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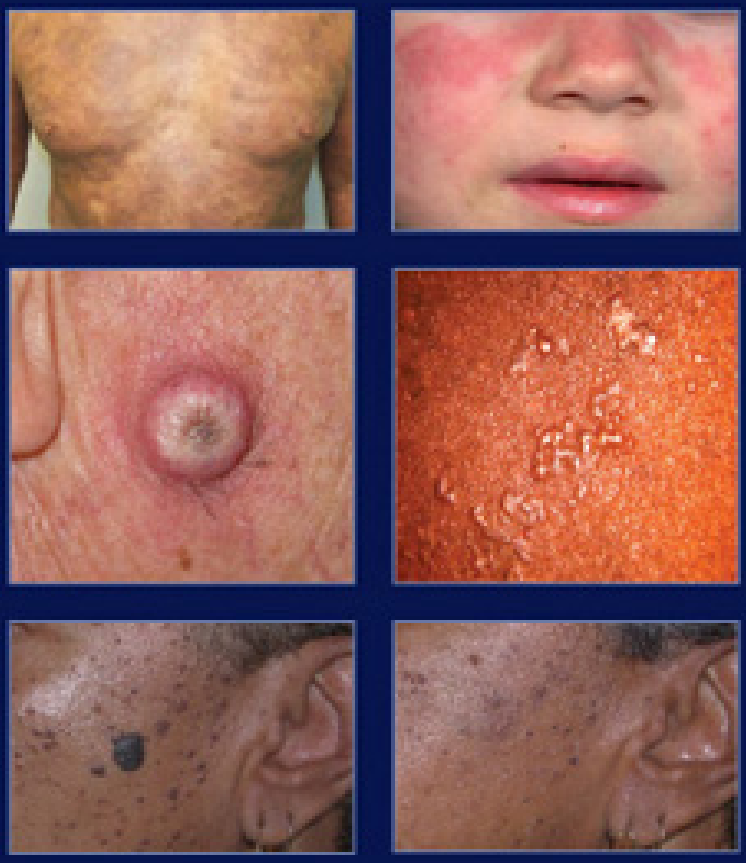
Sauer's Manual of Skin Diseases

TENTH EDITION



Brian J. Hall
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Wolters Kluwer Health | Lippincott Williams & Wilkins

Tenth Edition

SAUER'S MANUAL OF SKIN DISEASES

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Dedication

In memory of
Arnold and Fern Peterson,
Samantha Sue Hall,
and Gordon Sauer, MD whose book this will always be.

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Preface to the First Edition (Abridged)

Approximately 15% of all patients who walk into the general practitioner's office do so for the care of some skin disease or skin lesion. It may be for such a simple treatment as the removal of a wart, for the treatment of athlete's foot, or for something as complicated as severe cystic acne. There have been so many recent advances in the various fields of medicine that the medical school instructor can expect his or her students to learn and retain only a small percentage of the material that is taught. I believe that the courses in all phases of medicine, and particularly the courses of the various specialties, should be made as simple, basic, and concise as possible. If the student retains only a small percentage of what is presented, he or she will be able to handle an amazing number of walk-in patients. I am presenting in this book only the material that medical students and general practitioners must know for the diagnosis and the treatment of patients with common skin diseases. In condensing the material, many generalities are stated, and the reader must remember that there are exceptions to every rule. The inclusion of these exceptions would defeat the intended purpose of this book. More complicated diagnostic procedures or treatments for interesting problem cases are merely frosting on the cake. This information can be obtained by the interested student from any of several more comprehensive dermatologic texts.

This book consists of two distinct but complementary parts. The first part contains the chapters devoted to the diagnosis and the management of the important common skin diseases. In discussing the common skin diseases, a short introductory sentence is followed by a listing of the salient points of each disease in outline form. All diseases of the skin have primary lesions, secondary lesions, a rather specific distribution, a general course that includes the prognosis and the recurrence rate of the diseases, varying subjective complaints, and a known or unknown cause. Where indicated, a statement follows concerning seasonal incidence, age groups affected, family and sex incidence, contagiousness, relationship to employment, and laboratory findings. The discussion ends with a paragraph on differential diagnosis and treatment. Treatment, to be effective, has to be thought of as a chain of events. The therapy outlined on the first visit is usually different from the one given on subsequent visits or for cases that are very severe. The treatment is discussed with these variations in mind.

The second part consists of a very complete Dictionary–Index to the entire field of dermatology, defining the majority of rare diseases and the unusual dermatologic terms. The inclusion of this Dictionary–Index has a dual purpose. First, it enables me to present a concise first section on common skin diseases unencumbered by the inclusion of the rare diseases. Second, it provides rather complete coverage of all of dermatology for the more interested student. In reality, two books are contained in one.

Dermatologic nomenclature has always been a bugaboo for the new student. I heartily agree with many dermatologists that we should simplify the terminology, and that has been attempted in this text. Some of the changes are mine, but many have been suggested by others. However, after a diligent effort to simplify the names of skin diseases, one is left with the appalling fact that some of the complicated terms defy change. One of the main reasons for this is that all of our field, the skin, is visible to the naked eye. As a result, any minor alteration from normal has been scrutinized by countless physicians through the years and given countless names. The liver or heart counterpart of folliculitis ulerythematosa reticulata (ulerythema acneiform, atrophoderma reticulatum symmetricum faciei, atrophoderma vermiculatum) is yet to be discovered.

What I am presenting in this book is not specialty dermatology but general practice dermatology. Some of my medical educator friends say that only internal medicine, pediatrics, and obstetrics should be taught to medical students. They state that the specialized fields of medicine should be taught only at the internship, residency, or postgraduate level. That idea misses the very important fact that cases from all of the so-called specialty fields wander into the general practitioner's office. The general practitioner must have some basic knowledge of the varied aspects of all of medicine so that he or she can properly take care of his or her general everyday practice. This basic knowledge must be taught in the undergraduate years. The purpose of this book is to complement such teaching.

Gordon C. Sauer, MD

PREFACE

This is by far the most detailed edition of *Sauer's Manual of Skin Diseases.*

I think the most important change is the addition of new chapters that greatly enhance the completeness of the text. These new chapters are on sexually transmitted diseases, non-melanoma skin cancer, vascular tumors, cutaneous T-cell lymphoma, skin diseases in ethnic skin, obesity and dermatology, skin diseases in transplant patients, and nutrition and the skin.

All of the chapters have been updated, and new authors have been recruited. These new authors are all dermatology clinicians with expertise in their chapter topics which include immunology, atopic dermatitis, malignant melanoma, and general principles of skin aging.

Updated chapters written by outstanding authors from the previous edition include structures of the skin, laboratory procedures and tests, technologic applications in dermatology, fundamentals of cutaneous surgery, cosmetics for the physician, psoriasis, psychodermatology, virology, cutaneous diseases associated with immunodeficiency virus, diseases affecting the hair, diseases affecting the nail unit, collagen–vascular diseases, the skin and internal disease, dermatologic reactions to ultraviolet light and visible light, genodermatoses, pediatric dermatology, tropical diseases of the skin, sports dermatology, cutaneous signs of bioterrorism, and dermatoses of pregnancy.

Numerous color photographs have been added to this book of dermatology, the most visual of all medical specialties.

The chapters I have retained from the previous edition remain in the basic proven structure that Dr. Sauer has been recognized for worldwide during his outstanding dermatology teaching career.

It has been a pleasure and a learning experience working with my son, who has written a chapter and done the endless work of editing and acquiring new authors.

John C. Hall, MD

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I would like to thank Cindy Irey, a nurse par excellence, for helping with the editing. She somehow fit us into her busy schedule.

Marla Sussman was the best editor for any authors of a work of science. Her patience was endless.

The contributing chapter authors made this endeavor a true joy. They deserve kudos for the success we hope this book will achieve.

Charlotte is more than a wife. She is an inspiration, cheerleader, and friend.

Kim and Shelly, my daughters, and Tony and Tori, my grandchildren, give me my grounding and reasons for pursuit of scholarship.

My office staff kept my practice afloat while I was having fun writing. They are Christa Czysz, office manager; Kelly Howell, office administrator; Jennifer Phillips, receptionist; and Kelly Hedgens, nurse.

Thank you to Brent Johnson for his technical assistance with the photographs.

A big thank you goes to Dean Shepard, chief of photography services at St. Luke's Hospital in Kansas City, Missouri, whose photography has contributed greatly to this edition.

As brevity is the soul of wit, it is also the soul of understanding a complex subject. An overview is more priceless at the onset of learning than a mountain of detail. To stir one's interest and curiosity about a field of scientific endeavor, one needs to see that field as a whole. Therein lies the true genius of Gordon Sauer.

I frequently hear from dermatologists and nondermatologists alike that this book is their first exposure to the study of skin diseases. The tenth edition and all preceding editions are a tribute to Dr. Sauer's ability to open up the specialty of dermatology to those who wish to use its magic to help in the care of their patients.

CHAPTER 1

Structure of the Skin

Kenneth R. Watson, DO

The skin is the largest organ of the human body. It is composed of tissue that grows, differentiates, and renews itself constantly. Because the skin is a barrier between the internal organs and the external environment, it is uniquely subjected to noxious external agents and is also a sensitive reflection of internal disease. An understanding of the cause and effect of this complex interplay in the skin begins with knowledge of the basic structure of this organ.

Layers of the Skin

The skin is divided into three distinct layers. From the external surface inward, they are the epidermis, dermis, and subcutaneous tissue (Fig. 1-1). There are regional variations of these layers that probably represent adaptations to different functions, such as:

1. a thickened keratin layer of the epidermis on the palms and soles,
2. numerous nerve fibers within the fingertips for improved tactile function,
3. increased numbers of sebaceous glands on the face, and
4. thickened dermis on the back

Epidermis

The epidermis is the most superficial of the three layers of the skin and averages in thickness about the width of the mark of a sharp pencil (<1 mm). It contains several types of cells including keratinocytes, dendritic cells (melanocytes and Langerhans cells), and Merkel cells.

The keratinocytes, or keratin-forming cells, are by far the most common and develop into four identifiable layers of the epidermis (Fig. 1-2). From inside out, they are as follows:

Basal layer Spinous layer Granular layer	Living epidermis
Keratin layer	Dead end-product

The basal layer lies next to the dermis. This layer can be thought of as the stem cell layer of the epidermis, which is capable of progressive maturation into cell forms higher in the epidermis. It normally requires 3 or 4 weeks for the epidermis to replicate itself by the processes of division and differentiation. This cell turnover is greatly accelerated in diseases such as psoriasis in which the turnover rate may be as short as 2 to 3 days.

The spinous layer, or stratum malpighii, is made up of several layers of epidermal cells, which have a polyhedral shape. The cells of this layer are connected by intercellular bridges, which may be seen in routine sections.

The granular layer is composed of flatter cells containing protein granules called *keratohyalin granules*. In lichen planus, the granular cell layer is focally increased.

The outermost layer of the epidermis is the *keratin (cornified) layer*. It is made up of stratified layers of dead keratinized cells that are constantly shedding (Fig. 1-3). The protein in these cells is called *keratin* and is capable of absorbing vast amounts of water. This is readily seen during bathing, when the skin of the palms and the soles becomes white, swollen, and wrinkled. The keratin layer provides a major barrier of protection for the body. Mucous membranes, such as the oral and vaginal mucosa, do not have granular or keratin layers.

Immediately beneath the basal layer is the interface between the epidermis and the dermis known as the basement membrane zone or dermal–epidermal junction. It is difficult to visualize in routine hematoxylin and eosin stained sections but can clearly be seen as a thin band with periodic acid schiff (PAS) stains, due to the presence of mucopolysaccharides. Ultrastructurally, the basal cells are attached to the basement membrane by hemidesmosomes. Beneath the basal cells is an electron-clear layer known as the lamina lucida. Below this is a more electron-dense layer known as the lamina densa, which consists predominantly of type IV collagen. Anchoring filaments extend through the basement membrane zone to connect the surface membranes of the basal cells to the lamina densa. There are anchoring fibrils that attach the lamina densa to the papillary dermis.

Several blistering diseases occur due to defects in the basement membrane zone. Bullous pemphigoid antigens are present within the hemidesmosomes of the basement membrane zone. Circulating IgG antibodies bind to these antigens, resulting in the subepidermal blistering disease bullous pemphigoid. Epidermolysis bullosa represents a heterogeneous group of noninflammatory blistering disorders that can be divided into three subtypes based on the location of the blister. In epidermolysis bullosa simplex, the blister usually occurs through the basal cell layer. In the junctional

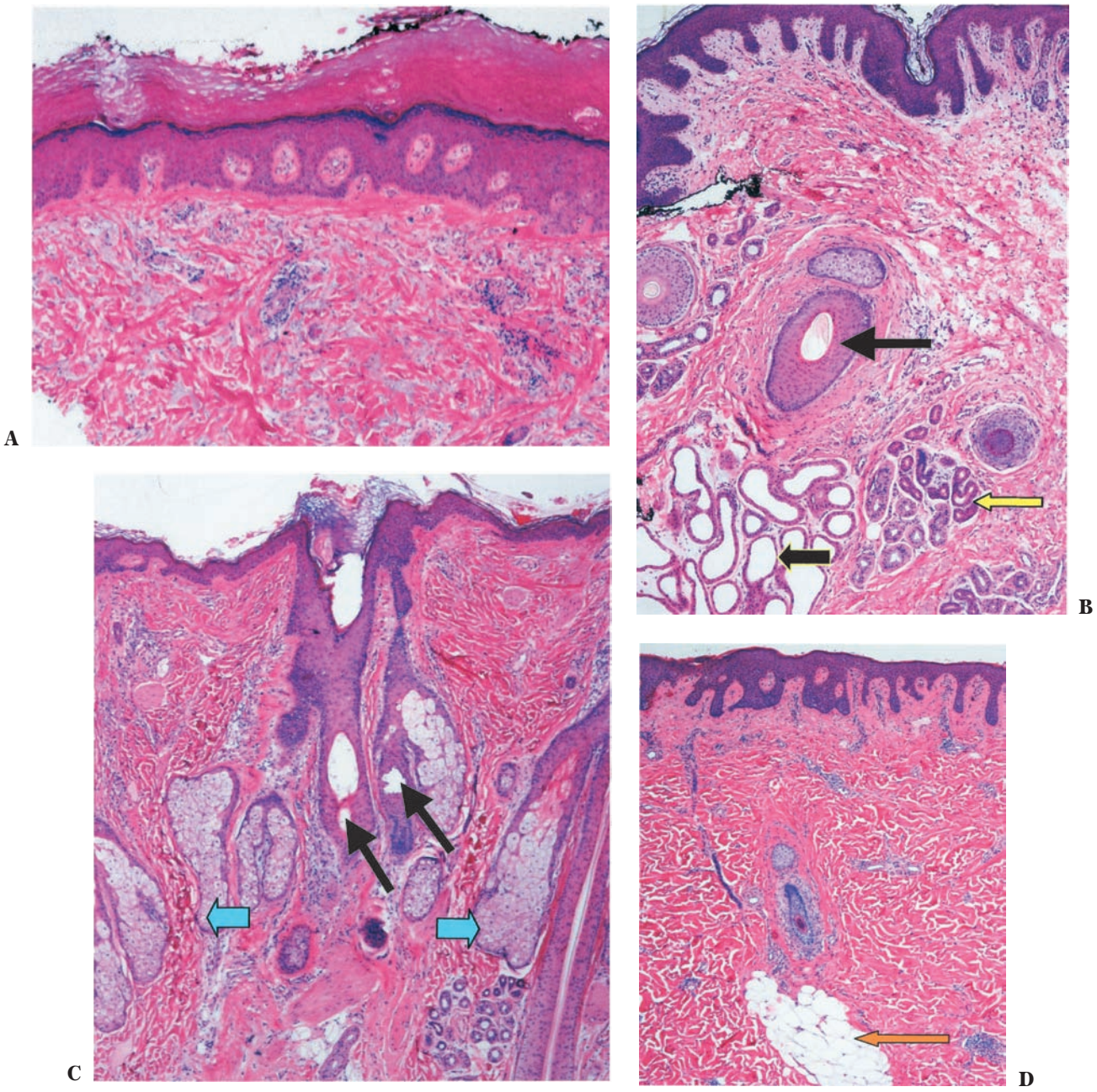

FIGURE 1-1 ■ Histology of the skin. Photomicrographs from four different areas of the body: palm **(A)**, axilla **(B)**, face **(C)**, and back **(D)**. Note the variations in the histologic features: thickened keratin layer from the palm (Fig. A), multiple (Fig. B) glandular elements from the axilla (hair follicle, *thin black arrow*; apocrine gland, *thick black arrow*; eccrine gland, *yellow arrow*), (Fig. C) numerous pilosebaceous units from the face (hair follicles, *thin arrows*; sebaceous glands, *thick arrows*), and (Fig. D) thick dermis from the trunk (subcutaneous fat, *arrow*). *(Courtesy of Dr. K. Watson.)*

form, it occurs between the basal cells and the lamina lucida, probably due to a defect in the hemidesmosomes. In the dermolytic form, the blister occurs beneath the lamina densa in the area of the anchoring fibrils.

The *melanin-forming cells*, or *melanocytes*, are sandwiched between the more numerous keratin-forming cells in the basal layer. In routine hematoxylin and eosin stained sections, melanocytes have small, dark nuclei and clear cytoplasm, which is the result of shrinkage artifact. Approximately 10% of the cells in the basal layer are melanocytes. However, this varies depending on the body site and ethnic background of the individual. These melanocytes are dopa-positive because they stain darkly after contact with a solution of levorotatory 3,4-dihydroxyphenylalanine, or *dopa*. This laboratory reaction closely simulates physiologic melanin formation, in which the amino acid tyrosine is oxidized by the enzyme tyrosinase to form dopa. Dopa is then further changed, through a series of complex metabolic processes, to melanin. In

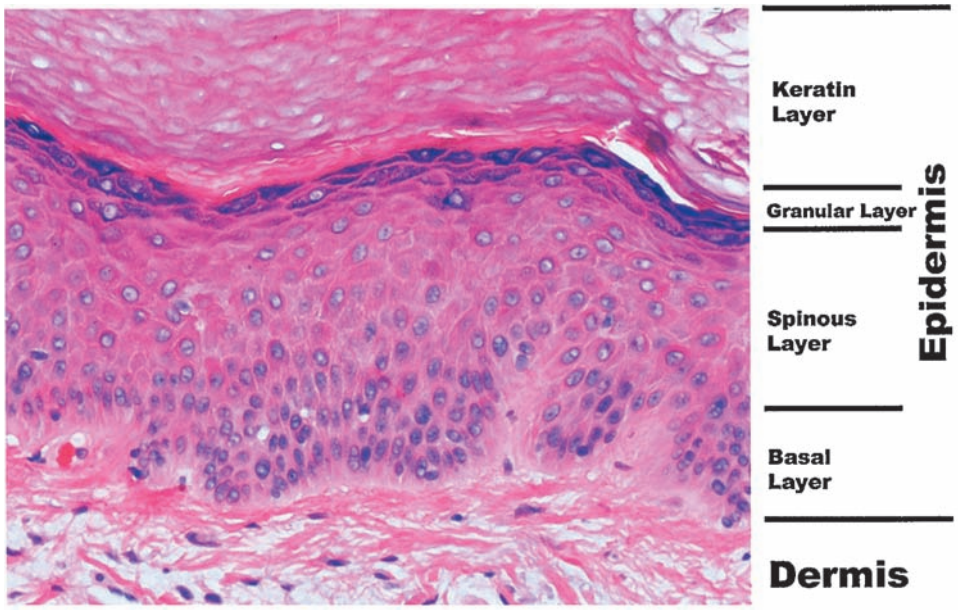

FIGURE 1-2 ■ Histology of the epidermis. A photomicrograph from the palm. *(Courtesy of Dr. K. Watson.)*

dermatopathology practices, melanocytes are most commonly recognized by showing positive immunoreactivity for S-100 protein, HMB-45, and Melan-A (MART-1), which may be useful in the diagnosis of melanocytic tumors such as malignant melanoma. Melanocytes may also be recognized using silver stains due to the fact that melanin is both argyrophilic and argentaffin. For example, the Fontana–Masson histochemical stain results in black cytoplasmic granules within melanocytes because of the ability of melanin to reduce ammoniated silver nitrate. Melanin may also be bleached, which is useful in identifying the neoplastic melanocytes that are obscured in heavily pigmented tumors.

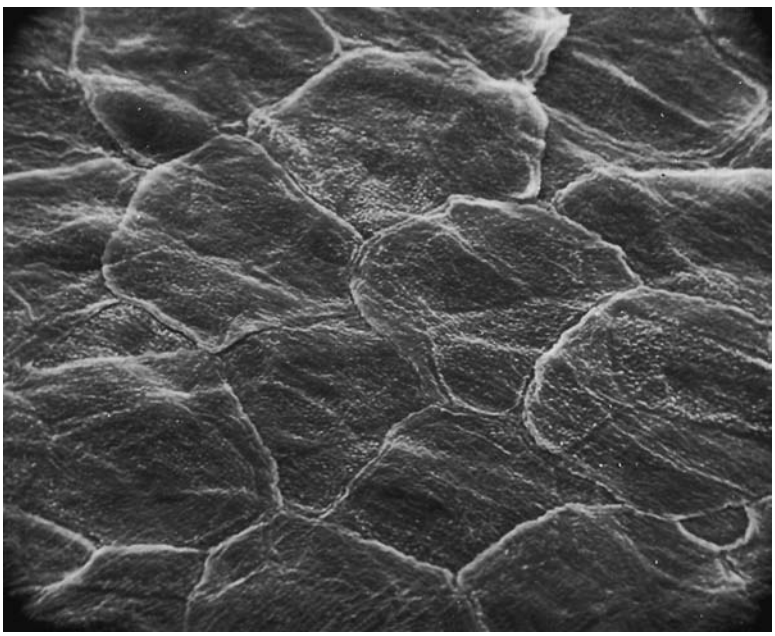

FIGURE 1-3 ■ Keratin-layer cells. Underside of the top layer of epidermal keratin-layer cells on Scotch tape stripping is seen with Cambridge Mark II Stereoscan at 1000X. *(Courtesy of Drs. J. Arnold, W. Barnes, and G. Sauer.)*

Melanin pigmentation of the skin, whether increased or decreased, is influenced by many local and systemic factors (see Chapter 30). Melanocyte-stimulating hormone from the pituitary is the most potent melanizing agent. Melanin is transferred from melanocytes to basal keratinocytes. Skin color is largely related to the amount of melanin present in basal cells. Exposure to ultraviolet light results in increased melanocyte concentration and function.

Langerhans cells are found scattered evenly throughout the epidermis. They are bone marrow–derived mononuclear cells. They are involved in cell-mediated hypersensitivity, antigen processing and recognition, stimulation of immune-competent cells, and graft rejection. Sunlight suppresses their immune function. Their number is decreased in certain skin diseases, such as psoriasis. Staining with membrane adenosine triphosphatase and monoclonal antibodies such as S-100 protein and CD-1 a can be done for identification. Electron microscopy reveals that these cells contain characteristic racquet-shaped Birbeck granules. These cells proliferate in the disease Langerhans cell histiocytosis (formerly known as histiocytosis X), which may be isolated to the skin or may be part of a larger systemic process.

Merkel cells are located within the basal layer but may also be found within hair follicles and sweat ducts. They are assumed to function as touch receptors and are associated with fine unmyelinated nerve fibers. They are inconspicuous in routine sections. They may be recognized using immunostains for the neuroendocrine markers neuron-specific enolase, chromogranin, and synaptophysin. Ultrastructurally, they contain dense core neurosecretory granules. They give rise to primary neuroendocrine carcinoma of the skin (Merkel cell carcinoma).

Dermis

The dermis consists of connective tissue, cellular elements, and ground substance. It has a rich vascular and nerve supply and contains pilosebaceous, apocrine, and eccrine structures. Anatomically, it is divided into two compartments. The first contains thin collagen fibers, delicate elastic fibers, numerous capillaries, and abundant ground substance, which form a thin layer beneath the epidermis (papillary dermis) and surrounding adnexal structures (periadnexal dermis). Together, these are regarded as a single unit called the *adventitial dermis.* This is an important unit because it is altered together with the adjacent epithelium in many inflammatory diseases. The second compartment, known as the *reticular* or *deep dermis,* is composed of thick collagen bundles with intertwined elastic fibers. The reticular dermis is thick and comprises the bulk of the dermis. It contains less ground substance, vascular spaces, and cellular elements than the thin adventitial dermis.

The *connective tissue* component of the dermis consists of collagen fibers, including reticulin fibers, and elastic fibers. These fibers contribute to the support and elasticity of the skin.

Two different types of collagen are present within the dermis. Type I collagen is predominantly found within the thick fibers of the reticular dermis. Type III collagen, also known as *reticulin,* is largely found within the thin fibers of the papillary and periadnexal dermis. These reticulin fibers are not visible in routine hematoxylin and eosin stained sections but can be identified with silver stains. They are abundant in certain pathologic conditions such as granulomas, syphilis, sarcoidosis, and some mesenchymal tumors. The proteins present in collagen fibers are responsible for nearly one-fourth of a person's overall protein mass. If tannic acid or the salts of heavy metals, such as dichromates, are combined with collagen, the result is leather.

Elastic fibers are thinner than most collagen fibers and are entwined among them. They are composed of the protein elastin. Elastic fibers do not readily take up acidic or basic stains, such as hematoxylin and eosin, but they can be identified with the Verhoeff–van Gieson stain.

Cellular elements of the dermis include fibroblasts, endothelial cells, mast cells, and a variety of miscellaneous cells, including smooth muscle, nerve, and hematopoietic cells. The hematopoietic cells include lymphocytes, histiocytes (macrophages), eosinophils, neutrophils, and plasma cells. These hematopoietic cells are increased in numerous inflammatory diseases of the skin.

Fibroblasts form collagen and produce ground substance. They are involved in immunologic and reparative processes. Fibroblasts are increased in numerous different skin disorders.

Mast cells are derived from bone marrow stem cells. They are present in normal skin in small numbers and are usually concentrated around blood vessels, particularly postcapillary venules. They have intracytoplasmic basophilic metachromatic granules containing heparin and histamine. The granules do not stain with routine hematoxylin and eosin but may be seen with colloidal iron, toluidine blue, and Alcian blue stains. Mast cells are increased in many different inflammatory dermatoses but play a particularly important role in urticarial eruptions. Urticaria occurs when mast cells and basophils are degranulated, resulting in vascular permeability and tissue edema. Mast cell degranulation also plays a role in activating other inflammatory cells to the area of tissue injury.

Neoplastic proliferations of mast cells may form papules, plaques, and nodules within the skin, known as cutaneous mastocytosis (*urticaria pigmentosa).* They may also have a telangiectatic appearance, as in telangiectasia macularis eruptiva perstans (TMEP). In addition to metachromatic staining mentioned in the previous paragraph, these proliferations of mast cells show positive immunoreactivity for human mast cell tryptase and CD117, which may be useful in differentiating mast cell proliferations from other cutaneous neoplasms, such as Langerhans cell histiocytosis and leukemia cutis.

Histiocytes (macrophages) are present in only small numbers in the normal skin. However, in pathologic conditions, they migrate to the dermis as tissue monocytes. They play a predominant role in the phagocytosis of particulate matter and bacteria. Under special pathologic conditions,

they may form giant cells. They are also involved in the immune system by phagocytizing antigens.

Lymphocytes and plasma cells are found in only small numbers in normal skin, but are significantly increased in pathologic conditions, such as increased plasma cells in syphilis.

The *ground substance* of the dermis is a gel-like amorphous matrix not easily seen in routine sections, but it may be identified with colloidal iron and Alcian blue stains. It is found in greatest concentration within the adventitial dermis, particularly around adnexal structures. There are variable amounts of ground substance in different areas of the body, with increased concentrations within the fingers and toes. The ground substance contains proteins, mucopolysaccharides, soluble collagens, enzymes, immune bodies, and metabolites. It has the capacity to bind water, allowing the movement of nutrients through the dermis, and it provides bulk, contributing to the malleability of the skin.

Subcutaneous Tissue

The subcutaneous tissue constitutes the largest volume of adipose tissue in the body. The adipose tissue is organized into lobules by fibrous septa, which contain most of the blood vessels, nerves, and lymphatics. The thickness of the subcutaneous fat varies from one area of the body to another. It is especially thick in the abdominal region and thin in the eyelids and scrotum. The subcutaneous tissue serves as a receptacle for the formation and storage of fat as well as a site of highly dynamic lipid metabolism for nutrition. It also provides protection from physical trauma and insulation to temperature changes.

Most of the fat in the body consists of white adipose tissue (Fig. 1-4). The white fat cells are derived from mesenchymal cells, as are fibroblasts. They store triglycerides, which can be broken down into fatty acids and used for energy by other tissues such as muscle. White adipose tissue is increased in obesity.

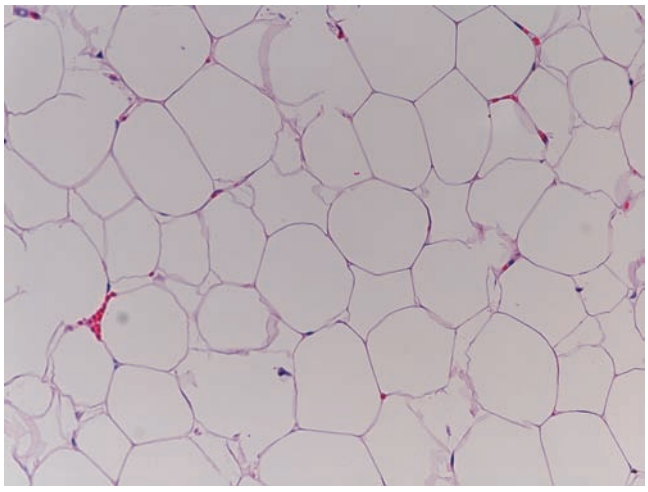

FIGURE 1-4 ■ White adipose tissue. *(Courtesy of Dr. K. Watson.)*

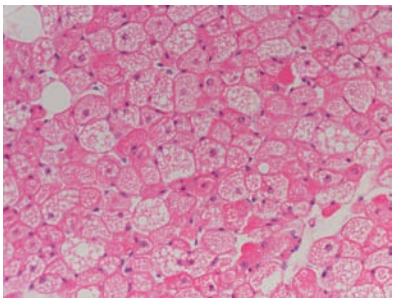

FIGURE 1-5 ■ Brown adipose tissue. *(Courtesy of Dr. K. Watson.)*

There is a second distinct type of adipose tissue, known as brown fat, which is found predominantly in human newborns and also hibernating animals (Fig. 1-5). Brown fat cells have a different appearance than white fat cells. They are smaller, contain multiple small lipid droplets, and have increased numbers of mitochondria. Recent studies using fluorodeoxyglucose positron emission tomography (PET) suggest that a significant percentage of adult humans have active brown adipose tissue. Brown fat has a different function than white fat. It is involved in energy expenditure that is responsible for generating heat, protecting body temperature in human newborns without shivering. It may also be useful in protecting against obesity. Brown fat may develop from a common precursor to skeletal myocytes.

Lipomas are benign tumors composed of mature fat cells identical to white adipose tissue in the subcutaneous fat. Hibernoma is a benign tumor composed of fat cells resembling brown fat.

Vasculature

The skin contains a rich vascular network that provides blood volume far exceeding its nutritional needs. In fact, the vascularization is so extensive that it has been postulated that its main function is to regulate heat and blood pressure of the body, with providing nutrition to the skin a secondary function. The vascular plexus arises from thick arteries within the subcutaneous fat. There are two major plexuses, which run parallel to the epidermis, one within the deep dermis near the dermal–subcutaneous junction and one within the superficial (papillary) dermis. There are vertically oriented perforating branches that connect the two plexuses and provide blood to surrounding dermal appendages. Perivascular inflammation surrounding the superficial and deep plexuses occurs in many types of "dermatitis," and this pattern of inflammation may be used as a method of classification of inflammatory disorders of the skin. Inflammatory reactions involving the superficial vascular plexus may result in erythema.

The vascular plexuses consist of a mixture of arterioles, venules, and capillaries. Most of the exchange of water, oxygen,

and nutrients with the skin occurs through thin capillaries and venules. The skin also contains an extensive lymphatic network that is independent of the vascular plexus. No blood vessels or lymphatics are present within the epidermis.

A special vascular body, the glomus, deserves mention. The glomus body is most commonly seen on the tips of the fingers and toes and under the nails. Each glomus body consists of a venous and arterial segment, called the *Sucquet–Hoyer canal.* This canal represents a short-circuit device that connects an arteriole with a venule directly, without intervening capillaries. The result is a marked increase in the blood flow through the skin. If this body grows abnormally, it forms an often painful, red, benign glomus tumor, commonly beneath the nail.

Nerve Supply

The skin is a major sensory organ with millions of nerve endings receiving stimulation from the surrounding environment. Sensory and autonomic nerves within the peripheral nervous system permeate the dermis with tiny nerve fibers, which may be myelinated or unmyelinated. These tiny nerve fibers are not visible in routine hematoxylin and eosin stained sections. Only larger myelinated nerve fibers and specialized nerve-end organs are discernable. Special stains are required to visualize the small nerve-fibers, such as silver impregnation techniques (Bielschowsky or Bodian stains) or immunoperoxidase stains such as neurofilament protein, which stains axons, and S-100 protein, which stains Schwann cells. The nerve fibers are variably distributed, resulting in regional variations in sensation. They are very prominent on the palms, soles, and fingers, and within mucocutaneous areas.

Numerous tiny unmyelinated sensory nerves with free nerve endings are present within the papillary dermis and surrounding hair follicles. They mediate the sensations of temperature, touch, pain, and itching. Some of the free nerve endings extend into the basal epidermis and contact Merkel cells.

SAUER'S NOTES

Itching is the most important presenting symptom of an unhappy patient. It may be defined simply as the desire to scratch. Itching apparently is a mildly painful sensation that differs from pain in having a lower frequency of impulse stimuli. The release of proteinases (such as follows itch-powder application) may be responsible for the itch sensation. The pruritus may be of a pricking or burning type and can vary greatly from one person to another. Sulzberger called abnormally sensitive people *itchish,* analogous to *ticklish.* Itching can occur without any clinical signs of skin disease or from circulating allergens or local superficial contactants. The skin of atopic or eczema patients tends to be more itchy. Scratching makes the itching worse. This results in a perpetual itch–scratch cycle.

Sensory nerves in hairless skin, such as the palms and soles, and at the mucocutaneous junction terminate in specialized end organs, known as Meissner corpuscles, Vater–Pacini corpuscles, and mucocutaneous end organs. Meissner corpuscles are most numerous on the fingertips, palms, and soles, where they sense touch and vibration. They are composed of S-100–positive laminated, flattened Schwann cells. Vater–Pacini corpuscles are most numerous within the deep dermis and subcutaneous fat of the feet and hands, and they sense pressure and tension. They are large, measuring up to 1 mm in diameter, and are composed of outer spherical layers of perineurial cells and an inner nerve fiber with accompanying Schwann cell.

Sympathetic autonomic nerve fibers supply blood vessels, arrector pili muscles, apocrine glands, and eccrine glands. Adrenergic fibers carry impulses to the arrector pili muscles, which produce gooseflesh if they are stimulated. This is caused by traction of the muscle on the hair follicles to which it is attached. Cholinergic fibers, if stimulated, increase sweating and may cause a specific type of hives called *cholinergic urticaria* (see Chapter 11). Sebaceous glands do not contain autonomic fibers but are controlled by endocrine stimulation.

Appendages

The appendages of the skin include both the cornified appendages (hairs and nails) and the glandular appendages.

Hair Follicles

Hairs are produced by the hair follicles, which develop from germinative cells of the fetal epidermis. Because no new hair follicles are formed after birth, the different types of body hairs are manifestations of the effect of location as well as external and internal stimuli. Hormones are the most important internal stimuli influencing the various types of hair growth. There are three main types of hairs: (1) *Lanugo hairs:* fine, lightly pigmented hairs covering the body of the fetus, (2) *Vellus hairs* ("peach fuzz"): short, fine hairs that replace lanugo hairs and cover most of the body but are barely noticeable, and (3) *Terminal hairs:* long, coarse hairs present in the adult, which are prominent on the scalp, beard, pubic, and axillary regions. Terminal hairs convert into vellus hairs in male pattern baldness. Vellus hairs develop into terminal hairs in hirsutism. The palms, soles, lips, and some genital areas do not contain hair follicles.

Hair growth is cyclic, with a growing (anagen) phase (**Fig. 1-6**) and a resting (telogen) phase. The *catagen cycle* is the transition phase between the growing and resting stages and lasts only a few weeks. The duration of hair growth varies in different areas of the body. Approximately 90% of normal scalp hairs are in the growing (anagen) stage, which can last between 3 to 6 years or more, depending on the location. Ten percent of hairs are in the resting (telogen) stage, which lasts approximately 60 to 90 days. However, systemic stresses, such as childbirth, or systemic anesthesia may cause

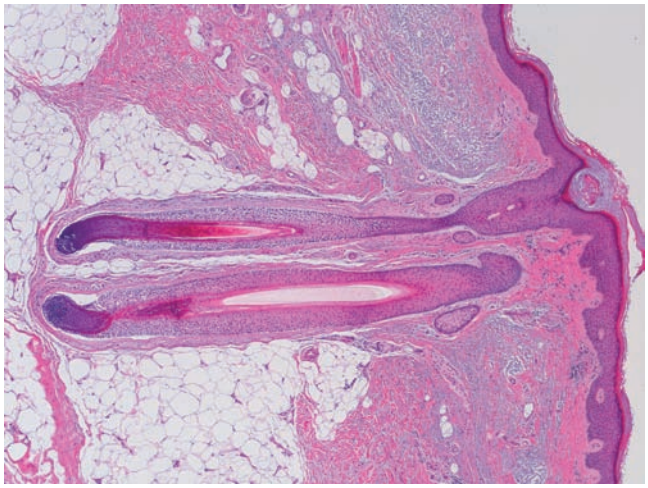

FIGURE 1-6 ■ Anagen hairs with hair bulb and matrix cells. *(Courtesy of Dr. K. Watson.)*

hairs to enter a resting stage prematurely. This *postpartum* or *postanesthetic effect* is noticed most commonly in the scalp when these resting hairs are depilated during combing or washing, and the thought of approaching baldness causes sudden alarm.

Hair follicles may be thought of as an invagination of the epidermis, with its different layers of cells. The hair follicle can be divided into three areas: (1) *infundibulum,* which extends from the follicular orifice to the entrance of the underlying sebaceous gland, (2) *isthmus,* which extends from the orifice of the sebaceous gland to the insertion of the erector pili muscle, and (3) *inferior segment,* which consists of the follicle below the insertion of the erector pili muscle.

The inferior portion of the follicle includes the hair bulb, which contains matrix cells. These cells perform a similar function to the basal cells of the epidermis. They are responsible for the development of the hair shaft. Melanocytes are present in the matrix and determine the color of hair.

There are approximately 100,000 anagen follicles on the normal scalp with tremendous protein-synthesizing capacity. At the rate of scalp hair growth of 0.35 mm/d, more than 100 linear feet of scalp hair is produced daily. The density of hairs in the scalp varies from 175 to 300 hairs per square centimeter. Up to 100 hairs may be normally lost daily.

Nail Unit

The nail unit consists of a nail plate and the surrounding soft tissues, which include the nail matrix, proximal and lateral nail folds, nail bed, cuticle, and hyponychium (Fig. 29-1). The nail plate covers the dorsal distal aspect of the fingers and toes and ranges between 0.3 and 0.75 mm in thickness. It inserts into grooves in the skin that are present proximally and laterally. The plate is produced by the nail matrix, which is located at the proximal end of the plate insertion, ventral to the proximal nail fold. The matrix extends distally to the lunula, which is a crescent-shaped white area under the proximal nail plate, particularly prominent on the thumb and less prominent on the remaining fingers. The nail plate lies on the nail bed. The epithelium of the nail bed produces a small amount of keratin, which tightly adheres to the overlying nail plate. The dermis of the nail bed is richly vascular, resulting in a pink appearance and blanching when compressed. Glomus bodies are also present, which aid in temperature control of the digits. The cuticle represents the cornified layer of the proximal nail fold and serves to seal off and protect the nail matrix. The hyponychium consists of cornified epidermis located at the distal end of the nail bed beneath the distal free edge of the nail plate.

The nail unit is an invagination of the epidermis, similar to the hair follicle. Both have a matrix that produces the protein keratin. The nail plate consists almost entirely of keratin, similar to the hair shaft and cornified layer of the epidermis. Unlike hair growth, which is periodic, nail growth is continuous. Nail growth proceeds at about one-third of the rate of hair growth, or about 0.1 mm/d. It takes about 3 months to restore a removed fingernail and about three times that long for the regrowth of a new toenail. Nail growth can be inhibited during serious illnesses or in old age, increased through nail biting or occupational trauma, and altered due to a variety of diseases and medications.

Glandular Appendages

The three types of glandular appendages of the skin are the sebaceous glands, apocrine glands, and eccrine glands (Fig. 1-7).

The *sebaceous glands* are present everywhere on the skin, except the palms and the soles. In most areas they are associated with hair follicles. There are sebaceous glands that are

SAUER'S NOTES

1. Shaving of excess hair, as women do on their legs and thighs, does not promote more rapid growth of coarse hair. The shaved stubs appear coarser, but if allowed to grow normally, the hairs appear and feel no different than before.
2. The value of intermittent massage to stimulate scalp hair growth has not been proved.
3. Hair cannot turn gray overnight. The melanin pigmentation, which is distributed throughout the length of the nonvital hair shaft, takes weeks to be shed through the slow process of hair growth.
4. Heredity is the greatest factor predisposing to baldness, and an excess of male hormone may contribute to hair loss. Male castrates do not become bald.
5. Common male pattern baldness cannot be reversed by over-the-counter "hair restorers." Minoxidil solution (Rogaine), which is sold over the counter, is beneficial for a limited percentage of patients, and finasteride (Propecia) pills, available by prescription, are helpful for most patients.

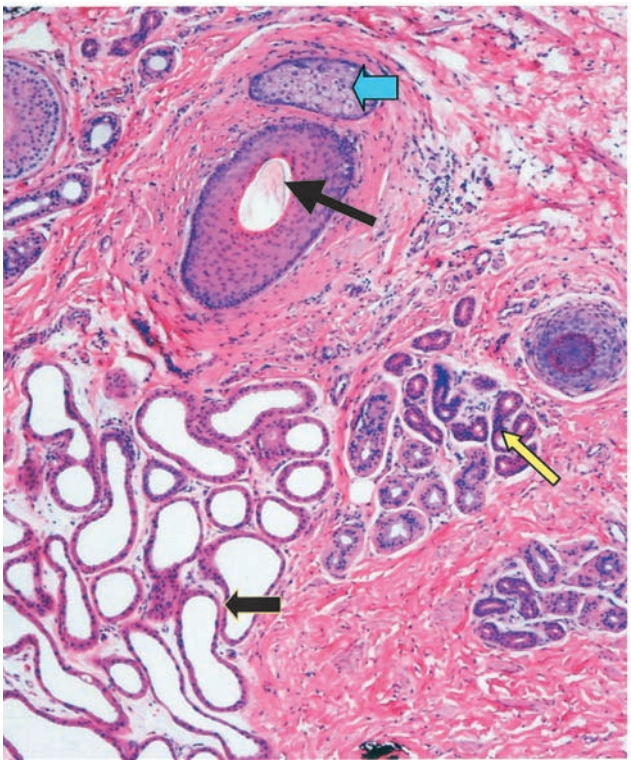

FIGURE 1-7 ■ Histology of the glands of the skin. A photomicrograph from the axilla (hair follicle, *long black arrow*; apocrine glands, *short black arrow*; sebaceous gland, *short blue arrow*; eccrine glands, *yellow arrow*). *(Courtesy of Dr. K. Watson.)*

not associated with hair follicles, such as the buccal mucosa and vermillion border of the lip, nipple and areola of the breast, labia minora, and eyelids (meibomian glands). The sebaceous glands are holocrine glands, forming their secretions through the disintegration of the entire glandular cell. The secretion from these glands is evacuated through the sebaceous duct to a follicle that may contain either a large terminal hair or a vellus hair. This secretion, known as *sebum*, is not under any neurologic control but is a continuous outflow of the material of cell breakdown. The sebum covers the skin with a thin lipoidal film that is mildly bacteriostatic and fungistatic and retards water evaporation. The scalp and the face may contain as many as 1,000 sebaceous glands per square centimeter. The activity of the gland increases markedly at the age of puberty, and, in certain people, it becomes plugged with sebum, debris, and bacteria to form the blackheads and pimples of acne.

Apocrine glands are found in the axillae, genital region, breast, external ear canal (ceruminous glands), and eyelid (Moll's glands). They do not develop until the time of puberty. They consist of a coiled secretory gland located in the deep dermis or subcutaneous fat and a straight duct that usually empties into a hair follicle. The function of the secretions is unknown; however, they may act as pheromones. They are responsible for the production of body odor (the infamous "BO"). Any emotional stresses that cause adrenergic sympathetic discharge produce apocrine secretion. This secretion is sterile when excreted but undergoes decomposition when contaminated by bacteria from the skin surface, resulting in a strong and characteristic odor. The purpose of the many cosmetic underarm preparations is to remove these bacteria or block the gland's excretion. The apocrine glands are involved in *hidradenitis suppurativa,* an inflammatory process that results from follicular obstruction and retention of follicular products, which usually occurs in patients with the acne–seborrhea complex.

Eccrine sweat glands are distributed everywhere on the skin surface, with the greatest concentration on the palms, soles, and forehead. They develop as a downgrowth from the primitive epidermis. They are composed of coiled secretory glands, a coiled duct, a straight duct, an intraepidermal coil, and an eccrine pore. The eccrine sweat glands and the vasculature of the skin serve in the maintenance of stable internal body temperature, despite marked environmental temperature changes. They flood the skin surface with water for cooling, and the blood vessels dilate or constrict to dissipate or conserve body heat. Their prime stimulus is heat, and their activity is under the control of the nervous system, usually through the hypothalamus. Both adrenergic and cholinergic fibers innervate the glands. Blockage of the eccrine ducts results in the disease known as *miliaria* (*prickly heat*). If eccrine glands are congenitally absent, as in *anhidrotic ectodermal dysplasia,* a life-threatening hyperpyrexia may develop.

Acknowledgment

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CHAPTER 2

Laboratory Procedures and Tests

Christopher J. Kligora, MD and Kenneth R. Watson, DO

In addition to the usual laboratory procedures used in the workup of medical patients, certain special tests are of importance in the field of dermatology. These include skin tests, fungus examinations, biopsies, and immunologic diagnosis. For special problems, additional testing methods are suggested in the sections on the various diseases.

Skin Tests

There are three types of skin tests:

- Intracutaneous
- Scratch
- Patch

The intracutaneous tests and the scratch tests can have two types of reactions: either an immediate wheal reaction or a delayed reaction. The immediate wheal reaction develops to a maximum in 5 to 20 minutes and is elicited in testing for the cause of urticaria, atopic dermatitis, and inhalant allergies. This is a type I or anaphylactoid type of immunity. The immediate wheal reaction test is seldom used for determining the cause of skin diseases.

The delayed reaction to intracutaneous skin testing is exemplified best by the tuberculin skin test. Tuberculin is available in two forms—as the purified protein derivative test and as a tuberculin tine test. The purified protein derivative test is performed by using tablets that come in two strengths and by injecting a solution of either one intracutaneously. If there is no reaction after the test with the first strength, then the second strength may be employed.

The tuberculin tine test (Mantoux) is a simple and rapid procedure using OTK. Nine prongs, or tines, covered with OTK are pressed into the skin. If at the end of 48 or 72 hours there is more than 2 mm of induration at the site of any prong insertion, the test is positive.

Patch tests are used commonly in dermatology and offer a simple and accurate method of determining whether a patient is allergic to any of the testing agents. There are two different reactions to this type of test: a primary irritant reaction and an allergic reaction. The primary irritant reaction occurs in most of the population if they are exposed to agents (in appropriate concentrations) that have skin-destroying properties. Examples of these agents include soaps, cleaning fluids, bleaches, "corn" removers, and counterirritants. The allergic reaction indicates that the patient is more sensitive than normal to the agent being tested. This test reaction is idiosyncratic and not necessarily related to concentration or dose. It also shows that the patient has had a previous exposure to that agent or a cross-sensitizing agent. This is a type IV or delayed type of immunity. It is often very helpful in cases of contact dermatitis.

The technique of the patch test is simple, but the interpretation of the test is not. For example, consider a patient presenting with dermatitis on top of the feet. It is possible that shoe leather or some chemical used in the manufacture of the leather is causing the reaction. The procedure for a patch test is to cut out a half-inch square piece of the material from the inside of the shoe, moisten the material with distilled water, place it on the skin surface, and cover it with an adhesive band or some patch-test dressing. The patch test is left on for 48 hours. When the patch test is removed, the patient is considered to have a positive patch test if there is any redness, papules, or vesiculation under the site of the testing agent. Delayed reactions to allergens can occur, and, ideally, a final reading should be made after 96 hours (4 days), that is, 2 days after the patch is removed.

The patch test can be used to make or confirm a diagnosis of poison ivy dermatitis, ragweed dermatitis, or contact dermatitis caused by medications, cosmetics, or industrial chemicals. Fisher (1995) and Adams (1990) compiled lists of chemicals, concentrations, and vehicles to be used for eliciting the allergic type of patch test reaction. Most tests can be performed very simply, however, as in the case of the shoe leather dermatitis. One precaution is that the patch must not be allowed to become wet in the 48-hour period. A patch test kit, T.R.U.E. Test (Glaxo), includes ready-to-apply self-adhesive allergen tapes. There are other more extensive patch test trays available.

A method of testing for food allergy is to use the Rowe elimination diet. The procedure is to limit the diet to the following basic foods, which are known to be hypoallergenic: lamb, lemon, grapefruit, pears, lettuce, spinach, carrots, sweet potatoes, tapioca, rice and rice bread, corn sugar, maple syrup, sesame oil, gelatin, and salt. The patient is to remain on this basic diet for 5 to 7 days. At the end of that time, one new food can be added every 2 days. The following foods can be added early: beef, white potatoes, green beans, milk (along with butter and American cheese), and white bread with puffed wheat. If there is a flare-up of the dermatitis, which should occur within 2 to 8 hours after ingestion of an offending food,

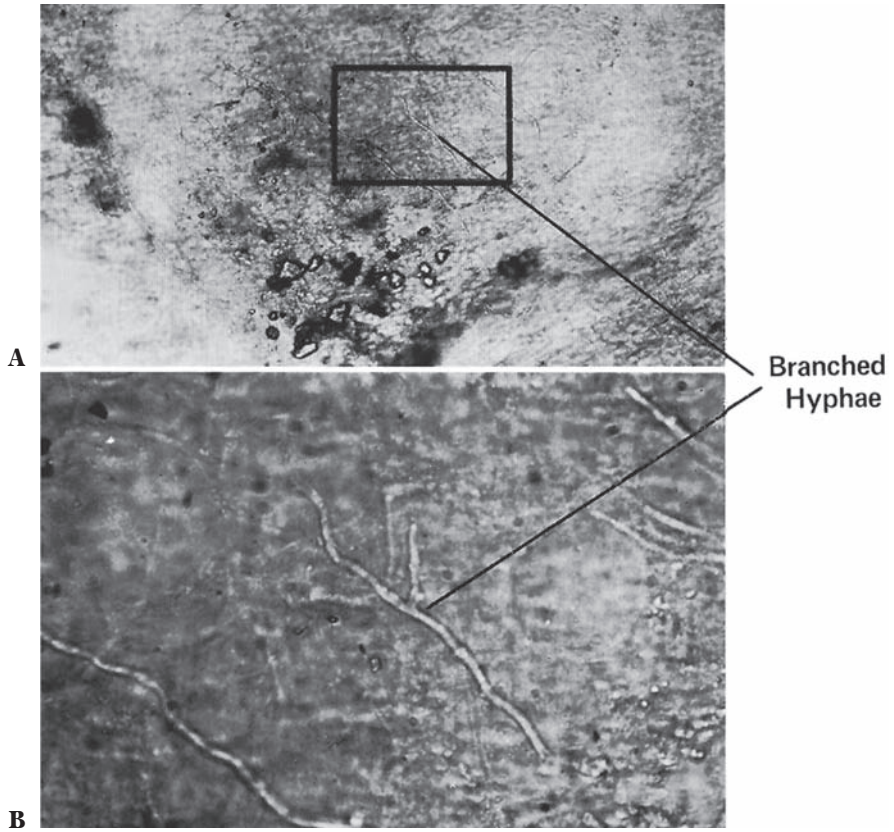

FIGURE 2-1 ■ Fungi from a skin scraping as seen with microscope in a KOH preparation. **(A)** Low-power lens (100X) view. **(B)** High-power lens (450X) view of area outlined above. *(Courtesy of Dr. D. Gibson.)*

the new food should be discontinued for the present. More new foods are added until the normal diet, minus the allergenic foods, is regained.

Keeping a "diet diary" of all foods, medicines, oral hygiene items, or anything injected or inhaled can sometimes be a retrospective way of identifying an allergen. The skin reaction usually occurs less than 8 hours after ingestion.

Fungus Examinations

The KOH preparation is a simple office laboratory procedure for the detection of fungal organisms present in skin and nails. It is performed by scraping the diseased skin and examining the material with the microscope. The skin scrapings are obtained by abrading a scaly diseased area with a scalpel. If a blister is present, the underside of the blister is examined. The material is deposited on a glass slide and then covered with 20% aqueous potassium hydroxide solution and a coverslip. The preparation can be gently heated or allowed to stand at room temperature for 15 to 60 minutes. The addition of dimethyl sulfoxide to the KOH preparation eliminates the need to heat the specimen. A diagnostically helpful pale violet stain can be imparted to the fungi if the 20% KOH solution is mixed with an equal amount of Parker Super Quik permanent blue-black ink. Other staining solutions are available. The slide is then examined microscopically for fungal organisms (Fig. 2-1).

For culture preparation, a portion of the material from the scraping can be implanted on several different types of agar, including mycobiotic agar, inhibitory mold agar (IMA), BHI (brain hear infusion) with blood, chloramphenicol, gentamicin agar, and Sabouraud's glucose agar. A white or variously colored growth is noted in approximately 1 to 3 weeks (Fig. 2-2).

The species of fungus can be identified by morphology on the culture plate, biochemical characteristics, and microscopic morphology with a lactophenol cotton blue stain of a smear from the fungal colony. A culture media is available that changes color when a pathogen is cultured.

Biopsies

The biopsy and microscopic examination of a questionable skin lesion may be invaluable. A definitive diagnosis is nearly always rendered with most pigmented lesions and other cutaneous tumors. In the case of inflammatory lesions, histologic findings may or may not be diagnostic, depending on the disease process, the age of the lesion, clinical description of the lesion(s) including extent of involvement, other symptoms and/or medical conditions, and a differential diagnosis.

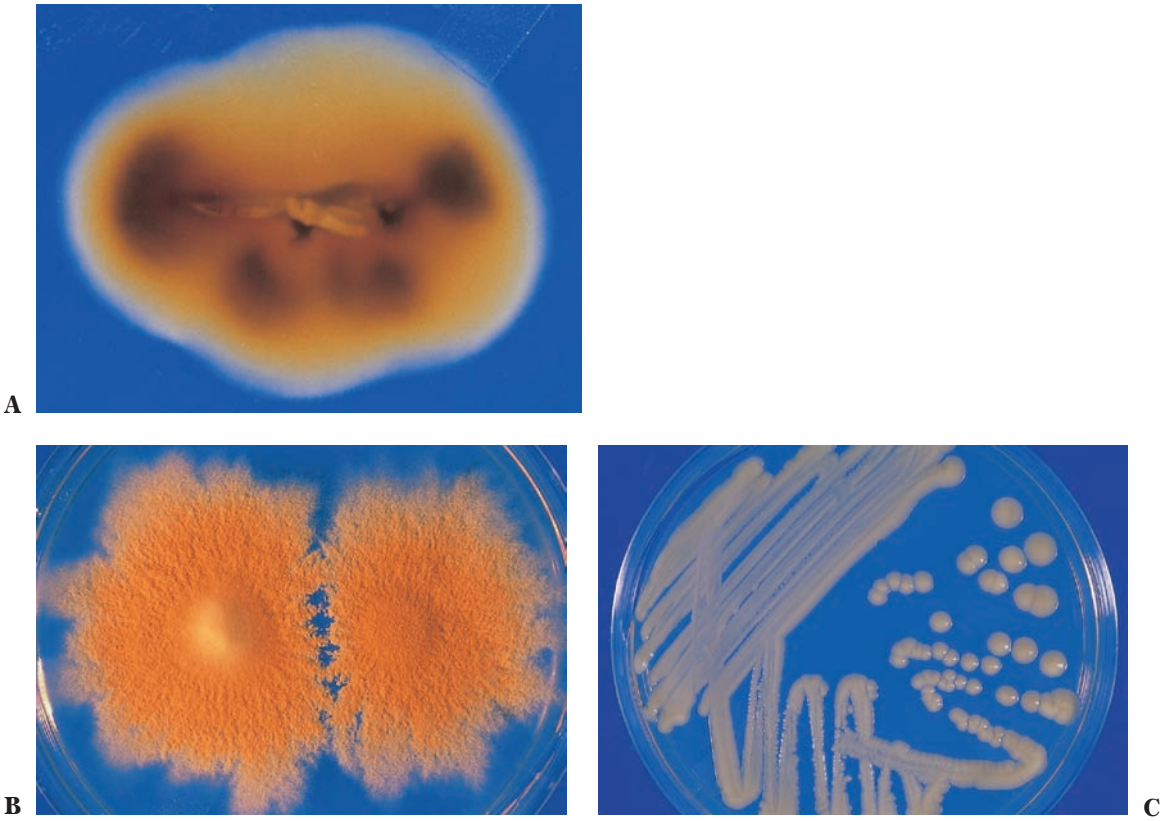

FIGURE 2-2 ■ Fungus cultures: subcultures grown on potato dextrose agar. **(A)** *Trichophyton rubrum.* **(B)** *Microsporum gypseum.* **(C)** *Candida albicans. (Courtesy of Dr. K. Watson.)*

In cases where histologic findings are not diagnostic, at the very least, many pertinent diagnoses on the clinical differential can be excluded. In addition to diagnosis, other useful parameters can be obtained with cutaneous lesions, such as depth of invasion, lymphovascular space invasion, perineural involvement, and adequacy of surgical margins. The quintessential example is malignant melanoma, where most of these factors plus several others may only be assessed histologically and are essential for staging and prognosis.

There are four principle techniques for performing skin biopsies:

1. Surgical excision with suturing
2. Punch biopsy
3. Excision with scissors
4. Shave biopsy

SAUER'S NOTES

1. The skin biopsy specimen must include adequate tissue for proper interpretation by the pathologist.
2. Communication between a pathologist knowledgeable in this disease and the clinician is mandatory for accurate tissue diagnosis.

The decision in favor of one method depends on factors such as location of the biopsy, desired cosmetic result, depth of the diseased tissue, type of lesion to be removed (flat or elevated), and simplicity of technique. For example, vesicles should be completely excised in an attempt to keep the roof intact. Scalp biopsy specimens should extend into the subcutis to include the bulbs of terminal follicles. The instruments and materials needed to perform a skin biopsy are discussed in Chapter 6.

Surgical Excision

The technique of performing surgical excision biopsies with suturing of the skin is well-known. In general, this type of biopsy is performed if a good cosmetic result is desired and if the entire lesion is to be removed. The disadvantage is that this procedure is the most time consuming of the three techniques, and it is necessary for the patient to return for removal of the sutures. Absorbable sutures can eliminate the need for a return visit. It is important that a sharp scalpel be used to reduce compression artifact and that care is taken not to crush the specimen with the forceps.

Punch Biopsy

Punch biopsies can be done rather rapidly, with or without suturing of the wound. A punch biopsy instrument of appropriate

size is needed. Disposable biopsy punches are available. A local anesthetic is usually injected at the site. The operator rotates the instrument until it penetrates to the subcutaneous level. The circle of tissue is then removed. Bleeding can be stopped with pressure or by the use of one or two sutures. An elliptical, like as compared better than versus a circular wound results in a neater scar after suturing. The elliptical punch can be produced by stretching the skin perpendicular to the desired suture line before the punch is rotated. Punch biopsies may be inadequate for evaluation of vesiculobullous diseases and must be deep enough to include subcutaneous fat if used for diagnosis of panniculitis or tumors in a subcutaneous location. In most instances, pigmented lesions should not be punched unless they can be completely excised.

Scissors Biopsy

The third way to remove skin tissue for a biopsy specimen is to excise the piece with sharp pointed scissors and stop the bleeding with light electrosurgery, Monsel solution, or aluminum chloride solution. This procedure is useful for certain types of elevated lesions and in areas in which the cosmetic result is not too important. The greatest advantage of this procedure is the speed and the simplicity with which it can be done.

Shave Biopsy

A scalpel or razor blade can be used to slice off a lesion. This can be performed superficially or deeply. Hemostasis can be accomplished by pressure, light electrosurgery, Monsel solution, or aluminum chloride solution. This method is generally not recommended for excision of melanocytic lesions or other potentially malignant tumors where margin assessment is required. However, it can be used for initial evaluation of pigmented tumors if done deep and wide enough.

Biopsy Handling

The biopsy specimen must be placed in an appropriate fixture, usually 10% formalin. If the specimen tends to curl, it can be stretched out on a piece of paper or cardboard before fixing. Mailing specimens in formalin during winter may result in freezing artifact. This can be avoided by the addition of 95% ethyl alcohol, 10% by volume. For some procedures, fresh tissue should be taken directly for pathologic processing (fresh tissue, Mohs surgery, direct immunofluorescence), put in sterile saline (for culture of fungi and bacteria, including acid-fast bacteria), viral transport media (for viral culture), Michel's solution (direct immunofluorescence), and occasionally sent frozen on dry ice for special procedures.

Cytodiagnosis

The Tzanck test is useful in identifying bullous diseases such as pemphigus and vesicular virus eruptions (herpes simplex and herpes zoster). The technique and choice of lesions are important. For best results, select an early lesion. In the case of a blister, remove the top with a scalpel or sharp scissors. Blot the excess fluid with a gauze pad, and then gently scrape the floor of the blister with a scalpel blade. Try not to cause bleeding. Make a thin smear of the cells on a clean glass slide. If you are dealing with a solid lesion, squeeze the material between two slides. The slide may be air dried, but it can also be fixed by placing it in 95% ethanol for 15 seconds. Stain the slide with Wright–Giemsa stain (stain for 30 seconds, rinse with water, let dry, and then observe under high-power oil immersion) or hematoxylin and eosin. Pap smear technique can also yield good results.

In addition to skin testing, fungus examination, biopsies, and cytodiagnosis, there are certain tests for specific skin conditions that are discussed in connection with their respective diseases.

Additional Studies

The deposition of immunoglobulin and complement may be detected by direct immunofluorescence. This is an extremely valuable technique for the diagnosis of lupus erythematosus and autoimmune bullous diseases. It is performed on a frozen section; therefore, the biopsy specimen must be received fresh or in Michel's solution.

Immunohistology is particularly helpful in the accurate diagnosis and classification of neoplasms. It is possible to identify specific antigens in a routinely processed tissue section by attaching a labeled antibody. For example, malignant melanoma may be identified using antibodies directed against S-100 protein as well as other more sensitive melanoma-specific antigens, such as MART-1 and tyrosinase (Fig. 2-3). Different cytokeratin subtypes may be used to

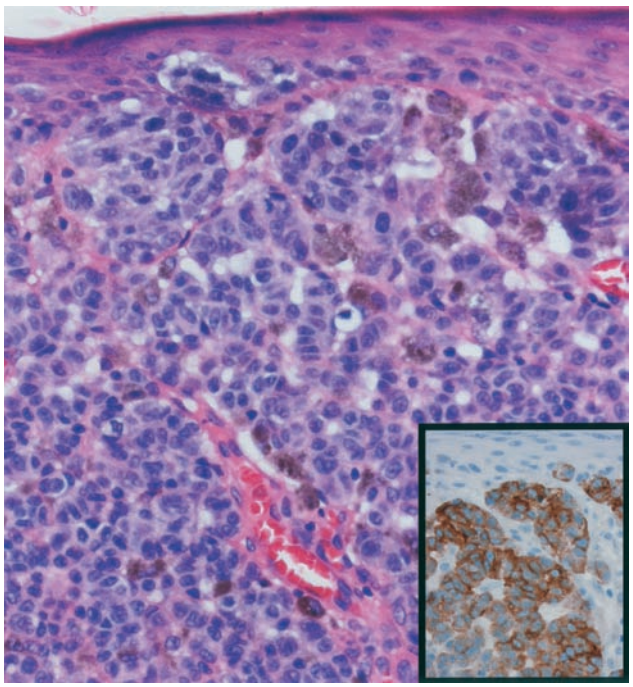

FIGURE 2-3 ■ Photomicrograph of malignant melanoma with positive immunoreactivity for Mart-1 (*inset*). *(Courtesy of Dr. K. Watson.)*

help differentiate certain epithelial tumors that are histologically similar. For example, cytokeratin 7 helps to differentiate metastatic small cell carcinoma of the lung from primary Merkel cell carcinoma of the skin, as well as both mammary and extramammary Paget's disease from squamous cell carcinoma in situ (Bowen's disease). Mesenchymal tumors, such as dermatofibroma, are generally immunoreactive for the intermediate filament vimentin, as well as a host of other markers, depending on the tumor type and cell origin. Leukocyte common antigen labels most lymphomas and leukemias. Multiple other antibodies can be used to distinguish the cell line, diagnosis, and prognosis. CD3, CD4, CD8, CD5, and CD7 are all T-cell markers that can be used to distinguish patch-stage mycosis fungoides from benign mimics, such as small plaque parapsoriasis and other forms of eczema.

DNA technology may be very useful. In situ hybridization allows recognition of specific DNA or RNA sequences using a gene probe in frozen or paraffin tissue sections. For example, a variety of different viruses, including herpes simplex, cytomegalovirus, and a human papillomavirus, can be identified using this technique.

Flow cytometry is another method of identifying specific cell antigens and is generally only useful with lymphomas and leukemias. This test is most commonly performed on lymph nodes, peripheral blood, and bone marrow, but may also be performed on solid organs, such as skin, provided the abnormal cell population is of sufficient quantity. A fresh specimen is needed. Following manipulation of the tissue to tease out the abnormal cells into a liquid media, the individual cells are labeled with antibodies (up to four at once) and passed through a light-scattering source that is able to measure cell size as well as antigen expression. The main advantage that flow cytometry has over tissue immunohistochemistry is the ability to characterize small populations of abnormal cells and to establish monoclonality via the analysis of immunoglobulin light chain expression. Polymerase chain reaction (PCR) may now also be used to establish monoclonality in both fresh and paraffin-embedded tissue. Disadvantages include lengthy time to diagnosis, high cost, and extreme sensitivity to DNA carryover/contamination problems from other specimens. PCR is able to pick up very small populations of clonal cells that may not be truly neoplastic or malignant.

Acknowledgments

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CHAPTER 3

Dermatologic Diagnosis

John C. Hall, MD

This chapter will discuss how to describe primary and secondary skin lesions, common dermatologic conditions associated with different anatomic locations, seasonal skin diseases, military dermatoses, and dermatoses found in patients of color.

Primary and Secondary Lesions

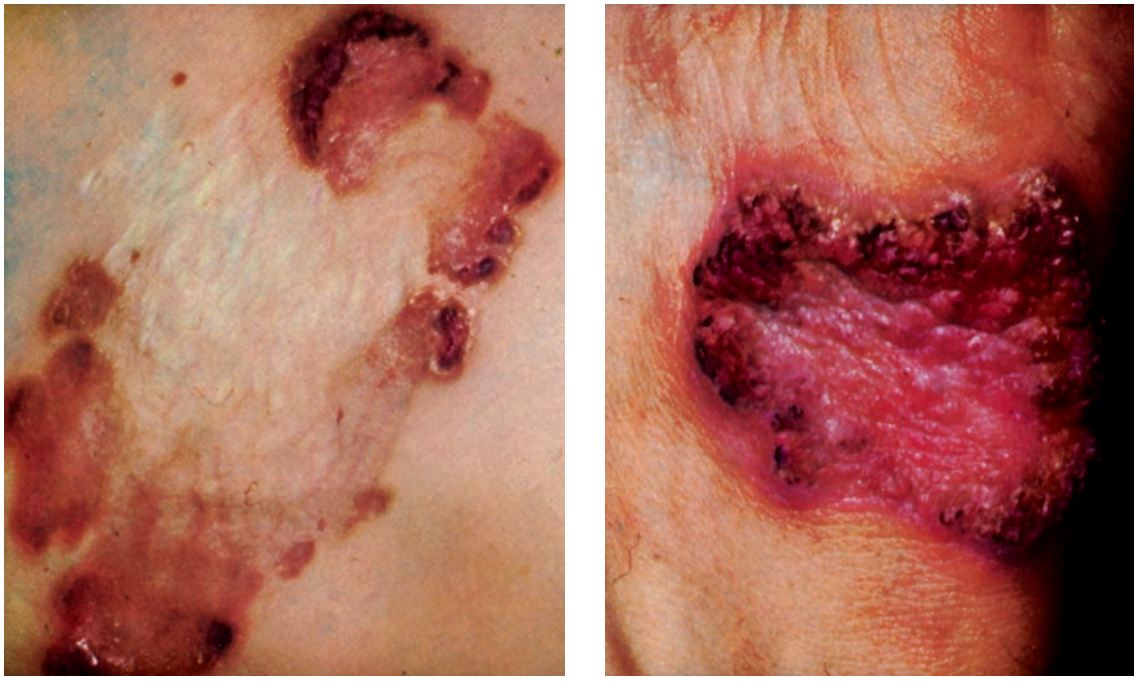
Most skin diseases have some characteristic primary lesions. It is important to examine the patient closely to find the primary lesion. Commonly, however, secondary lesions that are a direct result of overtreatment, excessive scratching, or infection have obliterated the primary lesions. Even in these cases, it is usually possible, through careful examination, to find some primary lesions at the edge of the eruption or on other, less irritated areas of the body (Fig. 3-1). Combinations of primary and secondary lesions also frequently occur.

Primary Lesions

- *Macules:* Up to 1 cm and are circumscribed, flat discolorations of the skin (Fig. 3-2A). Examples include freckles, flat nevi, and some drug eruptions.
- *Patches:* Larger than 1 cm and are circumscribed, flat discolorations of the skin. Examples include vitiligo, some drug eruptions, senile freckles, melasma, and measles exanthem.
- *Papules:* Up to 1 cm and are circumscribed, elevated, superficial, solid lesions (Fig. 3-2B). Examples include elevated nevi, some drug eruptions, warts, and lichen planus. A *wheal* (hive) is a type of papule that is edematous and transitory (present <24 hours). Causes of wheals include drug eruptions, food allergies, numerous underlying illnesses, and insect bites.
- *Plaques:* Larger than 1 cm and are circumscribed, elevated, superficial, solid lesions. Examples include mycosis fungoides and lichen simplex chronicus.
- *Nodules:* Range in size (up to 1 cm) and are solid lesions with depth. They may be above, level with, or beneath the skin surface (Fig. 3-2C, D). Examples are nodular secondary or tertiary syphilis, basal cell cancers, dermatofibromas, and xanthomas.
- *Tumors:* Larger than 1 cm and are solid lesions with depth. They may be above, level with, or beneath the skin surface (Fig. 3-2E). Examples include tumor stage of mycosis fungoides and larger basal cell cancers.
- *Vesicles:* Up to 1 cm in size and are circumscribed elevations of the skin containing serous fluid (Fig. 3-2F). Examples include poison ivy, early chickenpox, herpes zoster, herpes simplex, dyshidrosis, and contact dermatitis.
- *Bullae:* Larger than 1 cm and are circumscribed elevations containing serous fluid. Examples include pemphigus, bullous pemphigoid, poison ivy, and second-degree burns.

SAUER'S NOTES

1. One of the dermatologist's tools of the trade is a magnifying lens. *Use it.*
2. A complete examination of the entire body is a necessity when confronting a patient with a diffuse skin eruption or an unusual localized eruption.
3. Touch the skin and skin lesions. You learn a lot by palpating, and patients appreciate that you are not afraid of "catching" the problem. (For the uncommon contagious problem, use precaution.)
4. When in doubt of the diagnosis, verify your clinical impression with a biopsy. The most frequent reason for a successful malpractice suit in dermatology is failure to diagnose.
5. Do not underestimate the importance of adequate lighting.
6. Dermoscopy is a new tool that is mainly used to evaluate pigmented lesions. It combines diascopy and magnification and is useful in diagnosing melanoma as well as deciding which tumors need a biopsy. Diascopy is a test to observe change in color after compression of a skin condition with a clear plastic or glass slide. If an observer has significant experience in dermoscopy, it is useful when deciding whether a lesion is truly benign or not.
7. There are computerized systems that will soon be available to evaluate multiple variables of pigmented tumors in vivo to decide whether a biopsy is necessary.
8. Serial photography systems have been shown by some authors to be useful when determining which pigmented tumors have changed significantly enough over time to warrant a biopsy.



A B

FIGURE 3-1 ■ Nodular lesions. **(A)** Grouped nodular lesions with central scarring (tertiary syphilis). **(B)** Grouped warty, nodular lesions with central scarring (tuberculosis verrucosa cutis). *(Courtesy of Marion B. Sulzberger, Folia Dermatologica, No. 1, Geigy Pharmaceuticals.)*

- *Pustules:* Vary in size and are circumscribed elevations of the skin containing purulent fluid (Fig. 3-2G). Examples include acne, pustular psoriasis, and impetigo.
- *Petechiae:* Range in size (up to 1 cm) and are circumscribed deposits of blood or blood pigments. Examples are thrombocytopenia, vasculitis, and drug eruptions.
- *Purpura:* A circumscribed deposit of blood or blood pigment that is larger than 1 cm in the skin. Examples include senile purpura, drug eruptions, bleeding diatheses, chronic topical and systemic corticosteroid use, and vasculitis.

Secondary Lesions

- *Scales:* Shedding, dead epidermal cells that may be dry or greasy. Examples are seborrhea (greasy) and psoriasis (dry).
- *Crusts:* Variously colored masses of skin exudates of blood, serum, pus, or any combination of these (Fig. 3-3A). Examples include impetigo, infected dermatitis, nummular eczema, or any area of excoriation.
- *Excoriations:* Abrasions of the skin, usually superficial and traumatic. Examples are scratched insect bites, scabies, eczema, and dermatitis herpetiformis.
- *Fissures:* Linear breaks in the skin, sharply defined with abrupt walls. Examples include congenital syphilis, interdigital tinea pedis, and hand eczema.
- *Induration:* Woodiness or hardness as seen in infiltrating tumors such as dermatofibrosarcoma protuberans, cutaneous metastasis, lymphoma, scleroderma, or hypertrophic scars.
- *Ulcers:* Variously sized and shaped excavations in the skin extending into the dermis or often deeper that usually heal with a scar. Examples include stasis ulcers of legs, ischemic leg ulcers, pyoderma gangrenosum, and tertiary syphilis.
- *Scars:* Formations of connective tissue replacing tissue lost through injury or disease. Examples are discoid lupus, lichen planus in the scalp, and third-degree burns.
- *Keloids:* Hypertrophic scars beyond the borders of the original injury (Fig. 3-3B). They are elevated, can be progressive, and usually are the result of some sort of trauma in the skin. Keloids are more common in darker-skinned people. They are common on the upper torso, neck, and with body piercing (especially with piercings of the earlobe). Rarely, keloids can occur spontaneously. Any type of full-thickness skin trauma can heal with a keloidal scar. They are unsightly and can be numb, pruritic, or painful.
- *Lichenification:* A diffuse area of thickening and scaling with a resultant increase in skin lines and markings (Fig. 3-3C). It is often seen in atopic dermatitis or any area chronically rubbed or scratched.

Several combinations of primary and secondary lesions commonly exist on the same patient. Examples are *papulosquamous lesions* of psoriasis, *vesiculopustular lesions* in contact dermatitis, and *crusted excoriations* in scabies.

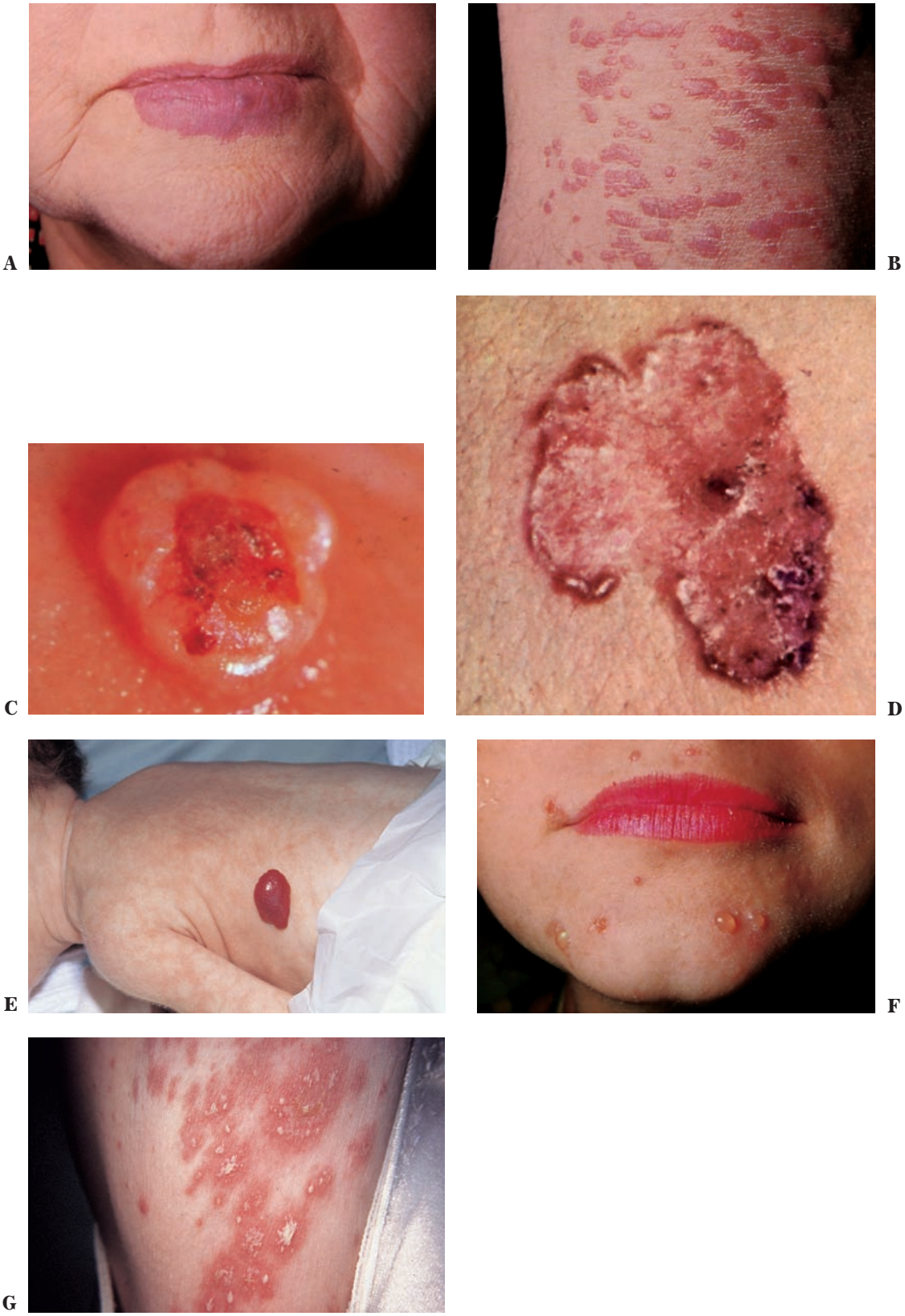


FIGURE 3-2 ■ Primary skin lesions. **(A)** Patch on lip (port wine hemangioma). **(B)** Papules on knee (lichen planus). **(C)** Nodule on lower eyelid (basal cell carcinoma). **(D)** Polycyclic nodular lesion (superficial basal cell carcinoma). **(E)** Tumor on the left side of an infant (hemangioma). **(F)** Vesicles on chin (pemphigus vulgaris). **(G)** Pustules, pretibial (pustular psoriasis). *(Courtesy of Geigy Pharmaceuticals.)*

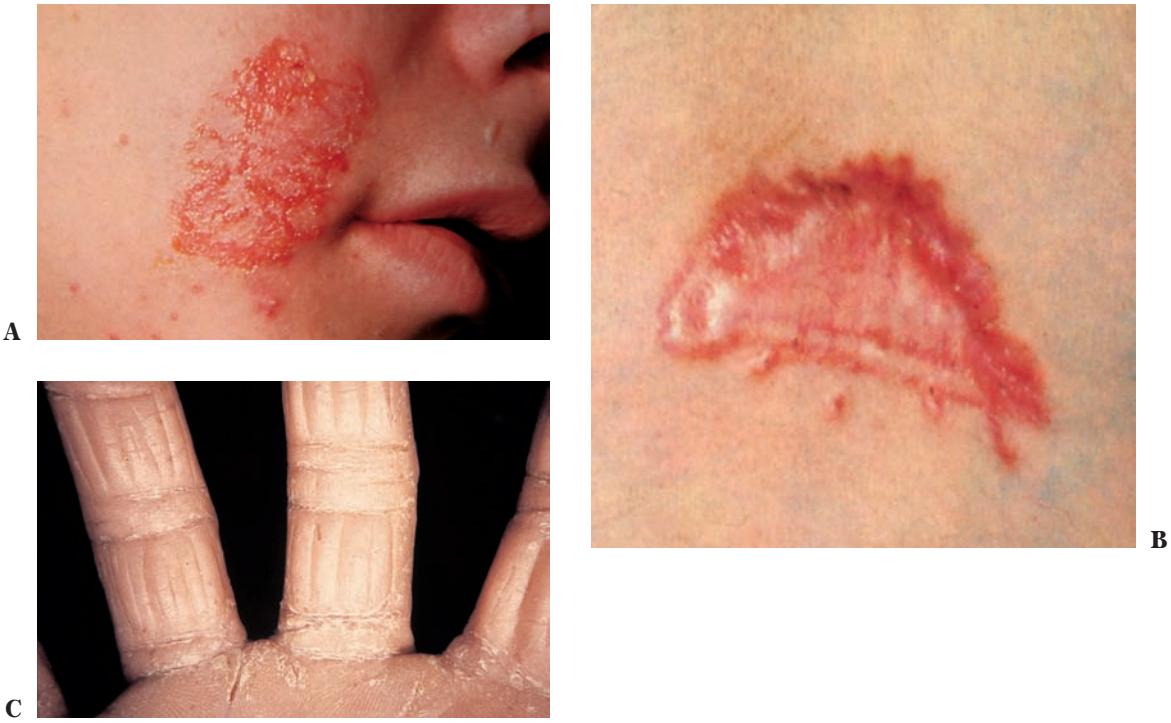


FIGURE 3-3 ■ Secondary lesions. **(A)** Crust on cheek (impetigo). **(B)** Keloid. **(C)** Lichenification of flexor fingers in a patient with chronic eczema.

Special Lesions

Some primary lesions, limited to a few skin diseases, can be called *specialized lesions.*

- *Burrows:* Very thin and short (in scabies) or tortuous and long (in creeping eruption) tunnels in the epidermis.
- *Comedones* or *blackheads:* Plugs of whitish (whiteheads or closed comedones) or blackish (blackheads or open comedones) sebaceous and keratinous material lodged in the pilosebaceous follicle, usually seen on the face, chest, or back and, rarely, on the upper part of the arms. Examples include acne and Favre–Racouchot on sun-damaged skin in the temporal areas. These are a hallmark of chloracne. Chloracne is caused by exposure to hydrocarbons such as those found in cutting oils and Agent Orange.
- *Cutaneous horn:* A localized spike-shaped area of marked overgrowth of keratin that can stick above the skin half an inch or more. It is quite localized (usually 0.5 to 1 cm in width or less). It most commonly overlies actinic keratoses, but can overlie seborrheic keratoses, squamous cell carcinomas, warts, porokeratoses, or, less likely, hyperkeratotic basal cell cancers.
- *Flagellate:* Linear whiplike red lesions most often associated with bleomycin therapy but also reported with peplomycin therapy, dermatomyositis, adult-onset Still's disease, and Shiitake mushroom dermatitis associated with eating this particular mushroom.
- *Follicular plugs:* Keratin plugs in the hair follicle that are 1 to 3 mm in size and most characteristically seen in lupus erythematosus ("carpet tack sign" seen on the underside of the scale) and lichen planus (more flask-shaped plugs).
- *Mal perforans ulcer:* Seen in diabetics and leprosy patients. There is an associated neuropathy, so the ulcers are painless despite being deep and destructive. They are circular and sharply marginated or "punched out." These ulcers are usually associated with vasculitis; however, ischemic (very painful) and factitial ulcers can also have the same appearance.
- *Milia:* Whitish papules, 1 to 2 mm in diameter, that have no visible opening onto the skin surface. Examples are found in healed burns or superficial trauma sites, healed bullous disease sites, or newborns. They are not uncommon on the face of adults and can become more widespread in newborns.
- *Chancre:* Rounded, usually single, erosions or ulcers often with an exudative surface. These include the following: anthrax, atypical mycobacterium, blastomycosis (primary cutaneous type), chancroid, coccidioidomycosis (primary cutaneous type), cowpox, cutaneous diphtheria, erysipeloid, furuncle, milker's nodule, orf, rat-bite fever (sodoku), sporotrichosis, syphilis (genital but also extragenital), tuberculosis (primary inoculation type), tularemia, and vaccinia.
- *Striae cutis distensae:* Red, resolving to white, linear areas of atrophy that may be indented. They are seen mainly on the thighs, buttocks, and breasts. They can be seen during rapid weight loss, prolonged use

of topical or systemic corticosteroids, bodybuilding (especially with androgen ingestion), Cushing's disease, and pregnancy, where it is most pronounced over the abdomen.

- *Telangiectasias:* Dilated superficial blood vessels. Examples include spider hemangiomas, chronic radiodermatitis, basal cell cancer, sebaceous hyperplasia, prolonged chronic sun exposure, necrobiosis lipoidica diabeticorum, and rosacea.

In addition, distinct and often diagnostic changes can occur in the nail plates and the hairs. These are discussed in the chapters relating to these appendages.

Diagnosis by Location

A physician is often confronted with a patient with skin trouble localized to one part of the body (**Figs. 3-4 to 3-7**). The following list of diseases with special locations is meant

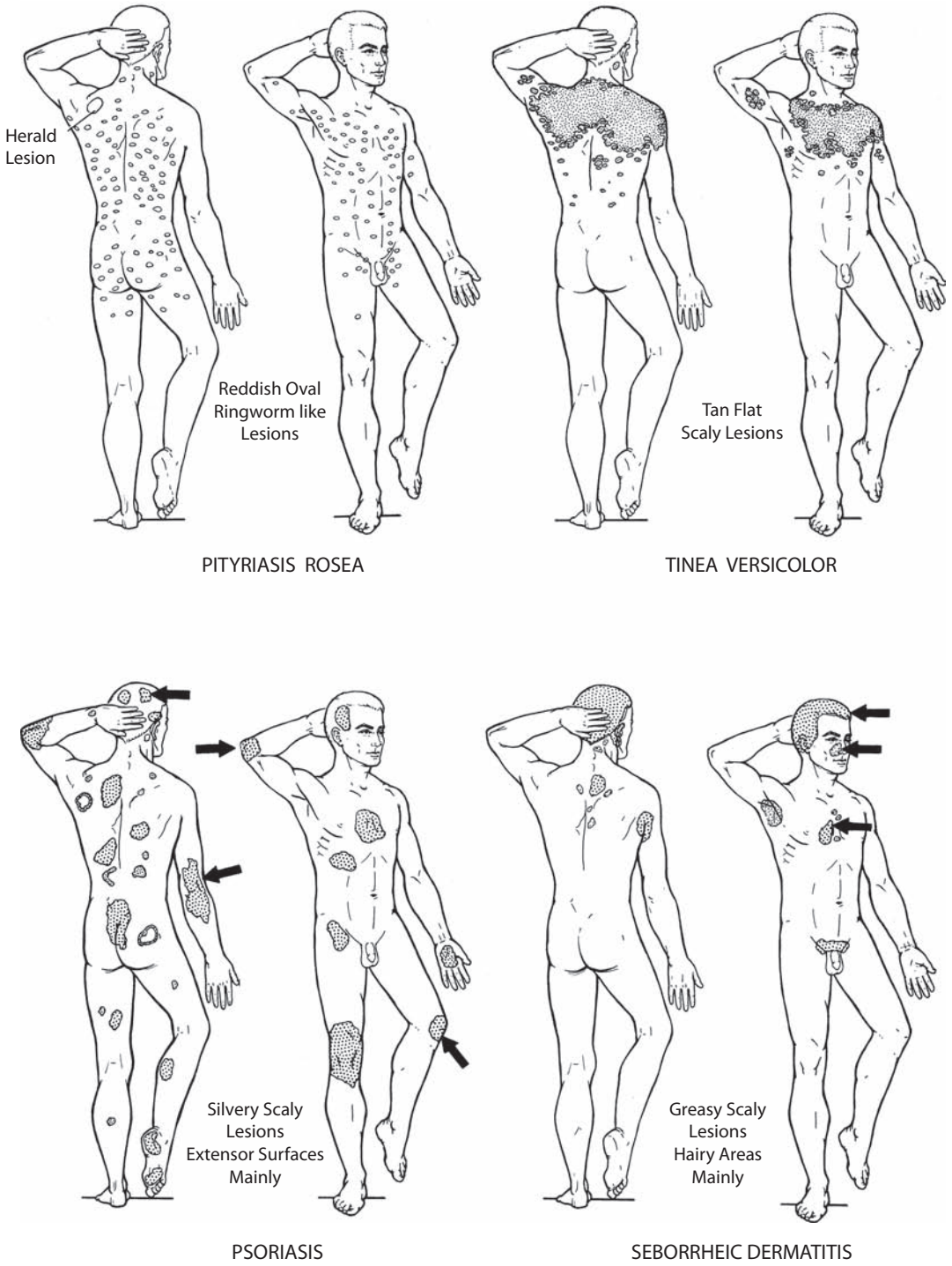


FIGURE 3-4 ■ Dermatologic silhouettes. Diagnosis by location.

to aid in the diagnosis of such conditions, but this list should not be considered exclusive. Generalizations are the rule, and many rare diseases are omitted. For further information concerning each particular disease, consult the Dictionary-Index located at the end of the book.

- *Scalp:* Seborrheic dermatitis, contact dermatitis, seborrheic keratoses, pilar cysts, psoriasis, folliculitis, pediculosis, and hair loss due to the following: male or female pattern alopecia areata, lichen planopilaris, tinea, chronic discoid lupus erythematosus, telogen effluvium (postpartum alopecia), or trichotillomania.
- *Ears:* Seborrheic dermatitis, psoriasis, atopic eczema, lichen simplex chronicus, actinic keratoses, melanoma, varix, seborrheic keratoses, and squamous cell carcinomas.
- *Face:* Acne, rosacea, impetigo, contact dermatitis, seborrheic dermatitis, folliculitis, herpes simplex, lupus erythematosus, dermatomyositis, nevi,

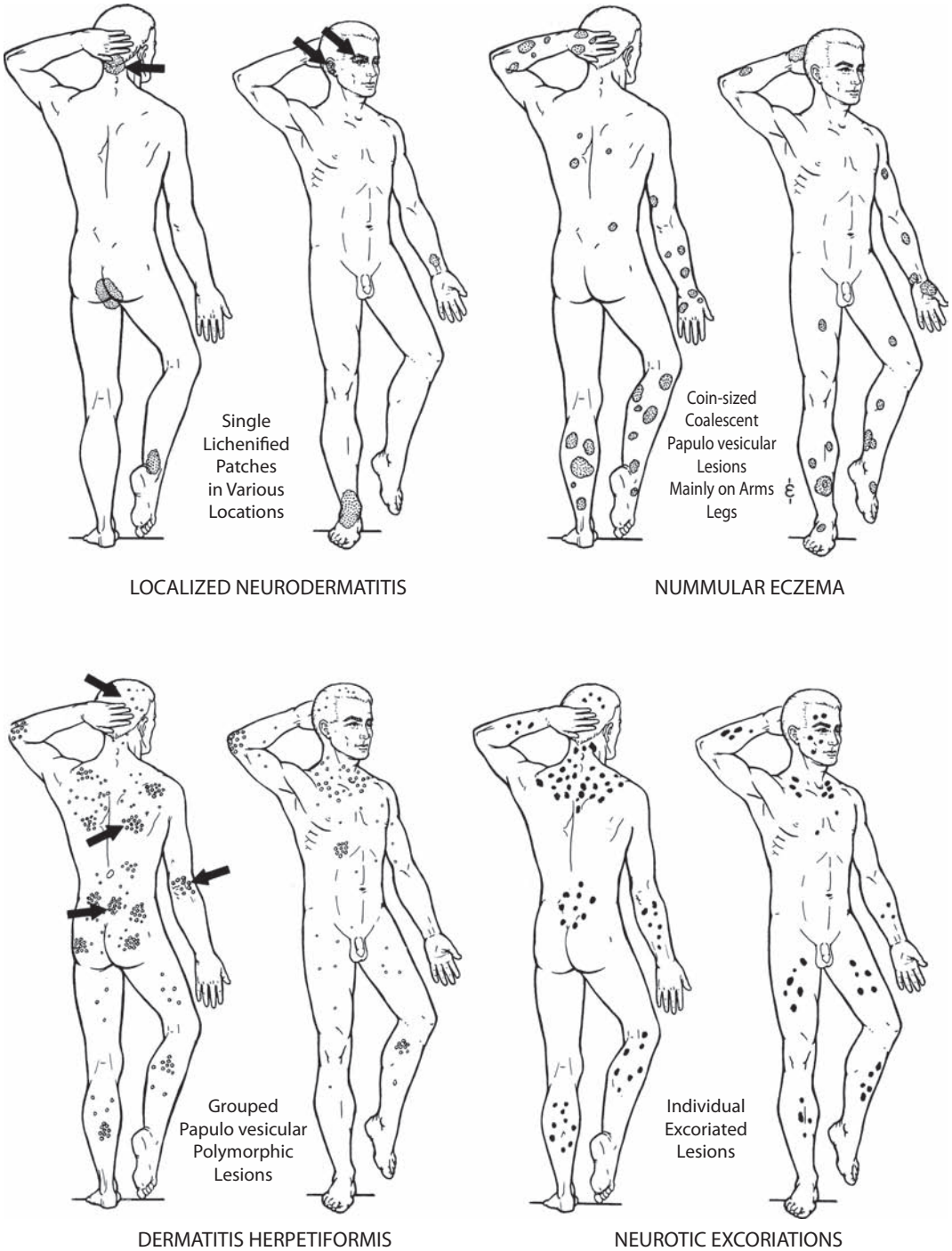


FIGURE 3-5 ■ Dermatologic silhouettes. Diagnosis by location.

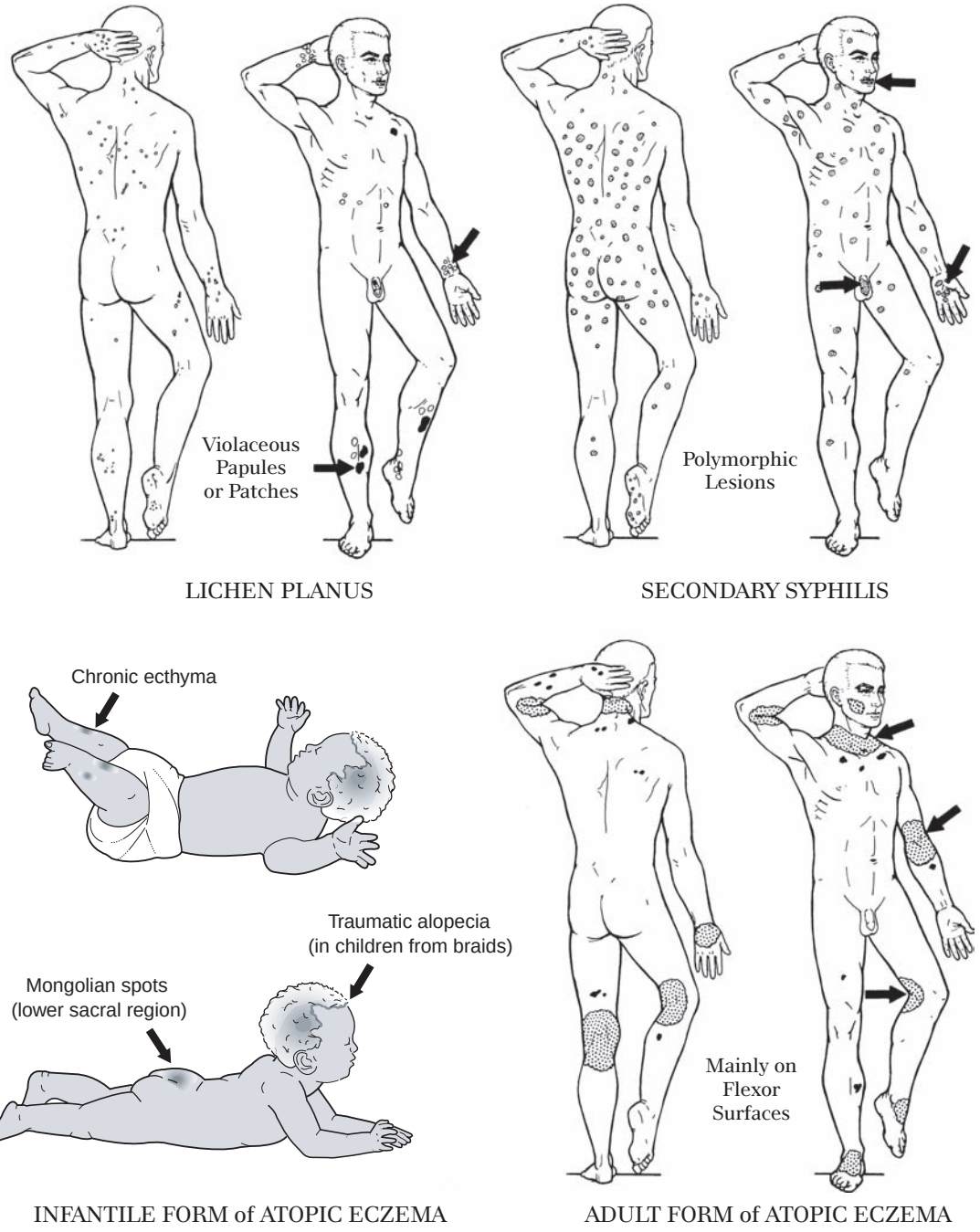


FIGURE 3-6 ■ Dermatologic silhouettes. Diagnosis by location.

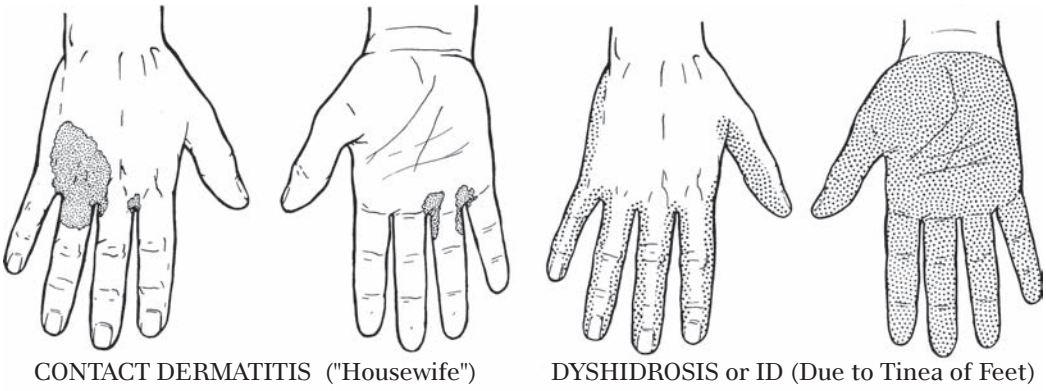
SAUER'S NOTES

In diagnosing a rather generalized skin eruption, the following three mimicking conditions must be considered first and ruled in or out by an appropriate history or examination:

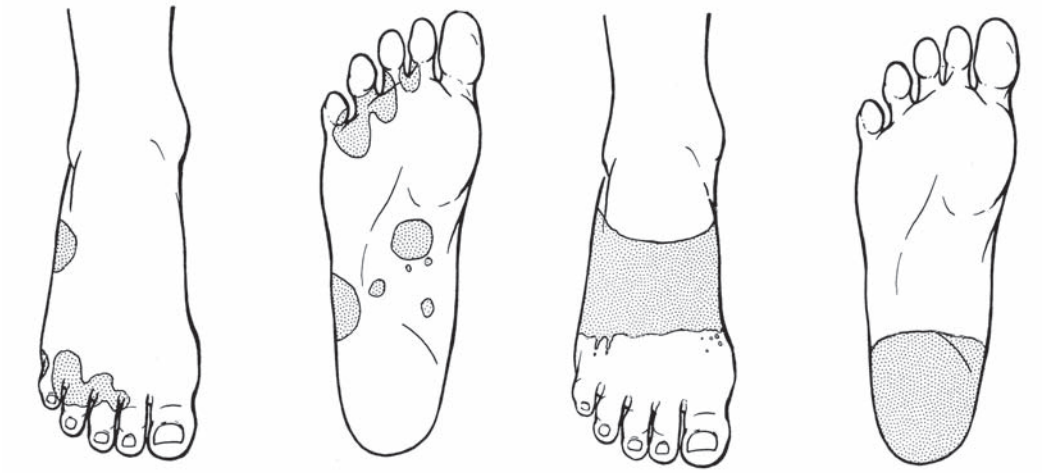
1. Drug eruption
2. Contact dermatitis
3. Infectious diseases, such as acquired immunodeficiency syndrome, other viral exanthems, and secondary syphilis

melasma, melanoma (especially lentigo maligna melanoma), basal cell cancer, actinic keratosis, seborrheic keratosis, squamous cell carcinoma, seborrheic keratosis, milia, and sebaceous hyperplasia.

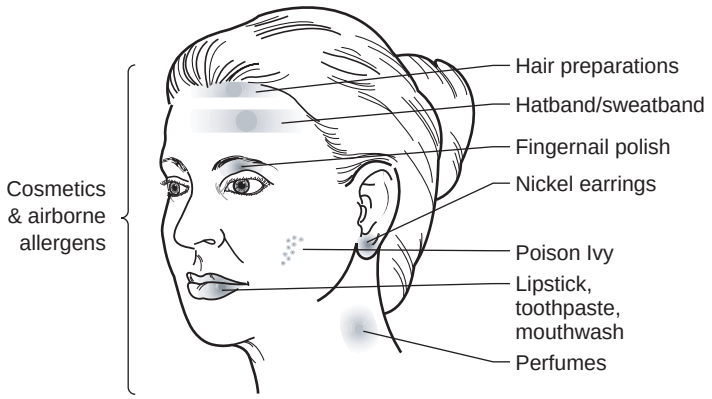
- *Eyelids:* Contact dermatitis due to cosmetics, especially fingernail polish or hair sprays, dermatomyositis, seborrheic dermatitis, atopic eczema, skin tags, syringomas, and basal cell cancer.
- *Posterior neck:* Neurodermatitis (lichen simplex chronicus), seborrheic dermatitis, psoriasis, folliculitis, contact dermatitis, cutis rhomboidalis



CONTACT DERMATITIS ("Housewife") DYSHIDROSIS or ID (Due to Tinea of Feet)



FUNGUS INFECTION CONTACT DERMATITIS (Shoes)



CONTACT DERMATITIS

FIGURE 3-7 ■ Dermatologic silhouettes. Diagnosis by location.

nuchae, and acne keloidalis nuchae especially in darker-skinned patients.

- *Mouth:* Aphthae, herpes simplex, geographic tongue, syphilis, lichen planus, traumatic fibromas, erythema multiforme, oral hairy leukoplakia, squamous cell cancer, candidiasis, and pemphigus.
- *Axillae:* Contact dermatitis, seborrheic dermatitis, hidradenitis suppurativa, erythrasma, acanthosis nigricans, and Fox–Fordyce disease.
- *Chest and back:* Tinea versicolor, pityriasis rosea, acne, seborrheic dermatitis, psoriasis, secondary syphilis, epidermoid cysts of the back, seborrheic keratoses, senile or cherry angiomas, nevi, and melanomas, especially on the backs of men.
- *Groin and crural areas:* Tinea infection, candida infection, bacterial intertrigo, scabies, pediculosis, granuloma inguinale, warts, skin tags, hidradenitis suppurativa, folliculitis, seborrhea, and inverse psoriasis.

- *Penis:* Contact dermatitis, fixed drug eruption, condyloma acuminata, candida balanitis, chancroid, herpes simplex, primary, actinic keratoses, secondary syphilis, scabies, balanitis xerotica obliterans, warts, psoriasis, seborrhea, and pearly penile papules.
- *Hands:* Contact dermatitis, id reaction to fungal infection of the feet, atopic eczema, psoriasis, verrucae, pustular psoriasis, nummular eczema, erythema multiforme, secondary syphilis (palms), fungal infection, dyshidrotic eczema, warts, and squamous cell carcinoma of the dorsal hands.
- *Cubital fossae and popliteal fossae:* Atopic eczema, contact dermatitis, and folliculitis.
- *Elbows and knees:* Psoriasis, xanthomas, dermatomyositis, granuloma annulare, and atopic eczema.
- *Feet:* Fungal infection, primary or secondary bacterial infection, contact dermatitis from footwear or foot care, atopic eczema, verrucae, psoriasis, erythema multiforme, dyshidrotic eczema, and secondary syphilis (soles of feet).

Seasonal Skin Diseases

Certain dermatoses have an increased incidence in various seasons of the year. In a busy dermatologist's office, a clinician can see "epidemics" of atopic eczema, pityriasis rosea, psoriasis, and winter itch, among others. Knowledge of this seasonal incidence associated with some skin conditions is helpful from a diagnostic standpoint. It is sufficient simply to list these seasonal diseases here, because more specific information concerning them can be found elsewhere in this text. Remember that there are always exceptions to every rule.

Winter

- Atopic eczema
- Irritant contact dermatitis of the hands
- Psoriasis
- Seborrheic dermatitis
- Nummular eczema
- Winter itch and dry skin (xerosis)
- Ichthyosis

Spring

- Pityriasis rosea
- Erythema multiforme
- Acne (flares)
- Viral exanthems

Summer

- Contact dermatitis due to poison ivy
- Tinea of the feet and the groin
- Candida intertrigo
- Miliaria or prickly heat
- Impetigo and other pyodermas
- Polymorphous light eruption
- Insect bites
- Tinea versicolor (noticed after suntan)
- Darier's disease (uncommon)
- Epidermolysis bullosa (uncommon)

Fall

- Winter itch
- Senile pruritus
- Atopic eczema
- Acne (less sun, more stress with school starting)
- Pityriasis rosea
- Contact dermatitis due to ragweed
- Seborrheic dermatitis
- Tinea of the scalp (schoolchildren)
- Viral exanthems

Military Dermatoses

Certain parts of the world continue to be at war, and under its ravages, the lack of good personal hygiene, the lack of adequate food, and the presence of overcrowding, injuries, and pestilence can result in the aggravation of any existing skin disease. In this setting, there is an increased incidence of the following skin diseases:

- Scabies
- Pediculosis
- Syphilis and other sexually transmitted diseases
- Bacterial dermatoses
- Tinea of the feet and the groin
- Pyoderma
- Miliaria
- Leishmaniasis (Middle East)

Dermatoses of Dark-skinned Patients

The following skin diseases are seen with greater frequency in people of color than in light-skinned patients (Figs. 3-8 and 3-9):

- Keloids
- Dermatosis papulosa nigra (variant of seborrheic keratoses that are dark, small, multiple, facial, and more common in women)
- Pigmentary disturbances from many causes, both hypopigmented and hyperpigmented
- Traumatic marginal alopecia (traction alopecia) (from braids and from heated irons used in hair straightening)
- Seborrheic dermatitis of the scalp, aggravated by grease on the hair
- Ingrown hairs of the beard (pseudofolliculitis barbae)
- Acne keloidalis nuchae
- Annular form of secondary syphilis
- Granuloma inguinale
- Mongolian spots
- Acral lentiginous melanomas
- Tinea capitis (in children who braid their hair)

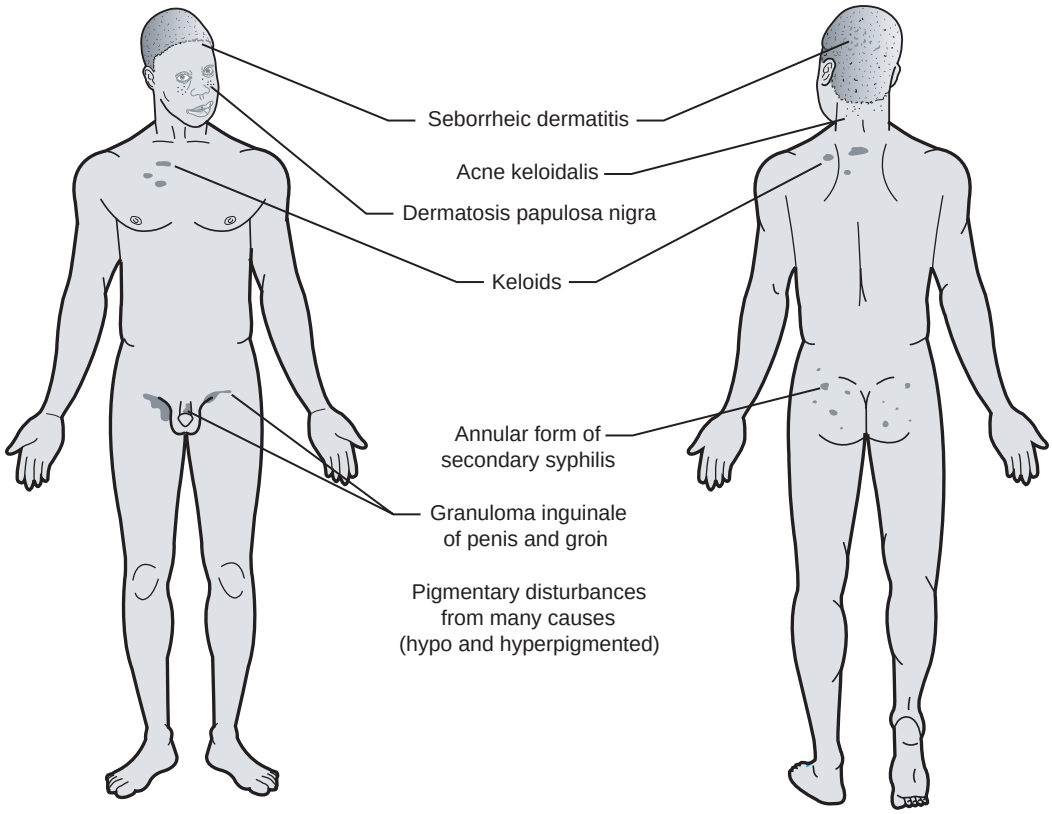


FIGURE 3-8 ■ Dermatologic silhouettes. Conditions more common among dark-skinned patients.

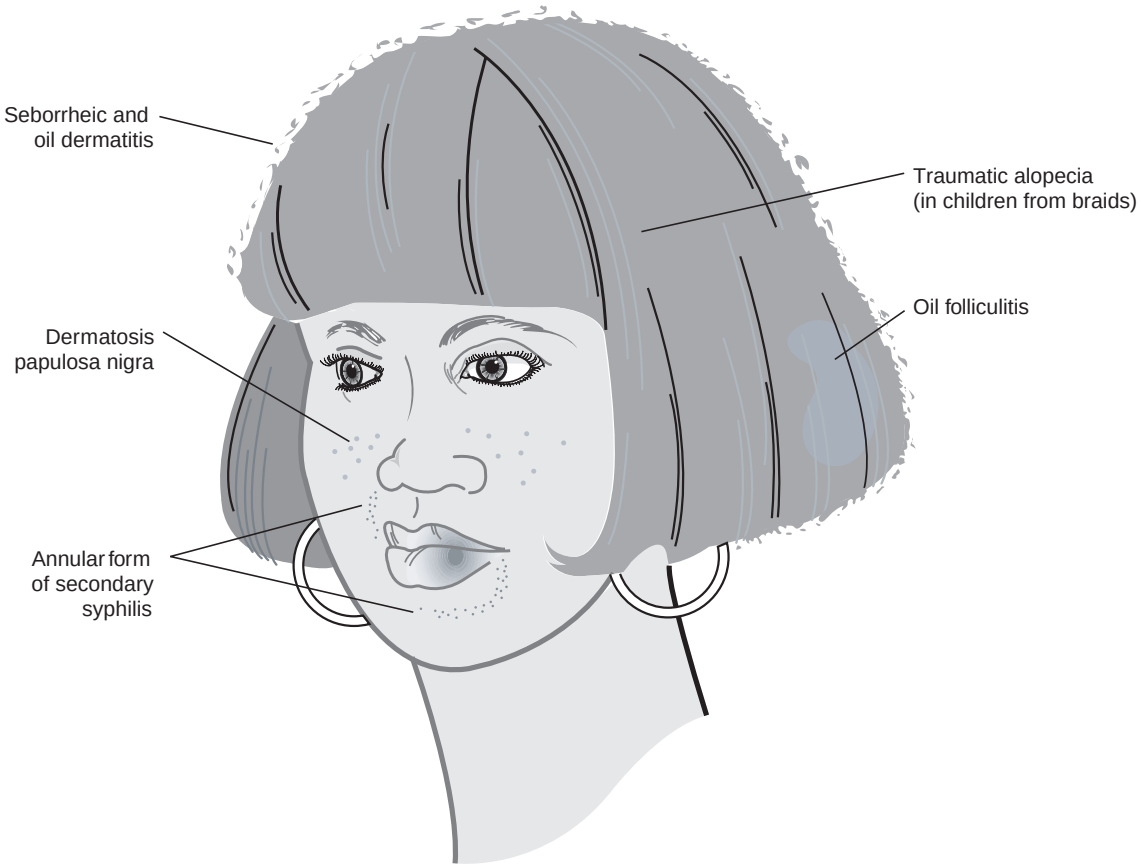


FIGURE 3-9 ■ Dermatologic silhouette. Conditions more common among dark-skinned patients.

On the other hand, certain skin conditions are rarely seen in dark-skinned people:

- Squamous cell or basal cell carcinomas
- Actinic keratoses
- Porokeratosis
- Psoriasis
- Superficial spreading, nodular, and lentigo maligna melanoma
- Scabies

Descriptive Terms Often Used in Dermatology

- *Acneiform:* Refers to a resemblance of acne as seen in acne, folliculitis, rosacea, and some drug eruptions such as those due to topical and systemic corticosteroids. It can also be seen with ingestion of iodides or bromides and as part of chloracne due to hydrocarbon exposure.
- *Agnimated:* refers to aggregated in a group such as a clustered group of blue nevi or agnimated blue nevi.
- *Annulare* or *arciform lesions:* Refer to a peripheral circular curving of the lesions seen in diseases such as erythema annulare centrifugum, erythema chronica migrans of Lyme disease, erythema marginatum of Scarlet fever, erythema gyratum perstans (which can be associated with underlying malignancy), tinea, impetigo, and psoriasis.
- *Atrophic:* Refers to a thinning of the skin as seen in mycosis fungoides with fine superficial "cigarette paper" wrinkling atrophy or deep with a resultant scarring formation as in discoid lupus erythematosus or third-degree burns.
- *Color alterations*
  Hyperpigmented: Increased pigment as seen in postinflammatory hyperpigmentation and residual lesions of lichen planus or dermatitis herpetiformis.
  Hypopigmented: Decreased pigment as seen in pityriasis alba, tinea versicolor after a tan, and postinflammatory hypopigmentation.
  Depigmented: Loss of pigment as in vitiligo or scar.
  Violaceous: Reddish-purple discoloration as seen in vasculitis and the tumors of mycosis fungoides.
  Apple-jelly colored: Reddish-brown color seen most often in sarcoidosis, particularly upon pressing the skin with a clear glass slide. This is called diascopy.
  Porcelain-white: Stark white color that can be seen in morphea, generalized cutaneous scleroderma, Degos disease, and atrophie blanche or livedoid vasculopathy.
  Heliotrope: Refers to violaceous color as seen on upper eyelids in dermatomyositis.
  Cayenne pepper: Tiny reddish-brown spots due to hemosiderin staining of the skin, seen most often in the pigmented purpuric dermatoses.
- *Cushingoid:* An appearance seen in patients on high-dose, long-term systemic corticosteroids in which the face has a round or moonlike appearance and there is increased central body fat particularly over the back with a "buffalo hump." Acne, striae, rosacea, and hirsutism are also often seen.
- *En cuirasse:* Refers to a shieldlike induration usually at the chest wall as seen in scleroderma and infiltrated malignancies, particularly breast cancer.
- *Exophytic:* Protruding from the skin such as in some squamous cell carcinomas, warts, advanced cutaneous lymphoma, and keloids.
- *Filiform:* Refers to tiny filamentous projections that come from a tumor, usually indicative of a filiform wart.
- *Forme fruste:* Refers to an atypical or partial example of a skin disease.
- *Herpetiform:* Means "in a group" and is seen in the blisters of herpes simplex, herpes zoster, varicella, and the autoimmune blistering diseases of dermatitis herpetiformis and impetigo herpetiformis (herpes gestationis).
- *Incognito:* Refers to a hidden skin disease such as in scabies patients who frequently bathe, in dermatitis herpetiformis that is so excoriated that no primary skin lesions can be seen, or in some cases of cutaneous T-cell lymphoma.
- *Keratotic:* Thickening of the horny layer causing dry, heaped up, hard skin such as in keratotic actinic keratosis, squamous cell cancer, keratoderma palmaris et plantaris, chronic palm and sole psoriasis, or eczema (especially lichen simplex chronicus).
- *Linear*: In a line as in poison ivy dermatitis, coup de sabre type of morphea, lichen striatus, and excoriations.
- *Leonine facies:* A lionlike look to the face with thickening of the normal furrows over the entire face, most often described with cutaneous T-cell lymphoma, leprosy, or tertiary syphilis.
- *Morbilliform:* Usually used to refer to a measles-like eruption that is symmetric, macular, and consists of 1 cm or smaller, usually red, macules that can become confluent. This is seen most often in morbilliform drug eruptions and viral exanthems, including measles, rubella, and HIV exanthem, among others.
- *Peau d' orange:* A bulging of the skin with an orange-peel look that has a mottled texture and is seen in cutaneous mucinosis such as myxedema, lymphoma of the skin such as T-helper cell lymphoma, other cutaneous malignancies such as breast cancer, and elephantiasis nostra verrucosa such as seen in chronic lymphedema of the lower extremities.
- *Pedunculated:* Ab narrow stalklike attachment to the skin as in skin tags.

- *Perifollicular:* Eruptions that seem to be around the hair follicle. This term is often used to describe folliculitis, keratosis pilaris, and follicular eczema.
- *Poikiloderma:* Has three components—fine "cigarette paper" wrinkling atrophy, alternating or lacey hyper- and hypopigmentation, and telangiectasias. This is seen as a result of radiation dermatitis, chronic corticosteroid use topically or systemically, and chronic sun exposure most often seen as poikiloderma of Civatte on the sides of the neck. It is also found in collagen–vascular disease (most commonly lupus erythematosus) as well as in dermatomyositis. It can be a generalized eruption, in which case it is called *poikiloderma atrophicans vasculare* and is considered by most authorities to be a cutaneous T-cell lymphoma.
- *Psoriasiform:* Resembling psoriasis, as in psoriasis or a psoriasiform plaque of cutaneous T-cell lymphoma.
- *Punched out:* Circular with sharply demarcated edges and full-thickness skin loss and is most often used in relation to an arterial ischemic ulcer, vasculitic ulcer, or mal perforans ulcer.
- *Purpuric:* Purple areas due to bleeding under the skin or purpura. This can be seen in vasculitis, pyoderma gangrenosum, drug eruptions, and brown recluse spider bites.
- *Reticulate:* A lacy distribution of skin lesions as seen in oral lichen planus, the pigmentation of poikiloderma, and the pigmentation seen in erythema ab igne. This can also be referred to as weblike.
- *Scalloped edges:* Circinate or rounded edges as in impetigo and ruptured blisters in bullous diseases.
- *Sclerodermoid:* Indurated skin, often with loss of pigment, and characteristic of scleroderma, scleredema, bleomycin injection sites, pentazocine (Talwin) injection sites, and chronic cutaneous graft-versus-host disease.
- *Telangiectatic:* Covered with telangiectasias as in rosacea, lupus erythematosus.
- *Umbilicated:* A tumor or plaque that has a central indentation or dell. This is often used to describe molluscum contagiosum and can also be seen in sebaceous hyperplasia, basal cell carcinoma, and sometimes in viral blisters such as in herpes simplex or herpes zoster.
- *Varicelliform:* To resemble chickenpox such as in smallpox, chickenpox, herpes zoster, Kaposi's varicelliform eruption, or pityriasis lichenoides et varioliformis of Muccha and Habermann.
- *Varioliformis:* to resemble smallpox as in pityriasis lichenoides et varioliformis acuta or smallpox.
- *Verrucous:* wartlike in appearance such as in a verrucous keratoacanthoma, squamous cell carcinoma, or wart.
- *Zosteriform:* refers to the distribution of cutaneous disease in a nerve root distribution. This is seen in herpes zoster, some epidermal and other hamartomatous nevi, and occasionally vitiligo, lichen planus, and others.

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CHAPTER 4

Dermatologic Therapy

John C. Hall, MD

Many hundreds of medications are available for use in treating skin diseases. Most physicians, however, have a few favorite prescriptions that they prescribe day in and day out. These few prescriptions may then be altered slightly to suit an individual patient or disease. Prescription pads printed with commonly used preparations can help save the clinician time and are always legible for the patient. Prescription pads that cannot be photocopied are mandatory.

Treatment of most of the common skin conditions is simpler to understand when the physician is aware of three basic principles:

1. The first principle is to treat the skin lesion by its *type of skin lesion,* more than the cause, influences the kind of local medication used. The old adage "If it's wet, dry it with a wet dressing, and if it's dry, wet it with an ointment" is true in most cases. For example, to treat a patient with an acute oozing, crusting dermatitis of the dorsum of the hand, whether due to poison ivy or soap, the physician should prescribe wet soaks. For a chronic-looking, dry, scaly patch of psoriasis on the elbow, an ointment is indicated because it holds moisture in the skin; an aqueous lotion or a wet dressing is more drying. Bear in mind, however, that the type of skin lesion can change rapidly under treatment. The patient must be followed closely after beginning therapy. An acute oozing dermatitis treated with water soaks can change, in 2 or 3 days, to a dry, scaly lesion that requires an ointment. Conversely, a chronic dry patch may become irritated with greasy ointment and begin to ooze.
2. The second basic principle in treatment is *first do no harm* and never overtreat. It is important for the physician to know which of the chemicals prescribed for local use on the skin are the greatest irritants and sensitizers. It is no exaggeration to say that a commonly seen dermatitis is actually due to patient overtreatment before coming to the office *(overtreatment contact dermatitis)*. The patient, many times has gone to the neighborhood drugstore, or to a friend, and used any, and many, of the medications available for the treatment of skin diseases. It is certainly not unusual to hear the patient tell of using an athlete's foot salve for the treatment of the lesions of pityriasis rosea.
3. The third principle is to *instruct the patient adequately regarding the application of the medicine prescribed.* The patient does not have to be told how to swallow a pill, but does have to be told how to put on a wet dressing. Most patients with skin disorders are ambulatory, so there is no nurse to help them; they are their own nurses. The success or the failure of therapy rests on adequate instruction of the patient or person responsible for the care. Even in hospitals, particularly when wet dressings or aqueous lotions are prescribed, it is wise for the physician to instruct the nurse regarding the procedure.

With these principles of management in mind, let us now turn to the medicine used. It is important to stress that we are endeavoring to present here only the most basic material necessary to treat most skin diseases. For instance, there are many solutions for wet dressings, but Domeboro solution is our preference. Other physicians have preferences different

SAUER'S NOTES

SKIN DISEASES ASSOCIATED WITH SMOKING (THERAPY IS QUITTING)

1. Smoker's wrinkles—deep facial wrinkles
2. Poor wound healing—especially for flaps and grafts
3. Psoriasis—especially associated with pustular psoriasis
4. Severity of skin cancer—increased risk of basal cancers becoming morpheaform
5. Atopic dermatitis in children whose mothers smoke
6. Arteriosclerotic vascular disease—Buerger's disease, ischemic leg ulcers
7. Leukoplakia and squamous cell cancer of the lip and oral mucosa
8. Condyloma and cervical cancer
9. Increased severity of Raynaud's phenomena and ischemic ulcers
10. Increased neuropathy (especially in diabetics) with mal perforans ulcers
11. Embolic phenomena—blue toes, livedo reticularis, necrosis with ulcers
12. Decreased effectiveness of antimalarials for cutaneous lupus erythematosus
13. Crohn's disease (15% with associated skin disease) incidence and activity
14. Increased nonlymphocytic leukemia systemically including skin
15. Possibly less incidence of aphthous ulcers and acne

from the drugs listed and their choices are respected, but to list all of them does not serve the purpose of this book.

Two factors have guided us in the selection of medications presented in this formulary. First, the medication must be readily available in most drugstores; second, it must be a very effective medication for one or several skin conditions. The medications listed in this formulary also are listed in a complete way in the treatment section for the particular disease. Instructions for more complete use of the medications, however, are as described in this formulary.

Formulary

A particular topical medication is prescribed to produce a specific beneficial effect.

SAUER'S NOTES

LOCAL THERAPY

1. The type of skin lesion (oozing, infected, or dry), more than the cause, should determine the local medication that is prescribed.
2. Do no harm. Begin local therapy for a particular case with mild drugs. The strength of the treatment can be increased if the condition worsens.
3. Do not begin local corticosteroid therapy with the "biggest gun" available, particularly for chronic dermatoses.
4. Carefully instruct the patient or nurse regarding the local application of salves, lotions, wet dressings, and baths. Thin coats of topical medications save money and are as effective as thick coats. Numbers of applications are more important than how thickly the medication is put on. Also, applying medications after hydration such as baths, showers, and hand washing increases penetration and makes topical therapy more effective. Effectiveness can also be increased by occlusion with Saran wrap or occlusions with cotton socks or cotton dermal gloves.
5. Prescribe the correct amount of medication for the area and the dermatosis to be treated. This knowledge comes with experience.
6. Change the therapy as the response indicates. If a new prescription is indicated and the patient has some of the first prescription left, instruct the patient to alternate using the old and new prescriptions.
7. If a prescription is going to be relatively expensive, explain this fact to the patient.
8. For many diseases, "therapy plus" is indicated. Advise the patient to continue to treat the skin problem for a specified period after the dermatosis has apparently cleared. This may prevent or slow down recurrences.
9. Instruct the patient to telephone you, your nurse, nurse practitioner, medical assistant, or physician assistant if there are any questions or if the medicine appears to irritate the dermatosis.

Effects of Locally Applied Drugs

Anesthetic agents are used in the skin to decrease pain when injections, laser, cryotherapy, electrolysis, excisions, or other procedures are performed. These include lidocaine hydrochloride 3% cream (LidaMantle), 30% to 40% lidocaine compounded in Velvachol or Acid Mantle cream, EMLA cream or disc (2.5% lidocaine and 2.5% prilocaine), and ethyl chloride spray. Anesthetic agents for mucous membranes are used to temporarily ameliorate discomfort from mucous membrane diseases. They include viscous solution of lidocaine (2%) and Hurricaine liquid or gel spray (20% benzocaine); for ophthalmic use, Alcaine solution (0.5% proparacaine) and Pontocaine (0.5% tetracaine) are used.

Antipruritic agents relieve itching in various ways. Commonly used chemicals include menthol (0.25%), phenol (0.5%), camphor (2%), pramoxine hydrochloride (1%), sulfur (2% to 5%), and coal tar solution (liquor carbonis detergens [LCD]) (2% to 10%). These chemicals are added to various bases for the desired effect. Numerous safe and unsafe proprietary preparations for relief of itching are also available. The unsafe preparations are those that contain sensitizing antihistamines, benzocaine, and related—*caine* derivatives. Itch-X gel and spray is over the counter (OTC) and contains 1% pramoxine hydrochloride and 10% benzyl peroxide (Itch-X lotion is OTC, 1% hydrocortisone).

Keratoplastic agents tend to increase the thickness of the horny layer. Salicylic acid (1% to 2%) is an example of a keratoplastic agent that will thicken the horny layer.

Keratolytics remove or soften the horny layer. Commonly used agents of this type include salicylic acid (4% [Salex lotion and cream] to 10%), resorcinol (2% to 4%), urea (20% to 50%), and sulfur (4% to 10%). A strong destructive agent is trichloroacetic acid. Urea in 5% to 10% concentration (Eucerin Plus lotion and Carmol) is moisturizing, whereas in 20% to 50% (Vanamide, Keralac, Carmol) concentration, it is keratolytic. Urea is also available in a nail stick applicator, Kerastick (50%), for onychoschizia and in a Redi-Cloth (Kerol [42%]). α-Hydroxy acids (lactic acid [Lac-Hydrin 5% or 12% cream and lotion or AmLactin and AmLactin XL 12% cream or lotion]), which are sold over the counter, and glycolic acid (Aqua Glycol is sold OTC in various concentrations and is available as facial cleanser, toner, face cream, shampoo, body cleanser, hand lotion, and body lotion) in 5% to 12% concentrations are moisturizers, whereas in higher concentrations up to 80%, are keratolytic and can be used in the office for facial peeling, with caution. Some moisturizers combine ureas and α-hydroxy acids such as U-Kera E (40% urea, 2% glycolic acid) and Eucerin Plus. Kerol Topical Suspension (50% urea with lactic acid and salicylic acid) is keratolytic and can be massaged into callosities for 60 seconds after bath or shower.

Antieczematous agents remove oozing and vesicular excretions by various actions. Soaks for 10 minutes twice a day or clean towels soaked in a solution for 10 minutes twice a day are very effective. The commonest agents include water soaks or compresses (lukewarm to cool), Domeboro solution

packets or dissolvable tablets that are nonprescription, coal tar solution (2% to 5%), hydrocortisone 0.5% to 2% (0.5% and 1% are available without a prescription), and more potent corticosteroid derivatives incorporated in solutions, foams, and creams.

Antiparasitic agents destroy or inhibit living infestations. Examples include permethrin (Elimite or Acticin) cream for scabies, γ-benzene hexachloride (Kwell) cream and lotion for scabies and pediculosis, crotamiton (Eurax) for scabies, and permethrin (Nix) for pediculosis. For scabies and lice, 10% sulfur can be mixed in petrolatum and is effective and very safe, even in infants and pregnant women, but is malodorous and stains.

Antiseptics destroy or inhibit bacteria, fungi, and viruses. Alcohol hand sanitizers are effective on hands and clorox-containing cleansers are very effective on inanimate fomites such as counters, floors, exam tables, and so on.

Antibacterial topical medications include gentamicin (Garamycin), retapamulin ointment (Altabax), mupirocin (Bactroban), bacitracin (recently found to cause a significant number of cases of contact dermatitis), Polysporin, and neomycin (Neosporin), which causes an appreciable (at least 1%) incidence of allergic contact sensitivity. Soaps, such as Lever 2000 and Cetaphil antibacterial soap, can have extra antibacterial additives.

Antifungal and anticandidal topical agents include miconazole (Micatin, Monistat-Derm), clotrimazole (Lotrimin, Mycelex), ciclopirox (Loprox), econazole (Spectazole), oxiconazole (Oxistat), naftifine (Naftin), ketoconazole (Nizoral), butenafine hydrochloride (Mentax, Lotrimin Ultra), and terbinafine (Lamisil). Sulfur (3% to 10%) is an older but effective antifungal and anticandida agent. Nystatin is anticandida but not antifungal.

Antiviral topical agents are acyclovir (Zovirax) ointment or cream and penciclovir (Denavir).

Emollients soften and moisturize the skin surface. Nivea oil, mineral oil, and white petrolatum are good examples. Newer emollients are more cosmetically elegant and effective.

Ointments moisturize the skin. Examples include Vaseline Petroleum Jelly, Lanolin, Aquaphor, Cetaphil, and Eucerin.

Creams dry the skin but are more cosmetically acceptable than ointments because they do not feel greasy and do not leave oil marks on paper products. Examples are Dermovan and Acid Mantle cream. Newer moisturizers attempt to restore the normal skin barrier for protection and to increase penetration of other topicals applied on top of these agents. Three examples are Mimyx, Atopiclair, and CeraVe.

Types of Topical Dermatologic Medications

Baths

1. Tar bath

   Coal tar solution (USP, LCD) 120.0 ml
   Or Cutar bath oil
   Sig: Add 2 tbsp to a tub of lukewarm water, 6- to 8-in deep.
   Actions: Antipruritic and antieczematous

2. Starch bath

   Limit or Argo starch, small box
   Sig: Add half box of starch to a tub of cool water, 6- to 8-in deep.
   Actions: Soothing; antieczematous and antipruritic
   Indications: Generalized itching and urticaria

3. Aveeno (regular and oilated) colloidal oatmeal bath

   Sig: Add 1 cup to the tub of water.
   Actions: Soothing and cleansing
   Indications: Oilated for generalized itching and dryness of skin, winter, and senile itch. Regular for oozing, draining, wet dermatitis.

4. Oil baths (see section on oils and emulsions) for dry skin.

5. Bleach baths and compresses. For baths, add 1 cup of bleach to full tub of water to soak for several minutes, and add 1 tablespoonful to 1 quart of water to use as compresses for several minutes b.i.d. to treat recurrent recalcitrant *Staphylococcus aureus* folliculitis and secondary infection in atopic dermatitis.

Soaps and Shampoos

1. Dove soaps, Neutrogena soaps, Cetaphil, Basis

   Action: Mild cleansing agents
   Indications: Dry skin or winter itch

2. Dial soap, Lever 2000, Cetaphil antibacterial soap

   Actions: Cleansing and antibacterial
   Indications: Acne, pyodermas

3. Capex shampoo 120.0
   Sig: Shampoo as needed.
   Actions: Anti-inflammatory, antipruritic, and cleansing
   Indications: Dandruff, psoriasis of scalp
   Comment: Contains fluocinolone acetonide, 0.01%

4. Selsun Suspension or Head and Shoulders Intensive Treatment shampoo 120.0

SAUER'S NOTES

LOCALLY APPLIED GENERIC PRODUCTS

Advantages: Lower cost—you can prescribe a larger quantity at relatively less expense, and patients appreciate your sharing their concern regarding cost.

Disadvantages: With a proprietary product, you are quite sure of the correct potency and bioavailability of the agent, and you know the delivery system and the ingredients in the base.

If you prescribe a proprietary medication when a less expensive generic is available, explain to the patient your reason for doing this.

Sig: Shampoo hair with two separate applications and rinses. You can leave the first application on the scalp for 5 minutes before rinsing off. Do not use another shampoo as a final cleanser. Contains selenium sulfide.
Actions: Cleansing and antiseborrheic
Indications: Dandruff, itching scalp (not toxic if used as directed but poisonous if swallowed, so keep out of reach of small children).

5. Tar shampoos: Tarsum (can be applied overnight or for several hours as a scalp oil and then shampooed out), Polytar, T/Gel (regular and maximum strength), Pentrax, Ionil T, and so on

Sig: Shampoo as necessary, even daily.
Actions: Cleansing and antiseborrheic
Indications: Dandruff, psoriasis, atopic eczema of the scalp

6. Nizoral shampoo 120.0 or Loprox shampoo

Sig: Shampoo two or three times a week.
Actions: Anticandidal and antiseborrheic
Indication: Dandruff, tinea versicolor, and tinea capitis infection
Comment: Nizoral is available as 1% OTC or as 2% with a prescription.
Loprox shampoo is similar, with ciclopirox as the active ingredient

7. T-Sal, Salex, and other salicylic acid shampoos
Indications: Psoriasis and seborrheic dermatitis

Wet Dressings or Soaks

1. Burow's solution, 1:20

Sig: Add 1 Domeboro tablet or packet to 1 pint of tap water. Cover affected area with sheeting wet with solution and tie on with gauze bandage or string. Do not allow any wet dressing to dry out. It can also be used as a solution for soaks.
Actions: Acidifying, antieczematous, and antiseptic
Indications: Oozing, vesicular skin conditions

2. Vinegar solution

Sig: Add ½ cup of white vinegar to 1 quart of water for wet dressings or soaks, as described for Burrow's solution.
Indications: Antieczematous, antiyeast, antifungal, antibacterial including antipseudomonas

3. Salt solution

Sig: Add 1 tbsp of salt to 1 quart of water for wet dressings or soaks, as above.
Indications: Antieczematous, cleansing

Powders

1. Purified talc (USP), ZeaSORB powder, or ZeaSORB-AF powder 60 (contains miconazole)

Sig: Dust on locally b.i.d. (supply in a powder can)
Actions: Absorbent, protective, and cooling
Indications: Intertrigo, diaper dermatitis

2. Tinactin powder, Micatin powder, ZeaSORB-AF powder, or Desenex powder

Sig: Dust on feet in the morning
Actions: Absorbent, antifungal, and antiyeast
Indications: Prevention and treatment of tinea pedis and tinea cruris as well as candida intertrigo
Comment: These powders are available OTC.

3. Mycostatin powder 15.0

Sig: Dust on locally b.i.d.
Action: Anticandida
Indication: Candida intertrigo

Shake Lotions

1. Calamine lotion (USP) 120

Sig: Apply locally to affected area t.i.d. with fingers or brush.
Actions: Antipruritic and antieczematous
Indications: Widespread, mildly oozing, inflamed dermatoses

2. Nonalcoholic white shake lotion

a. Zinc oxide 24.0
b. Talc 24.0
c. Glycerin 12.0
d. Distilled water q.s. ad 120.0

3. White shake lotion

a. Zinc oxide 24.0
b. Talc 24.0
c. Glycerin 12.0
d. Distilled water q.s. ad 120.0

4. Proprietary lotions

a. Sarna lotion (with menthol and camphor), Sarna for Sensitive Skin contains pramoxine
b. Cetaphil lotion
c. Aveeno anti-itch lotion (contains pramoxine)

Oils and Emulsions

1. Zinc oxide, 40%

Olive oil q.s. 120.0
Sig: Apply locally to affected area by hand or brush t.i.d.
Actions: Soothing, antipruritic, and astringent
Indications: Acute and subacute eczematous eruptions

SAUER'S NOTES

1. Shake lotions 1, 2, and 3 are listed for physicians who desire specially compounded lotions. One or two pharmacists near your office will be glad to compound them and keep them on hand.
2. To these lotions you can add sulfur, resorcinol, menthol, phenol, and so on, as indicated.

2. Bath oils

   Nivea skin oil, Alpha-Keri, Cutar bath oil (contains tar)
   Sig: Add 1 to 2 tbsp to a tub of water. *Caution:* Avoid slipping in tub.
   Actions: Emollient and lubricant
   Indications: Winter itch, dry skin, atopic eczema

3. Hand and body emulsions: A multitude of products are available OTC. Some have petrolatum or phospholipids (Moisturel), some have urea or α-hydroxy acids (Lac-Hydrin lotion 5% OTC, Lac-Hydrin cream and lotion 12% prescription, and AmLactin 12% cream and lotion OTC), Vaseline, (Curel) ceramides (CeraVe and EpiCeram Skin Body Emulsion), phospholipids (Moisturel), shea butter (Cetaphil hand cream), lanolin (Eucerin, Nivea, and Aquaphor), and some are lanolin free.

   Sig: Apply locally as necessary.
   Actions: Emollient and lubricant
   Indications: Dry skin, winter itch, atopic eczema

4. Scalp oil

   Derma-Smoothe/FS oil (fluocinolone acetonide 0.01%) 120.0
   Sig: Moisten scalp hair and apply lotion overnight; wear a plastic cap
   Indications: Scalp psoriasis, lichen simplex chronicus, severe seborrheic dermatitis

5. Baker's P and S Liquid (phenol and sodium chloride)

   Sig: Apply overnight under shower cap as needed for scaling
   Indications: Thick scaling psoriasis

Tinctures and Aqueous Solutions

1. Povidone–iodine (Betadine) solution (also in skin cleanser, shampoo, and ointment) 15

   Sig: Apply with swab t.i.d.
   Actions: Antibacterial, antifungal, and antiviral
   Indication: General antisepsis

2. Gentian violet solution

   Gentian violet, 1%
   Distilled water q.s. 30.0
   Sig: Apply with swab b.i.d.
   Actions: Antifungal and antibacterial
   Indications: Candidiasis, leg ulcers

3. Antifungal solutions

   a. Lotrimin, Mycelex, Loprox, Tinactin, Micatin, Monistat-Derm, and Lamisil spray, among others 30.0
   Sig: Apply locally b.i.d.

   b. Fungi-Nail 30.0
   Sig: Apply locally b.i.d.
   Comment: Contains resorcinol, salicylic acid, parachlorometaxylenol, and benzocaine in a base with acetic acid and alcohol

   c. Penlac nail lacquer (contains ciclopirox)
   Sig: Apply thin coat two times a week; contains ciclopirox

   d. Castellani's paint (can get as uncolored): Used for intertrigo

Pastes

1. Zinc oxide paste (USP)

   Sig: Apply locally b.i.d.
   Actions: Protective, absorbent, and astringent
   Indications: Localized crusted or scaly dermatoses

Creams and Ointments

A physician can write prescriptions for creams and ointments in two ways: (1) by prescribing proprietary creams and ointments already compounded by pharmaceutical companies or (2) by formulating one's own prescriptions by adding medications to certain bases, as indicated for the particular patient being treated. For the physician who uses the second method, two different types of bases are used:

1. *Water-washable cream bases:* These bases are pleasant for the patient to use, nongreasy, and almost always indicated when treating intertriginous and hairy areas. Their disadvantage is that they can be too drying. A number of medications, as specifically indicated, can be added to these bases (i.e., menthol, sulfur, tars, hydrocortisone, and triamcinolone).

   a. Unibase
   b. Vanicream
   c. Acid Mantle cream
   d. Dermovan
   e. Unscented cold cream (not water washable)

2. *Ointment bases:* These petroleum jelly–type bases are, and should be, the most useful in dermatology. Although not as pleasant for the patient to use as the cream bases, their greasy quality alleviates dryness, removes scales, and enables the medicaments to better penetrate skin lesions. Disadvantages are that they can flare or cause folliculitis, acne, or rosacea, and they are less cosmetically acceptable because of the greasy feel. Any local medicine can be incorporated into these bases.

   a. White petrolatum (USP)
   b. Zinc oxide ointment (USP), very protective
   c. Aquaphor (contains lanolin)
   d. Eucerin (contains lanolin)
   e. Moisturel (may sting when first applied)

SAUER'S NOTES

1. OTC 0.5% or 1.0% Cortaid has proved effective and well tolerated as an emergency nonprescription treatment.
2. Do not use group I topical agents for longer than 2 weeks or more than a 45 g tube per week. A rest period must follow for 2 weeks.
3. Do not overuse the more potent topical steroids because of possible side effects.

SAUER'S NOTES

COMPOUND PREPARATIONS

Compound proprietary preparations are frequently prescribed, particularly by family practice physicians and nondermatologic specialists. Physicians should know the ingredients in these compound preparations and should know the side effects. Here are some popular compounds:

Mycolog II cream: Contains Nystatin and triamcinolone. *Beware:* It is not beneficial for fungus (tinea) infections; the triamcinolone after long-term use can cause atrophy, striae, and telangiectasia of the skin, especially in intertriginous areas and on the face.

Lotrisone cream: Contains clotrimazole and betamethasone dipropionate. *Beware:* The betamethasone with long-term use can cause atrophy, striae, and dilated vessels, especially in intertriginous areas and on the face. It also can have significant enough absorption to cause systemic corticosteroid side effects.

Iodoquinol–hydrocortisone cream (Vytone): Contains iodoquin plus 1% hydrocortisone. *Beware:* The iodoquin causes a moderate yellow stain on skin and clothing.

Cortisporin ointment: Contains 1% hydrocortisone with neomycin, Polysporin, and bacitracin. *Beware:* Neomycin allergies can occur infrequently and bacitracin has now also become a significant allergen.

For the physician who wishes to prescribe ready-made, proprietary preparations, these are listed in groups:

3. *Antifungal ointments and creams:* Lotrimin cream, Lotrimin Ultra cream, Mycelex cream, Spectazole cream, Loprox cream, Tinactin cream, Lamisil cream, Oxistat cream, Naftin cream, Nizoral cream, Mentax cream, and others

   Action: Antifungal
4. *Antibiotic ointments and creams:* Bactroban ointment or Centany ointment (can get generic mupirocin) and cream, Altabax (retapamulin) ointment, gentamicin ointment and cream, Neosporin ointment, Mycitracin ointment, and Polysporin ointment (antibiotic solutions are discussed in Chapter 13 under Acne Treatment)
5. *Antiviral ointments for herpes simplex virus infections:* acyclovir ointment and penciclovir cream
6. *Corticosteroid ointments and creams*
   a. Hydrocortisone preparations (0.5% and 1% hydrocortisone creams and ointments are available OTC and generically)
      - Hytone 1% and 2.5% cream and ointment

   b. Desonide preparations (can be written generically)
      - Tridesilon cream and ointment
      - DesOwen cream and ointment

   c. Triamcinolone preparations (0.5%, 0.1%, 0.025%, 0.01%)
      - Kenalog ointment and cream
      - Aristocort ointment and creams
      - Also available generically

   d. Other fluorinated corticosteroid preparations (see Table 4-1 for a listing of these preparations, which are ranked according to potency)
7. *Corticosteroid antibiotic ointments and creams:* Cortisporin ointment
8. *Corticosteroid antifungal–antiyeast preparations:*
   a. Lotrisone (anticandidal and antifungal), contains betamethasone and clotrimazole
   b. Mycolog II cream and ointment (anticandida), contains triamcinolone and nystatin; generic available
9. *Antipruritic creams and lotions:*
   a. Eurax cream
   b. Sarna lotion
   c. Prax lotion
   d. PrameGel
   e. Doxepin (Zonalon) cream (may cause drowsiness)
   f. Aveeno anti-itch lotion
   g. Eucerin calming cream
   h. Eucerin anti-itch spray
10. *Retinoic acid products:*
    a. Retin-A cream (0.025%, 0.05%, 0.1%) and Retin-A gel (0.01% and 0.025%), Retin-A Micro (0.04% and 0.1%), Retin A Micro Pump (0.04% and 0.01%), and Renova (0.02%)

    Actions: Antiacne comedones and small pustules (especially the gel) and antiphotoaging

    Indications: Acne of comedonal and small pustular type; aging wrinkles on face; removal of mild actinic keratoses and prevention of actinic keratoses, and treatment of freckles, molluscum contagiosum, and flat warts

    b. Differin (adapalene gel and cream 0.1% and 0.03%)

    Action: Retinoic acid receptor binder

    Indications: Acne of comedonal and small pustular type

    c. Avita (tretinoin 0.025%) cream may be less drying

    Action: Antiacne

    Indications: Acne of comedonal and small pustular type

    d. Tazorac (tazarotene) 30 g 0.1% cream, 100 g 0.05% gel, and 100 g 0.1% gel

    Action: Used for treatment of acne, psoriasis, prevention and treatment of actinic keratoses, and prevention of skin cancer

    Comment: Expensive and may be irritating

    e. Avage (0.1% tazarotene) cream 30 g

    Action: Approved for acne and may be milder than the same concentration of Tazorac
11. *Miscellaneous creams, ointments, and gels:*
    a. MetroGel (metronidazole 0.75%) 15.0

    Noritate cream (metronidazole 1%) 30.0

    Indications: Rosacea, perioral dermatitis

    b. Dovonex ointment (also comes as cream and scalp solution) 30.0 or 100.0

    Action: Antipsoriatic

    Comment: Moderately expensive

TABLE 4-1 ■ Potency Ranking of Some Commonly Used Topical Corticosteroids*

Group I

Cordran tape
Diprolene AF cream 0.05%
Diprolene ointment 0.05%
Diprolene gel 0.05%
OLUX-E foam
Temovate cream 0.05%
Temovate ointment 0.05%
Temovate gel 0.05%
Temovate emollient 0.05%
Temovate solution 0.05%
Ultravate cream 0.05%
Ultravate ointment 0.05%
Vanos (fluocinonide) 0.1% cream

Group II

Cyclocort ointment 0.1%
Diprolene AF cream 0.05%
Diprosone ointment 0.05%
Halog cream 0.1%
Halog ointment 0.1%
Halog solution 0.1%
Halog-E cream 0.1%
Lidex cream 0.05%
Lidex gel 0.05%
Lidex ointment 0.05%
Lidex solution 0.05%
Maxiflor ointment 0.05%
Psorcon cream 0.05%
Psorcon ointment 0.05%
Topicort cream 0.25%
Topicort gel 0.05%
Topicort ointment 0.25%

Group III

Aristocort cream 0.5%
Aristocort ointment 0.5%
Aristocort A cream 0.5%
Aristocort A ointment 0.5%
Betamethasone ointment 0.1%
Cutivate ointment 0.005%
Cyclocort lotion 0.1%
Diprosone cream 0.05%
Elocon ointment 0.1%
Kenalog cream 0.5%
Kenalog ointment 0.5%
Maxiflor cream 0.05%
Topicort LP cream 0.5%

Group IV

Aristocort ointment 0.1%
Cordran ointment 0.05%
Cyclocort cream 0.1%
Desonide ointment 0.2%
Elocon cream 0.1%
Elocon lotion 0.1%
Fluocinolone ointment 0.025%
Fluocinolone cream 0.2%
Halog cream 0.025%
Halog ointment 0.025%
Kenalog cream 0.1%
Kenalog ointment 0.1%
Topicort LP cream

Group V

Aristocort cream 0.1%
Betamethasone valerate cream 0.1%
Betamethasone valerate lotion 0.1%
Cloderm cream 0.1%
Cordran cream 0.05%
Cordran lotion 0.5%
Cordran ointment 0.025%
Cutivate cream 0.1%
Dermatop cream 0.1%
DesOwen ointment 0.05%
Fluocinolone cream 0.025%
Kenalog cream 0.1%
Kenalog lotion 0.1%
Kenalog ointment 0.025%
Locoid cream 0.1%
Locoid ointment 0.1%
Tridesilon ointment 0.05%

Group VI

Aclovate cream 0.05%
Aclovate ointment 0.05%
Aristocort cream 0.1%
Betamethasone valerate 0.1%
DesOwen cream 0.05%
DesOwen ointment 0.05%
DesOwen lotion 0.05%
Fluocinolone cream 0.01%
Fluocinolone solution 0.01%
Kenalog cream 0.025%
Kenalog lotion 0.025%
Locoid solution 0.1%
Tridesilon cream 0.05%

Group VII

Epifoam 1.0%
Fluocinolone cream 1.0%, 2.5%
Hydrocortisone cream 1.0%, 2.5%
Hydrocortisone lotion 1.0%, 2.5%

*Group I is the superpotency category. Potency descends with each group, to group VII, which is the least potent (groups II and III are potent corticosteroids; IV and V are midstrength corticosteroids; VI and VII are mild corticosteroids). There is no significant difference between agents within groups II through VII. The compounds are arranged alphabetically within the groups. In group I, Temovate cream or ointment is most potent. *(Courtesy of the late Dr. Richard B. Stoughton and Dr. Roger C. Cornell.)*

c. Aczone (5% avlosulfone [dapsone])

Action: Antiacne and antirosacea
Comment: Some authors think a G6PD deficiency test should be done before therapy is initiated

12. *Scabicidal and pediculicidal preparations:*

a. Eurax cream and lotion (crotamiton)
Action: Scabicidal
Comment: It is antipruritic

b. Kwell (lindane) lotion and cream
Actions: Scabicidal and pediculicidal

c. Elimite or Acticin cream
Action: Scabicidal

d. Nix Crème rinse
Indications: Head lice, nits

e. Ovide (malathion) topical
Action: Pediculicidal
Indications: Head lice, nits

f. Ivermectin oral
Action: Scabicidal
Indications: Scabies

13. *Sunscreen creams and lotions:* Para-aminobenzoic acid (PABA) and its esters, such as octyl dimethyl PABA (padimate O), octocrylene, octyl salicylate, methyl anthranilate, avobenzone (Parsol 1789), cinnamates (octyl-methoxycinnamate), oxybenzone (benzophenone-3) are effective ultraviolet light absorbers. Zinc oxide and titanium oxide are light blockers. There are many products on the market. Any sunscreen with a sun protective factor (SPF) of 30 or above offers effective sun-damage protection against short-wavelength ultraviolet light or UVB (290 to 310 nm), if used correctly. There is no equivalent SPF number in the United States for long-wavelength ultraviolet light or UVA, which is also important in photoaging, development of skin precancers and skin cancers, lupus erythematosus, and porphyrias and is usually the most important wavelength for photoallergic reactions. Therefore, titanium dioxide, zinc oxide, or avobenzone (Parsol 1789) and probably the two best sunscreens, Antiehelios (Mexoryl SX[expensive] and Helioplex sufficiently screen out UVA and UVB in a possibly more cosmetically acceptable base.
Sig: Apply to exposed areas before going outside. This should be done at least a half hour in advance for the best effect. Reapplication is important if exposure to water or significant sweating occurs. After 1 hour, reapplication is advisable. Too thin of an application is a common mistake.
Action: Screening out ultraviolet rays
Indications: Polymorphous light eruption, photoaging, systemic and chronic lupus erythematosus, some cases of dermatomyositis, photoallergy from systemic or topical medications, some types of porphyria, and prevention of skin precancers and skin cancers, especially in light-complexioned people

14. *Antiyeast:* All products listed under *Antifungal* as well as products containing nystatin, which can be used orally, as a cream, as an ointment, with 0.1% triamcinolone (Mycolog II cream and ointment, which can be obtained generically), with various powders (ZeaSORB-AF, which also comes as a drying gel), and with any product which causes skin drying such as Domeboro compresses or ZeaSORB powder.

Aerosols and Foams

Various local medications have been incorporated in aerosol and foam-producing containers. These include corticosteroids (OLUX foam, LUXIQ foam), antibiotics (Evoclin foam), antiacne agents (Ovace foam), antirosacea agents (Ovace foam), antifungal agents (Lamisil spray), Retin A pump, antipruritic medicines (Eucerin spray), and so on. Clobex spray is a class 1 topical corticosteroid.

Kenalog spray (63-g can) and Diprosone aerosol are effective corticosteroid preparations for scalp psoriasis and seborrhea.

Triamcinolone (LUXIQ) and clobetasol (OLUX) are corticosteroid foams and Ovace foam is sodium sulfacetamide foam used for seborrhea, acne, and rosacea. Evoclin is erythromycin foam used to treat acne. Rogaine comes as a foam for hair loss and Verdeso (desonide 0.05%) is a class VI steroid now available as a foam.

Corticosteroid Medicated Tape

1. Cordran tape (also comes as a patch)

Indications: Small areas of psoriasis, neurodermatitis, lichen planus

Medicated Skin Patches

Several are available for transdermal delivery of such agents as nitroglycerin, EMLA patch, and lidocaine patch for topical anesthesia, nicotine antismoking patches, and hormones. More will be developed.

Imiquimod

Imiquimod (Aldara) is used topically for superficial basal cell cancers, actinic keratoses, cutaneous Kaposi's sarcoma, molluscum contagiosum, genital warts, and other warts under occlusion. It is being used experimentally for other skin diseases such as Bowen's disease, elastosis perforans serpiginosa, cutaneous leishmaniasis, alopecia areata, and lentigo maligna melanoma. Other indications may well become approved with more experience with this topical medication. See section on actinic keratosis therapy in Chapter 28 and wart therapy in Chapter 23.

Local Agents for Office Use

1. Podophyllum in compound tincture benzoin
Podophyllum resin (USP) 25%
Compound tincture benzoin q.s. ad 30.0

Sig: Apply small amount to warts with cotton-tipped applicator every 4 or 5 days until warts are gone.