp hair and general body surfaces are most frequent during childhood; hand, foot, or nail infections are much more common after puberty. Dermatophyte infections are more common in association with hot, humid environments, sweating or maceration of the skin, occlusive footwear, use of hair greases or oils, diabetes mellitus, and defects of cellular immunity (e.g., AIDS).

The various clinical presentations of dermatophyte infections are designated by use of the term tinea followed by the Latin term for the affected body area. Dermatophyte infections of scalp hairs and the skin of the scalp (**tinea capitis**) have a peak incidence in the first decade of life. In the U.S., tinea capitis most commonly results from the anthropophilic spread of *Trichophyton tonsurans*. There is currently an epidemic of *T. tonsurans* tinea capitis, with African-American children being at highest risk for acquiring the infection (this may be related to the practice of using hair grease and oils). Less common forms of tinea capitis may be acquired from pets or even soil.

The clinical features of tinea capitis can vary and may include scaling, localized alopecia, and follicular papules and pustules. Fragile broken hairs are characteristic of tinea capitis. Hairs broken off close to the skin surface result in a "black dot" appearance; when the hairs are broken off several millimeters above the scalp a "gray patch" alopecia is seen. Associated inflammation ranges from minimal to marked, with the latter including formation of tender, erythematous, boggy raised scalp nodules with pustules, crusting and extensive alopecia (known as **kerion**). Kerion formation may be accompanied by occipital and cervical adenopathy, low-grade fevers, and leukocytosis. The resulting alopecia may be scarring, and thus permanent.

Dermatophyte infections of general body surfaces (excluding the face, hands, feet, and groin) are referred to as **tinea corporis**. The latter begins as one or more scaly papules that enlarge or coalesce into plaques; the central portion of these plaques tends to clear, producing annular lesions (this typical configuration gives rise to the term "ringworm"). Infection of hair follicles occasionally occurs (known as **Majocchi's granuloma**) and may serve as a reservoir for recurrent disease. Dermatophyte infections of the groin or perineal area (**tinea cruris**) present as thin erythematous plaques with well-defined scaly borders. The most common location for tinea cruris is the medial thighs, but the infection may extend to the buttocks and gluteal cleft. It generally spares the penis and scrotum. This eruption must be differentiated from cutaneous candidiasis, erythrasma (caused by *Corynebacterium*), seborrheic dermatitis, and psoriasis.

Dermatophyte infection of the feet (**tinea pedis**) is the most common form of fungal infection. Tinea pedis can present in several patterns: (1) an interdigital type (macrated, scaly plaques in toe web spaces); (2) a "moccasin" type with dryness, scaling and erythema of the plantar and/or lateral foot; and (3) a vesicular type characterized by vesicles, pustules, or bullae on the feet. Dermatophyte infection of the hands (**tinea manuum**) presents as dryness, scaling and erythema involving the palm and occasionally the dorsum of the hand. "One hand, two foot disease" refers to a common clinical presentation of tinea involving one hand and both feet.

Tinea unguium (onychomycosis)

T. unguium is a dermatophyte infection of the nail plate and is often found in association with tinea pedis. Toenails (especially the great toenail) are affected more frequently than fingernails. Patterns of tinea unguium include the distal subungual form (most common), the proximal white subungual form (which may be a sign of HIV disease) and the white superficial form. The clinical findings of distal subungual tinea unguium include yellow discoloration, thickening, subungual debris and loss of attachment of the nail plate to the nail bed. An immunologic response to any one of these dermatophyte infections may result in a dermatophytid or "id" reaction, which often presents as a vesicular eruption on acral surfaces (especially the palms).

The diagnosis of a dermatophyte infection can be easily confirmed by obtaining material from an actively infected site (i.e., skin, hair, or nail), dissolving it in a potassium hydroxide (KOH) solution, and examining the material under a microscope. A KOH preparation that is positive for the presence of dermatophytes will demonstrate septate hyphae that branch at various angles in skin and nail specimens; in infected hairs, spores are generally present either on the hair surface or within the hair shaft. The presence of hyphae in hair is unusual. Although the KOH preparation confirms the presence or absence of a dermatophyte, it does not allow for identification of the responsible species. If this is necessary, infected material must be inoculated on a fungal culture medium such as Sabouraud's or Mycosel agar. A two- to four-week period is usually required for identification of the dermatophyte species.

In circumstances where a KOH prep and fungal culture are negative, but a dermatophyte infection remains a strong consideration on the basis of clinical findings, it may be appropriate to biopsy the affected site. The biopsy material can be stained with periodic acid-Schiff (PAS), which reveals the presence of fungal elements by staining them red. The Wood's lamp is an ultraviolet light source which is used to detect the coral red fluorescence of a skin infection called erythrasma, which is produced by the organism corynebacterium. This fluorescence helps differentiate erythrasma from tinea cruris. Wood's lamp illumination of some cases of *Microsporum*-induced tinea capitis also produces fluorescence, but the color is blue-green. However, the vast majority of tinea capitis is caused by *T. tonsurans*, and the infected hairs of these patients do not fluoresce under the Wood's light.

Treatment of dermatophyte infections generally requires patient education as to avoidance of factors which predispose to infection as well as the use of an antifungal

agent in either a topical or systemic form. Patients should be advised to avoid exposure to hot, humid conditions and situations which may result in excessive perspiration. Loose clothing, non-occlusive footwear (e.g., sandals), and the use of absorbent powders in intertriginous areas should be recommended. In order to avoid person-to-person spread, especially in the case of tinea capitis, direct contact with infected areas or use of potentially contaminated objects (e.g., caps, combs) should be avoided.

Topical antifungal agents are generally effective treatment for tinea pedis, tinea manuum, tinea cruris and limited tinea corporis. The majority of these agents are imidazoles or allylamines. They should be applied once or twice daily to affected areas and continue to be used for several weeks after the eruption has clinically resolved. For individuals with frequent recurrences, it may be appropriate to continue indefinite use of these agents on a prophylactic basis (e.g., application twice weekly.).

Tinea capitis and tinea unguium require systemic antifungals; widespread tinea corporis is usually also treated systemically. Griseofulvin remains the treatment of choice for tinea capitis. A dosage of 15-20 mg/kg/day is prescribed for a minimum 8-week course. Headache and nausea are the most common side effects. Hepatotoxicity is an uncommon side effect, and many practitioners do not routinely check liver function tests in otherwise healthy patients receiving a routine course of the medication. Terbinafine, itraconazole and fluconazole are alternatives for treatment of tinea capitis. As an adjunct to systemic therapy, tinea capitis patients and their household contacts should be advised to use an antifungal shampoo such as selenium sulfide or ketoconazole.

For treatment of tinea unguium, several newer antifungal agents are available and currently being used. Itraconazole, a triazole agent, can be used in either a "pulse" dosage (i.e., patient receives medication daily for one week, followed by three weeks off medication) or continuous (daily) dosage for the treatment of tinea unguium. Treatment duration is generally 2-3 months for fingernails and 3-4 months for toenails. Caution must be exercised in prescribing itraconazole in combination with certain medications metabolized by the cytochrome p-450 system (e.g., astemizole, midazolam, cisapride, HMG-CoA reductase inhibitors). Itraconazole can rarely cause hepatotoxicity, and thus many practitioners check baseline liver function tests and then recheck these periodically during treatment. Terbinafine, an allylamine, is prescribed in a continuous (daily) fashion for the treatment of fingernail (generally a 6-week course) and toenail (generally a 12-week course) dermatophyte infections. This medication does not interfere with the cytochrome p-450 system and thus avoids the drug interactions noted above. Rarely, hepatotoxicity and leukopenia have been associated with terbinafine use; thus, it may be prudent to check a baseline CBC and liver function tests and follow these periodically during treatment.

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Parasitic Infestations

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Scabies

Scabies is an ectoparasitic infection caused by the eight-legged mite *Sarcoptes scabiei*, var. *hominis*. The mites are spread by close human contact. In contrast to lice, which can be seen with the naked eye walking on the hair or skin, scabies mites are only visible with a microscope upon examination of a scraping from an unexcoriated lesion prepared with mineral oil or potassium hydroxide. The mites burrow no more deeply than the base of the stratum corneum. The itching is usually more impressive than the rash and results from a hypersensitivity to the organism. Skin lesions are widely scattered macules, papules, or pustules, with a predilection for the finger webs, axillae, breasts, and genitals. Burrows can sometimes be identified with the naked eye, or enhanced by the application of ink to the skin.

Nodular lesions of scabies are most common on the genitalia. These lesions are more impressive because of epithelial hyperplasia and inflammation from chronic scratching and reaction to the mites, which are usually no longer demonstrable in these types of lesions. Crusted scabies ("Norwegian" scabies) is a more extensive thick crusted or hyperkeratotic infestation by numerous mites, and is often not pruritic. Crusted scabies is most commonly seen in immunocompromised (such as HIV) patients, neurologically impaired individuals (such as those with Down syndrome), and in nursing homes.

Scabies is both over diagnosed and under diagnosed. Some physicians make the mistake of designating and treating any itchy rash as scabies. It is best to try to confirm the diagnosis by a microscopic examination of a skin scraping. Since scabies can resemble many other rashes, it can also be easily misdiagnosed as psoriasis, eczema, folliculitis, or urticaria.

Scabies mites die within 48 hours after removal from the body. They are therefore not typically found in clothing. Washing clothing and bedding in hot water is often recommended, but avoiding contact with such potentially contaminated fomites for 48 hours would suffice. It is important to treat all close human contacts to prevent "ping-pong" reinfections. Other varieties of the mite *Sarcoptes scabiei* are found in animals,

including dogs. Animal scabies mites may cause a rash or itching in humans, but these animal mites are not thought to infest humans.

The most common treatment for scabies is topical permethrin. It is important to apply the treatment to the entire body, at least from the neck to the toes. Topical lindane (gamma-benzene hexachloride) may cause neurotoxicity such as seizures, especially in infants, so it is less commonly used nowadays. Other treatments include topical benzyl benzoate, crotamiton, or precipitated sulfur. Oral ivermectin is very effective, but because of rare deaths in elderly patients, it is often reserved for refractory or extensive infections, such as crusted scabies.

Head and body lice

Head lice (*Pediculus humanus*, var. *capitis*) are six-legged insects that infest the scalp hair, and they are large enough to be seen with the naked eye. Children are affected more often than adults, and are often diagnosed by the school nurse. Most patients have itching or no symptoms, with no skin lesions. Eggs ("nits") are glued to hairs at an oblique angle. Sometimes microscopic examination is needed to distinguish real eggs from hair casts, scales and other "pseudonits." Like scabies, head lice are commonly overdiagnosed. Sometimes in the case of school epidemics, children with dandruff are mislabeled as having nits.

Body lice (*Pediculus humanus*, var. *corporis*) are similar morphologically to head lice. They infest individuals with poor hygiene or those living in crowded conditions. Body lice are more likely to be found in seams of clothing rather than on the body. Eggs are also more likely to be found on the clothing than cemented to hairs.

The treatment of head and body lice is similar. Over-the-counter treatment with topical pyrethrin with piperonyl butoxide is usually effective. Other treatments include topical sulfur, lindane, permethrin, or crotamiton. It is useful to remove nits using a special comb. Sanitizing the clothing and bedding is especially important in the treatment of body lice. Close contacts should be examined, especially children, who are more susceptible to head louse infestation. There is concern with the increasing resistance of head lice to standard treatment, whereas this problem has not generally been observed with scabies. Topical malathion, although more toxic, has been advocated for resistant cases.

Crab lice

Crab lice (*Phthirus pubis*) are shorter and rounder than head or body lice. They prefer the thicker hairs of the pubic area and eyelashes. Crab lice are usually sexually transmitted, and it may be worthwhile to examine and test patients for other sexually transmitted diseases. The nits are similar to those of head lice. Treatment is also the same. Eyelash infestations may be treated with petrolatum and/or mechanical removal.

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Urticaria and Drug Eruptions

Lisa A. Osberg, M.D., and Lee T. Nesbitt, Jr., M.D.

Urticaria

Urticaria, also popularly called "hives", represents a common vascular reaction pattern of the skin which affects as many as 15 to 20% of the population at some time during their lives. The lesions are erythematous to pale edematous papules and plaques that are often annular. Lesions may become confluent to form unusual serpiginous or polycyclic patterns. Pruritus is a constant feature but varies in intensity among affected persons. Individual urticarial lesions are transient, usually lasting less than twenty-four hours, representing the single most important diagnostic criterion. One classification of the disorder is based on its duration, with episodes persisting less than six weeks arbitrarily termed acute urticaria, and those persisting longer termed chronic urticaria. Lesions represent transudation of fluid from small cutaneous blood vessels into the dermis; they can result from either IgE-mediated hypersensitivity to an antigen with release of histamine and other mediators from mast cells and basophils, or from other immunologic or non-immunologic mechanisms.

Foods, drugs, insect bites, or acute infections are usually found to be the cause of acute urticaria. Any drug may be responsible, but penicillins are the most common class of drugs that induce IgE-mediated acute urticaria. Foods that may be implicated include nuts, shellfish, eggs, milk, chocolate, wheat, tomatoes and berries. Foods, drugs, and chronic infections are occasionally implicated as causes of chronic urticaria, but in the large majority of cases no definite etiology can be identified. Special forms of urticaria exist which are precipitated by physical stimuli such as brisk stroking of the skin (dermographism), exercise, pressure, cold, heat, vibration, sunlight exposure and contact with water. The mainstays of treatment for most types of urticaria are oral antihistamines.

Treatment also consists of the elimination of known causative factors when identified and avoidance of histamine-releasing agents such as aspirin and non-steroidal anti-inflammatory drugs. Systemic corticosteroids and epinephrine are sometimes utilized in severe urticaria and angioedema (edema of subcutaneous tissues), especially in the setting of anaphylaxis.

Drug eruptions

Drug eruptions are extremely diverse, with almost any drug sometimes capable of producing a specific type of reaction. A particular drug may also cause several different reaction patterns in different patients. Although most drug eruptions occur within days to weeks after beginning the offending agent, occasionally a reaction begins after long-term use of a particular drug. Certain types of drug reactions have immunologic mechanisms, but non-immunologic mechanisms are also involved, sometimes being dose-related. Exanthem-like (macular and papular) eruptions are the most frequent of all cutaneous reactions to drugs. Clinically, a widespread symmetrical eruption consisting of erythematous macules and papules is seen, usually accompanied by pruritus and sometimes fever. The drugs most commonly responsible for exanthematous eruptions include penicillins, cephalosporins, sulfonamides, carbamazepine, hydantoins, allopurinol, and gold. Occasionally, maculopapular eruptions are associated with interstitial nephritis, hepatitis, and lymphadenopathy.

The most dangerous types of drug reactions that have significant morbidity and mortality include toxic epidermal necrolysis, erythema multiforme major (Stevens-Johnson Syndrome), exfoliative dermatitis, and anaphylaxis. Other well-defined reaction patterns from drugs include urticaria, serum sickness reactions, photosensitivity, bullous eruptions, lichen planus-like (lichenoid) eruptions, fixed reactions, pustular and acneiform eruptions, vasculitis, and connective tissue disease syndromes. Management most commonly consists of stopping the offending drug, and administering symptomatic therapy until the reaction has subsided. Severe reactions may require treatment in a burn center.

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Leg Ulcers

William H. Eaglstein, M.D.

The most common lower extremity ulcers are the venous ulcer, the diabetic ulcer and the arterial ulcer. Approximately 70% of leg ulcers are *venous*, occurring mostly in the ankle area. They are usually secondary to damaged valves in the veins, which results in an inability of the venous pressure to fall as is normal when walking. This failure of the pressure to fall is called sustained venous hypertension. *Diabetic ulcers* occur almost exclusively on the foot. They are either secondary to large vessel atherosclerosis or to diabetic neuropathy and loss of feeling. The neuropathic diabetic ulcers occur on pressure areas of the foot and toes and can be thought of as pressure ulcers, while the atherosclerotic type can be considered ischemic ulcers. *Arterial ulcers* are secondary to large vessel atherosclerosis. Some leg ulcers are thought to be caused by concomitant arterial and venous disease and are called mixed.

Venous ulcers require compression wraps and, once healed, compression hose. There are many adjunctive therapies such as topical antimicrobials, occlusive dressings, skin grafting and tissue engineered skin. Both diabetic ischemic and arterial ulcers are treated with surgery to restore arterial blood flow. For diabetic neuropathic foot ulcers, the pressure must be relieved (off-loaded) by special shoes, casts or crutches.

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Skin Signs of Systemic Disease

Jeffrey P. Callen, M.D.

Entire books (see suggested reading list) have been written on the topic of skin signs of systemic disease. Rather than recapitulate these texts, four clinical cases are presented with detailed discussions of their differential diagnosis and evaluation.

Case 1.

A 53-year-old woman presents for evaluation of skin fragility involving her hands. The process began about seven months ago, when she noted blisters that occurred with minor trauma and healed very slowly. She states that she has no chronic illnesses and takes no medications on a regular basis. She drinks "moderately" and smokes regularly, but denies current use of intravenous drugs. On examination, you note only the hand lesions shown in online figure 1. The remainder of her physical examination is normal.

1. What is the differential diagnosis of this woman's problem?
2. Can the diagnosis be established at the time of this visit?
3. What evaluation should be done?

Case 2.

A 35-year-old woman presents one week after an upper respiratory tract infection for evaluation of the sudden onset of painful, erythematous lesions on her forehead. There was no prior history of similar lesions. Physical examination discloses a temperature = 100.7°F, blood pressure = 120/70 mm Hg, and a pulse = 88/min. Tender erythematous plaques on the forehead are observed (online **Figure 2**). No conjunctival, intraoral or additional skin lesions are seen. Laboratory evaluation reveals a normal hemoglobin and platelet count, but her white blood cell count is elevated at 18,000 per mm³ with a shift to the left.

1. What is the differential diagnosis?
2. What evaluation is needed?
3. What therapy should be prescribed?

Case 3.

A 30-year-old man presents with an eruption on his back, chest and arms. He has hypertension and takes hydrochlorothiazide. The eruption first started during a vacation to Florida. He has also noted fatigue and generalized achiness. His review of systems is otherwise unremarkable. Physical examination is normal other than the skin lesions shown in Online **Figure 3**.

1. What evaluation is needed?
2. What therapy should be prescribed?

Case 4.

A 22-year-old woman developed a rash approximately 1 month ago. She recently graduated from college where she was a member of the track team. Until now she has been completely healthy. She has also begun to note an unusual amount of fatigue and feels as though her "muscles are heavy". She is not taking any medications. Physical examination reveals facial erythema with violaceous, edematous upper eyelids (Online **Figure 4a**), poikiloderma on the extensor forearms, upper chest and upper back, and violaceous atrophic papules and plaques on her dorsal hands (Online **Figure 4b**). She has minimal weakness of her deltoid and quadriceps muscles, but is not weak elsewhere. The remainder of her examination is normal.

1. What is the differential diagnosis of the skin lesions?
2. What evaluation is needed?
3. What therapy should be prescribed?

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Skin Signs of Systemic Disease - Case 1

Case 1.

A 53-year-old woman presents for evaluation of skin fragility involving her hands. The process began about seven months ago, when she noted blisters that occurred with minor trauma and healed very slowly. She states that she has no chronic illnesses and takes no medications on a regular basis. She drinks "moderately" and smokes regularly, but denies current use of intravenous drugs.

On examination, you note only the hand lesions shown in Online **Figure 1**. The remainder of her physical examination is normal.

1. What is the differential diagnosis of this woman's problem?
2. Can the diagnosis be established at the time of this visit?
3. What evaluation should be done?

Comment:

1. There is a limited differential diagnosis for multiple blisters and erosions on the dorsal surface of both hands and it includes porphyria cutanea tarda (PCT), the process that this patient has, drug-induced pseudoporphyria, bullous lupus erythematosus, and epidermolysis bullosa acquisita.

2. Yes, a urine specimen that is dark in color (tea-colored) or fluoresces with Wood's light examination provides evidence in support of the diagnosis of PCT. The diagnosis is confirmed with a specific pattern of porphyrins on a 24 hour urine collection for quantitative porphyrins determinations.

3. Many patients (40%) may be carriers of hemochromatosis. Frequently, patients with PCT are found to be infected with the hepatitis C virus. Excessive alcohol intake is also a risk factor for the development of PCT and alcohol-induced hepatotoxicity can be exacerbated by the hepatitis C virus. This patient had hepatitis C, the source of which was previous intravenous drug use or possibly intranasal cocaine use. Patients often do not admit to such prior behavior until confronted with positive serologic findings. Treatment of PCT includes phlebotomy or low-dose antimalarials. The use of interferon for hepatitis C infections has led to improvement in some, but not all, patients.

Skin Signs of Systemic Disease - Case 2

Case 2. A 35-year-old woman presents one week after an upper respiratory tract infection for evaluation of the sudden onset of painful, erythematous lesions on her forehead. There was no prior history of similar lesions. Physical examination discloses a temperature = 100.7°F, blood pressure = 120/70 mm Hg, and a pulse = 88/min. Tender erythematous plaques on the forehead are observed (Online **Figure 2**). No conjunctival, intraoral or additional skin lesions are seen. Laboratory evaluation reveals a normal hemoglobin and platelet count, but her white blood cell count is elevated at 18,000 per mm³ with a shift to the left.

1. What is the differential diagnosis?
2. What evaluation is needed?
3. What therapy should be prescribed?

Comment:

1. This patient has acute febrile neutrophilic dermatosis, first described by Sweet in 1964 and therefore often called Sweet's syndrome. Other considerations would be cellulitis, an insect bite reaction, vasculitis, atypical pyoderma gangrenosum, Behcet's disease, and possibly a herpes virus infection.
2. Evaluation should include a skin biopsy, a careful history for possible inflammatory bowel disease, CBC for possible myeloproliferative disorder, and an assessment for associated arthritis or conjunctivitis of the skin. Sweet's syndrome is a reactive process which is uncommon. It has been described frequently in association with a prior upper respiratory tract infection, but may occur in patients with hematologic malignancies or pre-malignancies, in particular acute myelocytic leukemia.
3. Patients with acute neutrophilic dermatosis usually respond to a short course of oral corticosteroids. For patients with recurrent or chronic disease, steroid-sparing agents should be considered including antimicrobials or immunosuppressive agents.

Skin Signs of Systemic Disease - Case 3

Case 3. A 30-year-old man presents with an eruption on his back, chest and arms. He has hypertension and takes hydrochlorothiazide.

The eruption first started during a vacation to Florida. He has also noted fatigue and generalized achiness. His review of systems is otherwise unremarkable. Physical examination is normal other than the skin lesions shown in Online **Figure 3**.

1. What evaluation is needed?
2. What therapy should be prescribed?

Comment:

1. This patient has subacute cutaneous lupus erythematosus (SCLE). A potassium hydroxide preparation excluded a dermatophyte infection, and a skin biopsy specimen revealed a histologic pattern known as interface dermatitis. Many of the patients with SCLE have systemic disease and about half will fulfill the ACR criteria for classification of systemic lupus erythematosus. While severe systemic manifestations are possible, most authorities believe that these patients have a more benign course than unselected patients with systemic lupus erythematosus. They rarely manifest renal or neuropsychiatric involvement. Evaluation should include a complete blood count, urinalysis, tests of renal function, and serologic tests including ANA, anti-Ro (SS-A) antibodies, and anti-nDNA antibodies. Except for a positive ANA and the presence of anti-Ro antibodies, the results of these tests were normal in this patient. Note that while anti-Ro antibodies are prevalent in patients with SCLE, their presence is not a diagnostic test, and their absence does not exclude the diagnosis. SCLE skin lesions have been regularly reported in patients on hydrochlorothiazide and in some patients on calcium channel blockers. Withdrawal of the drug leads to a clinical clearing, but serologic abnormalities usually persist.

2. Therapy of SCLE should include daily application of sunscreens, topical corticosteroids preparations and often oral hydroxychloroquine (an antimalarial).

Skin Signs of Systemic Disease - Case 4

Case 4. A 22-year-old woman developed a rash approximately 1 month ago. She recently graduated from college where she was a member of the track team. Until now she has been completely healthy. She has also begun to note an unusual amount of fatigue and feels as though her “muscles are heavy”. She is not taking any medications.

Physical examination reveals facial erythema with violaceous, edematous upper eyelids (Online **Figure 4a**), poikiloderma on the extensor forearms, upper chest and upper back, and violaceous atrophic papules and plaques on her dorsal hands (Online **Figure 4b**). She has minimal weakness of her deltoid and quadriceps muscles, but is not weak elsewhere. The remainder of her examination is normal.

1. What is the differential diagnosis of the skin lesions?
2. What evaluation is needed?
3. What therapy should be prescribed?

Comment:

1. This patient has dermatomyositis (DM). The differential diagnosis for her skin lesions is limited to photo aggravated processes, in particular lupus erythematosus, and less likely a photo drug eruption. The presence of muscle symptoms and mild weakness further narrows the differential diagnosis.
2. The diagnosis of DM can be confirmed by abnormal muscle enzymes, electromyography, or muscle biopsy. The use of magnetic resonance imaging (MRI) will possibly supplant the need for electromyography or muscle biopsy; MRI also can be useful for the selection of a site for a muscle biopsy. Compared to the normal population, patients with DM have a greater chance of having an internal malignancy. This is particularly true in older individuals. A malignancy evaluation should be directed by the age sex and symptoms of the patient. For this young woman, it is appropriate to exclude the possibility of breast or gynecologic (particularly ovarian) cancer.
3. Therapy for the skin disease includes sunscreens, emollients, antimalarials, and/or methotrexate. For the muscle disease, oral corticosteroids (1 mg/kg/day) are an important component of the initial therapy. Steroid-sparing agents such as methotrexate or azathioprine should be considered early in the course of the disease. High-dose intravenous immune globulin has proven to be helpful in patients who fail to respond to conventional therapy.

Skin Signs of HIV Infection**Timothy G. Berger, M.D.**

HIV infection leads to progressive failure of cell-mediated immunity due to HIV-mediated loss of CD4 helper T cells. Virtually all HIV infected persons will develop some skin disorder during the course of their illness. Certain skin diseases typically appear at certain stages of HIV infection, sometimes allowing the practitioner to predict the stage of HIV infection. Mucocutaneous changes are frequently the initial clinical manifestation of HIV disease. The skin changes can be classified into three categories: infections, inflammatory diseases, and neoplasms.

Infections

Infections generally present when the helper T cell count is around $300/\text{mm}^3$. The most common infections are mucocutaneous candidiasis (thrush and vaginal candidiasis) and herpes zoster. As the helper T cell count approaches $200/\text{mm}^3$, the characteristic opportunistic infections of AIDS are seen. Some of these may appear on the skin in the form of disseminated fungal infections (cryptococcosis and histoplasmosis), mycobacterial infections (tuberculosis and *M. avium/intercellulare*), viral infections (chronic herpes simplex of the orolabial or genital areas, warts, and extensive molluscum contagiosum (Online **Figure 1**)), and parasitic infections (hyperkeratotic scabies). Many of these infectious complications are also seen in other immunosuppressed persons.

Effective treatment of HIV infection may reverse the immunosuppression and improve or prevent the infectious complications.

Inflammatory diseases

Inflammatory diseases occur throughout all stages of HIV infection. The most common are the papulosquamous diseases--seborrheic dermatitis (Online **Figure 2**), psoriasis, and Reiter's syndrome. They can have the same clinical appearance in the setting of HIV infection as they do in the immunocompetent host, but they are often much more extensive and severe. Drug eruptions are between 10 and 100 times more common in persons with HIV infection, especially those with helper T cells below 200/mm³. Up to 40% of AIDS patients treated with sulfamethoxazole/trimethoprim will develop a drug eruption. Pruritic folliculitis affects up to 25% of AIDS patients. These inflammatory conditions may not respond to improvements of the immune system with anti-HIV treatments.

Neoplastic complications

Neoplastic complications of HIV disease include Kaposi's sarcoma (Online **Figure 3**) and nonmelanoma skin cancer. Kaposi's sarcoma is associated with infection with human herpes virus 8 (HHV-8 or KSHV), a sexually transmitted virus. Typical lesions are purple, non-tender, symmetrical macules, plaques, or tumors on the skin or mucous membranes (especially the hard palate). Kaposi's sarcoma will usually improve or resolve if the helper T cell count increases and the viral load decreases with effective anti-HIV therapy. Nonmelanoma skin cancer, especially basal cell carcinomas, is very common in fair-skinned HIV-infected persons; they have the same clinical appearance and are managed in the same way as they are in immunocompetent hosts.

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Bedside Diagnostic Testing

M. Joyce Rico, M.D.

A few simple tests conducted at the bedside can confirm the suspected clinical diagnosis in several common transmittable dermatologic disorders. These tests require few

reagents, are easily learned, and can be performed by all practitioners. The tests include potassium hydroxide (KOH) examination to identify dermatophytes (Section 25) or yeast (Section 24), Gram stain for bacteria (Section 21), oil preparation for mites (Section 26), and Tzanck preparation for herpes simplex or varicella/zoster infections (Section 23).

KOH Examination

Materials: glass slide, cover slip, #15 scalpel (or second glass slide), 10-20% KOH dissolved in water or 40% dimethyl sulfoxide (DMSO), microscope.

The dermatologist's mantra, "If it scales, scrape it," acknowledges the prevalence of fungal (tinea) infections in patients presenting with scaly skin rashes. A KOH examination is performed by gently scraping across the active edge of a scaly rash and collecting the scale on a glass slide. A drop of KOH solution is applied to the scale to dissolve the cell walls followed by a cover slip. Either gently heating the slide, or waiting 10 - 15 minutes, or adding DMSO to the solution will hasten the dissolution of the keratin and make it easier to see the fungi. Examine the slide under low power of the microscope with the condenser lowered. In a dermatophyte (tinea) infection, look for branching hyphae (Online **Figure 1**). In candidiasis, look for budding yeast, although sometimes budding yeast are easier to see with a Gram stain (Online **Figure 2**). The following may present pitfalls: 1) In places where keratinocytes are piled up, overlapping cell walls may sometimes look like hyphae, however true hyphae are septate (have divisions) and cross over more than one cell. Exerting gentle pressure on the top of the slide (e.g., gently pushing on the cover slip with the end of a match) will disperse cell walls, but not hyphae 2) in partially treated dermatophyte infections, hyphae may be short, blunted and difficult to identify.

Gram Stain Evaluation

Materials: glass slide, #15 scalpel, crystal violet reagent (1% crystal violet, 5% sodium bicarbonate in water), Gram's iodine solution (1% iodine, 2% potassium iodide in water), 70% ethyl alcohol, counter stain (0.5% safranin O in water) , water, and microscope.

Gram stains are useful for identifying bacteria in patients suspected of having cutaneous infections such as impetigo, cellulitis, staphylococcal or streptococcal folliculitis, and venereal ulcers (chancroid). To perform a Gram stain, thinly smear a small amount of the test material on a microscope slide. Briefly 'heat fix' the material to the slide by holding the slide over a match or flame. Apply the 1) crystal violet solution for 1 minute, then rinse the slide with water. Fix the stain by incubating the slide with 2) Gram's iodine solution for 1 minute. 3) Decolorize with either a 50:50 mixture of 95% alcohol and acetone or 70% ethyl alcohol, then 4) counter-stain with safranin for 30 seconds. Rinse the slide in water, dry without blotting, and examine under the microscope. Identify bacteria by their shape (cocci vs rods), color (gram-negative organisms are red; gram-positive organisms are blue-purple) and note their location (intracellular vs extracellular).

In Online **Figure 3**, grouped gram-positive cocci, most likely *Staphylococcus* organisms, can be seen within cells.

Tzanck preparation

Materials: glass slide, #15 scalpel, Wright's stain, water, microscope.

Cutaneous herpes infections (simplex, varicella, and zoster) present early in their course as painful, single or grouped, tense blisters on a red base. The diagnosis can be confirmed by performing a Tzanck preparation to identify viral cytopathic changes in keratinocytes, in particular multinucleated giant cells. Test material is obtained by gently opening an early intact blister and scraping the underside of the blister roof with a #15 scalpel blade. The test material is smeared on a glass slide, air dried, then stained with Wright's stain or Paragon Multiple Stain for 30 seconds. If the above stains are not available, any reagent which stains nuclei can be used (e.g., urine Sedi-stain, Giemsa). Gently wash off excess stain, air dry, and examine with a light microscope. Low power magnification is used to scan for multinucleated giant cells (Online **Figure 4**). On higher power, alterations in cell morphology including graying of the cytoplasm and margination of the chromatin can be seen.

Oil Preparation

Materials: mineral oil, #15 blade, glass microscope slide, cover slip, microscope.

All patients presenting with pruritus and a rash warrant an oil preparation examination to rule out scabies. As a patient with active scabies may have only a few mites, selection of appropriate sites to perform the scraping is important. Burrows, non-excoriated papules, or vesicles are more likely to contain organisms than crusted papules or erosions; burrows appear as 5-10 mm long, skin-colored ridges. High-yield sites to scrape for scabies include the web spaces between the fingers, areas where clothing binds such as around the waist or axillae, and the buttocks. To perform an oil preparation for scabies, apply a drop of mineral oil to the skin and to the glass slide. With the blade perpendicular to the skin, gently scrape and deposit skin scrapings on the slide. The mites live in the stratum corneum; if you draw blood you are too deep! Coverslip the slide and examine it under low magnification with the microscope. In scabies infestations, you may see mites of varying sizes, eggs which are smaller than the live mite, or mite feces, which appear as small oval brown spheres (Online **Figure 5**).

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Questions to accompany clinical sections

Questions - Bedside Diagnostic Tests

Select the appropriate bedside diagnostic test for each clinical presentation:

- A) Tzanck preparation
- B) Oil preparation
- C) KOH preparation
- D) Gram stain

1. An otherwise healthy 42-year-old man presents with an itchy, scaly rash on the soles of his feet that extends between his toes.

2. You are asked to see an 82-year-old nursing home resident with an intensely pruritic rash characterized by excoriated papules in the web spaces of her fingers, on her buttocks, around her waist, and in her armpits.

3. A patient with chronic lymphocytic leukemia undergoing chemotherapy develops painful grouped vesicles on the upper lip.

4. An eight-year-old boy is brought to the clinic with a warm, red, tender plaque on his leg that is surmounted by a fluctuant pustule.

Questions - Acne Vulgaris, Folliculitis, and Acne Rosacea

1. A 16-year-old African-American girl presents with numerous comedones on the face. Papules and pustules are absent. Appropriate initial treatment would be:

- A) Adapalene
- B) Tetracycline
- C) Clearasil

- D) Topical steroids
- E) Oral isotretinoin

2. The same patient subsequently develops a moderate number of pustules on her face. The addition of what topical or oral medication should be considered in this case?

- A) Adapalene
- B) Tetracycline
- C) Clearasil
- D) Topical steroids
- E) Oral isotretinoin

2a. Why would isotretinoin NOT be considered as first-line therapy in this case?

3. A 30-year-old Asian male presents with papules and pustules around the hair follicles on his legs. What is the most likely etiology?

- A) Lubricating lotion
- B) Staphylococcus aureus
- C) Fungus infection
- D) Hot oil treatment
- E) Pseudomonas aeruginosa

3a. What is the most likely diagnosis in this case?

3b. What topical or oral medication should be prescribed?

4. A 45-year-old, Irish-American man presents with papules, pustules and diffuse erythema on his face. Comedones are absent. What is the most likely diagnosis?

- A) Photosensitivity

- B) Rosacea
- C) Lupus erythematosous
- D) Acne vulgaris
- E) Contact dermatitis

4a. What topical or systemic medication could be prescribed?

Questions Actinic Keratoses and Non-Melanoma Skin Cancer

1. A 58-year-old fair-skinned man presents with a 1 cm diameter pearly papule with central hemorrhagic crust and peripheral telangiectasias. The most like diagnosis is:

- A) melanoma
- B) basal cell carcinoma
- C) actinic keratosis
- D) psoriasis

2. In evaluating the patient described in question 1, what diagnostic test should be performed?

- A) KOH examination
- B) Gram stain
- C) Skin biopsy
- D) Frozen section
- E) Tzanck preparation

3. Appropriate treatment for actinic keratosis includes which of the following:

- A) Liquid nitrogen
- B) 5 - fluorouracil cream
- C) Imiquimod

- D) Photodynamic therapy
- E) All of the above

Questions - Pigmented Lesions

1. A small, tan macule located on sun-exposed skin is called a:

- A) Ephelis (freckle)
- B) Lentigo maligna
- C) Nevus
- D) Dysplastic nevus
- E) Melanoma

2. An increased familial risk for melanoma is associated with:

- A) Single dysplastic nevus
- B) Multiple dysplastic nevi
- C) Multiple ephelides
- D) Multiple junctional nevi
- E) Nevoid basal cell carcinoma syndrome

3. The most common form of melanoma in African-Americans is:

- A) Lentigo maligna
- B) Lentigo maligna melanoma
- C) Superficial spreading melanoma
- D) Acral lentiginous melanoma
- E) Nodular melanoma

4. The form of melanoma most associated with chronically sun-exposed skin is:

- A) Lentigo maligna melanoma
- B) Nodular melanoma
- C) Superficial spreading melanoma
- D) Acral lentiginous melanoma
- E) Amelanotic melanoma

Questions - Common Benign Cutaneous Growths

1. Eruptive seborrheic keratosis may be associated with:

- A) Parkinson's disease
- B) Addison's disease
- C) Internal malignancy
- D) Melanoma
- E) Hyperlipidemia

2. Epidermoid cysts are a feature of which syndrome:

- A) Gardner Syndrome
- B) Tuberous sclerosis
- C) Neurofibromatosis
- D) Gardner Diamond Syndrome
- E) Bloom's Syndrome

Questions - Dermatitis, including Atopic, Contact, Seborrheic, and Stasis

1. Individuals with atopic dermatitis are prone to develop:

- A) Bacterial infections
- B) Viral infections
- C) Fungal infections

- D) Allergic rhinitis
- E) All of the above

2. Irritant contact dermatitis is the result of:

- A) Type IV (delayed type) hypersensitivity
- B) Activation of Helper T-cells
- C) Type I hypersensitivity
- D) Chemical exposure
- E) Fixed drug eruption

3. Infantile seborrheic dermatitis frequently involves the:

- A) Scalp
- B) Eyebrows
- C) Nasolabial folds
- D) Central chest
- E) Antecubital fossa

Questions - Pruritus/xerosis

1. A 26-year-old man reports that he has been itching severely for almost two months. Over the past month he has also had fevers and drenching night sweats. On physical examination there are no recognizable signs of a specific skin disease such as scabies or atopic dermatitis, although there are some findings that look like they were caused by rubbing and scratching. Which of the following diagnoses is reasonably likely?

- A) Psoriasis
- B) Allergic contact dermatitis
- C) Hives (urticaria)
- D) Hodgkin's lymphoma

E) Ringworm

2. A 70 year-old professor who takes two 20 minute baths a day to ease what she calls her "rheumatism" reports that she has had generalized itching since November. She is a chemist and teaches undergraduates. On physical examination the skin at first appears entirely normal, but on closer inspection it is a little bit pink and scaly. The most likely diagnosis for her itching is:

- A) Eating too much citrus fruit
- B) Aggravation from students
- C) Hodgkin's lymphoma
- D) Primary biliary cirrhosis
- E) Winter itch

Questions - Psoriasis

1. The most common type of psoriasis is:

- A) Guttate psoriasis
- B) Plaque psoriasis
- C) Erythrodermic psoriasis
- D) Pustular psoriasis
- E) Nail psoriasis

2. Guttate psoriasis frequently follows:

- A) Streptococcal pharyngitis
- B) Staphylococcus infection
- C) Candida infection
- D) Tinea infection
- E) Haemophilus ear infection

Questions - Impetigo and Cellulitis

1. A 4-year-old girl develops vesicles with honey-colored crusts around her nose. The most likely organism is:

- A) Streptococcus pyogenes
- B) Staphylococcus aureus
- C) Haemophilus influenzae
- D) Pseudomonas aeruginosa
- E) Mixed bacterial flora

2. A 50-year-old man with renal failure develops blisters over his stomach followed by widespread erythema and superficial sloughing of his skin. Culture of lesions will show:

- A) Streptococcus pyogenes
- B) Staphylococcus aureus
- C) Haemophilus influenzae
- D) Pseudomonas aeruginosa
- E) No bacteria

3. Following a motor vehicle accident, a 22-year-old woman develops severe pain, swelling and a dusky blue discoloration of her right arm. What is your diagnosis?

- A) Erysipelas
- B) Cellulitis
- C) Impetigo
- D) Necrotizing fasciitis
- E) Undetected fracture

4. Predisposing factors for the development of necrotizing fasciitis include:

- A) Burns
- B) Surgical procedures
- C) Diabetes mellitus
- D) Childbirth
- E) All of the above

Questions - Syphilis

1. A 20-year-old man presents with a 2-day history of a painless ulcer of the penis. The best diagnostic test at this time is:

- A) Culture of the exudate
- B) Darkfield examination
- C) VDRL
- D) FTA
- E) Blood culture

2. Condyloma lata are found during which stage of syphilis:

- A) Primary
- B) Secondary
- C) Latent
- D) Tertiary
- E) Congenital

3. Foot-drop, a neurologic manifestation of syphilis is found in which stage:

- A) Primary

- B) Secondary
- C) Latent
- D) Tertiary
- E) Congenital

Questions - Viral Infections 1. Factors that predispose patients to warts include which of the following:

- A) Age
- B) Trauma
- C) Renal transplant
- D) Treatment for lymphoma
- E) All of the above

2. The most common time period of primary oral/facial infection with herpes simplex is:

- A) Childhood
- B) Teen years
- C) Adult years
- D) Elderly
- E) All of the above

3. An outbreak of recurrent herpes labialis may be precipitated by:

- A) Penicillin therapy
- B) Acne flare
- C) Sun burn
- D) Ear infection
- E) Strep throat infection

4. The incidence of post herpetic neuralgia increases with:

- A) Number of recurrences
- B) Involvement of the ventral nerve ganglion
- C) Involvement of the T3 dermatome
- D) Age
- E) Gender

Questions - Pityriasis and Candidiasis

1. An 18-year-old man comes to you with a complaint of a rash that has been present over the summer months. On your exam you find hypopigmented macular lesions with slight branny scaling involving primarily the trunk. A KOH examination will show:

- A) Yeast forms only
- B) Hypheal forms only
- C) Hyphae and yeast ("spaghetti and meatballs")
- D) Pseudomonas
- E) Sulfur granules

2. A 35-year-old, overweight woman has recurrent candida infections in the crural folds. Which of the following should be ruled out:

- A) Thyroid disease
- B) Diabetes
- C) Addison's disease
- D) Crohn's disease
- E) Concurrent strep infection

3. Systemic treatment for severe extensive tinea versicolor would include:

- A) Griseofulvin
- B) Terbinafine

- C) Ketoconazole
- D) Penicillin
- E) Amphotericin B

Questions - Tinea (dermatophyte) Infections

1. Tinea capitis has a peak incidence in:

- A) Children under 12
- B) Teenagers
- C) Young adults
- D) Middle age parents
- E) Elderly

2. The tinea infection of the nails that has been identified as a sign of HIV disease is:

- A) Distal subungual form
- B) White superficial form
- C) Proximal white subungual form
- D) Paronychia form
- E) Infection of the great toe

3. Woods light exam of tinea capitis may show:

- A) Coral red color
- B) Blue green color
- C) Yellow color
- D) Black grains
- E) Brown color

Questions - Parasitic Infestations

1. A patient presents with extensive thick, crusted plaques, and thick, dystrophic nails. A scraping shows numerous mites and eggs. Which of the following is most likely to be associated with this condition?

- A) Nits on the hairs
- B) HIV infection
- C) Thyroid disorder
- D) Syphilis
- E) Gastroenteritis

2. You choose to treat crusted scabies systemically because of extensive involvement. Which medication would you choose?

- A) Lindane
- B) Precipitated sulfur
- C) Ivermectin
- D) Crotamiton
- E) Permethrin

3. Which of the following is most likely to be found in the seams of clothing?

- A) Ear mite
- B) Scabies mite
- C) Head louse
- D) Body louse
- E) Crab louse

Questions - Urticaria and Drug Eruptions

1. Urticaria can be caused by:

- A) Penicillin
- B) Insect bite

- C) Infection
- D) Cheese
- E) All of the above

2. Histamine-releasing medications include:

- A) Penicillin
- B) Sulfa
- C) Hydrochlorothiazide
- D) Aspirin
- E) Hydroxyzine

Questions - Leg Ulcers

1. A patient with adult-onset diabetes has an ulcer on the plantar surface of the foot. The most likely diagnosis is which of the following?

- A) Venous hypertension
- B) Atherosclerosis of small vessels
- C) Peripheral neuropathy
- D) Bacterial infection
- E) Pyoderma gangrenosum

2. Which of the following is the most common leg ulcer?

- A) Diabetic
- B) Pyoderma gangrenosum
- C) Arterial ulcer
- D) Venous ulcer
- E) Mixed arterial/venous

3. Which of the following is the basic therapy for a patient with adult-onset diabetes and an ulcer near the ankle?

- A) Compression wraps or hose
- B) Topical or oral antibiotics
- C) Skin grafting
- D) Surgical restoration of blood flow
- E) Intralesional corticosteroid injections

Questions - Skin Signs of HIV Infection

1. A 32-year-old homosexual man presents with 5 days of pain and 3 days of a band of blisters on the left flank beginning in the midline posteriorly and ending abruptly in the midline anteriorly. What rapid test should you do to diagnose the blistering problem?

- A) Bacterial culture
- B) Gram stain
- C) KOH exam
- D) Tzanck smear
- E) CBC and blood culture

2. Regarding the same patient, what further testing is indicated first?

- A) Helper T cell count
- B) HIV testing
- C) CBC and blood culture
- D) Chest x-ray
- E) TB tine test

3. A 38-year-old homosexual man presents with about ten 0.5 to 1 cm purple plaques on

the body and feet for one month. The lesions are symmetrical, nontender, and smooth-surfaced. What is the correct diagnosis?

- A) Cherry hemangioma
- B) Hemangioma
- C) Basal cell carcinoma
- D) Kaposi's sarcoma
- E) Angiosarcoma

4. Which of the following is related to a low helper T cell count ($<200/\text{mm}^3$):

- A) Deep/disseminated cryptococosis infection
- B) BCC
- C) Pruritic folliculitis
- D) Oral thrush
- E) Seborrheic dermatitis

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Figures and answers to these questions are provided for on the AAD medical student core curriculum website. Please visit:

[http://www.aad.org/professionals/Residents/MedStudCoreCurr/ScienceCurriculum.
htm](http://www.aad.org/professionals/Residents/MedStudCoreCurr/ScienceCurriculum.htm)

Dermatology as an Advanced Residency Program¹

The NRMP offers three types of postgraduate training positions in the Main Match: categorical (C), preliminary (P), and advanced (A). Categorical programs begin training at the first postgraduate year (PGY-1) and are completed in approximately 3 or more years. Examples include internal medicine, pediatrics, obstetrics and gynecology, general surgery, and pathology. Preliminary programs last only 1 year and exist to satisfy the 1-year prerequisite for advanced specialty programs. They are available through internal medicine, surgery, or a transitional year. Dermatology is an advanced residency program. These positions begin at the second postgraduate year (PGY-2), after a year spent in a preliminary program. Students apply to both advanced and preliminary programs at the same time. Other examples include ophthalmology, anesthesiology, and radiology.

What is the difference between preliminary year options? Preliminary *medicine* consists of one year of training in internal medicine. Responsibilities and call schedule are similar to those of categorical (3-year) interns. This is a good choice for students entering advanced specialties, such as dermatology, that heavily emphasize internal medicine. Preliminary *surgery* offers the same experience as that of an intern in general surgery. Most of these positions are informally assigned outside the Match to students who have already matched into an early surgical specialty, such as otolaryngology, urology, and neurosurgery. The *transitional year* is usually offered by community hospitals with a typical curriculum of rotations in internal medicine, emergency medicine, ambulatory medicine, and sometimes surgery, obstetrics, or critical care. All transitional year internships allow for 2 to 6 flexible months of electives. Because the level of clinical duties and call responsibilities are on par with those of a “fifth-year medical student,” these internships are highly competitive.

Applying to Programs and Interviews

Dermatology is an extremely competitive specialty. Most programs interview about 30 candidates (out of hundreds of applicants) for only two or three positions. For this reason most students *apply* to nearly every program in the country. Estimates on the numbers of applications range from 40 to upwards of 65, even 70 programs. After 30, ERAS charges \$25 to apply to each additional program (\$250 for every additional 10). Those who secure 10 interviews have a good chance at matching². It is recommended to attend as many interviews as possible to maximize the chances of matching.

Enhancing Your Prospects

- ◇ identify your interest early in medical school
- ◇ get involved with the dermatology department as early as possible

¹ Freeman, Brian. The Ultimate Guide to Choosing a Medical Specialty. New York: Lange/McGraw, 2004.

² Long, Erin M. Dermatology Residency Board. 17 Sept. 2005
<<http://p204.ezboard.com/bdermatology>>.

- ◇ research and publications (basic science and/or clinical) are very important
- ◇ competitive Step I board scores are critical (average 240)³
 - passing score as of 9/17/2005 is 182
 - average score for all test-takers (estimated from score distribution) is 214
- ◇ membership in AOA (the medical school honor society)
- ◇ lots of honors grades in your third-year clerkships
- ◇ take several electives in dermatology early in your senior year
- ◇ externships at programs of highest interest
- ◇ networking – connections are important!
- ◇ very strong letters of recommendation from people you have worked with

Backup Plans

Everyone, regardless of their qualifications, needs to have a backup plan in case they don't match. Dermatology has one of the highest unmatched rates (24.3%)⁴, so facing the reality of possibly *not* matching is only good planning. There are several options:

- ◇ Another Specialty. Through ERAS you can choose to apply in both dermatology and another specialty, such as internal medicine or pediatrics. Try to avoid other super-competitive specialties like radiology, ophthalmology, and orthopedics.
- ◇ Pursue an Intensive Research (Basic/Clinical) Position. You may choose not to rank any programs below those in your dermatology rank list (i.e. not rank any preliminary programs). If you don't match, then, you will not be committed to an internship year, and you can begin research ASAP if that is your interest. In this way some applicants work to improve their resume and increase their chances of matching the second time around.
- ◇ Start an Internship. This is the choice of most applicants. By completing an internship year you will be eligible for clinical research fellowships since a lot of fellowships require you to be an M.D. Sometimes spots in dermatology open up during the year, and you will only be eligible if you will have completed an internship on time. Also, perhaps doing a preliminary year in medicine will sway you to switch into the categorical program (i.e. the 3-year internal medicine residency).

Externships

An externship, or away rotation, is a clinical rotation that is completed at a different medical school (anywhere in the country they are offered). Externships are a great way to check out a specific program to see if it's a good fit for you. They are also a great way to get acquainted with a different set of faculty who can write a recommendation, for example. Away rotations may be clinical or research oriented. Student may choose to do sub-specialty elective rotations in areas such as

³ As self-reported by applicants from the classes of 2001 and 2002 on the Dermatology Residency Board <<http://p204.ezboard.com/bdermatology>>

⁴ [M.D.tool.com: Guide for the Match and ERAS](http://www.M.D.tool.com/match.html). 18 Sept. 2005 <<http://www.M.D.tool.com/match.html>>.

dermatopathology to dermatologic surgery. The 'away' rotations provide the opportunity to see what it would be like to be a resident at that institution. Opportunities are limited so it is generally a good idea to start looking for externships in the spring of the 3rd year.

Internet Resources:

<http://www.aamc.org/students/eras/start.htm>

<http://www8.utsouthwestern.edu/utsw/cda/dept25717/files/169141.html>

<http://www.M.D.tool.com/match.html>

Printed Resources:

Tao, Le, Bhushan Vikas, and Amin Chirag. First Aid for the Match. 2nd ed. Appleton & Lange, 2000.

Timeline & Student-to-Student Guide by Tace Steele, M.D. (Derm Resident 2008-2011)

1st and 2nd Year

Meet with Dermatology Department advisor (if your school has one)
Research including summer projects
Attend Dermatology lectures/grand rounds

3rd Year

Sign up for Dermatology elective (if your school allows you to take it early)
Consider research rotation

Spring 3rd Year

Begin asking for letters of recommendation
Plan senior year schedule/Set up away rotations
Begin CV and personal statement
Consider internship programs for preliminary year
Select an advisor in the Dermatology department
Talk with graduating seniors about specific residency programs

Summer/Fall 4th Year

Rotation in Dermatology
Away rotations
Sub-I in medicine/surgery/pediatrics
Finish personal statement and CV
Pick up letters of recommendation
Take application photos
Complete and submit ERAS application (after September 1)
Register online for the NRMP Main Match (\$40)
Gather information and compare residency programs (i.e. using FREIDA)
Decide whether to apply for a backup specialty

Winter 4th Year

Leave open or with easier rotations for interviews
Finalize list of possible residency programs
Send letters to your highest-ranked programs
Decide rank list
Submit rank list online through the NRMP Main Match website
(due by midnight on the third Thursday in February)
Formulate a plan for scramble/not matching

3rd Week of March 4th Year

Monday: All applicants are notified of their match status (matched or unmatched).
Tuesday: Scramble Day – Unmatched applicants meet with advisor and contact programs with unfilled positions
Thursday: Match Day – All applicants find out where they have matched.

ERAS timeline

June	ERAS 2006 Applicant Manuals available
July	MyERAS Web site opens to applicants to begin their applications.
September	Applicants may begin applying to ACGME accredited programs
September	Programs begin downloading applications from ERAS
November	Dean's Letters are released
December	Application deadline
December	Military Match
January	Urology Match
February	Rank list deadline for NRMP match
Late January	Osteopathic Match
March	NRMP Match results will be available

NRMP: National residency match program

ERAS: Electronic Residency application service

ERAS application

The application is set up like the one for medical school. Be prepared to list all your activities, research, volunteer work and awards with descriptions of activities, duties, positions held and dates. This whole process might be easier if you compile your CV ahead of time with descriptive paragraphs.

Curriculum Vitae (CV)

You will need to submit your CV to your letter writers. It will help to have 2 versions. The first is for your reference. Keep it as long as you want. Include paragraph descriptions of what the organization does, mission statement, positions held and all duties. Also keep track of all the dates in which you were active in the organization or club. This version of the CV will help you fill out your ERAS application. It will also help you to review before all your interviews just in case you are asked about anything that you participated in during medical school. The second CV is to submit to your letter writers and programs. This one should be brief try to keep under two pages with your name at the top of all the pages.

The CV should include: Name, Address, Phone, email

USMLE scores; Education: university, degree, major; Honors/Awards; Research; Publications; Extra-Curricular Activities including volunteer projects and organizations

Letters of Recommendation

You will need to ask for letters of recommendation early in your 4th year or late 3rd year. Remember to keep this in mind when you are on your rotations. Try to keep in touch with the attending physicians that you worked well with. The best letters are from people who knew you well, for example people that you did research with. Keep this in mind early on when you are looking for research projects. Be able to provide the persons writing your recommendation letters with your personal statement and Curriculum Vitae. (The CV without descriptions is usually more professional) You can send up to 4 letters not

including your Dean's letter. You can use one of the 4 spots to include a resume. (this is recommended by some schools) **Remember thank you notes!**

Program Application Deadlines

ERAS does not set the application deadlines. All programs have their own deadlines with the majority in December. However, programs fill their interview spots, so the earlier the better!

Dean's Office

Each medical school's dean's office has their own timeline and procedure for handling your transcript, recommendation letters, Dean's Letter, and application photo. Processing files may take a few weeks, so try to give the dean's office ample time to process your transcripts etc. If there is a problem with transmissions, you could miss a deadline. Use the Application Document Tracking System (ADTS) on the ERAS website to verify document transmission to your programs. Follow up on any missing documents.

Rank Lists

The most important thing to remember about rank lists is to only rank programs that you would want to be a resident at! Given that dermatology is so competitive, some students choose to rank other specialty programs entirely and apply for entirely different programs and interview for two different specialties. Then, people rank their top choices in order (i.e. dermatology for their first few slots and then their second residency choice). This way they assure that if they do not match in dermatology, they would still match in a full residency program.

The second thing you could do is only rank dermatology along with ranking your internship programs. If you don't match for dermatology, you could still match for internship and then you will have another year to apply. Remember when you apply as an intern, you will have a year break in between the end of internship and the year you will hopefully start dermatology, so you will need to think of what you would like to do that year. Lastly, you could apply for dermatology and internship, but choose not to have an internship, unless you match in dermatology. The problem with this is if a spot in dermatology opens up then you will not have completed an internship and will not be eligible. The other plus in having completed an internship is it would allow you to apply for fellowships that require an M.D. and training. (possibly paid!)

Program information

AMA's search engine FRIEDA is a great resource to research different programs. All the individual programs list information on their websites and FRIEDA has links to most of the programs websites. It also allows you to search by area or specialty.

All information gathered from the following websites:

ERAS <http://www.aamc.org/students/eras/start.htm>

FREIDA <http://www.ama-assn.org/vapp/freida/srch/>

Programs

Below is a list of all the dermatology programs.

State	City	Program	# PGY1s	ACGME ID
AL	Birmingham	University of Alabama Medical Center Program	2	0800121010
AR	Little Rock	University of Arkansas for Medical Sciences Program	3	0800421013
AZ	Scottsdale	Mayo Clinic Scottsdale Program	2	0800321127
CA	Loma Linda	Loma Linda University Program		0800521111
CA	Los Angeles	UCLA Medical Center Program	4	0800521017
CA	Torrence	UCLA Medical Center Program	4	0800512137
CA	Los Angeles	University of Southern California Program	3	0800511015
CA	Orange	University of California Irvine		0800521014
CA	Sacramento	University of California (Davis) Program	3	0800521101
CA	San Diego	Naval Medical Center (San Diego) Program		0800531006
CA	San Diego	University of California (San Diego) Program	3	0800521018
CA	San Francisco	University of		0800521019

		California (San Francisco) Program		
CA	Stanford	Stanford University School of Medicine	5	0800521020
CO	Aurora	University of Colorado Program		0800721022
CT	New Haven	Yale-New Haven Medical Center Program	4	0800821023
CT	Farmington	University of Connecticut	2	0800831138
DC	Washington	Howard University Program		0801021025
DC	Washington	National Capital Consortium (NCC) Program		0801021123
DC	Washington	Washington Hospital Center Program		0801021106
FL	Gainesville	University of Florida Program	2	0801121115
FL	Jacksonville	Mayo Graduate School of Medicine (Jacksonville) Program	2	0801131125
FL	Miami	University of Miami-Jackson Memorial Medical Center Program	6	0801121026
FL	Tampa	University of South Florida Program	3	0801121096
GA	Atlanta	Emory University Program	5	0801221028
GA	Augusta	Medical College of Georgia Program	3	0801211029
IA	Iowa City	University of Iowa Hospitals and Clinics Program	3	0801821036
IL	Chicago	John H Stroger Hospital of Cook County	3	0801612030
IL	Chicago	McGaw Medical Center of Northwestern University Program	3	0801621031

IL	Chicago	Rush-Presbyterian-St Luke's Medical Center Program	2	0801611032
IL	Chicago	University of Chicago Program	4	0801611033
IL	Chicago	University of Illinois College of Medicine at Chicago Program		0801621034
IL	Maywood	Loyola University Program		0801612135
IL	Springfield	Southern Illinois University Program	2	0801621118
IN	Indianapolis	Indiana University School of Medicine Program	2	0801721035
KS	Kansas City	University of Kansas Medical Center Program		0801911037
KY	Louisville	University of Louisville Program	2	0802021038
LA	New Orleans	Louisiana State University Program	6	0802121109
LA	New Orleans	Tulane University Program	3	0802121108
MA	Boston	Boston University Medical Center/Tufts University Program	5	0802421044
MA	Boston	Harvard Medical School Program		0802431043
MA	Worcester	University of Massachusetts Program		0802421114
M.D.	Baltimore	Johns Hopkins University Program		0802321040
M.D.	Baltimore	University of Maryland Program		0802321041
M.D.	Bethesda	National Institutes of Health Clinical Center Program		0802312008
MI	Ann Arbor	University of Michigan Program	8 (½ 3 year spots & ½ 4 year spots)	0802531045
MI	Detroit	Henry Ford Medical Center	6 (4-3year positions and	0802512046

			2-4 year positions)	
MI	Detroit	Wayne State University/Detroit Medical Center Program		0802521047
MN	Minneapolis	University of Minnesota Program	4	0802631048
MN	Rochester	Mayo Graduate School of Medicine (Rochester) Program	7	0802621049
MO	Columbia	University of Missouri-Columbia Program		0802821050
MO	St Louis	St Louis University School of Medicine Program	2	0802821116
MO	St Louis	Washington University/B-JH/SLCH Consortium Program		0802821051
NC	Chapel Hill	University of North Carolina Hospitals Program	4	0803611066
NC	Durham	Duke University Program	3	0803621067
NC	Greenville	Pitt County Memorial Hospital/East Carolina University Program	2	0803613132
NC	Winston-Salem	Wake Forest University School of Medicine Program	3	0803621110
NH	Lebanon	Dartmouth-Hitchcock Medical Center Program	2	0803221053
NJ	CaM.D.en	UM.D.NJ-Robert Wood Johnson Medical School Program		0803321117
NJ	New Brunswick	UM.D.NJ-Robert Wood Johnson		0803331128

		Medical School Program		
NJ	Newark	UM.D.NJ-New Jersey Medical School Program		0803321107
NM	Albuquerque	University of New Mexico Program		0803421054
NY	Bronx	Albert Einstein College of Medicine Program	4	0803531058
NY	Brooklyn	SUNY Health Science Center at Brooklyn Program	6	0803521065
NY	Buffalo	SUNY at Buffalo Graduate Medical-Dental Education Consortium Program		0803521057
NY	New York City	New York Medical College		0803521063
NY	New York	Mount Sinai School of Medicine Program	3	0803521061
NY	New York	New York University Medical Center Dermatology Program	7	0803521064
NY	New York	New York and Presbyterian Hospital (Columbia Campus) Program	3	0803521104
NY	New York	New York and Presbyterian Hospital (Cornell Campus) Program		0803521062
NY	New York	St Luke's-Roosevelt Hospital Center Program	4	0803521124
NY	Rochester	University of Rochester Program		0803521102
NY	Stony Brook	SUNY @ Stony Brook Program	4	0803521113
OH	Cincinnati	University Hospital/University	4	0803821068

		of Cincinnati College of Medicine Program		
OH	Cleveland	Cleveland Clinic Foundation Program	3	0803812070
OH	Cleveland	University Hospitals of Cleveland/Case Western Reserve University Program		0803821120
OH	Columbus	Ohio State University Program	2	0803811072
OH	Dayton	Wright State University Program		0803821073
OK	Oklahoma City	University of Oklahoma Health Sciences Center Program	3	0803921074
OR	Portland	Oregon Health & Science University Program	4	0804021075
PA	Danville	Geisinger Health System Program	3	0804112076
PA	Hershey	Penn State University/Milton S Hershey Medical Center Program		0804121103
PA	Philadelphia	Drexel University College of Medicine, Department of Dermatology		0804121077
PA	Philadelphia	Thomas Jefferson University Program	4	0804111079
PA	Philadelphia	University of Pennsylvania Program	5	0804121080
PA	Pittsburgh	University of Pittsburgh Medical Center Medical Education Program in Dermatology		0804111081
PR	San Juan	University of Puerto Rico Program		0804221082

RI	Providence	Brown Medical School Program	4	0804321122
RI	Providence	Roger Williams Medical Center Program		0804321083
SC	Charleston	Medical University of South Carolina Program		0804521099
TN	Memphis	University of Tennessee Program	2	0804721084
TN	Nashville	Vanderbilt University Program	3	0804721098
TX	Dallas	University of Texas Southwestern Medical School Program	8	0804821085
TX	Fort Sam Houston	San Antonio Uniformed Services Health Education Consortium (SAUSHEC) Program		0804821121
TX	Galveston	University of Texas Medical Branch Hospitals Program	2	0804811086
TX	Houston	Baylor College of Medicine Program	3	0804821087
TX	Houston	University of Texas at Houston Program	4	0804821100
TX	Lubbock	Texas Tech University (Lubbock) Program	3	0804821105
TX	San Antonio	University of Texas Health Science Center at San Antonio Program		0804822088
TX	Temple	Scott and White Clinic	2	0804821133
UT	Salt Lake City	University of Utah Program		0804921112
VA	Charlottesville	University of Virginia Program	2	0805111089
VA	Norfolk	Eastern Virginia Medical School	2	0805121130
VA	Richmond	Medical College of Virginia/Virginia	2	0805121090

		Commonwealth University Program		
VT	Burlington	University of Vermont Program	1	0805013129
WA	Seattle	University of Washington Program	3	0805431091
WI	Madison	University of Wisconsin Program	2	0805621093
WI	Marshfield	Marshfield Clinic-St. Joseph's Hospital Program	1	0805622131
WI	Milwaukee	Medical College of Wisconsin Program		0805621095
WV	Morgantown	Department of Medicine, Section of Dermatology		0805511092

This guide is based on the 2005 University of Miami Medical Student Guide by Jacob, Zell, Berman and Schachner.

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