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**CLINICAL DERMATOLOGY: A COLOR GUIDE TO DIAGNOSIS AND THERAPY**  

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each drug to be administered to verify the recommended dose, the method and duration  
of administration, and contraindications. It is the responsibility of the licensed prescriber,
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Preface

Clinical Dermatology is intended to be a practical resource for the busy clinician. Over 1000 illustrations are combined with disease descriptions and current and comprehensive therapeutic information. Bold headings are used to facilitate rapid access to information. Diseases can be accessed in many ways.

The classic method of organizing skin diseases is used. Common diseases are covered in depth. Illustrations of classic examples of these disorders and photographs of variations seen at different stages are included. Basic dermatologic surgical techniques are covered in detail. Specialized techniques such as Mohs' micrographic surgery are described so that the physician can be better prepared to suggest referral. Theoretical information, disease mechanisms, and rare disease are found in comprehensive textbooks.

Rapid Access to the Text

1. List of disorders with page references—inside front cover.
2. List of common topical steroids—follows disorders pages.
3. List of diseases by region with page references—inside back cover.
4. List of diseases by lesion type with page references—page 3.
5. Formulary is located on pages 945 to 973.

How to Use This Book

Students in the classroom
Students should learn the primary and secondary lesions and the distribution of
diseases in Chapter 1 and study the differential diagnosis of each lesion. Select a few familiar diseases from each list and read about them. Study the close-up pictures carefully. Obtain an overview of the text. Turn the pages, look at the pictures, and read the captions.

Students in the clinic

You see skin abnormalities every day in the clinic. Try to identify these diseases, or ask for assistance. Study all diseases, especially tumors, with a magnifying glass or ocular lens. Read about what you see and you will rapidly gain a broad fund of knowledge.

Study Chapters 20 (Benign Skin Tumors), 21 (Premalignant and Malignant Nonmelanoma Skin Tumors), and 22 (Nevi and Malignant Melanoma). Skin growths are common, and it is important to recognize their features.

House officers are responsible for patient management. Read Chapter 2 carefully, and study all aspects of the use of topical steroids. These valuable agents are used to treat a great variety of inflammatory skin conditions. It is tempting to use these agents as a therapeutic trial and ask for a consultation only if therapy fails. Topical steroids mask some diseases, make some diseases worse, and create other diseases. Do not develop bad habits; if you do not know what a disease is, do not treat it.

The diagnosis of skin disease is deceptively easy. Do not make hasty diagnoses. Take a history, study primary lesions and the distribution, and be deliberate and methodical. Ask for help. With time and experience you will feel comfortable managing many common skin diseases.

The practicing clinician

Most skin diseases are treated by practitioners other than dermatologists. This includes primary care physicians, nurse practitioners and physician assistants. Clinicians involved in direct patient care should read the above guidelines for using this book. Learn a few topical steroids in each potency group. There are a great number of agents in the Formulary. Many in each table contain similar ingredients and have the same therapeutic effect. Develop an armamentarium of agents and gain experience in their use.

Inflammatory conditions are often confusing, and sometimes biopsies are of limited value in their diagnosis. Eczema is common, read Chapters 2 and 3. Acne is seen everyday, read Chapter 7. Managing acne effectively will provide a great service to many young patients who are very uncomfortable with their appearance. The clinical diagnosis of pigmented lesions is complicated. A dermatologist can often make a specific diagnosis without the need for a biopsy.
The dermatologist

Many dermatologists use the pictures as an aid to reassure patients. Examine the patient, make a diagnosis, and then show them an illustration of their disease. Many patients see the similarity and are reassured.

This book is designed to be a practical resource. All of the most current descriptive and therapeutic information that is practical and relevant has been included. All topics are researched on Medline. Details about basic science and complex mechanisms of disease can be found elsewhere. Rare diseases are found in larger textbooks.

Photography

The photographs were taken with medium format cameras, 35-mm macro cameras, and digital macro cameras. The digital images for this edition were taken by me with a Nikon D1 digital camera fitted with a 60?mm macro lens and a Canfield TwinFlash. The macro camera takes pictures that simulate the view through a hand lens. Therefore the distribution of the disease and the primary lesion can be accurately illustrated. Over 4000 new digital images were acquired in preparation for this edition. Alan N. Binnick, MD, Adjunct Assistant Professor of Medicine (Dermatology), Dartmouth Medical School, provided all of the new images taken with transparency film. He has 25 years of experience as a clinician, teacher, and expert photographer. His entire collection was available for this edition.

Production

The author writes the manuscript. The publishing company makes the book. Manufacturing a book is a complicated process. The key people involved in this effort are listed on the title page. As my first editor said 20 years ago, “if people ever realized what was involved in making a book, they would not believe that it could ever get done.”

The layout and design of each page in this book is done the “old fashion way,” by cutting and pasting images and strips of text by the layout artist. Page layout design is a science and an art. Jeanne Genz has designed all four editions of this book. This older, slower, noncomputerized technique created by an expert produces pages that are balanced and of maximum clarity. Computer layout programs are not capable of this art. The final “pasted” book is then converted to a digital file and printed on high-grade glossy paper on a sheetfed press. Glossy paper retains ink at the surface to enhance definition. Sheetfed presses print slowly and allow ink to be laid down precisely so that
exceptional sharpness and color balance are achieved.

2003

Thomas P. Habif
Chapter 1 - Principles of Diagnosis and Anatomy

Illustrated Skin Anatomy

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Skin Anatomy

The skin is divided into three layers: the epidermis, the dermis, and the subcutaneous tissue. The skin is thicker on the dorsal and extensor surfaces than on the ventral and flexor surfaces.

Epidermis

The epidermis is the outermost part of the skin; it is stratified squamous epithelium. The thickness of the epidermis ranges from 0.05?mm on the eyelids to 1.5?mm on the palms and soles. The microscopic anatomy of the epidermal-dermal junction is complex; it is discussed in detail in Chapter 16. The innermost layer of the epidermis consists of a single row of columnar cells called basal cells. Basal cells divide to form keratinocytes (prickle cells), which comprise the spinous layer. The cells of the spinous layer are connected to each other by intercellular bridges or spines, which appear histologically as lines between cells. The keratinocytes synthesize insoluble protein, which remains in the cell and eventually becomes a major component of the outer layer (the stratum corneum). The cells continue to flatten, and their cytoplasm appears granular (stratum granulosum); they finally die as they reach the surface to form the stratum corneum. There are three types of branched cells in the epidermis: the melanocyte, which synthesizes pigment (melanin); Langerhans' cell, which serves as a frontline element in immune reactions of the skin; and Merkel's cell, the function of which is not clearly defined.

Dermis

The dermis varies in thickness from 0.3?mm on the eyelid to 3.0?mm on the back; it is composed of three types of connective tissue: collagen, elastic tissue, and reticular fibers. The dermis is divided into two layers: the thin upper layer, called the papillary layer, is composed of thin, haphazardly arranged collagen fibers; the thicker lower layer, called the reticular layer, extends from the base of the papillary layer to the subcutaneous tissue and is composed of thick collagen fibers that are arranged parallel to the surface of the skin. Histiocytes are wandering macrophages that accumulate hemosiderin, melanin, and debris created by inflammation. Mast cells, located primarily about blood vessels, manufacture and release histamine and heparin.
Dermal nerves and vasculature

The sensations of touch and pressure are received by Meissner's and the Vater-Pacini corpuscles. The sensations of pain, itch, and temperature are received by unmyelinated nerve endings in the papillary dermis. A low intensity of stimulation created by inflammation causes itching, whereas a high intensity of stimulation created by inflammation causes pain. Therefore scratching converts the intolerable sensation of itching to the more tolerable sensation of pain and eliminates pruritus.

The autonomic system supplies the motor innervation of the skin. Adrenergic fibers innervate the blood vessels (vasoconstriction), hair erector muscles, and apocrine glands. Autonomic fibers to eccrine sweat glands are cholinergic. The sebaceous gland is regulated by the endocrine system and is not innervated by autonomic fibers. The anatomy of the hair follicle is described in Chapter 24.
Diagnosis of Skin Disease

What could be easier than the diagnosis of skin disease? The pathology is before your eyes! Why then do nondermatologists have such difficulty interpreting what they see?

There are three reasons. First, there are literally hundreds of cutaneous diseases. Second, a single entity can vary in its appearance. A common seborrheic keratosis, for example, may have a smooth, rough, or eroded surface and a border that is either uniform or as irregular as a melanoma. Third, skin diseases are dynamic and change in morphology. Many diseases undergo an evolutionary process: herpes simplex may begin as a red papule, evolve into a blister, and then become an erosion that heals with scarring. If hundreds of entities can individually vary in appearance and evolve through several stages, then it is necessary to recognize thousands of permutations to diagnose cutaneous entities confidently. What at first glance appeared to be simple to diagnose may later appear to be simply impossible.

Dermatology is a morphologically oriented specialty. As in other specialties, the medical history is important; however, the ability to interpret what is observed is even more important. The diagnosis of skin disease must be approached in an orderly and logical manner. The temptation to make rapid judgments after hasty observation must be controlled.

A methodical approach

The recommended approach to the patient with skin disease is as follows:

HISTORY.

Obtain a brief history, noting duration, rate of onset, location, symptoms, family history, allergies, occupation, and previous treatment.
DISTRIBUTION.

Determine the extent of the eruption by having the patient disrobe completely.

PRIMARY LESION.

Determine the primary lesion. Examine the lesions carefully; a hand lens is a valuable aid for studying skin lesions. Determine the nature of any secondary or special lesions.

DIFFERENTIAL DIAGNOSIS.

Formulate a differential diagnosis.

TESTS.

Obtain a biopsy and perform laboratory tests, such as skin biopsy, potassium hydroxide examination for fungi, skin scrapings for scabies, Gram stain, fungal and bacterial cultures, cytology (Tzanck test), Wood's light examination, patch tests, dark field examination, and blood tests.

Examination technique

DISTRIBUTION.

The skin should be examined methodically. An eye scan over wide areas is inefficient. It is most productive to mentally divide the skin surface into several sections and carefully study each section. For example, when studying the face, examine the area around each eye, the nose, the mouth, the cheeks, and the temples.

During an examination, patients may show small areas of their skin, tell the doctor that the rest of the eruption looks the same, and expect an immediate diagnosis. The rest of the eruption may or may not look the same. Patients with rashes should receive a complete skin examination to determine the distribution and confirm the diagnosis. Decisions about quantities of medication to dispense require visualization of the big picture. Many dermatologists now advocate a complete skin examination for all of their patients. Because of an awareness that some patients are uncomfortable undressing completely when they have a specific request such as treatment of a plantar wart, other dermatologists advocate a case-by-case approach.

PRIMARY LESIONS AND SURFACE CHARACTERISTICS.

Lesions should be examined carefully. Standing back and viewing a disease process provides valuable information about the distribution. Close examination with a magnifying device provides much more information. Often the primary lesion is identified
and the diagnosis is confirmed at this step. The physician should learn the surface characteristics of all the common entities and gain experience by examining known entities. A flesh-colored papule might be a wart, sebaceous hyperplasia, or a basal cell carcinoma. The surface characteristics of many lesions are illustrated throughout this book.

**Approach to treatment**

Most skin diseases can be managed successfully with the numerous agents and techniques available. If a diagnosis has not been established, medications should not be prescribed; this applies particularly to prescription of topical steroids. Some physicians are tempted to experiment with various medications and, if the treatment fails, to refer the patient to a specialist. This is not a logical or efficient way to practice medicine.

**Primary lesions**

Most skin diseases begin with a basic lesion that is referred to as a primary lesion. Identification of the primary lesion is the key to accurate interpretation and description of cutaneous disease. Its presence provides the initial orientation and allows the formulation of a differential diagnosis. Definitions of the primary lesions and their differential diagnoses are listed and illustrated on pp. 3 to 11.

**Secondary lesions**

Secondary lesions develop during the evolutionary process of skin disease or are created by scratching or infection. They may be the only type of lesion present, in which case the primary disease process must be inferred. The differential diagnoses of secondary lesions are listed and illustrated on pp. 12 to 16.

---

**Primary Lesions—Macules**
**Macule**
A circumscribed, flat discoloration that may be brown, blue, red, or hypopigmented

<table>
<thead>
<tr>
<th>Brown</th>
<th>Red</th>
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<tbody>
<tr>
<td>Becker's nevus (p. 780)</td>
<td>Drug eruptions (p. 485)</td>
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<td>Café-au-lait spot (p. 694)</td>
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<td>Stasis dermatitis (p. 73)</td>
<td>Radiation dermatitis</td>
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<td>Tinea nigra palmaris</td>
<td>Tinea versicolor (p. 451)</td>
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</table>

<table>
<thead>
<tr>
<th>Blue</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Ink (tattoo)</td>
<td></td>
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<tr>
<td>Maculae ceruleae (lice) (p. 508)</td>
<td>Tuberous sclerosis (p. 690)</td>
</tr>
<tr>
<td>Mongolian spot</td>
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<td>Primary Skin Lesions—Papules</td>
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**Papule**

An elevated solid lesion up to 0.5?cm in diameter; color varies; papules may become confluent and form plaques

<table>
<thead>
<tr>
<th>Flesh colored, yellow, or white</th>
<th>Red</th>
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<tbody>
<tr>
<td>Achrochordon (skin tag) (p. 706)</td>
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<td>Closed comedone (acne) (p. 171)</td>
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<td>Molluscum</td>
<td>Keratosis pilaris (p. 116)</td>
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<td>Disease</td>
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<tr>
<td>Lichen sclerosis et atrophicus</td>
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<tr>
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<td>Molluscum contagiosum</td>
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<td>Nevi (dermal)</td>
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<td>Neurofibroma</td>
<td>p. 906</td>
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<tr>
<td>Pearly penile papules</td>
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<td>Pseudoxanthoma elasticum</td>
<td>p. 916</td>
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<td>Sebaceous hyperplasia</td>
<td>p. 720</td>
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<td>Skin tags</td>
<td>p. 706</td>
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<td>Syringoma</td>
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**Blue or violaceous**

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<thead>
<tr>
<th>Disease</th>
<th>Page</th>
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<tr>
<td>Blue or violaceous Angiokeratoma</td>
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<td>Blue nevus</td>
<td>p. 782</td>
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<tr>
<td>Lichen planus</td>
<td>p. 250</td>
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<tr>
<td>Lymphoma</td>
<td></td>
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<td>Kaposi’s sarcoma</td>
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<td>Melanoma</td>
<td>p. 786</td>
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<td>Mycosis fungoides</td>
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<td>Venous lake</td>
<td>p. 825</td>
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<td></td>
<td>Wart (cylindrical projections)</td>
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<tr>
<td>Basal cell epithelioma</td>
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<tr>
<td>Nevi (dermal)</td>
<td>Lichen planus</td>
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<tr>
<td>Seborrheic keratosis</td>
<td>Seborrheic keratosis</td>
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<tr>
<td>Melanoma</td>
<td>Granuloma annulare</td>
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<tr>
<td>Flat warts</td>
<td>Molluscum contagiosum</td>
</tr>
<tr>
<td>Venous lake</td>
<td>Cherry angioma</td>
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</tbody>
</table>

Primary Skin Lesions—Plaques
**Plaque**

A circumscribed, elevated, superficial, solid lesion more than 0.5 cm in diameter, often formed by the confluence of papules

<table>
<thead>
<tr>
<th>Pityriasis rosea</th>
<th>Eczema</th>
<th>Seborrheic dermatitis</th>
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</thead>
<tbody>
<tr>
<td>Syphilis (secondary)</td>
<td>Psoriasis</td>
<td>Tinea corporis</td>
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<tr>
<td>Tinea pedis</td>
<td>Tinea versicolor</td>
<td>Pityriasis rosea</td>
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<tr>
<td>Tinea corporis</td>
<td>Tinea pedis</td>
<td>Tinea versicolor</td>
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<tr>
<td>Psoriasis</td>
<td>Paget's disease</td>
<td>Sweet's syndrome</td>
</tr>
</tbody>
</table>

**Primary Skin Lesions—Nodules**

**Nodule**
A circumscribed, elevated, solid lesion more than 0.5?cm in diameter; a large nodule is referred to as a tumor

- Basal cell carcinoma ([p. 724](#))
- Erythema nodosum ([p. 635](#))
- Furuncle ([p. 284](#))
- Hemangioma ([p. 815](#))
- Kaposi's sarcoma ([pp. 365, 827](#))
- Keratoacanthoma ([p. 711](#))
- Lipoma
- Lymphoma
- Melanoma ([p. 786](#))
- Metastatic carcinoma ([p. 766](#))
- Cutaneous T-cell lymphoma ([p. 754](#))
- Neurofibromatosis ([p. 906](#))
- Prurigo nodularis ([p. 68](#))
- Sporotrichosis
- Squamous cell carcinoma ([p. 744](#))
- Warts ([p. 371](#))
Primary Skin Lesions—Pustules

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<thead>
<tr>
<th>Basal cell carcinoma</th>
<th>Squamous cell carcinoma</th>
<th>Keratoacanthoma</th>
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<tbody>
<tr>
<td>Melanoma</td>
<td>Hemangioma</td>
<td>Kaposi's sarcoma</td>
</tr>
<tr>
<td>Cutaneous T-cell lymphoma</td>
<td>Prurigo nodularis</td>
<td>Neurofibromatosis</td>
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</table>

- Prurigo nodularis (p. 66)
- Sporotrichosis
- Squamous cell carcinoma (p. 744)
- Warts (p. 371)
- Xanthoma (p. 904)
**Pustule**
A circumscribed collection of leukocytes and free fluid that varies in size

<table>
<thead>
<tr>
<th>Chicken pox</th>
<th>Folliculitis</th>
<th>Gonococcemia</th>
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<tr>
<td>Acne (p. 172)</td>
<td>Candidiasis (p. 446)</td>
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<td>Gonococcemia (p. 333)</td>
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<td>Primary Skin Lesions—Vesicles and Bullae</td>
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<td>-----------------------------------------</td>
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**Vesicle**

A circumscribed collection of free fluid up to 0.5 cm in diameter

**Vesicles**

- Benign familial chronic pemphigus ([p. 575](#))
- Cat-scratch disease ([p. 528](#))
- Chicken pox ([p. 390](#))
- Dermatitis herpetiformis ([p. 554](#))
- Eczema (acute) ([p. 42](#))
- Erythema multiforme ([p. 629](#))
- Herpes simplex ([p. 382](#))
- Herpes zoster ([p. 395](#))
- Impetigo ([p. 268](#))
- Lichen planus
- Pemphigus foliaceus ([p. 568](#))
- Porphyria cutanea tarda ([p. 678](#))
- Scabies ([p. 500](#))
**Bulla**

A circumscribed collection of free fluid more than 0.5 cm in diameter

**Bullae**

- Bullae in diabetics (p. 559)
- Bullous pemphigoid (p. 568)
- Cicatricial pemphigoid (p. 571)
- Epidermolysis bullosa acquisita (p. 574)
- Fixed drug eruption (p. 492)
- Herpes gestationis (p. 573)
- Lupus erythematosus
- Pemphigus (p. 561)

<table>
<thead>
<tr>
<th>Eczema (acute)</th>
<th>Chicken pox</th>
<th>Dermatitis herpetiformis</th>
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<tbody>
<tr>
<td>Erythema multiforme</td>
<td>Herpes simplex</td>
<td>Herpes zoster</td>
</tr>
</tbody>
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*Primary Skin Lesions—Wheals (Hives)*
Wheal (hive)
A firm edematous plaque resulting from infiltration of the dermis with fluid; wheals are transient and may last only a few hours

<table>
<thead>
<tr>
<th>Angioedema</th>
<th>Angioedema</th>
<th>Dermographism</th>
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<td>Cholinergic urticaria</td>
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<th>Scales</th>
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<td>Excess dead epidermal cells that are produced by abnormal keratinization and shedding</td>
<td>Erythema craquele (p. 60)</td>
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<td>Ichthyosis—dominant (quadrangular) (p. 115)</td>
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<th>Scaling in sheets (desquamation)</th>
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<table>
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<tr>
<th>Erythema craquele (dense scale)</th>
<th>Ichthyosis—dominant (quadrangular)</th>
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<td>Ichthyosis—sex-linked (quadrangular)</td>
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<td>Psoriasis (silvery)</td>
<td>Pityriasis rosea (collarette)</td>
</tr>
<tr>
<td>--------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Scarlet fever (desquamation)</td>
<td>Kawasaki syndrome (desquamation)</td>
</tr>
</tbody>
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### Secondary Skin Lesions—Crusts

**Crust**

A collection of dried serum and cellular debris; a scab

- Acute eczematosus inflammation ([p. 42](#))
- Atopic (face) ([p. 109](#))
- Impetigo (honey colored) ([p. 270](#))
- Pemphigus foliaceus ([p. 563](#))
- Tinea capitis ([p. 431](#))

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<tr>
<th>Atopic (lips)</th>
<th>Impetigo (honey colored)</th>
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<tr>
<td>Pemphigus foliaceus</td>
<td>Tinea capitis</td>
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### Secondary Skin Lesions—Erosions and Ulcers
**Erosion**

A focal loss of epidermis; erosions do not penetrate below the dermoepidermal junction and therefore heal without scarring

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<th>Neurotic excoriations</th>
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<td></td>
<td>Vesiculobullous diseases (p. 547)</td>
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<th>Ulcer</th>
<th>Chancroid</th>
<th>Pyoderma gangrenosum</th>
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<td>Syphilis (chancre) (p. 316)</td>
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**Fissure**
A linear loss of epidermis and dermis with sharply defined, nearly vertical walls

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<td>--------</td>
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</tr>
<tr>
<td><strong>Atrophy</strong></td>
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<tr>
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<td></td>
<td>Topical and intralesional steroids (p. 35)</td>
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</tbody>
</table>

| Lichen sclerosis et atrophicus | Morphea | Topical and intralesional steroids |

**Secondary Skin Lesions—Scars**
Scar
An abnormal formation of connective tissue implying dermal damage; after injury or surgery scars are initially thick and pink but with time become white and atrophic

<table>
<thead>
<tr>
<th>Keloid</th>
<th>Herpes zoster</th>
<th>Porphyria</th>
</tr>
</thead>
</table>

**Special Skin Lesions**

**EXCORIATION**
An erosion caused by scratching; excoriations are often linear

**COMEDONE**
A plug of sebaceous and keratinous material lodged in the opening of a hair follicle; the follicular orifice may be dilated (blackhead) or narrowed (whitehead or closed comedone)

**MILIA**
A small, superficial keratin cyst with no visible opening
**CYST**  
A circumscribed lesion with a wall and a lumen; the lumen may contain fluid or solid matter

**BURROW**  
A narrow, elevated, tortuous channel produced by a parasite

**LICHENIFICATION**  
An area of thickened epidermis induced by scratching; the skin lines are accentuated so that the surface looks like a washboard

**TELANGIECTASIA**  
Dilated superficial blood vessels

**PETECHIAE**  
A circumscribed deposit of blood less than 0.5?cm in diameter

**PURPURA**  
A circumscribed deposit of blood greater than 0.5?cm in diameter

<table>
<thead>
<tr>
<th>Telangiectasia rosacea</th>
<th>Acne cyst</th>
<th>Lichenification</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. spider angioma</td>
<td>Pilar cyst</td>
<td>Epidermal cyst</td>
</tr>
</tbody>
</table>
Regional Differential Diagnoses

Most skin diseases have preferential areas of involvement. Disease locations are illustrated below; diseases are listed alphabetically by location on pp. 19 to 22. Common diseases that are obvious to most practitioners are not included. Diseases such as contact dermatitis and herpes zoster that can be found on any skin surface have also been left out of most of the lists.

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**Areolae (breast)**

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- Fox-Fordyce spots 169
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Pigmentary demarcation lines
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Striae distensae 37
Tinea versicolor 451
Transient acantholytic dermatosis (Grover's disease)

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Face

Actinic keratosis 742
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Hand (dorsa)

Acquired digital fibrokeratoma 888
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**Legs—lower**

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**Neck (back)**

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Scabies 501
Seborrheic keratosis 700

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CTCL (mycosis fungoides) 754
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Pityrosporum folliculitis 454
Poikiloderma vasculare atrophicans 756
Psoriasis (guttate) 212
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Extramammary Paget's disease 764
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Fox-Fordyce spots 169
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Granuloma inguinale 329
Herpes simplex/zoster 381
Hidradenitis suppurativa 202
Intertrigo 418
Leukoplakia 751
Lichen planus 255
Lichen sclerosis et atrophicus 258
Lichen simplex chronicus 54
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Molluscum contagiosum 344
Nevus 775
Pediculosis 506
Psoriasis 211
Squamous cell carcinoma 744
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Chapter 2 - Topical Therapy and Topical Corticosteroids

Topical Therapy

A wide variety of topical medications are available for treating cutaneous disease (see Dermatologic Formulary, p. 945). Specific medications are covered in detail in the appropriate chapters, and the basic principles of topical treatment are discussed here.

The skin is an important barrier that must be maintained to function properly. Any insult that removes water, lipids, or protein from the epidermis alters the integrity of this barrier and compromises its function. Restoration of the normal epidermal barrier is accomplished with the use of mild soaps and emollient creams and lotions. There is an old and often-repeated rule: “If it is dry, wet it; if it is wet, dry it”.

DRY DISEASES.

Dry skin or dry cutaneous lesions have lost water and, in many instances, the epidermal lipids and proteins that help contain epidermal moisture. These substances are replaced with emollient creams and lotions.

WET DISEASES.

Exudative inflammatory diseases pour out serum that leaches the complex lipids and proteins from the epidermis. A wet lesion is managed with wet compresses that suppress inflammation and debride crust and serum. Repeated cycles of wetting and drying eventually make the lesion dry. Excessive use of wet dressings causes severe drying and chapping. Once the wet phase of the disease has been controlled, the lipids and proteins must be restored with the use of emollient creams and lotions, and wet compressing should stop.
Emollient creams and lotions

Emollient creams and lotions restore water and lipids to the epidermis (see *Dermatologic Formulary*, pp. 945). Preparations that contain urea (e.g., Carmol 10, 20, 40, vanamide), or lactic acid (e.g., Lac-Hydrin, AmLactin) have special lubricating properties and may be the most effective. Creams are thicker and more lubricating than lotions; petroleum jelly and mineral oil contain no water.

Lubricating creams and lotions are most effective if applied to moist skin. After bathing is an ideal time to apply moisturizers. Wet the skin, pat dry, and immediately apply the moisturizer. Emollients should be applied as frequently as necessary to keep the skin soft. Chemicals such as menthol and phenol (e.g., Sarna Lotion) are added to lubricating lotions to control pruritus (see *Dermatologic Formulary*, p. 945).

Severe dry skin (xerosis)

Dry skin is more severe in the winter months when the humidity is low. “Winter itch” most commonly affects the hands and lower legs. Initially the skin is rough and covered with fine white scales; later, thicker tan or brown scales may appear. The most severely affected skin may be criss-crossed with shallow red fissures. Dry skin may itch or burn. Preparations listed in the *Formulary* on p. 945 should be used for mild cases; severe dry skin responds to 12% lactate lotion (Lac-Hydrin, AmLactin).

Wet dressings

Wet dressings, also called compresses, are a valuable aid in the treatment of exudative (wet) skin diseases (see *Box 2-1*). Their importance in topical therapy cannot be overstated. The technique for wet compress preparation and application is described in the list below.

1. Obtain a clean, soft cloth such as bedsheeting or shirt material. The cloth need not be new or sterilized. Compress material must be washed at least once daily if it is to be used repeatedly.
2. Fold the cloth so there are at least four to eight layers and cut to fit an area slightly larger than the area to be treated.
3. Wet the folded dressings by immersing them in the solution, and wring them out until they are sopping wet (neither running nor just damp).
4. Place the wet compresses on the affected area. Do not pour solution on a wet dressing to keep it wet because this practice increases the concentration of the solution and may cause irritation. Remove the compress and replace it with a new one.
5. Dressings are left in place for 30 minutes to 1 hour. Dressings may be used two to four times a day or continuously. Discontinue the use of wet compresses when the
skin becomes dry. Excessive drying causes cracking and fissures.

Wet compresses provide the following benefits:

- Antibacterial action: Aluminum acetate, acetic acid, or silver nitrate may be added to the water to provide an antibacterial effect (Table 2-1).
- Wound debridement: A wet compress macerates vesicles and crust, helping to debride these materials when the compress is removed.
- Inflammation suppression: Compresses have a strong antiinflammatory effect. The evaporative cooling causes constriction of superficial cutaneous vessels, thereby decreasing erythema and the production of serum. Wet compresses control acute inflammatory processes, such as acute poison ivy, faster than either topical applied or orally administered corticosteroids.
- Drying: Wet dressings cause the skin to become dry. Wetting something to make it dry seems paradoxical, but the effects of repeated cycles of wetting and drying are observed in lip chapping, caused by lip licking; irritant hand dermatitis, caused by repeated washing; and the soggy sock syndrome in children, caused by perspiration.

<table>
<thead>
<tr>
<th>Box 2-1. Diseases Treated With Wet Compresses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute eczematous inflammation (poison ivy)</td>
</tr>
<tr>
<td>Eczematous inflammation with secondary infection (pustules)</td>
</tr>
<tr>
<td>Bullous impetigo</td>
</tr>
<tr>
<td>Herpes simplex and herpes zoster (vesicular lesions)</td>
</tr>
<tr>
<td>Infected exudative lesions of any type</td>
</tr>
<tr>
<td>Insect bites</td>
</tr>
<tr>
<td>Intertrigo (groin or under breasts)</td>
</tr>
<tr>
<td>Nummular eczema (exudative lesions)</td>
</tr>
<tr>
<td>Stasis dermatitis (exudative lesions)</td>
</tr>
</tbody>
</table>
### TABLE 2-1 -- Wet Dressing Solutions

<table>
<thead>
<tr>
<th>Solution</th>
<th>Preparation</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>Tap water does not have to be sterilized.</td>
<td>Poison ivy, sunburn, any noninfected exudative or inflamed process</td>
</tr>
<tr>
<td>Burrow’s solution (aluminum acetate) Domeboro astringent powder packets Effervescent tablets</td>
<td>Dissolve one, two, or three packets of Domeboro powder in 16 ounces of water.</td>
<td>Mildly antiseptic; for acute inflammation. Poison ivy, insect bites, athlete’s foot</td>
</tr>
<tr>
<td>Silver nitrate, 0.1%–0.5% (prepared by some pharmacists and some hospitals)</td>
<td>Supplied as a 50% aqueous solution; stains skin dark brown and metal black.</td>
<td>Bactericidal, for exudative infected lesions (e.g., stasis ulcers and stasis dermatitis)</td>
</tr>
<tr>
<td>Acetic acid, 1%–2.5%</td>
<td>Vinegar is 5% acetic acid. Make a 1% solution by adding ½ cup of vinegar (white or brown) to 1 pint of water.</td>
<td>Bactericidal: for certain gram-negative bacteria (e.g., Pseudomonas aeruginosa), otitis externa, Pseudomonas intertrigo</td>
</tr>
</tbody>
</table>

The temperature of the compress solution should be cool when an antiinflammatory effect is desired and tepid when the purpose is to debride an infected, crusted lesion. Covering a wet compress with a towel or plastic inhibits evaporation, promotes maceration, and increases skin temperature, which facilitates bacterial growth.
Topical Corticosteroids

Topical corticosteroids are a powerful tool for treating skin disease. Understanding the correct use of these agents will result in the successful management of a variety of skin problems. Many products are available, but all have basically the same antiinflammatory properties, differing only in strength, base, and price.

Strength

POTENCY: GROUPS I THROUGH VII.

The antiinflammatory properties of topical corticosteroids result in part from their ability to induce vasoconstriction of the small blood vessels in the upper dermis. This property is used in an assay procedure to determine the strength of each new product. These products are subsequently tabulated in seven groups, with group I the strongest and group VII the weakest (see the Formulary and the inside front cover of this book). The treatment sections of this book recommend topical steroids by group number rather than by generic or brand name because the agents in each group are essentially equivalent in strength.

Lower concentrations of some brands may have the same effect in vasoconstrictor assays as much higher concentrations of the same product. One study showed that there was no difference in vasoconstriction between Kenalog 0.025%, 0.1%, or 0.5% creams.\[^{1}\]

CHOOSING THE APPROPRIATE STRENGTH.

Guidelines for choosing the appropriate strength and brand of topical steroid are presented in Box 2-2 and the diagram at the right. The best results are obtained when preparations of adequate strength are used for a specified length of time. Weaker, “safer” strengths often fail to provide adequate control. Patients who do not respond after 1 to 4 weeks of treatment should be reevaluated.
<table>
<thead>
<tr>
<th><strong>Groups I–II</strong></th>
<th><strong>Groups III–V</strong></th>
<th><strong>Groups VI–VII</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Psoriasis</td>
<td>Atopic dermatitis</td>
<td>Dermatitis (eyelids)</td>
</tr>
<tr>
<td>Lichen planus</td>
<td>Nummular eczema</td>
<td>Dermatitis (diaper area)</td>
</tr>
<tr>
<td>Discoid lupus†</td>
<td>Asteatotic eczema</td>
<td>Mild dermatitis (face)</td>
</tr>
<tr>
<td>Severe hand eczema</td>
<td>Stasis dermatitis</td>
<td>Mild anal inflammation</td>
</tr>
<tr>
<td>Poison ivy (severe)</td>
<td>Seborrheic dermatitis</td>
<td>Mild intertrigo</td>
</tr>
<tr>
<td>Lichen simplex chronicus</td>
<td>Lichen sclerosis et atrophicus (vulva)</td>
<td></td>
</tr>
<tr>
<td>Hyperkeratotic eczema</td>
<td>Intertrigo (brief course)</td>
<td></td>
</tr>
<tr>
<td>Chapped feet</td>
<td>Tinea (brief course to control inflammation)</td>
<td></td>
</tr>
<tr>
<td>Lichen sclerosis et atrophicus (skin)</td>
<td>Scabies (after scabicide)</td>
<td></td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Intertrigo (severe cases)</td>
<td></td>
</tr>
<tr>
<td>Nummular eczema (severe)</td>
<td>Anal inflammation (severe cases)</td>
<td></td>
</tr>
<tr>
<td>Atopic dermatitis (resistant adult cases)</td>
<td>Severe dermatitis (face)</td>
<td></td>
</tr>
</tbody>
</table>

* Stop treatment, change to less potent agent, or use intermittent treatment once inflammation is controlled.
† Use on the face may be justified.

**CHOOSING A TOPICAL STEROID**
MEGAPOTENT TOPICAL STEROIDS (GROUP I).

Cormax (clobetasol propionate), Ultravate (halobetasol propionate), Diprolene (betamethasone dipropionate), and Psorcon (diflorasone diacetate) are the most potent topical steroids available. Cormax and Ultravate are the most potent, and Psorcon and Diprolene are equipotent.

In general no more than 45 to 60?gm of cream or ointment should be used each week (see Table 2-2). Side effects are minimized and efficacy increased when medication is applied once or twice daily for 2 weeks followed by 1 week of rest. This cyclic schedule (pulse dosing) is continued until resolution occurs. Intermitent dosing (e.g., once or twice a week) can lead to a prolonged remission of psoriasis if used after initial clearing. Alternatively, intermittent use of a weaker topical steroid can be used for maintenance. Psorcon can be used with plastic dressing occlusion; Cormax, Ultravate, and Diprolene should not be used with occlusive dressings.

Patients must be monitored carefully. Side effects such as atrophy and adrenal suppression are a real possibility, especially with unsupervised use of these medications. Refills should be strictly limited.

CONCENTRATION.

The concentration of steroid listed on the tube cannot be used to compare its strength with other steroids. Some steroids are much more powerful than others and need be present only in small concentrations to produce the maximum effect. Nevertheless, it is difficult to convince some patients that Lidex cream 0.05% (group II) is more potent than hydrocortisone 1% (group VII).

It is unnecessary to learn many steroid brand names. Familiarity with one preparation from groups II, V, and VII gives one the ability to safely and effectively treat any steroid-responsive skin disease. Most of the topical steroids are fluorinated (i.e., a fluorine atom has been added to the hydrocortisone molecule). Fluorination increases potency and the possibility of side effects. Products such as Westcort Cream have increased potency without fluorination; however, side effects are possible with this midpotency steroid.

**TABLE 2-2 -- Restriction on the Use of Group I Topical Steroids:***

<table>
<thead>
<tr>
<th>Steroid</th>
<th>Length of therapy</th>
<th>Grams per week</th>
<th>Use under occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cormax (clobetasol propionate)</td>
<td>14 days</td>
<td>60</td>
<td>No</td>
</tr>
<tr>
<td>Cormax scalp solution</td>
<td>14 days</td>
<td>50?ml</td>
<td>No</td>
</tr>
<tr>
<td>Product</td>
<td>Duration</td>
<td>Strength</td>
<td>Prescription</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>----------</td>
<td>----------</td>
<td>--------------</td>
</tr>
<tr>
<td>Olux foam</td>
<td>14 days</td>
<td>50</td>
<td>No</td>
</tr>
<tr>
<td>Ultravate (halobetasol propionate)</td>
<td>14 days</td>
<td>60</td>
<td>No</td>
</tr>
<tr>
<td>Diprolene (betamethasone dipropionate)†</td>
<td>Unrestricted</td>
<td>45</td>
<td>No</td>
</tr>
<tr>
<td>Psorcon (diflorsone diacetate)†</td>
<td>Unrestricted</td>
<td>Unrestricted</td>
<td>Unrestricted</td>
</tr>
</tbody>
</table>

* Restrictions are listed in the package inserts.
† Generic form available.

**COMPOUNDING.**

Avoid having the pharmacist prepare or dilute topical steroid creams. The active ingredient may not be dispersed uniformly, resulting in a cream of variable strength. The cost of pharmacist preparation is generally higher because of the additional labor required. High-quality steroid creams, such as triamcinolone acetonide (Kenalog, Aristocort), are available in large quantities at a low cost.

**GENERIC VERSUS BRAND NAMES.**

Many generic topical steroid formulations are available (e.g., betamethasone valerate, betamethasone dipropionate, fluocinolone acetonide, fluocinonide, hydrocortisone, and triamcinolone acetonide). In many states, generic substitutions by the pharmacist are allowed unless the physician writes “no substitution.” Vasoconstrictor assays have shown large differences in the activity of generic formulations compared with brand-name equivalents: many are inferior, a few are equivalent, and a few are more potent than brand-name equivalents. Many generic topical steroids have vehicles with different ingredients (e.g., preservatives) than brand-name equivalents.

**Vehicle**

The vehicle, or base, is the substance in which the active ingredient is dispersed. The base determines the rate at which the active ingredient is absorbed through the skin. Components of some bases may cause irritation or allergy.

**CREAMS.**

The cream base is a mixture of several different organic chemicals (oils) and water, and it usually contains a preservative. Creams have the following characteristics:

- White color and somewhat greasy texture
- Components that may cause irritation, stinging, and allergy
• High versatility (i.e., may be used in nearly any area); therefore creams are the base most often prescribed
• Cosmetically most acceptable, particularly emollient bases (e.g., Aristocort-A, Cyclocort)

• Possible drying effect with continued use; therefore best for acute exudative inflammation
• Most useful for intertriginous areas (e.g., groin, rectal area, and axilla)

OINTMENTS.

The ointment base contains a limited number of organic compounds consisting primarily of greases such as petroleum jelly, with little or no water. Many ointments are preservative-free. Ointments have the following characteristics:

• Translucent (look like petroleum jelly)
• Greasy feeling persists on skin surface
• More lubrication, thus desirable for drier lesions
• Greater penetration of medicine than creams and therefore enhanced potency (see inside front cover; Synalar Cream in group V and Synalar Ointment in group IV)
• Too occlusive for acute (exudative) eczematous inflammation or intertriginous areas, such as the groin

GELS.

Gels are greaseless mixtures of propylene glycol and water; some also contain alcohol. Gels have the following characteristics:

• A clear base, sometimes with a jellylike consistency
• Useful for acute exudative inflammation, such as poison ivy, and in scalp areas where other vehicles mat the hair

SOLUTIONS AND LOTIONS.

Solutions may contain water and alcohol, as well as other chemicals. Solutions have the following characteristics:

• Clear or milky appearance
• Most useful for scalp because they penetrate easily through hair, leaving no residue
• May result in stinging and drying when applied to intertriginous areas, such as the groin
FOAMS.

A foam preparation of betamethasone valerate (Luxiq) and clobetasol propionate (Olux) is available. Olux contains a superpotent steroid. Treatment beyond 2 consecutive weeks is not recommended, and the total dosage should not exceed 50?gm per week because of the potential for the drug to suppress the hypothalamic-pituitary-adrenal (HPA) axis. Use in children under 12 years of age is not recommended. Foams spread between the strands of hair until they reach the scalp, where the foam melts and delivers the active drug. Foams are useful for treatment of scalp dermatoses and in other areas for acute eczematous inflammation such as poison ivy and plaque psoriasis.

Steroid-antibiotic mixtures

LOTIRISONE CREAM AND LOTION.

Lotrisone cream contains a combination of the antifungal agent clotrimazole and the corticosteroid betamethasone dipropionate. It is indicated for the topical treatment of tinea pedis, tinea cruris, and tinea corporis. This product is used by many physicians as their topical antiinflammatory agent of first choice. Most inflammatory skin disease is not infected or contaminated by fungus. Lotrisone is a marginal drug for cutaneous fungal infections. Brand-name Lotrisone cream is no longer available; it has been replaced by a brand-name lotion. The generic cream costs approximately $25.00 for 15?gm and $45.00 for 45?gm. Generic betamethasone dipropionate cream costs approximately $12.00 for 15?gm and $18.00 for 45?gm. Generic clotrimazole costs approximately $10.00 for 30?gm.

OTHER ANTIBIOTICS AND CORTICOSTEROID MIXTURES.

Mycolog II (Nystatin; Triamcinolone Acetonide) is indicated for the treatment of cutaneous candidiasis. Nystatin does not treat fungi that cause tinea pedis. The majority of steroid-responsive skin diseases can be managed successfully without topical antibiotics.

Amount of cream to dispense

The amount of cream dispensed is very important. Patients do not appreciate being prescribed a $90.00, 60-gm tube of cream to treat a small area of hand dermatitis. Unrestricted and unsupervised use of potent steroid creams can lead to side effects. Patients rely on the physician's judgment to determine the correct amount of topical medicine. If too small a quantity is prescribed, patients may conclude that the treatment did not work. It is advisable to allow for a sufficient amount of cream, and then to set limits on duration and frequency of application. Many steroids (e.g., triamcinolone, hydrocortisone) are available in generic form. They are purchased in bulk by the pharmacist and can be dispensed in large quantities at considerable savings.
The amount of cream required to cover a certain area can be calculated by remembering that 1?gm of cream covers 100 square cm of skin. The entire skin surface of the average-sized adult is covered by 20 to 30?gm of cream.

The fingertip unit and the rule of hand provide the means to assess how much cream to dispense and apply.

**FINGERTIP UNIT.**

A fingertip unit (FTU) is the amount of ointment expressed from a tube with a 5-mm diameter nozzle, applied from the distal skin crease to the tip of the index finger. One FTU weighs approximately 0.5?gm.

**THE RULE OF HAND.**

The hand area can be used to estimate the total area of involvement of a skin disease and to assess the amount of ointment required. The area of one side of the hand is defined as one hand area. One hand area of involved skin requires 0.5 FTU or 0.25?gm of ointment, or four hand areas equal 2 FTUs equal 1?gm. The area of one side of the hand represents approximately 1% of body surface area so it requires 1 FTU (2 hand units) to cover 2% of the body surface. Approximately 282?gm is required for twice-daily applications to the total body surface (except the scalp) for 1 week.

---

**Application**

**Frequency**

**TACHYPHYLAXIS.**

Tachyphylaxis refers to the decrease in responsiveness to a drug as a result of enzyme induction. The term is used in dermatology in reference to acute tolerance to the vasoconstrictive action of topically applied corticosteroids. Experiments have revealed that vasoconstriction decreases progressively when a potent topical steroid is applied to the skin three times a day for 4 days. The vasoconstrictive response returned 4 days after termination of therapy. These experiments support years of complaints by patients about initially dramatic responses to new topical steroids that diminish with constant use. It would therefore seem reasonable to instruct patients to apply creams on an interrupted schedule.

**INTERMITTENT DOSING**
Optimum dosing schedules for the use of potent topical steroids have not been determined. Studies show that steroid-resistant diseases, such as plaque psoriasis and hand eczema, respond most effectively when clobetasol (Cormax) is applied twice a day for 2 to 3 weeks. Treatment is resumed after 1 week of rest. The schedule of 2 weeks of treatment followed by 1 week of rest is repeated until the lesions have cleared.

Intermittent treatment of healed lesions can lead to prolonged remission. Psoriatic patients with lingering erythema remained clear with applications three times a day on 1 day a week. Twice-weekly applications of clobetasol kept 75% of psoriatic patients and 70% of hand eczema patients in remission.

Short weekly bursts of topical corticosteroids may play a role in keeping an adult's atopic dermatitis under control. Weekly applications of fluticasone ointment (Cutivate), applied once daily for 2 consecutive days each week maintained the improvements achieved after the initial treatment phase and delayed relapse.

Groups II through VII topical steroids.

The optimum frequency of application and duration of treatment for topical steroids have not been determined. Adequate results and acceptable patient compliance occur when the following steps are taken:

1. Apply groups II through VI topical steroids twice each day.
2. Limit the duration of application to 2 to 6 weeks.
3. If adequate control is not achieved, stop treatment for 4 to 7 days and begin another course of treatment.

Excellent control can be achieved with pulse dosing. These are general guidelines; specific instructions and limitations must be established for each individual case.

Methods

SIMPLE APPLICATION.

Creams and ointments should be applied in thin layers and slowly massaged into the site one to four times a day. It is unnecessary to wash before each application. Continue treatment until the lesion is clear. Many patients decrease the frequency of applications or stop entirely when lesions appear to improve quickly. Other patients are so impressed with the efficacy of these agents that they continue treatment after the disease has resolved in order to prevent recurrence; adverse reactions may follow this practice.

Different skin surfaces vary in the ability to absorb topical medicine. The thin eyelid skin
heals quickly with group VI or VII steroids, whereas thicker skin on palms and soles offers a greater barrier to the penetration of topical medicine and requires more potent therapy. Intertriginous (skin touches skin) areas (e.g., axilla, groin, rectal area, and underneath the breasts) respond more quickly to creams that are weaker in strength. The apposition of two skin surfaces performs the same function as an occlusive dressing, which greatly enhances penetration. The skin of infants and young children is more receptive to topical medicine and responds quickly to weaker creams. A baby's diaper has the same occlusive effect as covering with a plastic dressing. Penetration of steroid creams is greatly enhanced; therefore, only group V, VI, or VII preparations should be used under a diaper. Inflamed skin absorbs topical medicines much more efficiently. This explains why red, inflamed areas generally have such a rapid initial response when treated with weaker topical steroids.

OCLUSION.

Occlusion with a plastic dressing (e.g., Saran Wrap) is an effective method for enhancing absorption of topical steroids. The plastic dressing holds perspiration against the skin surface, which hydrates the top layer of the epidermis, the stratum corneum. Topical medication penetrates a moist stratum corneum from 10 to 100 times more effectively than it penetrates dry skin. Eruptions that are resistant to simple application may heal quickly with the introduction of a plastic dressing. Nearly any area can be occluded; the entire body may be occluded with a vinyl exercise suit, available at most sporting goods stores.

Discretion should be used with occlusion. Occlusion of moist areas may encourage the rapid development of infection. Occlusive dressings are used more often with creams than with ointments, but ointments may be covered if the lesions are particularly dry. Weaker, less expensive products (e.g., triamcinolone cream, 0.1%) provide excellent results. Large quantities of this medicine may be purchased at a substantial savings.

METHOD OF OCCLUSION.

The area should be cleaned with mild soap and water. Antibacterial soaps are unnecessary. The medicine is gently rubbed into the lesions, and the entire area is covered with plastic (e.g., Saran Wrap, Handi-Wrap, plastic bags, or gloves; Figures 2-1 to 2-3). The plastic dressing should be secured with tape so that it is close to the skin and the ends are sealed; an airtight dressing is unnecessary. The plastic may be held in place with an Ace bandage or a sock. The best results are obtained if the dressing remains in place for at least 2 hours. Many patients find that bedtime is the most convenient time to wear a plastic dressing and therefore wear it for 8 hours. More medicine is applied shortly after the dressing is removed and while the skin is still moist.
Dressings should not remain on the area continuously because infection or follicular occlusion may result. If an occluded area suddenly becomes worse or pustules develop, infection, usually with staphylococci, should be suspected (Figure 2-4). Oral antistaphylococcal antibiotics should be given (e.g., cephalexin [Keflex] 500?mg 2 to 4 times a day).

A reasonable occlusion schedule is twice daily for a 2-hour period or for 8 hours at bedtime, with simple application once or twice during the day.

Occluded areas often become dry, and the use of lubricating cream or lotion should be encouraged. Cream or lotion may be applied shortly after medicine is applied, when the plastic dressing is removed, or at other convenient times.

**Figure 2-1** Occlusion of the hand. A plastic bag is pulled on and pressed against the skin to expel air. Tape is wound snugly around the bag.

**Figure 2-2** Occlusion of the arm. A plastic sheet (e.g., Saran Wrap) is wound about the extremity and secured at both ends with tape. A plastic bag with the bottom cut out may be used as a sleeve and held in place with tape or an Ace bandage.

**Figure 2-3** Occlusion of the entire body. A vinyl exercise suit is a convenient way to occlude the entire body.

**Figure 2-4** Infection following occlusion. Pustules have appeared at the periphery of an eczematous lesion. Plastic dressing had been left in place for 24 hours.

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**Systemic absorption**

The possibility of producing systemic side effects from absorption of topical steroids is of concern to all physicians who use these agents. A small number of case reports have documented systemic effects after topical application of glucocorticoids for prolonged periods. Cataracts, retardation of growth, failure to thrive, and Cushing’s syndrome have all been reported.

**AVOID WEAKER, “SAFE” PREPARATIONS.**

In an attempt to avoid complications, physicians often choose a weaker steroid preparation than that indicated; these weaker preparations all too frequently fall short of expectations and fail to give the desired antiinflammatory effect. The disease does not
improve, but rather becomes worse because of the time wasted using the ineffective cream. Pruritus continues, infection may set in, and the patient becomes frustrated. Treatment of intense inflammation with hydrocortisone cream 0.5% is a waste of time and money. Generally, a topical steroid of adequate strength (see Box 2-2) should be used 2 to 4 times daily for a specific length of time, such as 7 to 21 days, in order to obtain rapid control. Even during this short interval adrenal suppression may result when groups I through III steroids are used to treat wide areas of inflamed skin. This suppression of the hypothalamic-pituitary-adrenal axis is generally reversible in 24 hours and is very unlikely to produce side effects characteristic of long-term systemic use.\[15\]

CHILDREN.

Many physicians worry about systemic absorption and will not use any topical steroids stronger than 1% hydrocortisone on infants. The group V topical steroid, fluticasone propionate cream 0.05% (Cutivate) appears to be safe for the treatment of severe eczema for up to 4 weeks in children 3 months of age and older. Children between 3 months and 6 years with moderate to severe atopic dermatitis (> or equal to 35% body surface area; mean body surface area treated, 64%) were treated with fluticasone propionate cream, 0.05% twice daily for 3 to 4 weeks. Mean cortisol levels were similar at baseline and at the end of treatment.\[16\] The relative safety of moderately strong topical steroids and their relative freedom from serious systemic toxicity despite widespread use in the very young has been clearly demonstrated. Patients should be treated for a specific length of time with a medication of appropriate strength. Steroid creams should not be used continually for many weeks, and patients who do not respond in a predictable fashion should be reevaluated.

Group I topical steroids should be avoided in prepubertal children. Use only group VI or VII steroids in the diaper area and for only 3 to 10 days. Monitor growth parameters in children on chronic topical glucocorticoid therapy.

ADULTS.

Suppression may occur during short intervals of treatment with group I or II topical steroids, but recovery is rapid when treatment is discontinued. Physicians may prescribe strong agents when appropriate, but the patient must be cautioned that the agent should be used only for the length of time dictated.

Adverse reactions

Because information concerning the potential dangers of potent topical steroids has been so widely disseminated, some physicians have stopped prescribing them. Topical steroids have been used for approximately 30 years with an excellent safety record. They do, however, have the potential to produce a number of adverse reactions. Once these are understood, the most appropriate-strength steroid can be prescribed confidently. The reported adverse reactions to topical steroids are listed below.
Rosacea, perioral dermatitis, acne

Skin atrophy with telangiectasia, stellate pseudoscars (arms), purpura, striae (from anatomic occlusion, e.g., groin)

Tinea incognito, impetigo incognito, scabies incognito

Ocular hypertension, glaucoma, cataracts

Allergic contact dermatitis

Systemic absorption

Burning, itching, irritation, dryness caused by vehicle (e.g., propylene glycol)

Miliaria and folliculitus following occlusion with plastic

Skin blanching from acute vasoconstriction

Rebound phenomenon (i.e., psoriasis becomes worse after treatment is stopped)

Nonhealing leg ulcers; steroids applied to any leg ulcer retard healing process

Hypopigmentation

Hypertrichosis of face

A brief description of some of the more important adverse reactions is presented in the following pages.

**Steroid rosacea and perioral dermatitis**

Steroid rosacea is a side effect frequently observed in fair-skinned females who initially complain of erythema with or without pustules, the “flusher blusher complexion.” In a typical example, the physician prescribes a mild topical steroid, which initially gives pleasing results. Tolerance (tachyphylaxis) occurs, and a new, more potent topical steroid is prescribed to suppress the erythema and pustules that may reappear following the use of the weaker preparation. This progression to more potent creams may continue until group II steroids are applied several times each day. Figure 2-5, A, shows a middle-aged woman who has applied a group V steroid cream once each day for 5 years. Intense erythema and pustulation occurs each time attempts are made to discontinue topical treatment (Figure 2-5, B, C). The skin may be atrophic and red with a burning sensation.

Perioral dermatitis (see Chapter 7) is sometimes caused by the chronic application of topical steroids to the lower face; pustules, erythema, and scaling occur about the nose,
mouth, and chin.

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STEROID ROSACEA

Figure 2-5 A, Numerous red papules formed on the cheeks and forehead with constant daily use of a group V topical steroid for more than 5 years.
B, Ten days after discontinuing use of group V topical steroid.
C, Two months after use of topical steroids was discontinued. Telangiectasia has persisted; rosacea has improved with oral antibiotics.

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MANAGEMENT.

Strong topical steroids must be discontinued. Doxycycline (100?mg twice a day) or erythromycin (250?mg four times a day) may reduce the intensity of the rebound erythema and pustulation that predictably occur during the first 10 days (Figures 2-6, 2-7, 2-8, and 2-9). Occasionally, cool, wet compresses, with or without 1% hydrocortisone cream, are necessary if the rebound is intense. Thereafter, mild noncomedogenic lubricants (those that do not induce acne, such as Curel lotion) may be used for the dryness and desquamation that occur. Erythema and pustules are generally present at a low level for months. Low dosages of doxycycline (50?mg twice a day) or erythromycin (250?mg two or three times a day) may be continued until the eruption clears. The pustules and erythema eventually subside, but some telangiectasia and atrophy may be permanent.

STEROID ROSACEA

Figure 2-6 A, Intense erythema and pustulation appeared 10 days after discontinuing use of a group V topical steroid. The cream had been applied every day for 1 year.
B, Patient shown in A 24 days after discontinuing the group V topical steroid. Pustules have cleared without any treatment. Gradual improvement followed over the next several months.

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Figure 2-7 Perioral dermatitis. Pustules and erythema have appeared in a perioral distribution following
several courses of a group III topical steroid to the lower face. The inflammation flares shortly after the topical steroid is discontinued.

**Figure 2-8** Steroid rosacea. A painful, diffuse pustular eruption occurred following daily application for 12 weeks of the group II topical steroid fluocinonide.

**Figure 2-9** Steroid acne. Repeated application to the entire face of a group V topical steroid resulted in this diffuse pustular eruption. The inflammation improved each time the topical steroid was used but flared with increasing intensity each time the medication was stopped.

**Figure 2-10** Steroid-induced telangiectasia. The patient in Figure 2-12 stopped all topical steroids. One year later he has permanent telangiectasia on the cheeks. His intraocular pressure was elevated but returned to near normal levels 3 months after stopping the fluocinonide.

**Figure 2-11** Atrophy and telangiectasia after continual use of a group IV topical steroid for 6 months. Atrophy may improve after the topical steroid is discontinued, but telangiectasia often persists.

**Atrophy**

Long-term use of strong topical steroids in the same area may result in thinning of the epidermis and regressive changes in the connective tissue in the dermis. The affected areas are often depressed slightly below normal skin and usually reveal telangiectasia, prominence of underlying veins, and hypopigmentation. Purpura and ecchymosis result from minor trauma. The skin becomes lax, wrinkled, and shiny. The face (Figures 2-10, 2-11, 2-12, and 2-13), dorsa of the hands (Figure 2-14), extensor surfaces of the forearms and legs, and intertriginous areas are particularly susceptible. In most cases atrophy is reversible and may be expected to disappear in the course of several months.\(^{19}\) Diseases (such as psoriasis) that respond slowly to strong topical steroids require weeks of therapy; some atrophy may subsequently be anticipated (Figure 2-15).

**Figure 2-12** Steroid-induced erythema. This patient used the group II topical steroid fluocinonide almost constantly for 12 years. Erythema rather than pustules occurred each time the medication was stopped.

**STEROID ATROPHY**
Figure 2-13 **A**, Daily application of the group II topical steroid desoximetasone to the lids resulted in almost complete atrophy of the dermis. The lids bleed spontaneously when touched. The intraocular pressure was elevated. There was marked improvement in the atrophy and intraocular tension 8 weeks after stopping the topical steroid. **B**, Daily application for months of a group II topical steroid to the skin on the abdomen produced severe atrophy with telangiectasia.

Figure 2-14 Severe steroid atrophy after continual occlusive therapy over several months. Significant improvement in the atrophy occurs after topical steroids are discontinued.

Figure 2-15 Steroid atrophy. Atrophy with prominence of underlying veins and hypopigmentation following use of Cordran Tape applied daily for 3 months to treat psoriasis. Note that small plaques of psoriasis persist. Atrophy improves after topical steroids are discontinued, but some hypopigmentation may persist.

OCCLUSION.

Occlusion enhances penetration of medicine and accelerates the occurrence of this adverse reaction. Many patients are familiar with this side effect and must be assured that the use of strong topical steroids is perfectly safe when used as directed for 2 to 3 weeks. Patients must also be assured that if some atrophy does appear, it resolves in most cases when therapy is discontinued.

MUCOSAL AREAS.

Atrophy under the foreskin (Figure 2-16) and in the rectal and vaginal areas may appear much more quickly than in other areas. The thinner epidermis offers less resistance to the passage of corticosteroids into the dermis. These are intertriginous areas where the apposition of skin surfaces acts in the same manner as a plastic dressing, retaining moisture and greatly facilitating absorption. These delicate
tissues become thin and painful, sometimes exhibiting a susceptibility to tear or bleed with scratching or intercourse. The atrophy seems to be more enduring in these areas. Therefore careful instruction about the duration of therapy must be given (e.g., twice a day for 10 days). If the disease does not resolve quickly with topical therapy, reevaluation is necessary.

STEROID INJECTION SITES.

Atrophy may appear very rapidly after intralesional injection of corticosteroids (e.g., for treatment of acne cysts or in attempting to promote hair growth in alopecia areata). The side effect of atrophy is used to reduce the size of hypertrophic scars and keloids. When injected into the dermis, 5?mg/ml of triamcinolone acetonide (Kenalog) may produce atrophy; 10?mg/ml of triamcinolone acetonide almost always produces atrophy. For direct injection into the skin, stronger concentrations should probably be avoided.

LONG-TERM USE.

Long-term use (over months) of even weak topical steroids on the upper inner thighs or in the axillae results in striae similar to those on the abdomens of pregnant women (Figure 2-17). These changes are irreversible. Pruritus in the groin area is common, and patients receive considerable relief when prescribed the less potent steroids. Symptoms often recur after treatment is terminated. It is a great temptation to continue topical treatment on an “as needed” basis but every attempt must be made to determine the underlying process and discourage long-term use.

Figure 2-17 A, Striae of the axillae appeared after using Lotrisone cream continuously for 3 months. B, Striae of the groin after long-term use of group V topical steroids for pruritus. These changes are irreversible.

Alteration of infection

Cortisone creams applied to cutaneous infections may alter the usual clinical presentation of those diseases and produce unusual atypical eruptions. Cortisone cream suppresses the inflammation that is attempting to contain the infection and allows unrestricted growth.

TINEA INCOGNITO.

Tinea of the groin is characteristically seen as a localized superficial plaque with a
well-defined scaly border (Figure 2-18). A group II corticosteroid applied for 3 weeks to this common eruption produced the rash seen in Figure 2-19. The fungus rapidly spreads to involve a much wider area, and the typical sharply defined border is gone. Untreated tinea rarely produces such a florid eruption in temperate climates. This altered clinical picture has been called tinea incognito.

Figure 2-20 shows a young girl who applied a group II cream daily for 6 months to treat “eczema.” The large plaques retain some of the characteristics of certain fungal infections by having well-defined edges. The red papules and nodules are atypical and are usually observed exclusively with an unusual form of follicular fungal infection seen on the lower legs.

Boils, folliculitis, rosacea-like eruptions, and diffuse fine scaling resulting from treatment of tinea with topical steroids have been reported. If a rash does not respond after a reasonable length of time or if the appearance changes, the presence of tinea, bacterial infection, or allergic contact dermatitis from some component of the steroid cream should be considered.

Figure 2-18 Typical presentation of tinea of the groin before treatment. Fungal infections of this type typically have a sharp, scaly border and show little tendency to spread.

Figure 2-19 Tinea incognito. A bizarre pattern of widespread inflammation created by applying a group II topical steroid twice daily for 3 weeks to an eruption similar to that seen in Figure 2-18. A potassium hydroxide preparation showed numerous fungi.

Figure 2-20 Tinea incognito. A plaque of tinea initially diagnosed as eczema was treated for 6 months with a group II topical steroid. Red papules have appeared where only erythema was once present.

INFESTATIONS AND BACTERIAL INFECTIONS.

Scabies and impetigo may initially improve as topical steroids suppress inflammation. Consequently, both diseases become worse when the creams are discontinued (or, possibly, continued). Figure 2-21 shows numerous pustules on a leg; this appearance is characteristic of staphylococcal infection after treatment of an exudative, infected plaque of eczema with a group V topical steroid.

Contact dermatitis

Topical steroids are the drugs of choice for allergic and irritant contact dermatitis, but occasionally topical steroids cause such dermatitis. Allergic reactions to various components of steroid creams (e.g., preservatives [parabens], vehicles [lanolin], antibacterials [neomycin], and perfumes) have all been documented. Figure 2-22 shows
allergic contact dermatitis to a preservative in a group II steroid gel. The cream was prescribed to treat seborrheic dermatitis. Allergic reactions may not be intense. Inflammation created by a cream component (e.g., a preservative) may be suppressed by the steroid component of the same cream and the eruption simply smolders, neither improving nor worsening, presenting a very confusing picture.

**Topical steroid allergy**

Of patch-tested patients with dermatitis, 4% to 5% are allergic to corticosteroids. Patients affected by chronic dermatoses are at high risk for the development of sensitization to corticosteroids. Patients with any condition that does not improve or that deteriorates after administration of a topical

![Figure 2-21](image) Impetiginized eczema with satellite pustules after treatment of exudative, infected eczema with a group V topical steroid.

steroid may be allergic to a component of the base or to the medication itself. Patients with stasis dermatitis and leg ulceration who are apt to use several topical medications for extended periods are more likely to be allergic to topical steroids. The over-the-counter availability of hydrocortisone makes long-term, unsupervised use possible. Allergy to topical steroids is demonstrated by patch or intradermal testing.

**MANAGEMENT.**

When a patient does not respond as predicted or becomes worse while using topical corticosteroids, all topical treatment should be stopped. If corticosteroid therapy is absolutely necessary, one of the corticosteroids with a low sensitizing potential (e.g., mometasone furoate [Elocon], fluticasone propionate [Cutivate], betamethasone esters) could be used, and then only in an ointment base to avoid other allergens.  

![Figure 2-22](image) Acute contact allergy to a preservative in a group II steroid gel.

**PATCH TESTING.**

Allergy to a component of the vehicle or the steroid molecule may occur. Patch testing for steroid cream allergy is complicated and usually performed by patch-test experts.

Four groups of corticosteroids are recognized, where substances from the same group may cross-react. The four groups are: group A (hydrocortisone type), group B (triamcinolone acetonides), group C (betamethasone type-nesterified) and group D (hydrocortisone-17-butyrate type). The latter group is subclassified into two groups, group D1 (halogenated and with C16 substitution) and group D2 (the “labile” prodrug...
esters without the latter characteristics).

Tixocortol pivolate, hydrocortisone-17 butyrate, and budesonide are the screening agents of choice. Patients should be patch tested to screen for corticosteroid allergy. If a corticosteroid sensitivity is detected, a more extensive corticosteroid series should be tested to determine cross-reactivity patterns. Cross-reactivity among topically administered corticosteroids is frequent.

**Glaucoma**

There are isolated case reports of glaucoma occurring after the long-term use of topical steroids about the eyes. Glaucoma induced by the chronic use of steroid-containing eyedrops instilled directly into the conjunctival sac is encountered more frequently by ophthalmologists. The mechanism by which glaucoma develops from topical application is not understood, but presumably, cream applied to the lids seeps over the lid margin and into the conjunctival sac. It also seems possible that enough steroid could be absorbed directly through the lid skin into the conjunctival sac to produce the same results.

Inflammation about the eye is a common problem. Offending agents that cause inflammation may be directly transferred to the eyelids by rubbing with the hand, or they may be applied directly, as with cosmetics. Women who are sensitive to a favorite eye makeup often continue using that makeup on an interrupted basis, not suspecting the obvious source of allergy. Patients have been known to alternate topical steroids with a sensitizing makeup. Unsupervised use of over-the-counter hydrocortisone cream might also induce glaucoma.

No studies have yet determined what quantity or strength of steroid cream is required to produce glaucoma. The patient shown in Figure 2-13 used a group II topical steroid on the eyelids daily for 3 years. Severe atrophy and bleeding with the slightest trauma occurred, and ocular pressure was elevated.

It is good practice to restrict the use of topical steroids on the eyelids to a 2- to 3-week period and use only groups VI and VII preparations.
References


Chapter 3 - Eczema and Hand Dermatitis

Eczema (eczematous inflammation) is the most common inflammatory skin disease. Although the term dermatitis is often used to refer to an eczematous eruption, the word means inflammation of the skin and is not synonymous with eczematous processes. Recognizing a rash as eczematous rather than psoriasiform or lichenoid, for example, is of fundamental importance if one is to effectively diagnose skin disease. Here, as with other skin diseases, it is important to look carefully at the rash and to determine the primary lesion.

It is essential to recognize the quality and characteristics of the components of eczematous inflammation (erythema, scale, and vesicles) and to determine how these differ from other rashes with similar features. Once familiar with these features, the experienced clinician can recognize a process as eczematous even in the presence of secondary changes produced by scratching, infection, or irritation. With the diagnosis of eczematous inflammation established, a major part of the diagnostic puzzle has been solved.

THREE STAGES OF ECZEMA.

There are three stages of eczema: acute, subacute, and chronic. Each represents a stage in the evolution of a dynamic inflammatory process (Table 3-1). Clinically, an eczematous disease may start at any stage and evolve into another. Most eczematous diseases, if left alone (i.e., neither irritated, scratched, nor medicated), resolve in time without complication. This ideal situation is almost never realized; scratching, irritation, or attempts at topical treatment are almost inevitable. Some degree of itching is a cardinal feature of eczematous inflammation.
### TABLE 3-1 -- Eczematous Inflammation

<table>
<thead>
<tr>
<th>Stage</th>
<th>Primary and secondary lesions</th>
<th>Symptoms</th>
<th>Etiology and clinical presentation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Vesicles, blisters, intense redness</td>
<td>Intense itch</td>
<td>Contact allergy (poison ivy), severe irritation, id reaction, acute nummular eczema, stasis dermatitis, pompholyx (dyshidrosis), fungal infections</td>
<td>Cold wet compresses, oral or intramuscular steroids, topical steroids, antihistamines, antibiotics</td>
</tr>
<tr>
<td>Subacute</td>
<td>Redness, scaling, fissuring, parched appearance, scalded appearance</td>
<td>Slight to moderate itch, pain, stinging, burning</td>
<td>Contact allergy, irritation, atopic dermatitis, stasis dermatitis, nummular eczema, asteatotic eczema, fingertip eczema, fungal infections</td>
<td>Topical steroids with or without occlusion, lubrication, antihistamines, antibiotics, tar</td>
</tr>
<tr>
<td>Chronic</td>
<td>Thickened skin, skin lines accentuated (lichenified skin), excoriations, fissuring</td>
<td>Moderate to intense itch</td>
<td>Atopic dermatitis, habitual scratching, lichen simplex chronicus, chapped fissured feet, nummular eczema, asteatotic eczema, fingertip eczema, hyperkeratotic eczema</td>
<td>Topical steroids (with occlusion for best results), intralesional steroids, antihistamines, antibiotics, lubrication</td>
</tr>
</tbody>
</table>

**Figure 3-1** Acute eczematous inflammation. Numerous vesicles on an erythematous base. The vesicles may become confluent with time.

**Figure 3-2** Acute eczematous inflammation. Vesicle appeared during a 24-hour period in this patient with chronic hand eczema. Episodes of acute inflammation had occurred several times in the past.
Stages of Eczematous Inflammation

Acute eczematous inflammation

ETIOLOGY.

Inflammation is caused by contact with specific allergens such as Rhus (poison ivy, oak, or sumac) and chemicals. In the id reaction, vesicular reactions occur at a distant site during or after a fungal infection, stasis dermatitis, or other acute inflammatory processes.

PHYSICAL FINDINGS.

The degree of inflammation varies from moderate to intense. A bright red, swollen plaque with a pebbly surface evolves in hours. Close examination of the surface reveals tiny, clear, serum-filled vesicles (Figures 3-1 and 3-2). The eruption may not progress or it may go on to develop blisters. The vesicles and blisters may be confluent and are often linear. Linear lesions result from dragging the offending agent across the skin with the finger during scratching. The degree of inflammation in cases caused by allergy is directly proportional to the quantity of antigen deposited on the skin. Excoriation predisposes to infection and causes serum, crust, and purulent material to accumulate.

SYMPTOMS.

Acute eczema itches intensely. Patients scratch the eruption even while sleeping. A hot shower temporarily relieves itching because the pain produced by hot water is better tolerated than the sensation of itching; however, heat aggravates acute eczema.

COURSE.

Lesions may begin to appear from hours to 2 to 3 days after exposure and may continue to appear for a week or more. These later-occurring, less inflammatory lesions are
confusing to the patient, who cannot recall additional exposure. Lesions produced by small amounts of allergen are slower to evolve. They are not produced, as is generally believed, by contact with the serum of ruptured blisters, because the blister fluid does not contain the offending chemical. Acute eczematous inflammation evolves into a subacute stage before resolving.

**TREATMENT**

Cool wet dressings.

The evaporative cooling produced by wet compresses causes vasoconstriction and rapidly suppresses inflammation and itching. Burrow's powder, available in a 12-packet box, may be added to the solution to suppress bacterial growth, but water alone is usually sufficient. A clean cotton cloth is soaked in cool water, folded several times, and placed directly over the affected areas. Evaporative cooling produces vasoconstriction and decreases serum production. Wet compresses should not be held in place and covered with towels or plastic wrap because this prevents evaporation. The wet cloth macerates vesicles and, when removed, mechanically debrides the area and prevents serum and crust from accumulating. Wet compresses should be removed after 30 minutes and replaced with a freshly soaked cloth. It is tempting to leave the drying compress in place and to wet it again by pouring solution onto the cloth. Although evaporative cooling will continue, irritation may occur from the accumulation of scale, crust, serum, and the increased concentration of aluminum sulfate and calcium acetate, the active ingredients in Burrow's powder.

Oral corticosteroids.

Oral corticosteroids such as prednisone are useful for controlling intense or widespread inflammation and may be used in addition to wet dressings. Prednisone controls most cases of poison ivy when it is taken in 20-mg doses twice a day for 7 to 14 days (for adults); however, to treat intense or generalized inflammation, prednisone may be started at 30?mg or more twice a day and maintained at that level for 3 to 5 days. Sometimes 21 days of treatment are required for adequate control. The dosage should not be tapered for these relatively short courses because lower dosages may not give the desired antiinflammatory effect. Inflammation may reappear as diffuse erythema and may even be more extensive if the dosage is too low or is tapered too rapidly. Commercially available steroid dose packs taper the dosage and provide treatment for too short a time and so should not be used. Topical corticosteroids are of little use in the acute stage because the cream does not penetrate through the vesicles.

Antihistamines.

Antihistamines, such as diphenhydramine (Benadryl) and hydroxyzine (Atarax), do not alter the course of the disease, but they relieve itching and provide enough sedation so patients can sleep. They are given every 4 hours as needed.
Antibiotics.

The use of oral antibiotics may greatly hasten resolution of the disease if signs of superficial secondary infection, such as pustules, purulent material, and crusts, are present. Staphylococcus is the usual pathogen, and cultures are not routinely necessary. Deep infection (cellulitis) is rare with acute eczema. Erythromycin, cephalexin, and dicloxacillin are effective; topical antibiotics are much less effective.

Subacute eczematous inflammation

PHYSICAL FINDINGS.

Erythema and scale are present in various patterns, usually with indistinct borders (Figures 3-3 and 3-4). The redness may be faint or intense (Figures 3-5 through 3-8). Psoriasis, superficial fungal infections, and eczematous inflammation may have a similar appearance (Figures 3-9 through 3-11). The borders of the plaques of psoriasis and superficial fungal infections are well defined. Psoriatic plaques have a deep, rich red color and silvery white scales.

SYMPTOMS.

These vary from no itching to intense itching.

Figure 3-3 Subacute and chronic eczematous inflammation. The skin is dry, red, scaling, and thickened.

COURSE.

Subacute eczematous inflammation may be the initial stage or it may follow acute inflammation. Irritation, allergy, or infection can convert a subacute process into an acute one. Subacute inflammation resolves spontaneously without scarring if all sources of irritation and allergy are withdrawn. Excess drying created from washing or continued use of wet dressings causes cracking and fissures. If excoriation is not controlled, the subacute process can be converted to a chronic one. Diseases that have subacute eczematous inflammation as a characteristic are listed in Box 3-1.

Figure 3-4 Subacute and chronic eczematous inflammation. The ear canal is red, scaling, and thickened from chronic excoriation.
Figure 3-5 Red, scaling, nummular (round) superficial plaques occurred during the winter months from excessive washing.

<table>
<thead>
<tr>
<th>Box 3-1. Diseases Presenting as Subacute Eczematous Inflammation</th>
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<tbody>
<tr>
<td><strong>Allergic contact dermatitis</strong></td>
</tr>
<tr>
<td><strong>Asteatotic eczema</strong></td>
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<tr>
<td><strong>Atopic dermatitis</strong></td>
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<tr>
<td><strong>Chapped fissured feet (sweaty sock dermatitis)</strong></td>
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<tr>
<td><strong>Circumileostomy eczema</strong></td>
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<tr>
<td><strong>Diaper dermatitis</strong></td>
</tr>
<tr>
<td><strong>Exposure to chemicals</strong></td>
</tr>
</tbody>
</table>

Figure 3-6 Erythema and scaling are present, the surface is dry, and the borders are indistinct.

Figure 3-7 The areolae of both breasts are red and scaly. Inflammation of one areola is characteristic of Paget’s disease.
Figure 3-8 Wetting the lip by licking will eventually cause chapping and then eczema.

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SUBACUTE ECZEMATOUS INFLAMMATION

Figure 3-9 Acute vesicular eczema has evolved into subacute eczema with redness and scaling.

Figure 3-10 Acute and subacute eczematous inflammation. Acute vesicular eczema is evolving into subacute eczema. Vesicles, redness, and scaling are all present in this lesion undergoing transition.

Figure 3-11 Subacute eczematous inflammation. Erythema and scaling in a round or nummular pattern.

TREATMENT.

It is important to discontinue wet dressings when acute inflammation evolves into subacute inflammation. Excess drying creates cracking and fissures, which predispose to infection.

Topical corticosteroids.

These agents are the treatment of choice (see Chapter 2). Creams may be applied 2 to 4 times a day or with occlusion. Ointments may be applied 2 to 4 times a day for drier lesions. Subacute inflammation requires groups III through V corticosteroids for rapid control. Occlusion with creams hastens resolution, and less expensive, weaker products such as triamcinolone cream 0.1% (Kenalog) give excellent results. *Staphylococcus aureus* colonizes eczematous lesions, but studies show their numbers are significantly reduced following treatment with topical steroids.[1]

Topical macrolide immune suppressants.

Tacrolimus ointment (Protopic) and pimecrolimus cream (Elidel) are the first topical macrolide immune suppressants that are not hydrocortisone derivatives. They inhibit the production of inflammatory cytokines in T cells and mast cells and prevent the release of preformed inflammatory mediators from mast cells. Dermal atrophy does not occur. These agents are effective for the treatment of inflammatory skin diseases, such as atopic dermatitis, allergic contact dermatitis, and irritant contact dermatitis. They are approved for use in children 2 years or older. Response to these agents is slower than
the response to topical steroids. Topical steroids may be used for several days before the use of these agents to obtain rapid control.

Pimecrolimus (Elidel) cream.

Pimecrolimus permeates through skin at a lower rate than tacrolimus, indicating a lower potential for percutaneous absorption. The cream is applied twice a day and may be used on the face. There are no restrictions on duration of use.

Tacrolimus ointment (Protopic).

Tacrolimus is effective in the treatment of children (aged 2 years and older) and adults with atopic dermatitis and eczema. The most prominent adverse event is application site burning and erythema. It is available in 0.03% and 0.1% ointment formulations. Some clinicians find the 0.03% concentration to be marginally effective.

Doxepin cream.

A topical form of the antidepressant doxepin (doxepin 5% cream; Zonalon) is effective for the relief of pruritus associated with eczema in adults and children aged over 12 years. The two most common adverse effects are stinging at the site of application and drowsiness. The medication can be applied four times a day as needed.

Lubrication.

This is a simple but essential part of therapy. Inflamed skin becomes dry and is more susceptible to further irritation and inflammation. Resolved dry areas may easily relapse into subacute eczema if proper lubrication is neglected. Lubricants are best applied a few hours after topical steroids and should be continued for days or weeks after the inflammation has cleared. Frequent application (1 to 4 times a day) should be encouraged. Applying lubricants directly after the skin has been patted dry following a shower seals in moisture. Lotions or creams with or without the hydrating chemicals urea and lactic acid may be used. Bath oils are very useful if used in amounts sufficient to make the skin feel oily when the patient leaves the tub.

Lotions.

Curel, DML, Lubriderm, Cetaphil or any of the other lotions listed in the Formulary are useful.

Creams.

DML, Moisturel, Neutrogena, Nivea, Eucerin, and Acid Mantle or any of the other creams listed in the Formulary are useful.
Mild soaps.

Frequent washing with a drying soap, such as Ivory, delays healing. Infrequent washing with mild or superfatted soaps (e.g., Dove, Cetaphil, Basis—see the Formulary) should be encouraged. It is usually not necessary to use hypoallergenic soaps or to avoid perfumed soaps. Although allergy to perfumes occurs, the incidence is low.

Antibiotics.

Eczematous plaques that remain bright red during treatment with topical steroids may be infected. Infected subacute eczema should be treated with appropriate systemic antibiotics, which are usually those active against staphylococci. Systemic antibiotics are more effective than topical antibiotics or antibiotic-steroid combination creams.

Tar.

Tar ointments, baths, and soaps were among the few effective therapeutic agents available for the treatment of eczema before the introduction of topical steroids. Topical steroids provide rapid and lasting control of eczema in most cases. Some forms of eczema, such as atopic dermatitis and irritant eczema, tend to recur. Topical steroids become less effective with long-term use. Tar is sometimes an effective alternative in this setting. Tar ointments or creams may be used for long-term control or between short courses of topical steroids.

Chronic eczematous inflammation

Etiology.

Chronic eczematous inflammation may be caused by irritation of subacute inflammation, or it may appear as lichen simplex chronicus.

Physical findings.

Chronic eczematous inflammation is a clinical-pathologic entity and does not indicate simply any long-lasting stage of eczema. If scratching is not controlled, subacute eczematous inflammation can be modified and converted to chronic eczematous inflammation (Figure 3-12). The inflamed area thickens, and surface skin markings may become more prominent. Thick plaques with deep parallel skin marking (“washboard lesion”) are said to be lichenified (Figure 3-13). The border is well defined but not as sharply defined as it is in psoriasis (Figure 3-14). The sites most commonly involved are those areas that are easily reached and associated with habitual scratching.
(e.g., dorsal feet, lateral forearms, anus, and occipital scalp), areas where eczema tends to be long-lasting (e.g., the lower legs, as in stasis dermatitis), and the crease areas (antecubital and popliteal fossa, wrists, behind the ears, and ankles) in atopic dermatitis (Figures 3-15, 3-16, and 3-17).

SYMPTOMS.

There is moderate to intense itching. Scratching sometimes becomes violent, leading to excoriation and digging, and ceases only when pain has replaced the itch. Patients with chronic inflammation scratch while asleep.

Figure 3-12 Subacute and chronic eczema. Dermatitis of the lids may be allergic, irritant, or atopic in origin. This atopic patient rubs the lids with the back of the hands.

COURSE.

Scratching and rubbing become habitual and are often done unconsciously. The disease then becomes self-perpetuating. Scratching leads to thickening of the skin, which itches more than before. It is this habitual manipulation that causes the difficulty in eradicating this disease. Some patients enjoy the feeling of relief that comes from scratching and may actually desire the reappearance of their disease after treatment.

TREATMENT.

Chronic eczematous inflammation is resistant to treatment and requires potent steroid therapy.

Topical steroids.

Groups II through V topical steroids are used with occlusion each night until the inflammation clears—usually in 1 to 3 weeks; group I topical steroids are used without occlusion.

Intralesional injection.

Intralesional injection (Kenalog, 10?mg/ml) is a very effective mode of therapy. Lesions that have been present for years may completely resolve after one injection or a short series of injections. The medicine is delivered with a 27- or 30-gauge needle, and the entire plaque is infiltrated until it blanches white. Resistant plaques require additional injections given at 3- to 4-week intervals.

Figure 3-13 Chronic eczematous inflammation. Chronic excoriations thicken the epidermis, which results in accentuated skin lines. Chronic eczema created by picking is called lichen simplex chronicus.
CHRONIC ECZEMATOUS INFLAMMATION

**Figure 3-14** Erythema and scaling are present, and the skin lines are accentuated, creating a lichenified or "washboard" lesion.

**Figure 3-15** Atopic dermatitis. Atopic dermatitis is common in the crease areas. Atopic patients scratch, lichenify the skin, and often create a chronic process.

**Figure 3-16** Picking and rubbing thickened the skin behind the ear.

**Figure 3-17** A plaque of lichen simplex chronicus created by excoriation is present. Accentuated skin lines and eczematous papules beyond the border help to differentiate this process from psoriasis.
Hand Eczema

Inflammation of the hands is one of the most common problems encountered by the dermatologist. Hand dermatitis causes discomfort and embarrassment and, because of its location, interferes significantly with normal daily activities. Hand dermatitis is common in industrial occupations: it can threaten job security if inflammation cannot be controlled. Box 3-2 lists instructions for patients with irritant hand dermatitis.
### Box 3-2. Irritant Hand Dermatitis
#### Instructions for Patients

<table>
<thead>
<tr>
<th>1. Wash hands as infrequently as possible. Ideally, soap should be avoided and hands simply washed in lukewarm water.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Shampooing must be done with rubber gloves or by someone else.</td>
</tr>
<tr>
<td>3. Avoid direct contact with household cleaners and detergents. Wear cotton, plastic, or rubber gloves when doing housework.</td>
</tr>
<tr>
<td>4. Do not touch or do anything that causes burning or itching (e.g., wool; wet diapers; peeling potatoes or handling fresh fruits, vegetables, and raw meat).</td>
</tr>
<tr>
<td>5. Wear rubber gloves when irritants are encountered. Rubber gloves alone are not sufficient because the lining collects sweat, scales, and debris and can become more irritating than those objects to be avoided. Dermal white cotton gloves</td>
</tr>
</tbody>
</table>
debris and can become more irritating than those objects to be avoided. Dermal white cotton gloves should be worn next to the skin under unlined rubber gloves. Several pairs of cotton gloves should be purchased so they can be changed frequently. Try on the rubber gloves over the white cotton gloves at the time of purchase to ensure a comfortable fit.

EPIDEMIOLOGY.

A large study provided the following statistics: the prevalence of hand eczema was approximately 5.4% and was twice as common in females as in males. The most common type of hand eczema was irritant contact dermatitis (35%), followed by atopic hand eczema (22%), and allergic contact dermatitis (19%). The most common contact allergies were to nickel, cobalt, fragrance mix, balsam of Peru, and colophony. Of all the occupations studied, cleaners had the highest prevalence at 21.3%. Hand eczema was more common among people reporting occupational exposure. The most harmful exposure was to chemicals, water and detergents, dust, and dry dirt. A change of occupation was reported by 8% and was most common in service workers. Hairdressers had the highest frequency of change. Hand eczema was shown to be a long-lasting disease with a relapsing course; 69% of the patients had consulted a physician, and 21% had been on sick leave at least once because of hand eczema. The mean total sick-leave time was 18.9 weeks; the median was 8 weeks. The most important predictive factors for hand eczema are listed in Box 3-3.
Box 3-3. Predictive Factors for Hand Eczema

<table>
<thead>
<tr>
<th>Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of childhood eczema (most important predictive factor)</td>
</tr>
<tr>
<td>Female sex</td>
</tr>
<tr>
<td>Occupational exposure</td>
</tr>
<tr>
<td>History of asthma and/or hay fever</td>
</tr>
<tr>
<td>Service occupation (cleaners, etc.)</td>
</tr>
</tbody>
</table>

*From Meding B, Swanbeck G: Contact Dermatitis 23:154, 1990.*

**TABLE 3-2 -- Hand Dermatitis: Differential Diagnosis and Distribution**

<table>
<thead>
<tr>
<th>Location</th>
<th>Redness and scaling</th>
<th>Vesicles</th>
<th>Pustules</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back of hand</td>
<td>Atopic dermatitis</td>
<td>Id reaction</td>
<td>Bacterial infection</td>
</tr>
<tr>
<td></td>
<td>Irritant contact dermatitis</td>
<td>Scabies (web spaces)</td>
<td>Psoriasis</td>
</tr>
<tr>
<td></td>
<td>Lichen simplex chronicus</td>
<td></td>
<td>Scabies (web spaces)</td>
</tr>
<tr>
<td>Nummular eczema</td>
<td></td>
<td></td>
<td>Tinea</td>
</tr>
<tr>
<td>Psoriasis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tinea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palmar surface</td>
<td>Fingertip eczema</td>
<td>Allergic contact dermatitis</td>
<td>Bacterial infection</td>
</tr>
<tr>
<td></td>
<td>Hyperkeratotic eczema</td>
<td>Pompholyx (dyshidrosis)</td>
<td>Pompholyx (dyshidrosis)</td>
</tr>
<tr>
<td>Recurrent focal palmar peeling</td>
<td></td>
<td></td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Psoriasis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
DIAGNOSIS.

The diagnosis and management of hand eczema is a challenge. There is almost no association between clinical pattern and etiology. No distribution of eczema is typically allergic, irritant, or endogenous. Not only are there many patterns of eczematous inflammation (Table 3-2), but there are other diseases, such as psoriasis, that may appear eczematous. The original primary lesions and their distribution become modified with time by irritants, excoriation, infection, and treatment. All stages of eczematous inflammation may be encountered in hand eczema (Box 3-4).

Irritant contact dermatitis

Irritant hand dermatitis (housewives’ eczema, dishpan hands, detergent hands) is the most common type of hand inflammation. Some people can withstand long periods of repeated exposure to various chemicals and maintain normal skin. At the other end of the spectrum, there are those who develop chapping and eczema from simple hand washing. Patients whose hands are easily irritated may have an atopic diathesis.

PATHOPHYSIOLOGY.

The stratum corneum is the protective envelope that prevents exogenous material from entering the skin and prevents body water from escaping. The stratum corneum is composed of dead cells, lipids (from sebum and cellular debris), and water-binding organic chemicals. The stratum corneum of the palms is thicker than that of the dorsa and is more resistant to irritation. The pH of this surface layer is slightly acidic. Environmental factors or elements that change any component of the stratum corneum interfere with its protective function and expose the skin to irritants. Factors such as cold winter air and low humidity promote water loss. Substances such as organic solvents and alkaline soaps extract water-binding chemicals and lipids. Once enough of these protective elements have been extracted, the skin decompensates and becomes eczematous.
CLINICAL PRESENTATION.

The degree of inflammation depends on factors such as strength and concentration of the chemical, individual susceptibility, site of contact, and time of year. Allergy, infection, scratching, and stress modify the picture.

STAGES OF INFLAMMATION.

Dryness and chapping are the initial changes (Figure 3-18). Very painful cracks and fissures occur, particularly in joint crease areas and around the fingertips. The backs of the hands become red, swollen, and tender. The palmar surface, especially that of the fingers, becomes red and continues to be dry and cracked. A red, smooth, shiny, delicate surface that splits easily with the slightest trauma may develop. These are subacute eczematous changes (Figures 3-19 and 3-20).

Acute eczematous inflammation occurs with further irritation creating vesicles that ooze and crust. Itching intensifies, and excoriation leads to infection (Figures 3-21 and 3-22).

Necrosis and ulceration followed by scarring occur if the irritating chemical is too caustic.

Figure 3-18 Early irritant hand dermatitis with dryness and chapping.
IRRITANT HAND DERMATITIS

Figure 3-19 Subacute eczematous inflammation appeared on the dry, chapped third and fourth fingers.

Figure 3-20 Subacute and chronic eczematous inflammation with severe drying and splitting of the fingertips.

Figure 3-21 Numerous tiny vesicles suddenly appeared on these chronically inflamed fingers.

Figure 3-22 Chronic eczematous inflammation. Scratching has thickened the skin. Crusts are signs of infection.

PATIENTS AT RISK.

Individuals at risk include mothers with young children (changing diapers), individuals whose jobs require repeated wetting and drying (e.g., surgeons, dentists, dishwashers, bartenders, fishermen), industrial workers whose jobs require contact with chemicals (e.g., cutting oils), and patients with the atopic diathesis.

PREVENTION.

One study revealed that hospital staff members who used an emulsion cleanser (e.g., Cetaphil lotion, Duosoft [in Europe]) had significantly less dryness and eczema than those who used a liquid soap. Regular use of emollients prevented irritant dermatitis caused by a detergent.

BARRIER-PROTECTANT CREAMS.

Loss of skin barrier function by mechanical or chemical insults may result in water loss and hand eczema. Barrier creams (see Box 3-5) applied at least twice a day on all exposed areas protect the skin and are formulated to be either water-repellent or oil-repellent. The water-repellent types offer little protection against oils or solvents.

TREATMENT.
The inflammation is treated as outlined in the section on stages of eczematous inflammation. Lubrication and avoidance of further irritation helps to prevent recurrence. A program of irritant avoidance should be carefully outlined for each patient (see Box 3-2).

<table>
<thead>
<tr>
<th>Box 3-5. Barrier Creams—Applied at Least Twice a Day on All Exposed Areas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Water repellent</strong></td>
</tr>
<tr>
<td>• North 201</td>
</tr>
<tr>
<td>• SBS-44</td>
</tr>
<tr>
<td>• Kerodex #71</td>
</tr>
<tr>
<td><strong>For oil- or solvent-based materials</strong></td>
</tr>
<tr>
<td>• Kerodex #51</td>
</tr>
<tr>
<td>• SBS-46</td>
</tr>
<tr>
<td>• North 222</td>
</tr>
<tr>
<td>• Dermashield (both oil- and water-based materials)</td>
</tr>
<tr>
<td><strong>General purpose barrier protective creams</strong></td>
</tr>
<tr>
<td>• SBR-Lipocream</td>
</tr>
<tr>
<td>• TheraSeal</td>
</tr>
</tbody>
</table>

**Atopic hand dermatitis**

Hand dermatitis may be the most common form of adult atopic dermatitis (see Chapter 5). Hand eczema is significantly more common in people with a history of atopic dermatitis than in others. The following factors predict the occurrence of hand eczema
in adults with a history of atopic dermatitis

• Hand dermatitis before age 15
• Persistent eczema on the body
• Dry or itchy skin in adult life
• Widespread atopic dermatitis in childhood

Many people with atopic dermatitis develop hand eczema independently of exposure to irritants, but such exposure causes additional irritant contact dermatitis.

The backs of the hands, particularly the fingers, are affected (Figure 3-23). The dermatitis begins as a typical irritant reaction with chapping and erythema. Several forms of eczematous dermatitis evolve; erythema, edema, vesiculation, crusting, excoriation, scaling, and lichenification appear and are intensified by scratching. Management for atopic hand eczema is the same as that for irritant hand eczema.

Allergic contact dermatitis

Allergic contact dermatitis of the hands is not as common as irritant dermatitis. However, allergy as a possible cause of hand eczema, no matter what the pattern, should always be considered in the differential diagnosis; it may be investigated by patch testing in appropriate cases. The incidence of allergy in hand eczema was demonstrated by patch testing in a study of 220 patients with hand eczema. In 12% of the 220 patients, the diagnosis was established with the aid of a standard screening series now available in a modified form (T.R.U.E. TEST). Another 5% of the cases were diagnosed as a result of testing with additional allergens. The hand eczema in these two groups (17%) changed dramatically after identification and avoidance of the allergens found by patch testing. Table 3-3 lists some possible causes of allergic hand dermatitis.

PHYSICAL FINDINGS.

The diagnosis of allergic contact dermatitis is obvious when the area of inflammation corresponds exactly to the area covered by the allergen (e.g., a round patch of eczema under a watch or inflammation in the shape of a sandal strap on the foot). Similar clues may be present with hand eczema, but in many cases allergic and irritant hand eczemas cannot be distinguished by their clinical presentation. Hand inflammation, whatever the source, is increased by further exposure to irritating chemicals, washing, scratching, medication, and infection. Inflammation of the dorsum of the hand is more
often irritant or atopic than allergic.

TREATMENT.

Allergy may initially appear as acute, subacute, or chronic eczematous inflammation and is managed accordingly.

### TABLE 3-3 -- Allergic Hand Dermatitis: Some Possible Causes

<table>
<thead>
<tr>
<th>Allergens</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nickel</td>
<td>Door knobs, handles on kitchen utensils, scissors, knitting needles, industrial equipment, hairdressing equipment</td>
</tr>
<tr>
<td>Potassium dichromate</td>
<td>Cement, leather articles (gloves), industrial machines, oils</td>
</tr>
<tr>
<td>Rubber</td>
<td>Gloves, industrial equipment (hoses, belts, cables)</td>
</tr>
<tr>
<td>Fragrances</td>
<td>Cosmetics, soaps, lubricants, topical medications</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Wash-and-wear fabrics, paper, cosmetics, embalming fluid</td>
</tr>
<tr>
<td>Lanolin</td>
<td>Topical lubricants and medications, cosmetics</td>
</tr>
</tbody>
</table>

### Nummular eczema

Eczema that appears as one or several coin-shaped plaques is called *nummular eczema*. This pattern often occurs on the extremities but may also present as hand eczema. The plaques are usually confined to the backs of the hands (Figure 3-24). The number of lesions may increase, but once they are established they tend to remain the same size. The inflammation is either subacute or chronic and itching is moderate to intense. The cause is unknown. Thick, chronic, scaling plaques of nummular eczema look like psoriasis; treatment for nummular eczema is the same as that for subacute or chronic eczema.
Lichen simplex chronicus

A localized plaque of chronic eczematous inflammation that is created by habitual scratching is called lichen simplex chronicus or localized neurodermatitis. The back of the wrist is a typical site. The plaque is thick with prominent skin lines (lichenification) and the margins are fairly sharp. Once established, the plaque does not usually increase in area. Lichen simplex chronicus is treated in the same manner as chronic eczematous inflammation.

Figure 3-24 Nummular eczema. Eczematous plaques are round (coin-shaped).

Recurrent focal palmar peeling

Keratolysis exfoliativa or recurrent focal palmar peeling is a common, chronic, asymptomatic, noninflammatory, bilateral peeling of the palms of the hands and occasionally soles of the feet; its cause is unknown[13] (Figure 3-25). The eruption is most common during the summer months and is often associated with sweaty palms and soles. Some people experience this phenomenon only once, whereas others have repeated episodes. Scaling starts simultaneously from several points on the palms or soles with 2 or 3?mm of round scales that appears to have originated from a ruptured vesicle; however, these vesicles are never seen. The scaling continues to peel and extend peripherally, forming larger, roughly circular areas that resemble ringworm whereas the central area becomes slightly red and tender. The scaling borders may coalesce. The condition resolves in 1 to 3 weeks and requires no therapy other than lubrication.

Figure 3-25 Keratolysis exfoliativa. Noninflammatory peeling of the palms that is often associated with sweating. The eruption must be differentiated from tinea of the palms.

Hyperkeratotic eczema

A very thick, chronic form of eczema that occurs on the palms and occasionally the soles is seen almost exclusively in men. One or several plaques of yellow-brown, dense scale increase in thickness and form deep interconnecting cracks over the surface, similar to mud drying in a river bed (Figure 3-26). The dense scale, unlike callus, is moist below the surface and is not easily pared with a blade. Patients discover that the scale is firmly adherent to the epidermis when they attempt to peel off the thick scale and this exposes tender bleeding areas of dermis. Hyperkeratotic eczema may result from allergy or excoriation and irritation, but in most cases the cause is not apparent. The disease is chronic and may last for years. Psoriasis and lichen simplex chronicus must be considered in the differential diagnosis. The disease is treated like chronic
eczema; although the plaques respond to group II steroid cream and occlusion, recurrences are frequent. Patch testing is indicated for recurrent disease.

**Figure 3-26** Hyperkeratotic eczema. Patches of dense yellow-brown scale occur on the palms. This patient was allergic to a steering wheel.

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**FINGERTIP ECZEMA**

**Figure 3-27** A, An early stage. The skin is moist. A vesicle is present. Redness and cracking have occurred in the central area. B, A more advanced stage. Peeling occurs constantly. The skin lines are lost.

**Figure 3-28** Asteatotic eczema. Excessive washing produced this advanced case with cracking and fissures.

---

**Fingertip eczema**

A very dry, chronic form of eczema of the palmar surface of the fingertips may be the result of an allergic reaction (e.g., to plant bulbs or resins) or may occur in children and adults as an isolated phenomenon of unknown cause. One finger or several fingers may be involved. Initially the skin may be moist and then may become dry, cracked, and scaly (Figures 3-27). The skin peels from the fingertips distally, exposing a very dry, red, cracked, fissured, tender, or painful surface without skin lines (Figures 3-27, 3-28, and 3-29). The process usually stops shortly before the distal interphalangeal joint is reached (Figures 3-29 and 3-31). Fingertip eczema may last for months or years and is resistant to treatment. Topical steroids with or without occlusion give only temporary relief. Once allergy and psoriasis have been ruled out, fingertip eczema should be managed the same way as subacute and chronic eczema, by avoiding irritants and lubricating frequently. Elidel or Protopic is sometimes effective; tar creams such as Fototar applied twice each day have at times provided relief.

**Figure 3-29** Fingertip eczema. Inflammation has been present for months and responded poorly to topical steroids.
Figure 3-30 Severe chronic inflammation. The skin lines are lost. The dry skin is fragile and cracks easily. Patients are tempted to peel away the dry loose scale.

Figure 3-31 The fingers are dry and wrinkled, and the skin is fragile. The skin peels but does not form the thick scale shown in Figures 3-29 and 3-30.

POMPHOLYX (DYSHIDROSIS)

Figure 3-32 Vesicles have evolved into pustules. The eruption has persisted for many weeks.

Figure 3-33 Vesicles have become infected. Pustular lesions occurred and then became more numerous.

Figure 3-34 The acute process ends as the skin peels, revealing a red, cracked base with brown spots. The brown spots are sites of previous vesiculation.

Figure 3-35 A severe form (with large, deep vesicles and blisters) that is indistinguishable from pustular psoriasis of the palms and soles.

Pompholyx

Pompholyx (dyshidrosis) is a distinctive reaction pattern of unknown etiology presenting as symmetric vesicular hand and foot dermatitis (Figure 3-32). Moderate to severe itching precedes the appearance of vesicles on the palms and sides of the fingers (Figure 3-33). The palms may be red and wet with perspiration, hence the name dyshidrosis. The vesicles slowly resolve in 3 to 4 weeks and are replaced by 1- to 3-mm rings of scale (Figures 3-34 and 3-35). Chronic eczematous changes with erythema, scaling, and lichenification may follow. Waves of vesiculation may appear indefinitely. Pustular psoriasis of the palms and soles may resemble pompholyx, but the vesicles of psoriasis rapidly become cloudy with purulent fluid, and pain rather than itching is the chief complaint. Pustular psoriasis is chronic and the pustules do not evolve and disappear as rapidly as those of pompholyx. Patients with atopic dermatitis are affected as frequently as others.

The cause of pompholyx is unknown, but there seems to be some relationship to stress. Pompholyx is a disease that disrupts the skin and allows sensitization to contact
allergens to occur, but direct contact with the allergen does not seem to be the cause of
the disease. Ingestion of allergens such as chromate, neomycin, quinoline, or nickel
may cause some cases. Ingestion of nickel, cobalt, and chromium can elicit
pompholyx in patients who are patch test negative to these metals. The
perspiration volume of pompholyx patients was found to be 2.5 times higher than that of
age-matched normal control; 20% of patients showed sensitivity to chromate, 16% to
cobalt, and 28% to nickel on patch testing. Some patients with positive results who are
challenged orally with nickel, cobalt, or chromium show vesicular reactions on their
hands. Sensitivity to orally ingested metal compounds in combination with local
hyperhidrosis may contribute to the development of vesicular lesions in pompholyx.

TREATMENT.

Topical steroids, cold wet compresses, and possibly oral antibiotics are used as the
initial treatment, but the response is often disappointing. Short courses of oral steroids
are sometimes needed to control acute flares. Resistant cases might respond to
PUVA. Patients (64%) who flared after oral challenge to metal salt cleared or
markedly improved on diets low in the incriminated metal salt, and 78% of those
patients remained clear when the diet was rigorously followed (a suggested diet for
nickel-sensitive patients with pompholyx appears on p. 94). Attempts to control
pompholyx with elimination diets may be worth a trial in difficult cases.

Patients with severe pompholyx who did not respond to conventional therapy or who
had debilitating side effects from corticosteroids were treated with low-dose
methotrexate (15 to 22.5?mg per week). This led to significant improvement or clearing,
and the need for oral corticosteroid therapy was substantially decreased or
eliminated.

Complete remission of severe dyshidrotic eczema was achieved with low-dose external
beam megavoltage therapy.

Id reaction

Intense inflammatory processes, such as active stasis dermatitis or acute fungal
infections of the feet, can be accompanied by an itchy, dyshidrotic-like vesicular
eruption (“id reaction”; Figure 3-36). These eruptions are most common on the sides of
the fingers but may be generalized. The eruptions resolve as the inflammation that
initiated them resolves. The id reaction may be an allergic reaction to fungi or to some
antigen created during the inflammatory process. Almost all dyshidrotic eruptions are
incorrectly called id reactions. The diagnosis of an id reaction should not be made
unless there is an acute inflammatory process at a distant site and the id reaction
disappears shortly after the acute inflammation is controlled.

Figure 3-36 Id reaction. An acute vesicular eruption most often seen on the lateral aspects of the fingers.
Figure 3-37 Asteatotic eczema (xerosis). The skin is extremely dry, cracked, and scaly. This pattern appears in the winter months when the air is dry.

Figure 3-38 Asteatotic eczema (xerosis). Excessive washing of the dry skin shown in Figure 3-37 may result in horizontal, parallel cracks.
Eczema: Various Presentations

Asteatotic eczema

Asteatotic eczema (eczema craquele) occurs after excess drying, especially during the winter months and among the elderly. Patients with an atopic diathesis are more likely to develop this distinctive pattern. The eruption can occur on any skin area, but it is most commonly seen on the anterolateral aspects of the lower legs. The lower legs become dry and scaly and show accentuation of the skin lines (xerosis) (Figure 3-37). Red plaques with thin, long, horizontal superficial fissures appear with further drying and scratching (Figure 3-38). Similar patterns of inflammation may appear on the trunk and upper extremities as the winter progresses. A cracked porcelain or “crazy paving” pattern of fissuring develops when short vertical fissures connect with the horizontal fissures. The term eczema craquele is appropriately used to describe this pattern. The severest form of this type of eczema shows an accentuation of the above pattern with deep, wide, horizontal fissures that ooze and are often purulent (Figure 3-39). Pain, rather than itching, is the chief complaint with this condition. Scratching or treatment with drying lotions such as calamine aggravates the eczematous inflammation and leads to infection with accumulation of crusts and purulent material.

**Figure 3-39** Asteatotic eczema (eczema craquele). Excessive drying on the lower legs may eventually become so severe that long, horizontal, superficial fissures appear. The fissures eventually develop a cracked porcelain or “crazy paving” pattern when short vertical fissures connect with the horizontal fissures.

The initial stages are treated as subacute eczematous inflammation with groups III or IV topical steroid ointments. The severest form may have to be treated as acute eczema. The treatment involves wet compresses and antibiotics to remove crust and suppress infection before group V topical steroids and lubricants are applied. Wet compresses should be used only for a short time (one or two days). Prolonged use of wet
compresses results in excessive drying. Lubricating the dry skin during and after topical steroid use is essential. The use of oral steroids should be avoided; the disease flares within 1 or 2 days once they are discontinued.

**Nummular eczema**

Nummular eczema is a common disease of unknown cause that occurs primarily in the middle-aged and elderly. The typical lesion is a coin-shaped, red plaque that averages 1 to 5 cm in diameter (Figure 3-40). The lesions can itch, and scratching often becomes habitual. In these cases, the term *nummular neurodermatitis* has been used (Figure 3-41). The plaque may become thicker and vesicles appear on the surface; vesicles in ringworm, if present, are at the border. Unlike the thick, silvery scale of psoriasis, this scale is thin and sparse. The erythema in psoriasis is darker. Once the disease is established, lesions may become more numerous, but individual lesions tend to remain in the same area and do not increase in size. The disease is worse in the winter. The back of the hand is the most commonly involved site; usually only one lesion or a few lesions are present (see Figure 3-23). Other frequently involved areas are the extensor aspects of the forearms and lower legs, the flanks, and the hips. Lesions in these other sites tend to be more numerous. An extensive form of the disease can occur suddenly in patients with dry skin that is exposed to an irritating medicine or chemical, or in patients who have an active eczematous process at another site, such as stasis dermatitis on the lower legs. The lesions in these cases are round, faintly erythematous, dry, cracked, superficial, and usually confluent.

The course is variable, but it is usually chronic, with some cases resisting all attempts at treatment. Many cases become inactive after several months. Lesions may reappear at previously involved sites in recurrent cases.

**TREATMENT.**

Treatment depends on the stage of activity; all stages of eczematous inflammation may be present simultaneously. The red vesicular lesions are treated as acute, the red scaling plaques as subacute, and the habitually scratched thick plaques as chronic eczematous inflammation.

**ADULT-ONSET RECALCITRANT ECZEMA AND MALIGNANCY.**

Generalized eczema or erythroderma may be the presenting sign of cutaneous T-cell lymphoma. Intractable pruritus has been associated with Hodgkin’s lymphoma. Unexplained eczema of adult onset may be associated with an underlying
lymphoproliferative malignancy. Patients may have widespread erythematous plaques that are poorly responsive to therapy. When a readily identifiable cause (e.g., contactants, drugs, or atopy) is not found, a systematic evaluation should be pursued.[22]

**Figure 3-41** Nummular eczema. Round, eczematous plaques formed on the trunk and arms become confluent.
Chapped Fissured Feet

CLINICAL PRESENTATION.

Chapped fissured feet (sweaty sock dermatitis, peridigital dermatitis, juvenile plantar dermatosis) are seen initially with scaling, erythema, fissuring, and loss of the epidermal ridge pattern. The tendency to severe chapping declines with age and is gone around the age of puberty. The mean age of onset is 7.3 years; the mean age of remission is 14.3 years. Onset is in early fall when the weather becomes cold and heavy socks and impermeable shoes or boots are worn. An artificial intertrigo is created when moist socks are kept in contact with the soles. The skin in pressure areas, toes, and metatarsal regions becomes dry, brittle, and scaly, and then fissured (Figure 3-42, A). The chapping extends onto the sides of the toes. Eventually, the entire sole may be involved; sometimes the hands are also affected (Figure 3-42, B).

The eruption lasts throughout the winter, clears without treatment in the late spring, and predictably recurs the next fall. Earlier descriptions referred to this entity as atopic winter feet in children, but the name has been changed to include patients who do not have atopic dermatitis. Atopic dermatitis of the feet in children occurs on the dorsal toes and usually not on the plantar surface, and it is itchy. The role of atopy is not yet defined. Children with chapped fissured feet complain of soreness and pain. Affected individuals must be

Figure 3-42 Chapped fissured feet. A, An early stage with erythema and cracking on pressure areas. B, An advanced case in which the entire plantar surface is severely dried and fissured.

predisposed to chapping because their wearing of moist socks and impermeable boots does not differ from that of unaffected children.

DIFFERENTIAL DIAGNOSIS.
The differential diagnosis includes psoriasis, tinea pedis, and allergic contact dermatitis. The erythema in psoriasis is darker and the scales shed; the scales in chapped fissured feet are adherent, and removal of the scales causes bleeding. Tinea of the feet in children is rare. Feet with the rare case of familial *Trichophyton rubrum* are pale brown and have a fine scale. Fissuring is minimal, and there is little seasonal variation. Allergic contact dermatitis to shoes usually affects the dorsal aspect and spares the soles, webs, and sides of the feet. The eruption is bright red and scaly rather than pale red and chapped.

**TREATMENT.**

Treatment is less than satisfactory. Topical steroids and lubrication provide some relief. Group II or III topical steroids are applied twice each day or, preferably, with plastic wrap occlusion at bedtime. Elidel cream or Protopic ointment may be effective. Lubricating creams are applied several times each day, especially directly after removing moist socks to seal in moisture. The feet should not be allowed to remain moist inside shoes. Preventive measures include changing into light leather shoes after removing boots at school and changing cotton socks 1 or 2 times each day.
Self-Inflicted Dermatoses

A number of skin disorders are created or perpetuated by manipulation of the skin surface [25][26][27][28][29][30][31][32][33][34]. Patients may benefit from both dermatologic and psychiatric care. The most common self-inflicted dermatoses are discussed here.

Lichen simplex chronicus

Lichen simplex chronicus (Figures 3-43 through 3-48), or circumscribed neurodermatitis, is an eczematous eruption that is created by habitual scratching of a single localized area. The disease is more common in adults, but may be seen in children. The areas most commonly affected are those that are conveniently reached. These are listed in Box 3-6 in approximate order of frequency. Patients derive great pleasure in the relief that comes with frantically scratching the inflamed site. Loss of this pleasurable sensation or continued subconscious habitual scratching may explain why this eruption frequently recurs.

A typical plaque stays localized and shows little tendency to enlarge with time. Red papules coalesce to form a red, scaly, thick plaque with accentuation of skin lines (lichenification). Lichen simplex chronicus is a chronic eczematous disease, but acute changes may result from sensitization with

Figure 3-43 Lichen simplex chronicus of the vulva. The skin lines are markedly accentuated from years of rubbing and scratching.
Box 3-6. Lichen Simplex Chronicus:
Areas Most Commonly Affected Listed
in Approximate Order of Frequency

<table>
<thead>
<tr>
<th>Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outer lower portion of lower leg</td>
</tr>
<tr>
<td>Scrotum, vulva, anal area, pubis</td>
</tr>
<tr>
<td>Wrists and ankles</td>
</tr>
<tr>
<td>Upper eyelids</td>
</tr>
<tr>
<td>Back (lichen simplex nuchae) and side of neck</td>
</tr>
<tr>
<td>Orifice of the ear</td>
</tr>
<tr>
<td>Extensor forearms near elbow</td>
</tr>
<tr>
<td>Fold behind the ear</td>
</tr>
<tr>
<td>Scalp-picker's nodules</td>
</tr>
</tbody>
</table>

Figure 3-44 Anal excoriations. Scratching has produced focal erosions and thickening of the skin about the anus.

topical medication. Moist scale, serum, crusts, and pustules are signs of infection.

Lichen simplex nuchae occur almost exclusively in women who reach for the back of the neck during stressful situations (see Figure 3-46). The disease may spread beyond the initial well-defined plaque. Diffuse dry or moist scale, crust, and erosions extend into the posterior scalp beyond the neck. Secondary infection is common. Nodules, usually less than 1 cm and scattered randomly in the scalp, occur in patients who frequently pick at the scalp; there may be few nodules or many.
CHRONIC VULVAR ITCHING.

Women who have chronic vulvar itching usually have eczema. The degree of itching may not correspond to the appearance of the skin. Scratching begins a cycle that makes the skin rough, red and irritated, producing more itching. Lichen sclerosus, contact dermatitis, lichen planus, psoriasis and Paget's disease are other causes of itching. A 2 to 4 week course of a group I topical steroid is usually very effective.

RED SCROTUM SYNDROME.

Lichen simplex of the scrotum is a common finding, and thickened skin with accentuated skin markings is typical. Some patients present with persistent redness of the anterior half of the scrotum that may involve the base of the penis. There is persistent itching, burning or pain. The cause is unknown and it is resistant to treatment.

TREATMENT.

The patient must first understand that the rash will not clear until even minor scratching and rubbing is stopped. Scratching frequently takes place during sleep, and the affected area may have to be covered. Lichen simplex chronicus is chronic eczema and is treated as outlined in the section on eczematous inflammation. Treatment of the anal area or the fold behind the ear does not require potent topical steroids as do other forms of lichen simplex; rather, these intertriginous areas respond to group V or VI topical steroids. Lichen simplex nuchae, because of its location, is difficult to treat. Dry inflammation that extends into the scalp may be treated with a group II steroid gel such as fluocinonide (Lidex) applied twice each day. Moist, secondarily infected areas respond to oral antibiotics and topical steroid solutions (e.g., Cormax Scalp Solution). A 2- to 3-week course of prednisone (20?mg twice daily) should be considered when an extensively inflamed scalp does not respond rapidly to topical treatment. Nodules caused by picking at the scalp may be very resistant to treatment, requiring monthly intralesional injections of triamcinolone acetonide (Kenalog 10?mg/ml). Botulinum toxin A injected intradermally into lichenified lesions may block acetylcholine release and control pruritus. Pruritus subsided within 3 to 7 days and lesions cleared in 2 to 4 weeks.

<table>
<thead>
<tr>
<th>TABLE 3-4 -- Self-Inflicted and Self-Perpetuated Dermatoses</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dermatologic complaint that is a primary psychiatric symptom</strong></td>
</tr>
<tr>
<td>-------------------------------------------------------------</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>


| Psychogenic parasitosis (Delusions of parasitosis) | Focal erosions and scars  
Patients convinced they are infested and angry with doctors because “no one believes them” | Delusional disorder, somatic type, or monosymptomatic hypochondriacal psychosis; shared psychotic disorder with another person (folie a deux); major depressive disorder with psychotic features; incipient schizophrenia | Most patients are women over 50 | Antipsychotics (pimozide [Orap])  
Antidepressants may be used for comorbid depressive disease  
Anxiolytics and hypnotics may be used in conjunction with antipsychotics in some cases |
|---|---|---|---|---|
| Factitial dermatitis | Cutaneous lesions are wholly self-inflicted; patient denies their self-inflicted nature  
Wide range of lesions, blisters, ulcers, burns  
Bizarre patterns not characteristic of any disease  
Often a diagnosis of exclusion  
Adolescents, young adults | Personality disorder, cutaneous lesions are an appeal for help  
Posttraumatic stress disorder  
Rule out sexual and child abuse  
Depression, psychosis, obsessive-compulsive disorder, malingering, and Munchausen's syndrome | Ratio of female to male patients is 4 to 1  
Sudden appearance of lesions “Hollow history”; patient cannot describe how lesion evolved | Empathic, supportive approach  
None in most cases  
Antipsychotics and antidepressants may be used in posttraumatic stress disorder |
| Neurotic excoriations and acne excoriée | Possibly initiated by itchy skin disease  
Repetitive self-excoriation—patient admits self-inflicted nature  
Linear excoriations in easily reached areas  
Groups of round or linear scars | Depression, obsessive-compulsive disorder, perfectionistic traits, presence of significant psychosocial stressor in 33%–98% of patients  
Body image problems, including eating disorders, in acne excoriée | Exclude systemic causes of itching  
Patient admits self-inflicted nature | Empathic, supportive approach  
Antidepressants, especially SSRIs; antianxiety and antipsychotic drugs may be used as adjunctive therapies where indicated |
| Trichotillomania | Compulsive extraction of hair  
Nonscarring alopecia as a result of self-plucking of hair; patients deny that their alopecia is self-induced in 43% of cases  
Hairs of various lengths  
Area not completely devoid of hair | A variant of obsessive-compulsive disorder  
Many causes  
Depressive illness  
Disturbed parent-child relationship  
Stressful life situation  
Usually not a primary psychiatric disorder | Many patients are girls between 5 and 12  
Patients may deny pulling hair  
KOH exam rules out tinea  
Biopsy shows no hair in follicle  
Hair pluck shows 100% of hairs in anagen | Antidepressants, especially the SSRIs, for some patients. Underlying psychiatric pathology should be diagnosed before psychotropic agents are used  
Psychotherapy and family therapy |
|---|---|---|---|
| Lichen simplex chronicus | Created and perpetuated by constant scratching and rubbing  
Very thick oval plaques  
Usually just one lesion  
Severe itching  
Lasts indefinitely  
Recurs frequently | No known psychopathology  
Triggered by stress | Biopsy shows eczematous inflammation or resembles psoriasis | Topical steroids and plastic occlusion  
Cordran tape  
Intralesional steroids |
| Prurigo nodularis | 0.5–1?cm itchy nodules on arms and legs; lasts for years | Severe pruritus interferes with life activities and sleep | Biopsy shows very thick epidermis and hyperplasia of nerve fibers | Intralesional steroids  
Cryotherapy  
Excision  
Capsaicin cream  
Calcipotriol ointment |


SSRIs, Selective serotonin reuptake inhibitors.
LICHEN SIMPLEX CHRONICUS

Figure 3-45 This localized plaque of chronic eczematous inflammation was created by rubbing with the opposite heel.

Figure 3-46 Lichen simplex nuchae occurs almost exclusively in women who scratch the back of their neck in stressful situations.

LICHEN SIMPLEX CHRONICUS

Figure 3-47 Lichen simplex chronicus of the scrotum. The skin is thickened and skin lines are accentuated, unlike the adjacent scrotal skin.

Figure 3-48 Two linear areas are picked and scratched, causing the skin to become very thick. The patient scratches during the day and while asleep.

Prurigo nodularis

Prurigo nodularis is an uncommon disease of unknown cause that may be considered a nodular form of lichen simplex chronicus. There is intractable pruritus. It resembles picker's nodules of the scalp except that the few to 20 or more nodules are randomly distributed on the extensor aspects of the arms and legs (Figures 3-49 and 3-50). They are created by repeated scratching. The nodules are red or brown, hard, and dome-shaped with a smooth, crusted, or warty surface; they measure 1 to 2 cm in diameter. Hypertrophy of cutaneous papillary dermal nerves is a relatively constant feature. Complaints of pruritus vary. Some patients claim there is no itching and that scratching is only habitual, whereas others complain that the pruritus is intense.

TREATMENT.

Prurigo nodularis is resistant to treatment and lasts for years. As with picker's nodules of the scalp, repeated intralesional steroid injections may be effective. Excision of individual nodules is sometimes helpful. Cryotherapy is sometimes successful. Capsaicin 0.025% (Zostrix cream) and capsaicin 0.075% - HP (Zostrix-HP cream)
interferes with the perception of pruritus and pain by depletion of neuropeptides

**Figure 3-49** Prurigo nodularis. Thick, hard nodules usually present on the extensor surfaces of the forearms and legs from chronic picking.

in small sensory cutaneous nerves. Application of the cream 4 to 6 times daily for up to 10 months resulted in cessation of burning and pruritus within 12 days. Lesions gradually healed. Pruritus returned within 2 months in some patients who stopped treatment.[39] Calcipotriol ointment applied twice a day to nodules was effective. The use of combination or sequential topical calcipotriol with topical steroids might maximize the benefits and decrease the potential adverse effects of both drugs.[40] Naltrexone (50?mg daily), an orally active opiate antagonist, was found to be effective therapy for pruritic symptoms in many diseases.[41]

**Neurotic excoriations**

Neurotic excoriations are patient-induced linear excoriations. Patients dig at their skin to relieve itching or to extract imaginary pieces of material that they feel is imbedded in or extruding from the skin. Itching and digging become compulsive rituals. Most patients are aware that they create the lesions. The most consistent psychiatric disorders reported are perfectionistic and compulsive traits; patients manifest repressed aggression and self-destructive behavior.

**Figure 3-50** Prurigo nodularis. Thick papules and linear excoriations are features of both prurigo nodularis and neurotic excoriations.

**Figure 3-51** Neurotic excoriations. The upper back is one of the most common sites attacked by chronic pickers. Several white, round scars are evidence of past activity.

**Figure 3-52** Neurotic excoriations. Lesions appear on any area of the trunk and extremities that is easily reached.

**CLINICAL APPEARANCE.**

Repetitive scratching and digging produces few to several hundred excoriations; all lesions are of similar size and shape. They tend to be grouped in areas that are easily reached, such as the arms, legs, and upper back (Figures 3-51 through 3-54). Recurrent picking at crusts delays healing. Groups of white scars surrounded by brown hyperpigmentation are typical; their presence alone can indicate past difficulty.
TREATMENT.

The use of group I topical steroids applied twice a day or group V topical steroids under plastic wrap occlusion combined with systemic antibiotics produces gratifying results. Frequent lubrication and infrequent washing and with only mild soaps should be encouraged once areas are healed. Patients should try to substitute the ritual of applying lubricants for the ritual of digging. An empathic, supportive approach has been reported to be significantly more effective than insight-oriented psychotherapy, which often exacerbates the symptoms.

Figure 3-53 Neurotic excoriations. Severe involvement of the upper back. Picking causes shallow erosions and small, round scars. Long, linear scars occur from deep gouging.

Figure 3-54 Neurotic excoriations. Deep scars occurred after long periods of aggressive picking.
Psychogenic Parasitosis

Patients with psychogenic parasitosis believe they are infested with parasites. They move from one physician to another looking for someone who will believe them. A variety of psychiatric disorders may be associated with this disorder, but suggesting psychiatric referral may offend the patient. A supportive, therapeutic relationship is essential.\cite{42}

THE DELUSION.

Patients report seeing and feeling parasites. Involvement of the ears, eyes, and nose is common. They present with the “matchbox” sign, in which small bits of excoriated skin, dried blood, debris or insect parts are brought in matchboxes or other containers as “proof” of infestation. Body fluids thought to contain parasites are brought in jars. Pest control officers may have been hired to rid the house of parasites.

THE SKIN.

Excoriations and ulcers and linear scars are common on easily reached areas of the forearms, legs, trunk, and face (Figure 3-55).

CLASSIFICATION.

Classify patients (see diagram on the facing page) with psychogenic parasitosis into four groups: anxiety/hypochondriasis, anxiety/hypochondriasis with depression, delusional parasitosis, and delusional parasitosis with depression.\cite{43} Patients suffering from anxiety/hypochondriasis may believe that they are infested by parasites but may also express doubt about their infestation, express fears of “going crazy,” and agree that parasites may not be present. Patients with anxiety/hypochondriasis and depression may agree to undergo a psychiatric evaluation. Patients who have a true delusion are convinced that they have a parasitic infestation that none of the doctors can find; these
patients may have an underlying major depression.

MANAGEMENT.

A majority of patients with short-term illness can be cured with suggestion; the remainder have a true delusion. Patients with symptoms for over 3 months are usually not cured by suggestion alone. Listen and show concern; examine the skin with magnification and prepare scrapings; rule out true infestation. Animal and bird mites and scabies may actually be present. Do not suggest that the diagnosis is obvious on the first visit. The second visit can be lengthy. Collect specimens brought in by the patient and set them aside for later evaluation. Conduct a thorough examination and listen for indicators of depression. After two or more visits patients may suggest that this may be “all in my head.” At this point, explain that some patients actually see and feel parasites that are not real. Explain that this is an illness that affects sane people. The clinician may sense that the patient doubts the existence of the infestation (i.e., the belief is shakable). The patient is then offered a benzodiazepine to help with anxiety while waiting for the next visit 2 weeks later; a psychiatric referral is suggested at that 2-week follow-up visit.

Patients whose belief is unshakable are considered to have a delusional disorder. Suggest psychiatric referral. If that is refused, offer a neuroleptic medication such as pimozide (Orap) or haloperidol (Haldol). Intramuscular Haldol is available. Explain that many other patients with similar symptoms have been helped with this treatment and that the medication can be taken as a “therapeutic trial.” Psychiatric referral may be more acceptable after a short course of medication.

Figure 3-55 Psychogenic parasitosis. Attempts to pick “bugs” out of the skin produce focal erosions on easily accessed areas such as the arms and legs.

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TREATMENT FOR PSYCHOGENIC PARASITOSIS
**Stasis Dermatitis and Venous Ulceration: Postphlebitic Syndromes**

**Stasis dermatitis**

**ETIOLOGY.**

Stasis dermatitis is an eczematous eruption that occurs on the lower legs in some patients with venous insufficiency. The dermatitis may be acute, subacute, or chronic and recurrent, and it may be accompanied by ulceration. Most patients with venous insufficiency do not develop dermatitis, which suggests that genetic or environmental factors may play a role. The reason for its occurrence is unknown. Some have speculated that it represents an allergic response to an epidermal protein antigen created through increased hydrostatic pressure, whereas others believe that the skin has been compromised and is more susceptible to irritation and trauma.

**ALLERGY TO TOPICAL AGENTS.**

Patients with stasis dermatitis have significantly more positive reactions when patch tested with components of previously used topical agents. Topical medications that contain potential sensitizers such as lanolin, benzocaine, parabens, and neomycin should be avoided by patients with stasis disease. Allergy to corticosteroids in topical medication is also possible.

*Figure 3-56* Stasis dermatitis in an early stage. Erythema and erosions produced by excoriations are shown.

**Types of eczematous inflammation**
Subacute inflammation

Subacute inflammation usually begins in the winter months when the legs become dry and scaly. Brown staining of the skin (hemosiderin) may have appeared slowly for months (Figure 3-56). The pigment is iron left after disintegration of red blood cells that leaked out of veins because of increased hydrostatic pressure. Scratching induces first subacute and then chronic eczematous inflammation (Figure 3-57). Attempts at self-treatment with drying lotions (calamine) or potential sensitizers (e.g., neomycin-containing topical medicines) exacerbate and prolong the inflammation.

Acute inflammation

A red, superficial, itchy plaque may suddenly appear on the lower leg. This acute process may be eczematous inflammation, cellulitis, or both. Weeping and crusts appear (Figure 3-57). A vesicular eruption (id reaction) on the palms, trunk, and/or extremities sometimes accompanies this acute inflammation. The inflammation responds to systemic antibiotics, wet compresses, and group III to V topical steroids. Wet compresses should be discontinued before excessive drying occurs. The id reaction resolves spontaneously as the primary site improves.

Figure 3-57 Stasis dermatitis (severe inflammation). A red, itchy plaque may suddenly develop acute inflammation and/or cellulitis. Weeping, crusts, and fissuring may be extensive.

Chronic inflammation

Recurrent attacks of inflammation eventually compromise the poorly vascularized area, and the disease becomes chronic and recurrent (Figures 3-58 and 3-59). The typical presentation is a cyanotic red plaque over the medial malleolus. Fibrosis following chronic inflammation leads to permanent skin thickening. The skin surface in these irreversibly changed areas may have a bumpy, cobblestone appearance that results from fibrosis and venous and lymph stasis. The skin remains thickened and diffusely dark brown (postinflammatory hyperpigmentation) during quiescent periods.

Figure 3-58 Stasis dermatitis. Severe, painful, exudative, weeping, infected eczema with moist crust. Oral antibiotics and cool compresses are initial treatment followed in a few days with group II to V topical steroid creams or ointments.

TREATMENT OF STASIS DERMATITIS

Topical steroids and wet dressings.
The early, dry, superficial stage is managed as subacute eczematous inflammation with group II to V topical steroid creams or ointments and lubricating creams or lotions. Oral antibiotics (usually those active against staphylococci, e.g., cephalaxin) hasten resolution if cellulitis is present. Moist exudative inflammation and moist ulcers respond to tepid wet compresses of Burrow's solution or just saline or water for 30 to 60 minutes several times a day. Wet dressings suppress inflammation while debriding the ulcer. Adherent crust may be carefully freed with blunt-tipped scissors. Group V topical steroids are applied to eczematous skin at the periphery of the ulcer. Patients must be warned that steroid creams placed on the ulcer stop the healing process. Elevation of the legs encourages healing.

Figure 3-59 Stasis dermatitis. Cycles of inflammation, ulceration, and healing produce scarring. The surrounding chronically inflamed skin is hyperpigmented from deposits of hemosiderin from extravasated erythrocytes.

### Venous leg ulcers

The three main types of lower-extremity ulcers are venous, arterial, and neuropathic. Most leg ulcers are venous; foot ulcers are more often caused by arterial insufficiency or neuropathy. Most venous ulcers are located over the medial malleolus and are often larger than other ulcers. Diabetes is a common underlying condition.

**Differential diagnosis of leg ulcers.**

Many diseases cause leg ulcers. Biopsy (basal cell carcinomas, squamous cell carcinomas) and culture (fungal, atypical mycobacterial) chronic ulcers that do not respond to conventional therapy. Vasculitis, pyoderma gangrenosum, rheumatoid arthritis, and systemic lupus erythematosus may be associated with lower-extremity ulcers.

**Pathophysiology of venous insufficiency.**

The leg has superficial, communicating, and deep veins. The superficial system contains the long (medial) and short (lateral) saphenous veins. Perforator veins connect the superficial veins to the deep venous system. Normally blood flows from the superficial to the deep system. Venous hypertension

<p>| Table 3-5 -- The Three Common Types of Leg Ulcers |
|-----------------|-----------------|-----------------|
| Venous          | Arterial        | Neuropathic     |</p>
<table>
<thead>
<tr>
<th>Ulcer location</th>
<th>Medial malleolus; trauma or infection may localize ulcers laterally or more proximally</th>
<th>Distal, over bony prominences; trauma may localize ulcers proximally</th>
<th>Pressure points on feet (e.g., junction of great toe and plantar surface, metatarsal head, heel)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcer appearance</td>
<td>Shallow, irregular borders; base may be initially fibrinous but later develops granulation tissue</td>
<td>Round or punched-out, well-demarcated border; fibrinous yellow base or true necrotic eschar; bone and tendon exposure may be seen</td>
<td>Callus surrounding the wound and undermined edges are characteristic; blister, hemorrhage, necrosis, and exposure of underlying structures are commonly seen</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Varicose veins, leg edema, atrophie blanche, dermatitis, lipodermatosclerosis, pigmentary changes, purpura</td>
<td>Loss of hair, shiny, atrophic skin, dystrophic toenails, cold feet, femoral bruit, absent or decreased pulses, prolonged capillary refill time</td>
<td>No sensation to monofilament; bone resorption, claw toes, flat foot, Charcot joints</td>
</tr>
<tr>
<td>Frequent symptoms</td>
<td>Pain, odor, and copious drainage from the wound; pruritus</td>
<td>Claudication, resting ischemic pain</td>
<td>Foot numbness, burning, paresthesia</td>
</tr>
<tr>
<td>Ankle-to-brachial blood pressure ratio (ankle/brachial index [ABI]) measured by Doppler ultrasonography</td>
<td>&gt;0.9</td>
<td>ABI &lt;0.7 suggests arterial disease; calcification of vessels gives falsely high Doppler readings</td>
<td>Normal, unless associated with arterial component</td>
</tr>
<tr>
<td>Risk factors</td>
<td>Deep venous thrombosis, significant leg injury, obesity</td>
<td>Diabetes, hypertension, cigarette smoking, hypercholesterolemia</td>
<td>Diabetes, leprosy, frostbite</td>
</tr>
<tr>
<td>Complications</td>
<td>Allergic contact dermatitis, cellulitis</td>
<td>Gangrene</td>
<td>Underlying osteomyelitis</td>
</tr>
</tbody>
</table>
Treatment pearl

| Compression therapy, leg elevation | Pentoxifylline, vascular surgery assessment if ABI <5 | Vigorous surgical debridement, pressure avoidance |


(“chronic venous insufficiency”) occurs if any of the valves dysfunction, a thrombosis blocks the deep system, or there is calf muscle pump failure. Increased pressure causes diffusion of substances, including fibrin, out of capillaries. Fibrotic tissue may predispose the tissue to ulceration.

ETIOLOGY AND LOCATION.

Venous insufficiency followed by edema is the fundamental change that predisposes to dermatitis and ulceration. Venous insufficiency occurs when venous return in the deep, perforating, or superficial veins is impaired by vein dilation and valve dysfunction. Deep vein thrombophlebitis, which may have been asymptomatic earlier, is the most frequent precursor of lower leg venous insufficiency. Blood pools in the deep venous system and causes deep venous hypertension and dilation of the perforators that connect the superficial and deep venous systems. Venous hypertension is then transmitted to the superficial venous system. The largest perforators are posterior and superior to the lateral and medial malleoli. These are the same areas where dermatitis and ulceration are most prevalent. Superficial varicosities alone are unlikely to produce venous insufficiency.

VENOUS ULCER—CLINICAL FEATURES.

Ulceration is almost inevitable once the skin has been thickened and circulation is compromised. Ulceration may occur spontaneously or after the slightest trauma (Figure 3-60). The ulcer may remain small or may enlarge rapidly without any further trauma. A dull, constant pain that improves with leg elevation is present. Pain from ischemic ulcers is more intense and does not improve with elevation.

Ulcers have a sharp or sloping border and are deep or superficial. Removal of crust and debris reveals a moist base with granulation tissue. The base and surrounding skin is often infected. Healing is slow, taking several weeks or months. After healing, it is not uncommon to see ulcers rapidly recur. The ulcers are replaced with ivory-white sclerotic scars. Despite the pain and the inconvenience of treatment, most patients tolerate this disease well and remain ambulatory.