

Pediatric Dermatology

4th
Edition

A QUICK REFERENCE GUIDE



EDITORS

Anthony J. Mancini, MD, FAAP, FAAD

Daniel P. Krowchuk, MD, FAAP

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN®



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Section on Dermatology
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Published by the American Academy of Pediatrics

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Printed in the United States of America

MA0997

3-356/1020 1 2 3 4 5 6 7 8 9 10

ISBN: 978-1-61002-458-7

eISBN: 978-1-61002-459-4

EPUB: 978-1-61002-493-8

Cover and publication design by LSD DESIGN, LLC

Library of Congress Control Number: 2020933085

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The editors wish to thank J. Thomas Badgett, MD, PhD, FAAP, for his contributions to the first 2 editions of this book and Amy Jo Nopper, MD, FAAP, FAAD, and Michael L. Smith, MD, FAAP, FAAD, for their contributions to the first 3 editions of this book.

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To our families—Nicki, Mallory, Christopher, Mackenzie,
and Alexander; Heidi and Will—whose understanding and
support made this project possible.

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Foreword

Concerns relating to the skin are common reasons for parents to seek medical care for their children. Data from several sources indicate that up to 20% of child visits to pediatricians or family physicians involve a dermatologic problem as the primary reason for the visit, a secondary concern, or an incidental finding on physical examination. The volume of skin-related concerns and the supply-demand crunch for dermatologic referrals mandate that primary care physicians who care for children are prepared to recognize, diagnose, and treat common cutaneous disorders.

This guide was originally designed to be a practical, easy-to-use tool for the busy practitioner, and we hope that this fourth edition continues to meet these goals. It is not an exhaustive reference; instead, it provides a concise summary of many common dermatologic disorders, with a standardized format that includes a brief background, physical findings, diagnostic modalities, and treatment approaches. Each chapter includes a useful Look-alikes table to assist in differential diagnosis and, when applicable, a Resources for Families section that provides links to patient information or support groups. Chapters to help enhance skills in recognizing and describing skin disorders, performing and interpreting diagnostic tests, and managing skin disease also are included. The accompanying color photographs have been selected to illustrate some cardinal features of each disorder. In this edition, we have added new chapters on cercarial dermatitis, confluent and reticulated papillomatosis, ectodermal dysplasia, hyperhidrosis, pilomatricoma, pityriasis rubra pilaris, Spitz nevus, and subcutaneous fat necrosis. We have also updated the text throughout, supplied new links to useful patient resources, and added or replaced numerous clinical images.

We hope this guide continues to fulfill an important need for the pediatric practitioner who wants a quick dermatology reference.

A.J.M.

D.P.K.

Editors' Note

The information contained in this text has been gleaned from reviews of multiple scientific papers and textbooks. The materials have been synthesized into what we hope is a coherent, easy-to-read style. Individual references have not been included in an effort to keep the size of this work practical for a quick reference guide. Some textbook references are listed here, and we invite the reader to refer to updated medical publications for further information or contemporary scientific updates.

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Textbook References

Bolognia JL, Schaffer JV, Cerroni L, eds. *Dermatology*. 4th ed. Elsevier; 2018

Eichenfield LF, Frieden IJ, eds. *Neonatal and Infant Dermatology*. 3rd ed. Elsevier Saunders; 2015

Paller AS, Mancini AJ. *Hurwitz Clinical Pediatric Dermatology*. 5th ed. Elsevier; 2016

Schachner LA, Hansen RC. *Pediatric Dermatology*. 4th ed. Mosby Elsevier; 2011

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All figures not included in the following list are courtesy of the American Academy of Pediatrics. Special thanks to Anthony J. Mancini, MD, FAAP, FAAD; Daniel P. Krowchuk, MD, FAAP; J. Thomas Badgett, MD, PhD, FAAP; Anna L. Bruckner, MD, FAAP, FAAD; Fred Ghali, MD; Amy Jo Nopper, MD, FAAP, FAAD; Ingrid Polcari, MD, FAAP; Steven D. Resnick, MD; Michael L. Smith, MD, FAAP, FAAD; and Patricia A. Treadwell, MD, FAAP, FAAD.

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Figure 1.10

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Figure 87.5

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Figures 7.8, 96.5

Approach to the Patient With a Rash

Introduction

- ▶ Recognizing and describing skin lesions accurately is essential to the diagnosis and differential diagnosis of skin disorders.
- ▶ The first step is to identify the primary lesion, defined as the earliest lesion and the one most characteristic of the disease.
- ▶ Next note the distribution, arrangement, and color of primary lesions, along with any secondary change (eg, crusting or scaling).

Types of Primary Lesions

- ▶ Flat lesions
 - Macule: a small (<1 cm), circumscribed area of color change without elevation or depression of the skin (Figure 1.1)
 - Patch: a larger (≥ 1 cm) area of color change without skin elevation or depression (Figure 1.2)
- ▶ Elevated lesions
 - Solid lesions
 - Papules (<1 cm in diameter) (Figure 1.3)
 - Nodules: lesion measuring 0.5 to 2.0 cm in diameter, most of which resides below the skin surface (Figure 1.4)
 - Tumor: deeper than a nodule and measuring larger than 2 cm in diameter
 - Wheals: pink, rounded, or flat-topped elevations due to edema in the skin (Figure 1.5)
 - Plaques: plateau-shaped structures often formed by the coalescence of papules; larger than 1 cm in diameter (Figure 1.6)



Figure 1.1. Café au lait macules (spots) in a patient who has neurofibromatosis type 1.



Figure 1.2. A port-wine stain—a vascular patch.



Figure 1.3. Molluscum contagiosum. There are erythematous and skin-colored papules.



Figure 1.4. Nodules representing neurofibromas in a patient who has neurofibromatosis type 1.



Figure 1.5. Pink wheals in a patient who has urticaria.



Figure 1.6. Scaling plaques, plateau-like lesions, are observed in psoriasis.

- Fluid-filled lesions
 - Vesicles: smaller than 1 cm in diameter and filled with serous or clear fluid (Figure 1.7)
 - Bullae: 1 cm or larger in diameter and typically filled with serous or clear fluid (Figure 1.8)
 - Pustules: smaller than 1 cm in diameter and filled with purulent material (Figure 1.9)
 - Abscess: 1 cm or larger and filled with purulent material
 - Cysts: 0.5 cm or larger in diameter; represent sacs containing fluid or semisolid material (unlike in bullae, the material within a cyst is not visible from the surface)
- ▶ Depressed lesions
 - Erosions: superficial loss of epidermis with a moist base (Figure 1.10)
 - Ulcers: deeper lesions extending into the dermis or below (Figure 1.11)



Figure 1.7. Vesicles, as seen here in varicella, are filled with clear or serous fluid.



Figure 1.8. Bullae, filled with clear fluid, are observed in chronic bullous disease of childhood.

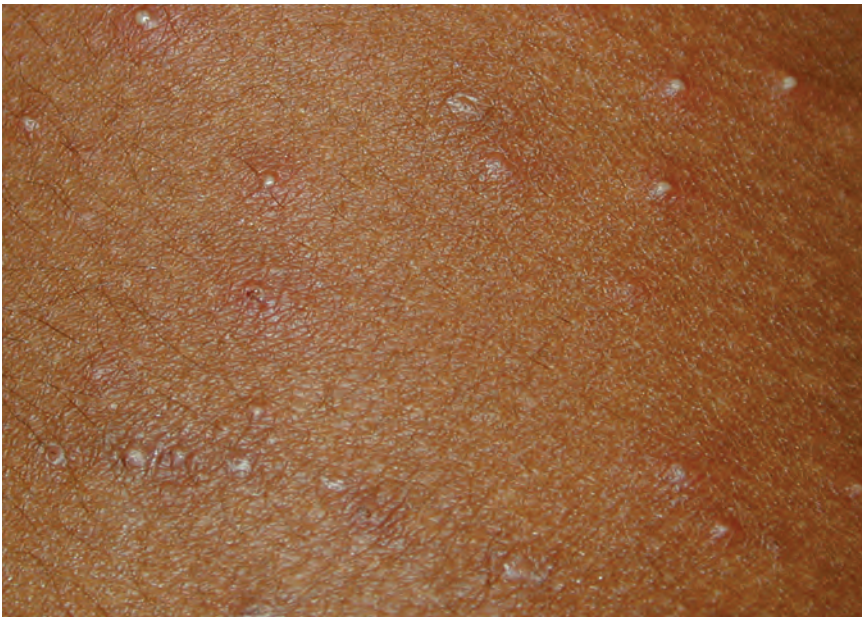


Figure 1.9. Pustules are filled with purulent material. This patient has folliculitis.

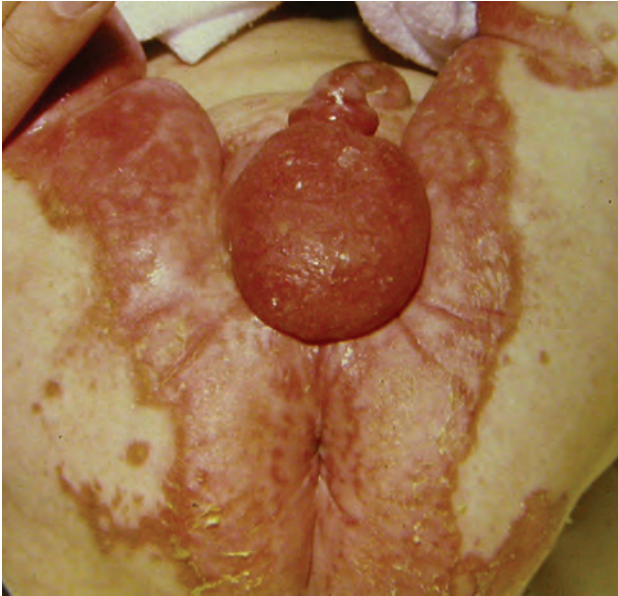


Figure 1.10. Erosions, as seen in this infant who has acrodermatitis enteropathica, represent a superficial loss of epidermis.



Figure 1.11. An ulcer occurs when there has been loss of epidermal and dermal tissues. In the patient shown here, the ulcer is the result of pyoderma gangrenosum.

Distribution of Lesions

Certain disorders are characterized by unique patterns of lesion distribution. For example,

- ▶ Atopic dermatitis in children and adolescents typically involves the antecubital or popliteal fossae.
- ▶ Seborrheic dermatitis in adolescents commonly involves not only the scalp but also the eyebrows and nasolabial folds.
- ▶ Lesions of psoriasis are often seen in areas that are traumatized, such as the extensor surfaces of the elbows and knees.
- ▶ Acne is limited to the face, back, shoulders, and chest, sites of the highest concentrations of pilosebaceous follicles.

Arrangement of Lesions

The arrangement of lesions also may provide a clue to diagnosis. Some examples include

- ▶ Linear: contact dermatitis due to plants (eg, poison ivy) (Figure 1.12), lichen striatus, and incontinentia pigmenti; may also occur in epidermal nevi, psoriasis, and warts
- ▶ Grouped: herpes simplex virus infection (Figure 1.13), warts, molluscum contagiosum, microcystic lymphatic malformation
- ▶ Dermatomal: herpes zoster (Figure 1.14)
- ▶ Annular (ie, ring-shaped with central clearing): tinea corporis (Figure 1.15), granuloma annulare, erythema migrans, lupus erythematosus



Figure 1.12. A linear arrangement of papules or vesicles often occurs in contact dermatitis due to poison ivy.



Figure 1.13. Grouped vesicles are characteristic of herpes simplex virus infection on the skin.



Figure 1.14. The lesions of herpes zoster appear in a dermatomal distribution.



Figure 1.15. An annular (ring-shaped with central clearing) plaque is typical of tinea corporis.

Color

- ▶ Erythematous: pink or red. When erythematous lesions are observed, it is important to note if they blanch. If the red cells are within vessels, as occurs in urticaria, compression of the skin forces the cells into deeper vessels and blanching occurs. However, if the cells are outside vessels, as occurs in forms of vasculitis, blanching will not occur. Non-blanching lesions are termed *petechiae*, *purpura*, or *ecchymoses*. Also note that in individuals with more deeply pigmented skin, erythema may be more difficult to appreciate.
- ▶ Hyperpigmented: tan, brown, or black.
- ▶ Hypopigmented: amount of pigment decreased but not entirely absent (as seen with postinflammatory pigmentary alteration).
- ▶ Depigmented: all pigment absent (as occurs in vitiligo).

Secondary Changes

Alterations in the skin that may accompany primary lesions include

- ▶ Excoriation: a superficial loss of skin (ie, an erosion) caused by scratching, picking, or rubbing.
- ▶ Crusting: dried fluid; commonly seen following rupture of vesicles or bullae (as occurs with the “honey-colored” crust of impetigo).
- ▶ Scaling: represents epidermal fragments that are characteristic of several disorders, including fungal infections (eg, tinea corporis) and psoriasis.
- ▶ Atrophy: an area of surface depression due to absence of the epidermis, dermis, or subcutaneous fat; atrophic skin often is thin and wrinkled. Examples include steroid atrophy, morphea, and atrophoderma.
- ▶ Lichenification: thickening of the skin from chronic rubbing or scratching (as occurs in atopic dermatitis); as a result, normal skin markings and creases appear more prominent (Figure 1.16).

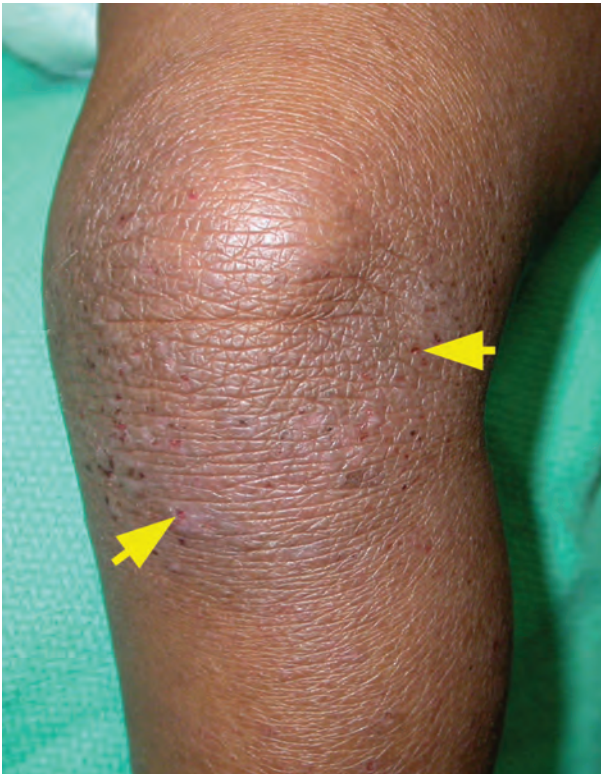
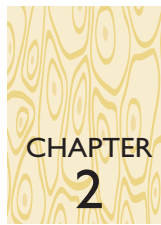


Figure 1.16. Lichenification. The normal skin markings are very prominent due to chronic scratching. Also note the tiny erosions (arrows), some of which have formed crust.



Diagnostic Techniques

Introduction

Several procedures can assist the clinician in diagnosing skin problems. Discussed here are the potassium hydroxide (KOH) preparation, fungal culture, mineral oil preparation for scabies, and Wood light examination.

Potassium Hydroxide (KOH) Preparation

Used to identify fungal elements (eg, spores, hyphae, pseudohyphae) in skin, hair, or nail samples. The procedure is as follows:

- ▶ Using the edge of a glass microscope slide or #15 scalpel blade, scrape the skin and collect fragments or hair remnants on a second glass microscope slide. Preparing the area first with alcohol may be useful in helping debris stick to blade or slide.
- ▶ If sampling a nail, use a scalpel blade to scrape the underside of the nail (or its surface if superficial infection is suspected) and collect the debris obtained.
- ▶ Cover the specimen on the glass slide with a cover slip.
- ▶ Apply 1 to 2 drops of 10% to 20% KOH to the edge of the cover slip. Capillary action will draw the liquid under the entire cover slip.
- ▶ Gently heat the slide with an alcohol lamp or match, taking care to avoid boiling (which causes the KOH to crystallize and makes interpretation of the preparation difficult).
- ▶ Gently compress the cover slip to further separate skin fragments.
- ▶ Scan the preparation initially under low power (using the 10× objective lens).

- ▶ Examine any suspicious areas under higher power (using a 40× objective lens) for
 - Branching hyphae or spores: characteristic of dermatophyte infections of the skin or nails (eg, tinea corporis, tinea pedis, tinea cruris, onychomycosis) (Figure 2.1).
 - Spores within hair fragments (ie, an endothrix infection): characteristic of the most common form of tinea capitis in the United States caused by *Trichophyton tonsurans* (Figure 2.2). If tinea capitis is caused by *Microsporum canis* (approximately 5% of all cases), hyphae or spores will be seen on the outside of hair shafts (ie, an ectothrix infection).
 - Pseudohyphae and spores: seen in infections with *Candida* species (Figure 2.3).
 - Spores and short hyphae (ie, “spaghetti and meatballs”): seen in tinea versicolor (Figure 2.4).

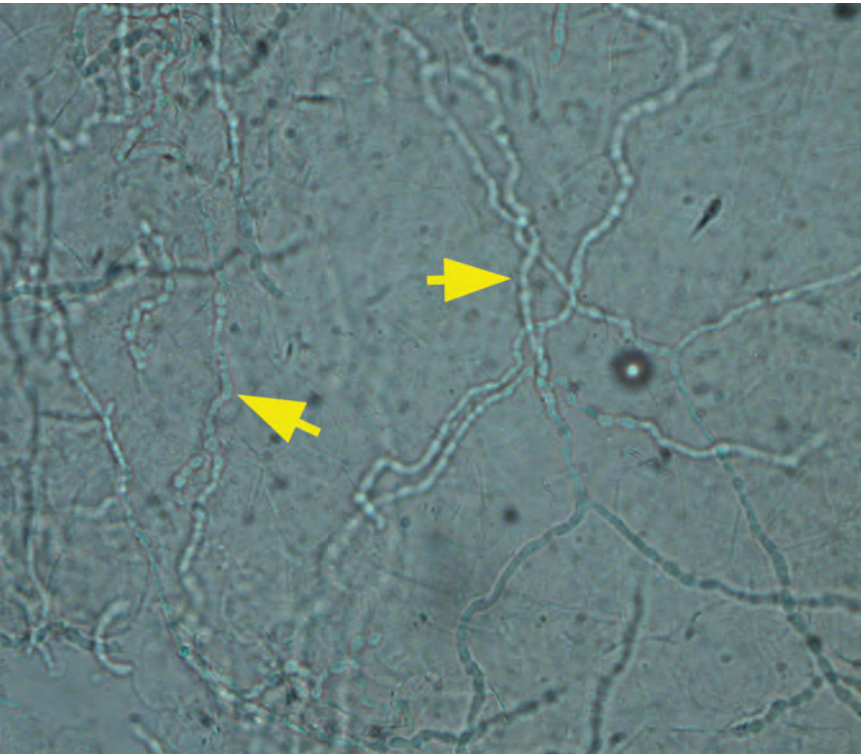


Figure 2.1. Potassium hydroxide preparation showing branching hyphae (arrows).

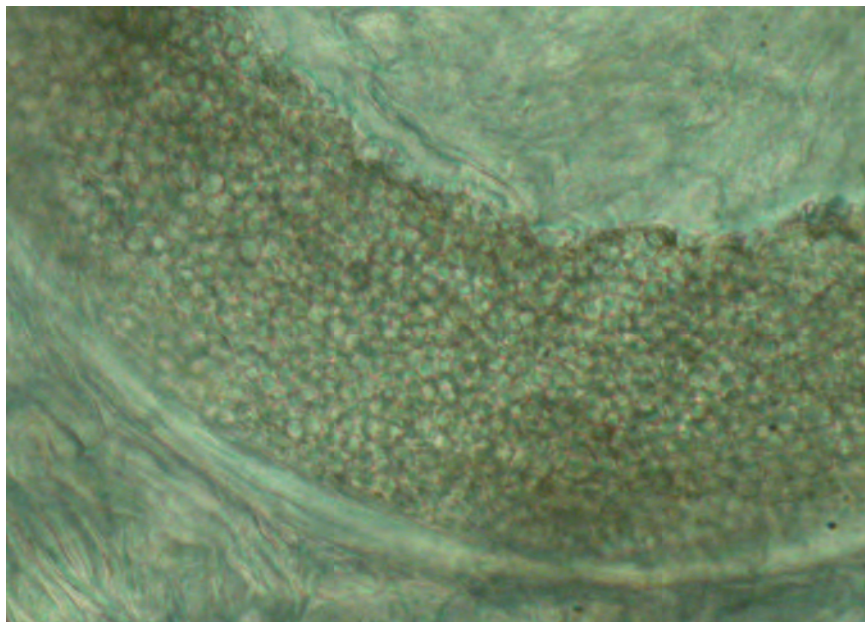


Figure 2.2. Potassium hydroxide preparation in tinea capitis caused by *Trichophyton tonsurans*. The hair fragment is filled with small spheres (ie, arthrospores).

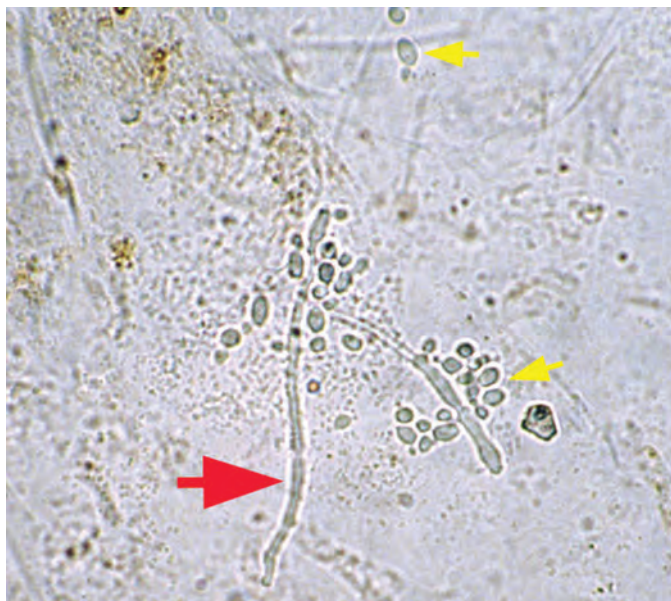


Figure 2.3. Pseudohyphae (red arrow) and spores (yellow arrows) are characteristic of infection caused by *Candida* species.

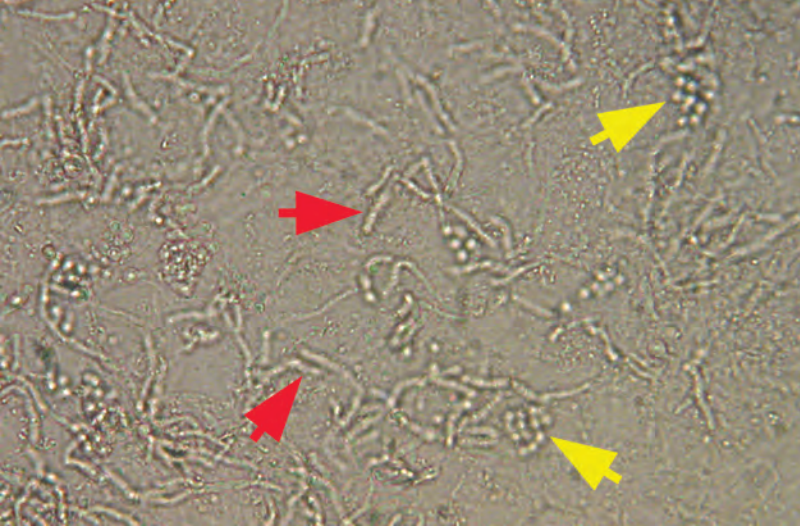


Figure 2.4. In tinea versicolor, the potassium hydroxide preparation reveals short hyphae (red arrows) and spores (yellow arrows) (“spaghetti and meatballs”).

Fungal Culture

- ▶ Sampling techniques
 - If sampling the skin: Using the edge of a glass microscope slide or #15 scalpel, scrape the lesion and collect scale on a glass microscope slide.
 - If sampling a nail: Use a scalpel blade to scrape the underside of the nail (or its surface if superficial infection is suspected), and collect the debris on a glass microscope slide or folded sheet of paper; alternatively, use a nail clipper to obtain nail clippings.
 - If sampling the scalp: Moisten a cotton-tipped applicator with tap water, rub the affected area of the scalp, and inoculate the fungal culture medium with the swab. (If fungal culture medium is not available, a culturette swab system may be used to collect and transport the specimen to the laboratory.)

- ▶ Transfer the material collected to the fungal medium (typically dermatophyte test medium [DTM] or Mycosel agar) and process appropriately.
 - Leave the cap slightly loose to permit air entry.
 - If fungal culture medium is not available, transfer the specimen in a sterile glass tube or other container to the laboratory.
- ▶ In the presence of a pathogenic fungus, DTM will change from yellow to red in 1 to 2 weeks (Figure 2.5).



Figure 2.5. Uninoculated dermatophyte test medium is yellow (left). In the presence of a pathogenic fungus the medium becomes red (right).

Mineral Oil Preparation for Scabies

- ▶ Place a small drop of mineral oil on a suspicious burrow, papule, or vesicle that has not been traumatized by the patient.
- ▶ Using a #15 scalpel blade oriented parallel to the skin surface, scrape the lesion. Because scabies mites live in the epidermis, it is not necessary to scrape deeply; however, some bleeding is common with the procedure.
- ▶ Transfer the material to a drop of mineral oil on a glass microscope slide.
- ▶ Repeat the process for several other suspicious lesions.
- ▶ Cover the sample on the glass slide with a cover slip (add a few more drops of mineral oil if necessary for uniform distribution).
- ▶ Examine at low power for the presence of mites, eggs, or fecal material (Figures 2.6 and 2.7).

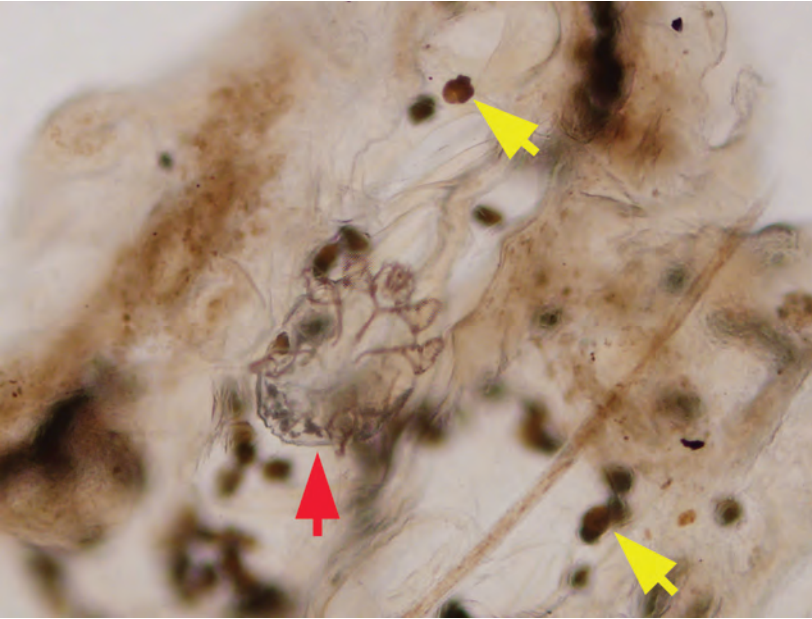


Figure 2.6. Newly hatched mite (red arrow) and fecal material (yellow arrows) on a mineral oil preparation.

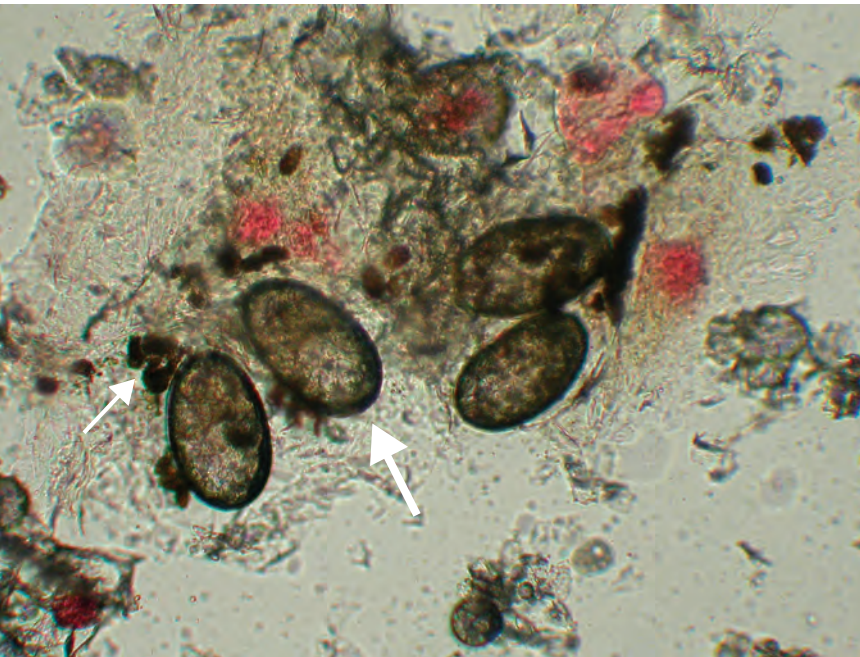


Figure 2.7. A mineral oil preparation in a patient who has scabies reveals eggs (large arrow) and mite fecal material (ie, scybala) (small arrow).

Wood Light Examination

Examination of the skin using Wood light in a darkened room may assist in the diagnosis of several conditions.

- ▶ Erythrasma (a superficial *Corynebacterium* infection): Affected areas fluoresce a coral-red color.
- ▶ Tinea capitis: Wood light examination is only useful in the recognition of a minority of cases (perhaps 5%) of tinea capitis caused by *Microsporum* species (Figure 2.8). Green fluorescence does not occur when infections are caused by *T tonsurans*.
- ▶ Tinea versicolor (caused by yeasts of the genus *Malassezia* [formerly *Pityrosporum*]): Affected areas may fluoresce a yellow-gold color.
- ▶ Diseases characterized by hypopigmentation or depigmentation: In lightly pigmented individuals, examining the skin with Wood light may assist in identifying lesions of vitiligo or ash-leaf macules of tuberous sclerosis.



Figure 2.8. Wood light examination in tinea capitis caused by *Microsporum canis*. There is green fluorescence of affected hairs.

Therapeutics

I. Selection and Use of Topical Corticosteroids

Introduction

- ▶ Topical corticosteroids exert their effect through many mechanisms, including anti-inflammatory, immunosuppressive, antiproliferative, and vasoconstrictive effects.
- ▶ Preparations may be grouped according to relative potency (Table 3.1). Differences in potency between groups are not linear. For example, hydrocortisone (group 7) has a relative potency of less than 1; triamcinolone (eg, Kenalog, group 4), 75; and clobetasol propionate (eg, Temovate, group 1), 1,869.

Table 3.1. Selected Topical Corticosteroids by Potency

Group	Generic Name (Brand Name, Vehicle, Concentration)
Group 1 (most potent)	Betamethasone dipropionate (Diprolene, ointment, 0.05%) Clobetasol propionate (Temovate, cream or ointment, 0.05%; Olux, foam, 0.05%) Diflorasone diacetate (Psorcon, ointment, 0.05%) Fluocinonide (Vanos, cream, 0.1%) Halobetasol propionate (Ultravate, cream or ointment, 0.05%)
Group 2	Amcinonide (Cyclocort, ointment, 0.1%) Betamethasone dipropionate (Diprosone, cream or ointment, 0.05%) Betamethasone valerate (Luxiq, foam, 0.12%) Fluocinonide (Lidex; cream, ointment, gel, or solution; 0.05%) Mometasone furoate (Elocon, ointment, 0.1%)
Group 3	Amcinonide (Cyclocort, cream or lotion, 0.1%) Diflorasone diacetate (Psorcon, cream, 0.05%) Fluticasone propionate (Cutivate, ointment, 0.005%) Triamcinolone acetonide (Aristocort, ointment, 0.1%)
Group 4	Fluocinolone acetonide (Synalar, ointment, 0.025%) Hydrocortisone valerate (Westcort, ointment, 0.2%) Mometasone furoate (Elocon, cream, lotion, 0.1%) Triamcinolone acetonide (Kenalog, cream, 0.1%)

Table 3.1 (continued)

Group	Generic Name (Brand Name, Vehicle, Concentration)
Group 5	Betamethasone valerate (Valisone, cream, 0.1%) Fluocinolone acetonide (Synalar, cream, 0.025%) Fluticasone propionate (Cutivate, cream, 0.05%) Hydrocortisone valerate (Westcort, cream, 0.2%) Prednicarbate (Dermatop, cream, 0.1%)
Group 6	Alclometasone dipropionate (Aclovate, cream or ointment, 0.05%) Desonide (Tridesilon, cream, 0.05%; DesOwen, cream or ointment, 0.05%; Desonate, gel, 0.05%; Verdeso, foam, 0.05%) Fluocinolone acetonide (Synalar, solution, 0.01%; Derma-Smoothe/FS, oil, 0.01%)
Group 7 (least potent)	Hydrocortisone (Hytone, cream or ointment, 1%, 2.5%)

Adapted with permission from Eichenfield LF, Friedlander SF. Coping with chronic dermatitis. *Contemp Pediatr.* 1998;15(10):53–80 and Eichenfield LF, Boguniewicz M, Simpson EL, et al. Translating atopic dermatitis management guidelines into practice for primary care providers. *Pediatrics.* 2015;136(3):554–565 PMID: 26240216 <https://doi.org/10.1542/peds.2014-3678>.

Selecting and Prescribing a Topical Corticosteroid

Consider the following factors when selecting a topical corticosteroid (Table 3.2):

- ▶ How old is the patient?
 - In general, a less potent preparation is required in infants than in older children or adolescents. For example, for the management of flares of atopic dermatitis, a low-potency preparation (eg, hydrocortisone ointment 1% or 2.5%) usually is sufficient in an infant, while in an adolescent, a mid-potency (eg, triamcinolone 0.1%) or high-potency (eg, mometasone 0.1%) product is needed.
- ▶ What area will be treated?
 - Absorption of steroids varies with the thickness of the skin in various regions of the body.
 - Absorption is greatest in areas where the skin is thin (eg, face, perineum) and lowest where the skin is thick (eg, palms, soles). Thus, only a low-potency preparation should be used on the face, while a mid-potency (or high-potency) product will be needed to manage dermatitis on the feet.
 - Absorption is also increased in occluded or warm and opposed areas of skin. Hence, in areas such as the axillae, groin, or diapered area of an infant, low-potency preparations are typically recommended.

- ▶ What vehicle should be selected?
 - Creams: tolerated by most patients but can be drying and, occasionally, their ingredients may cause burning or contact dermatitis.
 - Ointments: the most effective vehicle, especially for thickened or lichenified skin; increase the absorption and potency ranking of the steroid; generally are preservative-free and less likely to cause contact or irritant dermatitis; have a greasy feel that may not be tolerated by some patients.
 - Lotions: cosmetically pleasing because they do not leave a greasy feel; tend to sting on open or damaged skin.
 - Gels: usually for hair-bearing areas; may cause stinging or burning.
- ▶ How much should you dispense?
 - For treatment of a self-limited condition involving a small area, prescribing a small tube (eg, 15 g) will be sufficient; however, if the process is more extensive or chronic, larger amounts will be needed. Some rules that will help include the following:
 - One gram of product will cover a 10-cm by 10-cm area (perhaps 30% more coverage if an ointment is used rather than a cream). Note that 0.5 g is the amount of cream dispensed from a standard tube that extends from the tip of the adult finger to the flexural crease overlying the volar distal interphalangeal joint.
 - In an older child (6–10 years of age), it takes
 - 1 g to cover the face and neck
 - 1.25 g to cover the hand and arm
 - 1.75 g to cover the chest and abdomen
 - 2.25 g to cover the foot and leg
 - Thus, when managing a chronic condition like atopic dermatitis that involves a significant portion of the body, prescribing amounts of 0.5 or 1 lb (227 or 454 g), rather than small tubes, may be necessary.
- ▶ Cost
 - As with other medications, the cost of topical corticosteroids varies widely and often is influenced by the patient's insurance formulary. Although proprietary corticosteroids typically are more expensive than generics, generics are not always inexpensive. There are insufficient data, however, to enable direct comparison of efficacy and bioavailability of branded versus generic preparations.

Table 3.2. Guidelines for Selecting Corticosteroid Potency

Potency	Guideline
Low	<ul style="list-style-type: none"> • Infant (any body site) or young child • Face, perineum, axillae in patient of any age
Moderate	<ul style="list-style-type: none"> • Child (exclusive of face) with moderate to severe disease • Adolescent (exclusive of face or anatomically occluded areas [eg, axillae, genitalia])
High	<ul style="list-style-type: none"> • Used primarily by dermatologists • Most often used in the management of severe or lichenified dermatoses or those involving the feet or hands

Adverse Effects

When used appropriately topical corticosteroids are very safe; however, using too potent a preparation, particularly in an inappropriate location or for too long, may result in adverse effects.

- ▶ Local adverse effects: atrophy, striae, pigmentary changes, easy bruising, hypertrichosis, and acne-like eruptions. To prevent these effects, use only low-potency preparations on the face, axillae, and groin (including the diaper area); limit the duration of use of all corticosteroids; and use high-potency preparations very discriminately.
- ▶ Systemic adverse effects: hypothalamic-pituitary-adrenal axis suppression, Cushing syndrome, growth retardation, glaucoma, and cataracts. Systemic adverse effects are most likely to occur when very potent agents are used (even for short periods) or when moderately potent preparations are used over large areas of the body for long periods, especially in young infants, where the ratio of skin to body surface area is larger.

Frequency of Application

- ▶ Typically twice daily as needed

II. Selection and Use of Moisturizers

Introduction

- ▶ Moisturizers (also known as emollients or lubricants) are designed to hydrate the skin by creating a barrier and preventing evaporation.
- ▶ In patients who have atopic dermatitis, moisturizers can reduce the need for corticosteroids.

Selecting a Moisturizer

Traditional moisturizers are available as ointments, creams, or lotions. Barrier repair agents also are available.

- ▶ Ointments
 - Water-in-oil emulsions are most occlusive and are the best moisturizers.
 - Have a greasy feel that some patients find unpleasant.
 - Because they generally are preservative-free, they are less likely to cause contact or irritant dermatitis.
 - Some examples include Aquaphor ointment, CeraVe healing ointment, and petrolatum (eg, Vaseline petroleum jelly).
- ▶ Creams
 - Oil-in-water emulsions that often are more cosmetically pleasing than ointments.
 - Some examples include CeraVe cream, Cetaphil cream, Eucerin cream, and Vanicream.
- ▶ Lotions
 - Oil-in-water emulsions containing more water than creams.
 - Cosmetically pleasing but least effective as moisturizers.
 - Some examples include CeraVe lotion, Cetaphil lotion, Currel lotion, DML lotion, Eucerin lotion, Keri lotion, Lubriderm lotion, and Moisturel lotion.
- ▶ Barrier repair agents
 - A variety of over-the-counter (eg, CeraVe, Cetaphil RestoraDerm) and prescription (eg, Atopiclair, EpiCeram, Hylatopic) barrier repair agents exist that may help reduce the severity of atopic dermatitis and play an adjunctive therapeutic role. These agents include products with ceramides, filaggrin degradation products, natural moisturizing factor, avenanthramides, glycyrrhetic acid, shea nut derivatives, and palmitamide monoethanolamine.

- While the exact role of these agents is unclear, they may play a role in active disease (usually in conjunction with anti-inflammatory agents such as corticosteroids and calcineurin inhibitors) and as maintenance agents.
- Prescription barrier repair agents typically are expensive.

Adverse Effects

Preservatives, antimicrobial agents, or fragrances contained in moisturizers, or products that are lanolin-based, may cause allergic or irritant contact dermatitis.

Frequency of Application

- ▶ Apply 2 to 3 times daily if needed (application should immediately follow a bath or shower while the skin is still damp).
- ▶ Lotions and creams may need to be applied more often than ointments.
- ▶ If the patient is being treated with a topical corticosteroid, calcineurin inhibitor, or phosphodiesterase 4 inhibitor, apply these agents first, followed by the moisturizer.

III. Cryotherapy

Introduction

Cryotherapy employs liquid nitrogen (or another cryogen) to destroy skin lesions through tissue necrosis. In pediatrics it is commonly used to treat warts.

Selecting a Cryogen

- ▶ Liquid nitrogen is the most effective cryogen, with an achieved temperature of approximately -195°C (-319°F).
- ▶ If cryotherapy will be performed infrequently, products that employ other cryogens (eg, dimethyl ether and propane [eg, Histofreezer]) may prove more economical for a practice because they have a long shelf life, although their effectiveness and freeze effect (temperature around -57°C [-70.6°F]) are significantly lower than liquid nitrogen.
- ▶ Some cryotherapy devices can also be purchased by patients without a prescription. They also contain dimethyl ether and propane.

Procedure

- ▶ Liquid nitrogen usually is applied with a spray device or a cotton swab that is dipped into the liquid nitrogen and then applied to the skin.
 - Standard cotton-tipped applicators do not work well because the tight wrap of the cotton does not allow liquid nitrogen to be absorbed.
 - To make an applicator, wrap additional cotton onto the tip of an applicator, shaping it to a point.
- ▶ Liquid nitrogen should be applied to the lesion until a white ring (the ice ball) extends 1 to 3 mm beyond the margin of the wart. The freeze should be maintained for 10 to 30 seconds. Some experts advise a second treatment following initial thawing.
- ▶ Patients should be advised that within 1 to 2 days a blister may form. Once the blister ruptures, the area should be cleansed twice daily and a topical antibiotic and a bandage applied.
- ▶ Any remaining wart should be treated with a keratolytic that contains salicylic acid. Repeat cryotherapy may be performed in 2 to 3 weeks if necessary.

IV. Sun Protection

Elements of Sun Protection

- ▶ Minimize prolonged outdoor activities between 10:00 am and 4:00 pm when possible.
- ▶ Wear protective clothing, such as a wide-brimmed hat, long-sleeved shirt, and long pants. Many manufacturers produce sun-protective clothing with a UV protection factor (approximately equivalent to the sun protection factor [SPF]) of 30 or more.
- ▶ Use a sunscreen regularly.
 - Choose a product with an SPF of 30 or more that has UV-A and UV-B protection (ie, is labeled “broad spectrum”). Sun protection factor is a measure of protection from UV-B. At present, there is no rating system for UV-A protection (although zinc oxide and avobenzone are the ingredients most active against UV-A). With respect to sunscreen active ingredients
 - Zinc oxide and titanium dioxide (physical sunscreen ingredients) are generally recognized as safe and effective by the US Food and Drug Administration (FDA); trolamine salicylate and para-aminobenzoic acid are not. For the remaining 12 chemical sunscreens, data are insufficient to determine their status.
 - Of note, in a recent study, plasma concentrations of the chemical sunscreens avobenzone, oxybenzone, octocrylene, and ecamsule were found to exceed the FDA threshold for waiving toxicology studies. These agents were applied in “maximal use” conditions (ie, consistent with FDA recommendations for use): liberal application (2 mg/1 cm²) every 2 hours to areas not covered by a swimsuit for 4 days, as might occur during a vacation at the beach. The clinical significance of these findings is unknown, but additional study is needed to understand the implications of sunscreen absorption. Pending this, individuals should not stop using sunscreen (*JAMA*. 2019;321[21]:2082–2091 PMID: 31058986 <https://doi.org/10.1001/jama.2019.5586>).

- Oxybenzone can act as an endocrine disruptor with an estrogen-like effect. It also may inhibit the migration of neural crest cells during embryogenesis. Women with high levels of urine oxybenzone had a greater than expected risk of giving birth to neonates with Hirschsprung disease (congenital megacolon). Pending further study, oxybenzone should be avoided in infants, children, and adolescents and during pregnancy.
- Consider a product that is not alcohol-based (ie, will not cause stinging) and that is labeled non-acnegenic or noncomedogenic (ie, to prevent worsening acne in adolescents).
- Apply liberally (using too little may reduce the SPF), ideally 30 minutes before beginning outdoor activities, even on cloudy days.
- Apply every 2 hours, as well as after swimming or activities resulting in significant sweating.
- Although there are limited data on the safety of sunscreen use in infants younger than 6 months, there is no evidence that applying small amounts is associated with adverse long-term effects. Therefore, in situations in which other sun protection strategies may be inadequate or unfeasible, it is reasonable to apply sunscreen to exposed areas of the skin in young infants.
- ▶ To prevent cataracts and ocular melanoma, wear sunglasses that are labeled as blocking 100% of UV-A and UV-B rays.

Dermatitis

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Atopic Dermatitis

Introduction/Etiology/Epidemiology

- ▶ Most common chronic pediatric skin disorder, affecting as many as 15% of children.
- ▶ Cause unknown but appears to be the result of a complex interplay between immune dysregulation, barrier dysfunction, and the environment.
- ▶ Strong genetic predisposition; many patients have personal or family history of atopy.
- ▶ Generally begins during infancy or childhood; 90% of those ultimately affected present before 5 years of age.
- ▶ Children who have atopic dermatitis are susceptible to certain bacterial and viral infections.
 - Increased adherence of *Staphylococcus aureus* to the skin and reduced production of antimicrobial peptides may explain the high rates of colonization with and infection due to this bacterium.
 - Altered T-cell function may explain the predisposition of children to develop molluscum contagiosum, eczema herpeticum, and eczema vaccinatum.

Signs and Symptoms

- ▶ Characterized by pruritus with resultant scratching that leads to excoriations and lichenification.
- ▶ The appearance of lesions varies with the patient's age and racial background.
 - Infants and toddlers: involvement of the face, trunk, and extensor extremities (Figures 4.1 and 4.2).
 - Childhood: Lesions are concentrated in flexural areas, such as the antecubital and popliteal fossae, wrists, and ankles (Figures 4.3 and 4.4). Some children exhibit round, crusted lesions (ie, nummular [coin-shaped] eczema, Figure 4.5); in older children, the feet may be involved (Figure 4.6).