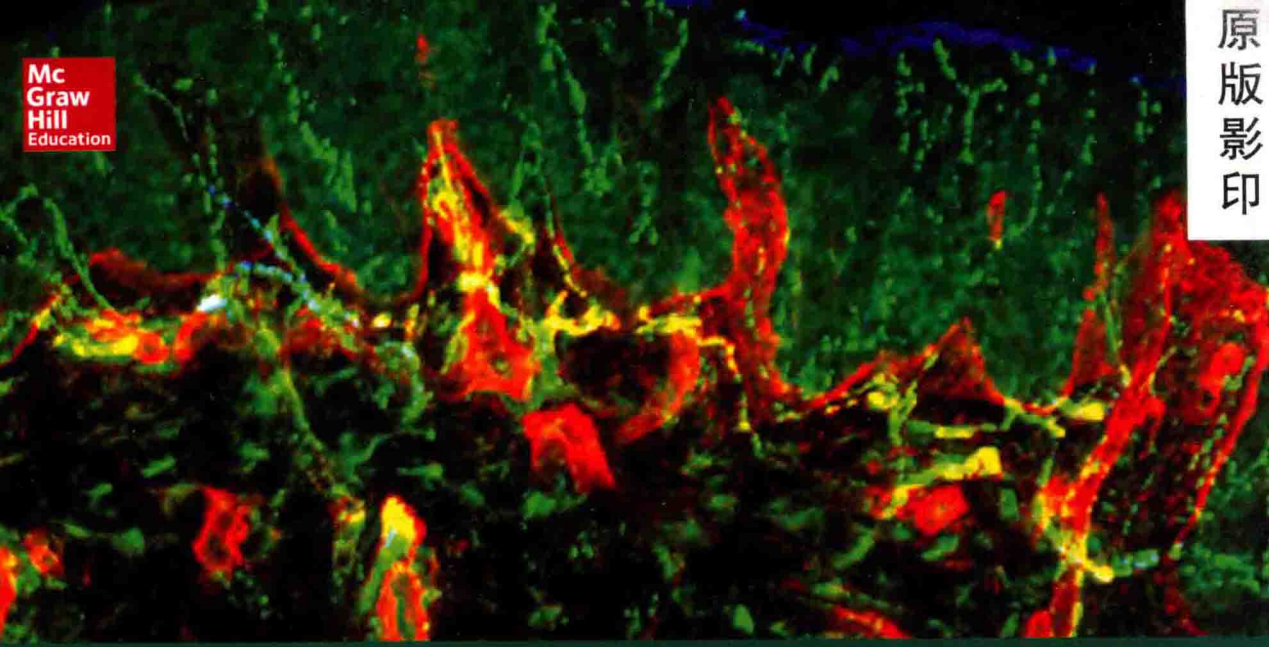


原版影印

临床皮肤病学

CLINICAL DERMATOLOGY

Carol Soutor • Maria Hordinsky

北京大学医学出版社

临床皮肤病学
Clinical Dermatology

Carol Soutor, MD
Clinical Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota

Maria K. Hordinsky, MD
Chair and Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota

北京大学医学出版社

图书在版编目（CIP）数据

临床皮肤病学：Clinical Dermatology：英文/（美）苏特（Soutor,C.），（美）霍登斯凯（Hordinsky,M.K.）编．—北京：北京大学医学出版社，2013.11
ISBN 978-7-5659-0701-2

Ⅰ．①临… Ⅱ．①苏… ②霍… Ⅲ．①皮肤病学－英文 Ⅳ．① R751

中国版本图书馆 CIP 数据核字（2013）第 268625 号

Carol Soutor, Maria K. Hordinsky
Clinical Dermatology
ISBN 978-0-07-176915-0

北京市版权局著作权合同登记号：01-2013-8434

临床皮肤病学

编　　写：Carol Soutor, Maria K. Hordinsky
出版发行：北京大学医学出版社（电话：010-82802230）
地　　址：（100191）北京市海淀区学院路38号　北京大学医学部院内
网　　址：http://www.pumpress.com.cn
E-mail：booksale@bjmu.edu.cn
印　　刷：北京画中画印刷有限公司
经　　销：新华书店
责任编辑：冯智勇　　**责任校对**：齐　欣　　**责任印制**：张京生
开　　本：787mm × 1092mm　1/16　　**印张**：22　　**字数**：574千字
版　　次：2014年1月第1版　2014年1月第1次印刷
书　　号：ISBN 978-7-5659-0701-2
定　　价：99.00元

Contributors

Rehana L. Ahmed, MD, PhD
Assistant Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 26

Bruce Bart, MD
Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 11, 12

Andrea Bershow, MD
Assistant Professor
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 10, 20

Kimberly Bohjanen, MD
Associate Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 1, 22

Caleb Creswell, MD
Clinical Assistant Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 14

Bart Endrizzi, MD, PhD
Assistant Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 7, 16

John Fenyk, MD
Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 24

Neal Foman, MD, MS
Clinical Associate Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 29

Noah Goldfarb, MD
Internal Medicine and Dermatology Resident
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 31, 32, 33, 34, 35, 36, and 37

Khaled M. Hassan, MD
Mohs Micrographic Surgery
Procedural Dermatology Fellow
Dermatologic SurgiCenter
Philadelphia, Pennsylvania
Chapter 40

H. Spencer Holmes, MD
Clinical Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 15

Kristen Hook, MD
Assistant Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 8, 27

Maria K. Hordinsky, MD
Chair and Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 19, 30

Laura E. Keck, MD
Dermatology Resident
Department of Dermatology
University of New Mexico School of Medicine
Albuquerque, New Mexico
Chapter 9

Phoebe Koch, MD
Park Nicollet Clinic
Dermatology Department
St. Louis Park, Minnesota
Chapter 39

Ioannis G. Koutlas, DDS, MS
Associate Professor
Division of Oral and Maxillofacial Pathology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 38

Peter K. Lee, MD, PhD
Associate Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 17

Steven W. Lin, MD
Dermatology Resident
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 31, 32, 33, 34, 35, 36, and 37

Sarah Nakib, MD, MS
Assistant Professor
Department of Dermatology
Johns Hopkins University
Baltimore, Maryland
Chapter 25

R. Steven Padilla, MD
Chair and Professor
Department of Dermatology
University of New Mexico School of Medicine
Albuquerque, New Mexico
Chapter 9

Amit G. Pandya, MD
Professor
Department of Dermatology
University of Texas Southwestern Medical Center
Dallas, Texas
Chapter 21

Scott Prawer, MD
Clinical Assistant Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 10, 12

Steven Prawer, MD
Clinical Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 10

Monica Rani, MD
Instructor
Departments of Dermatology and Medicine
Northwestern University
Feinberg School of Medicine
Chicago, Illinois
Chapter 25

April T. Sanchez, MD
Dermatology Resident
Department of Dermatology
University of Texas Southwestern Medical Center
Dallas, Texas
Chapter 21

Cindy Firkins Smith, MD
Clinical Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 13, 23

Carol Soutor, MD
Clinical Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapters 2, 3, 4, 5, and 6

David L. Swanson, MD
Associate Professor
Department of Dermatology
Mayo Clinic Scottsdale
Scottsdale, Arizona
Chapter 18

Erin Warshaw, MD, MS
Professor
Department of Dermatology
University of Minnesota Medical School
Minneapolis, Minnesota
Chapter 8

Barbara D. Wilson, MD
Associate Professor
Department of Dermatology
Medical College of Wisconsin
Milwaukee, Wisconsin
Chapter 28

Christopher B. Zachary, MBBS, FRCP
Chair and Professor
Department of Dermatology
University of California
Irvine, California
Chapters 40

Barrett Zlotoff, MD
Assistant Professor
Department of Dermatology
University of New Mexico School of Medicine
Albuquerque, New Mexico
Chapter 9

Preface

Clinical Dermatology is the product of decades of interaction with our primary care and dermatology colleagues and residents. It features concise, practical information on the diagnosis and management of common skin disorders. Diagnostic features, cost-effective management, evidence-based medicine, and patient-centered care are emphasized.

INTENDED AUDIENCE

Clinicians, residents, and medical students will find this textbook helpful in expanding their understanding and management of skin disorders. Our advisory group, consisting of primary care physicians, residents, nurse practitioners, physician assistants, and medical students, was instrumental in the design and review of this textbook.

ORGANIZATION AND CONTENT

Clinical Dermatology is divided into three sections.

- **Section One** covers the principles of diagnosis, management of common skin disorders, and diagnostic and surgical procedures.
- **Section Two** covers common skin disorders and selected less common disorders with high morbidity. The information on each disease is formatted into ten sections: introduction, pathogenesis, history, physical examination, laboratory findings, diagnosis, differential diagnosis, management, indications for consultation, and patient information. Evidence-based reviews and national and international guidelines are used when available in the management sections.
- **Section Three** focuses on the differential diagnosis of diseases in specific body regions based on history and physical examination. This section also includes the differential diagnosis of purpura, fever and rash, hospital-acquired rashes, pruritus, and skin ulcers. A chapter on cosmetic dermatology completes this section.

The online learning center (www.LangeClinicalDermatology.com) for this textbook contains multimedia presentations that complement and expand the content in the textbook. It contains the following.

- Videos with detailed demonstrations of common cutaneous diagnostic and surgical procedures.
- Clinical unknown cases with self-assessment questions that cover challenging diagnostic and management problems.
- PowerPoint presentations that cover the diagnosis, evaluation, and management of common skin disorders. These PowerPoints can be used for a rapid review of cutaneous disease or by educators for teaching.

ACKNOWLEDGMENTS

We would like to thank our editors, Anne Sydor and Christina M. Thomas, the production supervisor, Sherri Souffrance, and the project manager, Asheesh Ratra. We are grateful for contributions of photographs from Doctors Whitney Tope, Spencer Holmes, Bruce Bart, Richard Steinberg, Cindy Vehe, Dan Kaplan, Marna Ericson, Valda Kaye, Mark Wilke, Warren Goodman, David Ahrenholz, and the residents of the University of Minnesota dermatology residency programs. We thank our reviewers Jamie Santilli, MD, Assistant Professor, University of Minnesota Department of Family Medicine and Community Health; Catherine Manabat, MD, medicine/dermatology resident; Sanober Amin, MD, PhD, dermatology resident; Cynthia Olson, MD, Associate Professor, University of Minnesota Department of Dermatology; our medical student reviewers James Dorrian, Kathleen P. Lane, Matthew Majerus, Julie Fox, Jaime Loso, Maria Smith, and Heather Haberman PA-C and Victoria Eddy NP.

We want to especially acknowledge Robert W. Goltz, MD, who was our mentor and chairman of the Department of Dermatology at the University of Minnesota (1971 to 1985). His mastery of the art, science, and practice of medicine influenced all who were privileged to train with him.

We are especially grateful for our husbands' support and patience during this project.

Contents

1 Structure and Functions of the Skin

Kimberly Bohjanen

INTRODUCTION TO CHAPTER

The skin is the site of many complex and dynamic processes as demonstrated in Figure 1-1 and Table 1-1. These processes include barrier and immunologic functions, melanin production, vitamin D synthesis, sensation, temperature regulation, protection from trauma and aesthetics.

BARRIER FUNCTION

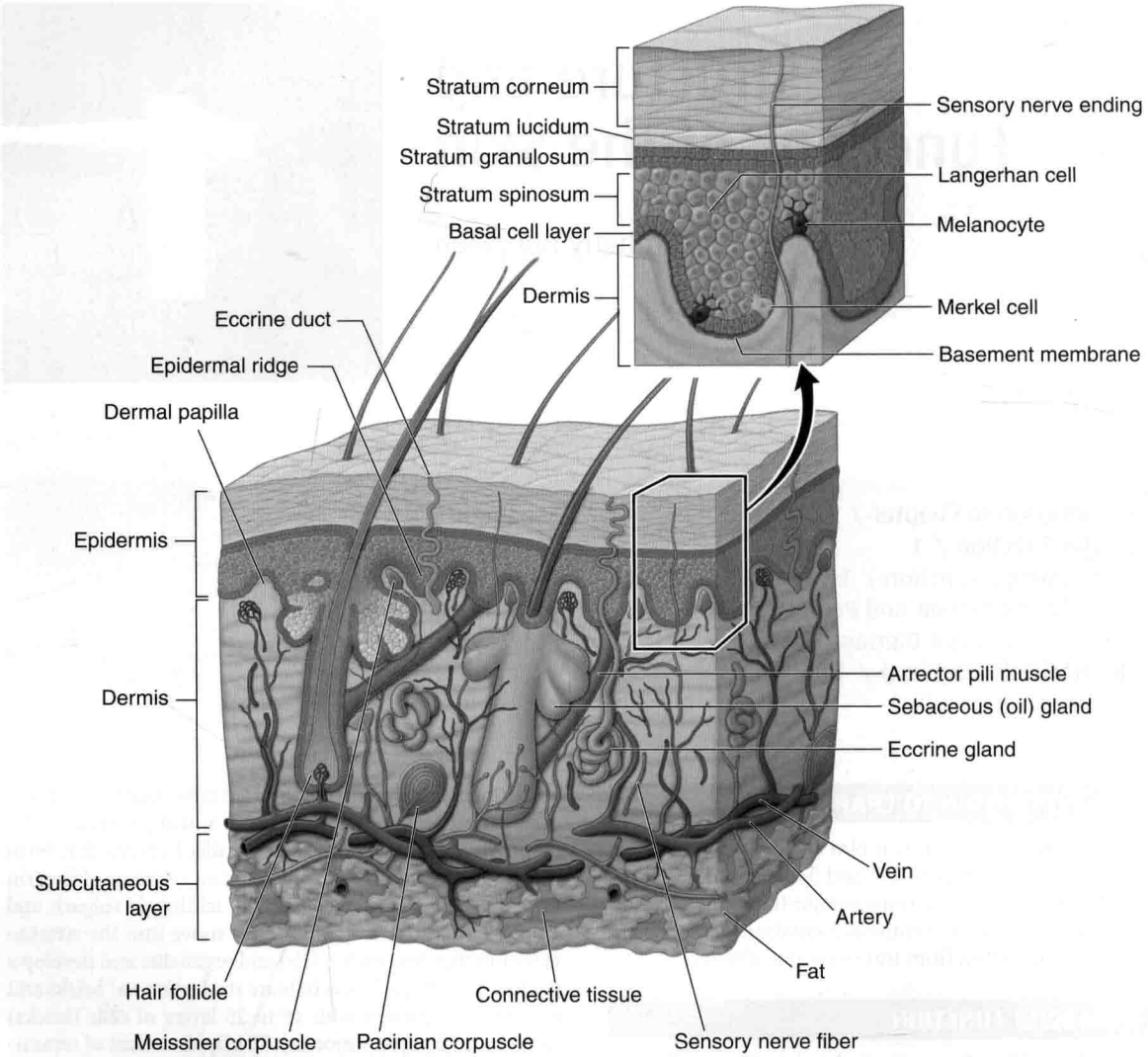
The epidermal barrier protects the skin from microbes, chemicals, physical trauma, and desiccation due to transepidermal water loss.[1–3] This barrier is created by differentiation of keratinocytes as they move from the basal cell layer to the stratum corneum. The keratinocytes of the epidermis are produced and renewed by stem cells in the basal layer resulting in replacement of the epidermis approximately every 28 days. It takes 14 days for these cells to reach the stratum corneum and another 14 days for the cells to desquamate.

Keratinocytes produce keratins, structural proteins that form filaments that are part of the keratinocyte cytoskeleton. In the stratum spinosum keratin filaments radiate outwards from the nucleus and connect with desmosomes which are prominent under the microscope giving a "spiny" appearance to cells. As cells move into the stratum granulosum, keratohyalin granules composed of keratin and profilaggrin are formed. Profilaggrin is converted into filaggrin (*fila*ment *aggr*egation prote*in*) that aggregates and aligns keratin filaments into tightly compressed parallel bundles that form the matrix for the cells of the stratum corneum. Filaggrin gene mutations are associated with ichthyosis vulgaris and atopic dermatitis. As keratinocytes move into the stratum corneum they lose their nuclei and organelles and develop a flat hexagon shape. These cells are stacked into a "bricks and mortar"–like pattern with 15 to 25 layers of cells (bricks) surrounded by lipids (mortar). The lipids consist of ceramides, free fatty acids, and cholesterol.

IMMUNOLOGIC FUNCTION

Epithelial cells at the interface between the skin and the environment provide the first line of defense via the innate immune system.[4–6] Epithelial cells are equipped to respond to the environment through a variety of structures including Toll-like receptors (TLRs) of which there are at least 10, nucleotide-binding oligomerization domain-like receptors, C-type lectins, and peptidoglycan recognition proteins. TLR-mediated activation of epithelial cells is also associated with the production of defensins and cathelicidins, families of antimicrobial peptides.

Dendritic cells bridge the gap between the innate and adaptive immune systems. Dermal dendritic cells can induce autoproliferation of T cells and production



▲ **Figure 1-1.** Cross-section of skin.

of cytokines as well as nitric oxide synthase. The exact function of dendritic epidermal Langerhans cells is an area of rapidly evolving research suggesting that these cells are very important to the modulation of the adaptive immune response.[7]

MELANIN PRODUCTION AND PROTECTION FROM ULTRAVIOLET LIGHT DAMAGE

Melanocytes comprise 10% of the cells in the basal cell layer.[8] There is another population of melanocytes in the hair follicle that is responsible for hair color and replacing epidermal melanocytes as needed (Figure 1-2). Melanocytes produce melanin, a pigmented polymer that absorbs UV light. Melanin is synthesized from tyrosine in several steps that require the enzyme tyrosinase. As melanin is produced it is then packaged into melanosomes, a specialized organelle. Melanosomes are phagocytosed by keratinocytes and moved to an area above the keratinocyte's nucleus acting as a protective shield from UV light. One melanocyte provides melanosomes for as many 30 to 40 keratinocytes. All humans have the same number of melanocytes. The variation in the degree of skin color is due to variations in melanosomes. Individuals with darker brown skin tones have

Table 1-1. Structure and function of the skin.

Component	Structure and Function
Stratum corneum	Semipermeable barrier with "bricks" (stacked cornified cells) and "mortar" (ceramides, cholesterol, and fatty acids) like construction
Stratum granulosum	Contains keratohyalin granules that produce profilaggrin
Stratum spinosum	Contains desmosomes for intercellular adhesion
Langerhans cells	Dendritic cells, important in the modulation of the adaptive immune response
Merkel cells	Specialized cells with neuroendocrine function
Melanocytes	Dendritic cells that produce melanin for ultraviolet light protection
Basal cell layer	Contains the stem cells that divide and produce the rest of the keratinocytes in the epidermis
Basement membrane	Interface between the epidermis and dermis
Ground substance	Amorphous gel of mucopolysaccharides that is the substrate for the dermis
Collagen	Network of fibrous proteins for skin tensile strength
Elastic fibers	Fibrous proteins responsible for skin elasticity
Fibroblasts	Cells that produce ground substance, collagen, and elastic fibers
Mast cells	Leukocytes that release histamine and heparin
Histiocytes/macrophages	Leukocytes that phagocytize and present antigen
Eccrine glands	Sweat glands that help with temperature regulation
Apocrine glands	Axillary and anogenital glands responsible for body odor
Sebaceous glands	Component of pilosebaceous unit that produces sebum
Hair follicle	Component of pilosebaceous unit that produces the hair fiber
Somatic sensory and sympathetic autonomic nerves	Supply blood vessels, glands, and hair follicles
Meissner corpuscles	Specialized nerve receptors for light touch
Pacinian corpuscles	Specialized nerve receptors for pressure and vibration
Blood vessels	Two horizontal plexies in the dermis that are connected and can shunt blood flow
Lymphatics	Parallel to blood vessels with 2 plexuses for flow of plasma
Fat	Provides protection from cold and trauma; Essential for storage of energy and metabolism of sex hormones and glucocorticoids

▲ **Figure 1-2.** Melanocytes in the basal cell layer and in the hair bulb region. Confocal image of nerves (aqua) and melanocytes (yellow) in the epidermis and the hair bulb region of a human anagen scalp hair follicle. Montage of 3 fields of view. Sample was immunostained with antibodies to a pan-neuronal marker PGP9.5 (aqua) and melanocytes (Mels-5) (yellow). (Reproduced with permission from Marna Ericson, PhD.)

more abundant, larger, and more dispersed melanosomes. Exposure to UV light stimulates the production of melanin within melanosomes producing a "tan." Tyrosinase deficiency is associated with albinism and vitiligo is associated with absence of melanocytes.

SYNTHESIS OF VITAMIN D

The main sources for vitamin D are dietary intake and production of vitamin D precursors by the skin. With exposure to UV light provitamin D_3 (7-dehydrocholesterol) in the epidermis is converted into previtamin D that converts into vitamin D_3. Vitamin D_3 is converted to its metabolically active form in the liver and kidneys.[8]

SENSATION

The skin is one of the principal sites of interaction with the environment and many types of stimuli are processed by the peripheral and central nervous systems.[9,10] Initially, cutaneous nerves were classified as being either "afferent" controlling sweat gland function and blood flow or "efferent" transmitting sensory signals to the central nervous system, but after the discovery of the neuropeptide substance P (SP) and other neuropeptides in sensory nerves, many trophic properties of nerve fibers and neuropeptides have been reported.

There are 3 major nerve types in the skin:

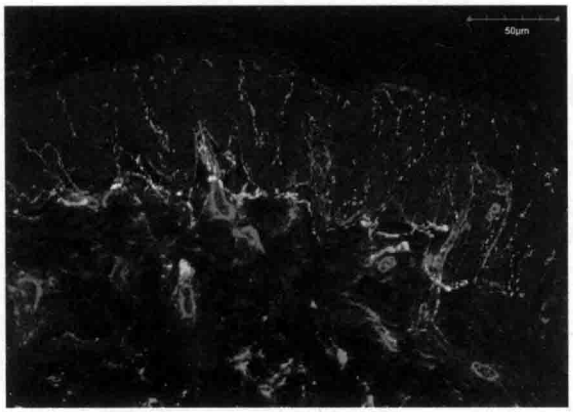
- Aβ fibers—large, heavily myelinated nerve fibers that transmit tactile sensation
- Aδ fibers—thinly myelinated nerve fibers involved in the transmission of short and fast painful stimuli
- C-fibers—unmyelinated nerves that transmit pain and itch sensations

Mixed nerve fiber bundles form a plexus from which individual nerve fibers extend toward their specific targets. The first tier is underneath the epidermis and innervates the epidermis and cutaneous mechanoreceptors or the upper dermis (Figure 1-3).

The second and third tiers are located between the dermis and subcutis or in the deep subcutis and innervate hair follicles, the arrector pili muscles, and sweat glands as well as the lower dermis and subcutis. All 3 plexi innervate blood vessels, smooth muscle cells, and mast cells, thereby connecting different skin cell populations with the brain.

TEMPERATURE REGULATION

The skin helps to regulate and maintain core body temperature through regulation of sweating and varying the blood flow in the skin. Evaporation of sweat contributes to temperature control of the body. Under normal conditions 900 mL of sweat is produced daily. With increased physical activity or increased environmental temperature, 1.4 to 3 L of sweat per hour can be produced.[11]



▲ **Figure 1-3.** Epidermal nerve fibers and blood vessels. Confocal image of epidermal nerve fibers (green), collagen type IV (red), and the neuropeptide calcitonin gene-related peptide (CGRP) (blue) in human scalp skin. The dermal/epidermal boundary and blood vessels are delineated by collagen type IV (red). Sample was immunostained with antibodies to protein gene product (PGP) 9.5 (green), collagen type IV (red), and CGRP (blue). (Reproduced with permission from Marna Ericson, PhD.)

The regulation of blood flow in the capillaries in the dermal papillae and other cutaneous vessels plays an important role in convective heat loss and heat conservation. Normally the blood flow in the skin is approximately 5% of the cardiac output, but in extremely cold temperatures it can drop to almost zero and in severe heat stress it can be as high as 60%.[12] Dysfunction of thermoregulation can lead to hyperthermia and hypothermia.

PROTECTION FROM TRAUMA

The dermis varies in thickness from 1 to 4 mm. It protects and cushions underlying structures from injury and provides support for blood vessels, nerves, and adnexal structures. It is separated from the epidermis by the basement membrane, which is created by the basal layer of the epidermis. Collagen is responsible for the tensile strength of the skin and comprises 75% of the dry weight of the dermis. Defects in collagen synthesis are associated with diseases such as Ehlers–Danlos syndrome (hyperextensible joints and skin). Elastic fibers are responsible for the elasticity and resilience of the skin and are 2% to 3% of the dry weight of the skin. Defects in elastic fibrils can be associated with cutis laxa and Marfan syndrome.

IDENTITY AND AESTHETICS

The perception of an individual's ethnicity, age, state of health, and attractiveness is affected by the appearance of his or her skin and hair. Sun-damaged skin, rashes, hair disorders, pigment disorders, and acne can have a profound effect on how individuals perceive themselves and others.

REFERENCES

1. Baroni A, Buommino E, De Gregorio V, Ruocco E, Ruocco V, Wolf R. Structure and function of the epidermis related to barrier properties. *Clin Dermatol.* 2012;30(3):257–262. PMID: 22507037.
2. Hwa C, Bauer EA, Cohen DE. Skin biology. *Dermatol Ther.* 2011;24(5):464–470. PMID: 22353152.
3. Brown SJ, McLean WH. One remarkable molecule: filaggrin. *J Invest Dermatol.* 2012;132(3):751–762. PMID: 22158554.
4. Gallo RL, Hooper LV. Epithelial antimicrobial defence of the skin and intestine. *Nat Rev Immunol.* 2012;12(7):503–516. PMID: 22728527.
5. Di Meglio P, Perera GK, Nestle FO. The multitasking organ: recent insights into skin immune function. *Immunity.* 2011;35(6):857–869. PMID: 22195743.
6. Nestle FO, Kaplan DH, Barker J. Mechanisms of disease: psoriasis. *N Engl J Med.* 2009;361(5):496–509. PMID: 19641206.
7. Kaplan DH. Langerhans cells: not your average dendritic cell. *Trends Immunol.* 2010;31(12):437. PMID: 21075684.
8. Brenner M, Hearing VJ. The protective role of melanin against UV damage in human skin. *Photochem Photobiol.* 2008;84(3):539–549. PMID: 18435612.
9. Peters EM, Ericson ME, Hosi J, et al. Neuropeptide control mechanisms in cutaneous biopsy: physiological mechanism and clinical significance. *J Invest Dermatol.* 2006;126(9):1937–1947. PMID: 16912691.
10. Davidson S, Giesler GJ. The multiple pathways for itch and their interactions with pain. *Trends Neurosci.* 2010;33(12):550–558. PMID: 21056479.
11. Shibasaki M, Wilson TE, Crandall CG. Neural control and mechanisms of eccrine sweating during heat stress and exercise. *J Appl Physiol.* 2006;100(5):1692–1701. PMID: 16614366.
12. Charkoudian N. Mechanisms and modifiers of reflex induced cutaneous vasodilation and vasoconstriction in humans. *J Appl Physiol.* 2010;109(4):1221–1228. PMID: 20448028.

2 Morphology and Terminology of Skin Lesions

Carol Soutor

INTRODUCTION TO CHAPTER

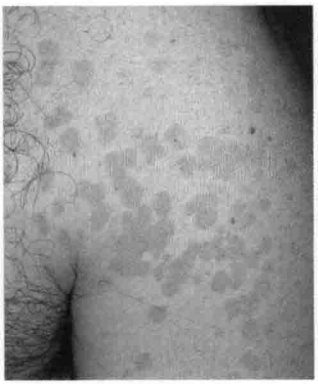
Identification and classification of a patient's skin lesions are important steps in the diagnosis of any skin disorder. The numerous descriptive terms used in dermatology can be overwhelming and at times confusing as there are some variations in the use and meaning of these words in the literature.[1] However, a few simple terms can be used to describe the cutaneous findings in most skin diseases. Using proper terminology to describe skin findings is essential for both documentation and communication with other clinicians. The effort to use precise descriptive terms also encourages a clinician to look with more care and more closely at a patient's skin lesions. The key features of skin lesions are (1) the type of lesion, (2) secondary changes to the surface of the lesion, (3) the color of the lesion, (4) the shape of the lesion, and (5) the arrangement and distribution of the lesions.

TYPES OF LESIONS

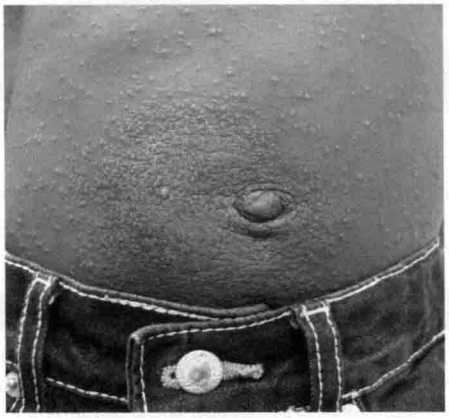
The first step is categorization of the primary skin lesion(s). This may be difficult if the lesions are excoriated or if the examination takes place late in the disease process. The lesion may need to be lightly touched or deeply palpated to accurately assess its features. Table 2-1 lists the 10 most

Table 2-1. Primary lesions and their morphology.

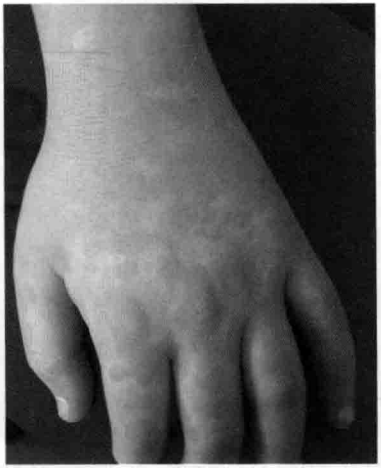
Terminology	Diameter	Morphology	Example
Macule Patch	<0.5 cm >0.5 cm	Flat, level with surface of skin	Tinea versicolor (Figure 2-1)
Papule Plaque	<0.5 cm >0.5 cm	Solid, elevated lesion	Dermatitis (Figure 2-2)
Wheal	Any size	White to pink edematous papule or plaque that lasts less than 24 h	Urticaria (Figure 2-3)
Nodule	>0.5 cm	Dermal or subcutaneous solid, elevated lesion	Amelanotic melanoma (Figure 2-4)
Vesicle Bulla	<0.5 cm >0.5 cm	Blister containing fluid or blood	Pemphigus vulgaris (Figure 2-5)
Pustule	<0.5 cm	Cavity filled with pus, may be sterile	Pustular psoriasis (Figure 2-6)
Cyst	>0.5 cm	Cavity filled with pus or keratin	Epidermal cyst (Figure 2-7)



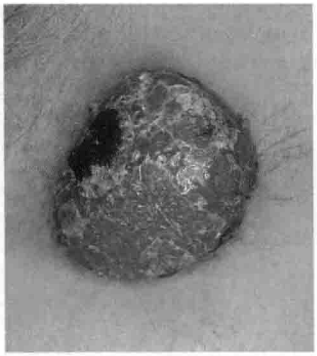
▲ **Figure 2-1.** Macules and patches. Tinea versicolor.



▲ **Figure 2-2.** Papules and a plaque. Contact dermatitis due to nickel in metal button in a child with atopic dermatitis.

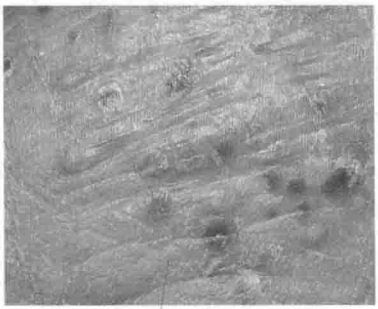


▲ **Figure 2-3.** Wheal. Urticaria.

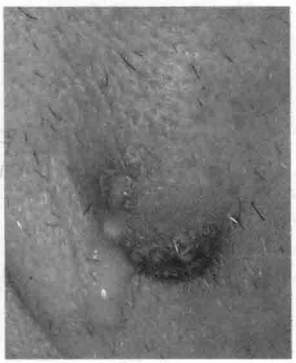


▲ **Figure 2-4.** Nodule. Nodular amelanotic melanoma.

▲ **Figure 2-5.** Vesicle and bulla. Pemphigus vulgaris.



▲ **Figure 2-6.** Pustules. Pustular psoriasis.



▲ **Figure 2-7.** Cyst. Staphylococcal boil.

Table 2-2. Examples of surface changes in skin lesions.

Terminology	Surface Changes in Lesions	Example
Scale	Loose or adherent flake composed of stratum corneum cells. The term hyperkeratotic is used for small areas of thick adherent scale	Psoriasis (Figure 2-8)
Crust	Yellow, brown, black, or green surface deposits of serum, pus, and/or blood	Pemphigus vulgaris (Figure 2-9)
Lichenification	Thickening of the epidermis with accentuation of skin markings	Atopic dermatitis (Figure 2-10)
Fissure	Linear, sharply defined, deep crack in the skin	Callous (Figure 2-11)
Erosion Excoriation	Localized loss of the superficial epidermis Linear or punctate, superficial, erosions in the skin caused by fingernails and sharp objects	Drug rash (Figure 2-12)
Ulcer	Defect in epidermis and dermis due to loss of tissue	Pyoderma gangrenosum (Figure 2-13)
Eschar	Black, hard crust resulting from tissue necrosis of the epidermis and/or dermis	Self-induced injury (Figure 2-14)
Atrophy	Depression and/or surface change in skin as the result of diminution of a component(s) of the epidermis, dermis, or fat	Lichen sclerosis (Figure 2-15)
Scar	Depressed or elevated proliferation of connective tissue that has replaced inflamed or traumatized skin	Depressed scar (Figure 2-16) Hypertrophic scar (Figure 2-17)

common morphological terms for types of skin lesions. These are based on:

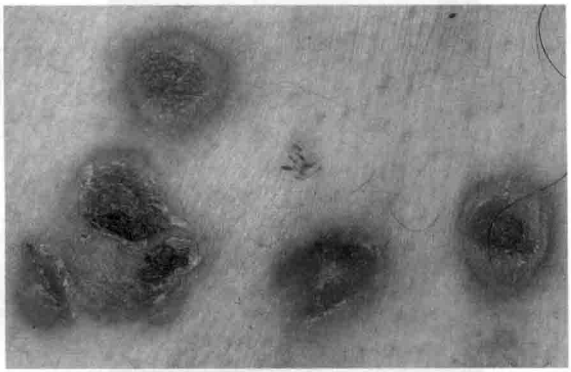
- Diameter of the lesion.
- Relationship of the lesion to the surface of the skin—is the lesion flat or elevated above the surface of the skin?
- Composition of the lesion—is it fluid filled or solid?

Most textbooks use a lesion diameter of either 0.5 or 1 cm to distinguish between various lesion types. This textbook uses 0.5 cm. It is not uncommon for a skin disease to have multiple types of lesions. Therefore, terms such as maculopapular or vesiculobullous are commonly used.

SURFACE CHANGES

Some lesions have a smooth surface, but surface changes frequently quickly develop during the course of a skin disorder. Table 2-2 lists common surface changes. Papulosquamous is a term used to describe papules/plaques that have scale.

▲ **Figure 2-8.** Scale. Psoriasis.



▲ **Figure 2-9.** Crust on collapsed bullae of pemphigus vulgaris.