Robert G. Micheletti
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Patrick J. McMahon

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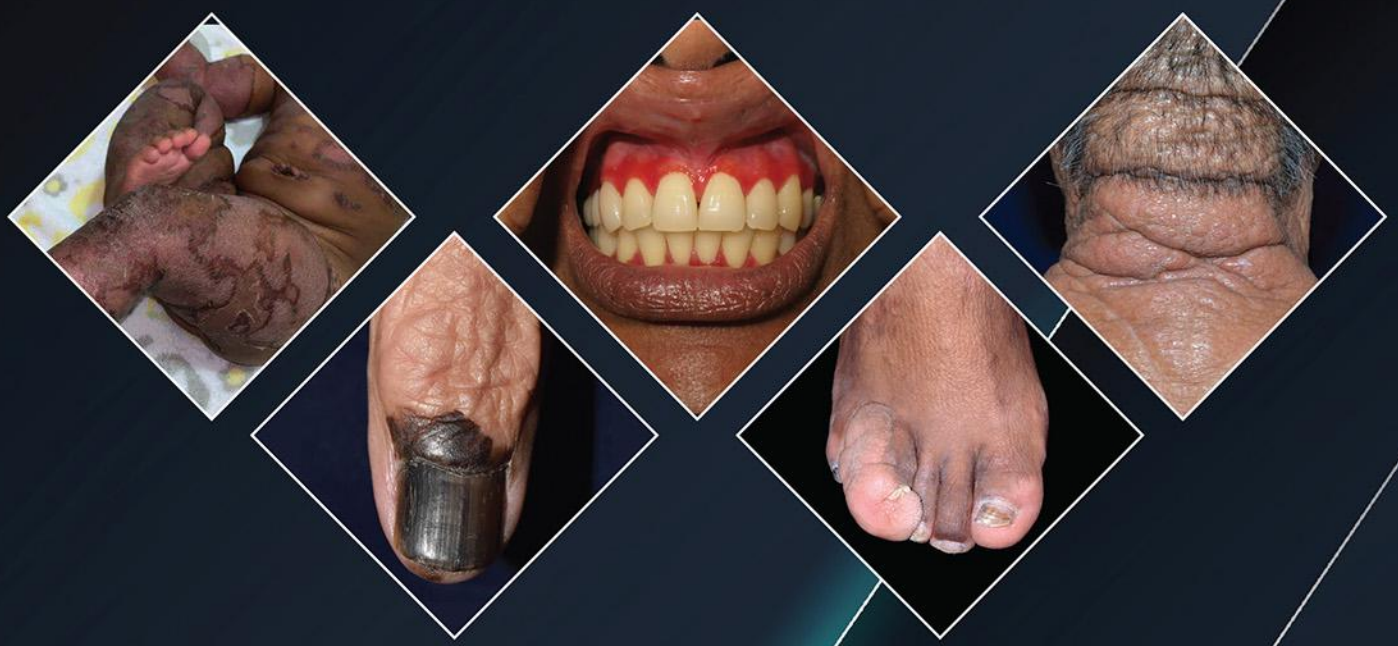
Andrews' DISEASES of the SKIN CLINICAL ATLAS



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Andrews' Diseases of the Skin
Clinical Atlas

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Andrews' Diseases *of the* Skin

CLINICAL ATLAS

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To my loving family:
My wife Ann, son Dan, daughter Becca, daughter-in-law Wynn, grandsons Declan and Driscoll, and sister Judy and her husband Cal.
You have given me a wonderful life!
–Bill James

Dorothy, Andrew, and Elisa: I am so proud of you all. You are a true source of love, joy, and inspiration.
To my dad Gil, a dermatologist: thank you for setting me on this path.
–Robert Micheletti

To my family—you are my greatest joy—and to my teachers and students, past and present, who have made every day a pleasure.
–Dirk Elston

Thank you to my wonderful wife Kate and loving children Bridget, Brendan, Colin, and Molly for their support and for being up for any adventure.
–Pat McMahon

PREFACE

It is our pleasure to bring you this atlas of skin disorders. We are hopeful that by seeing this wide array of images, you may more easily recognize and diagnose skin conditions in your own patients. If our efforts result in a better outcome for one patient, then they were worthwhile. Our atlas is an accompanying volume to *Andrews' Diseases of the Skin.* The chapters and entities follow the organization of *Andrews',* making explanatory text unnecessary and allowing you to enjoy the superior photographs in an uncluttered fashion.

Dermatologic diagnostic abilities are learned through repetitive exposures to patient presentations. The present volume of over 3000 pictures, combined with the main textbook, will be an outstanding resource to learn the depth and breadth of our specialty.

The four authors have benefitted by spending a combined 60 years in academic medicine, taking photographs along the journey. Additionally, the resources of our institutions and many friends have allowed a stunning array and variety of presentations to be represented. Bill James recognizes Richard Odom, MD, my teaching chief, who allowed myself and fellow resident Robert Horn to make copies of his best slides. Bill also thanks past residents and faculty of the Walter Reed Army Medical Center. Tim Berger has shared photographs from his experiences at the University of California at San Francisco. Bill and Rob both wish to thank the faculty and residents at the University of Pennsylvania, who generously shared their expertise. Dirk thanks the members of Brooke Army Medical Center who have contributed to the image collection in San Antonio. Pat wishes to recognize that the considerable resources of Paul Honig, MD's personal collection, as well as the combined image database of the current faculty at Children's Hospital of Philadelphia, were made fully available. These photos of pediatric patients enhance the atlas considerably. Finally, James Fitzpatrick, MD, also generously shared photographs taken by the faculty and staff of Fitzsimmons Army Medical Center. We call out all of these wonderful people, as they are not recognized individually in most of the photographs. In some cases when a photograph was obtained from a specific faculty member or resident, their names appear in a "Courtesy" line under the image.

You will find over 50 contributors individually recognized in the figure legends. These range from institutions in Taiwan, Brazil, and Japan, to individuals in Singapore, India, Thailand, Portugal, and the Philippines, to the National Institutes of Health and faculty from a variety of academic centers around the country. Several individuals deserve special mention: Steven Binnick, MD, is a superb dermatologist from Plymouth Meeting, Pennsylvania. When asked if he would make available some of his excellent photographs for this atlas, he simply donated his collection and was happy they would be used to educate others. Curt Samlaska, MD, trained at Walter Reed with Bill. He is an outstanding dermatologic photographer and contributed many photos of his patients from Hawaii and his practice in Henderson, Nevada. Doctors Shyam Verma and Archana Singal, close friends from India, shared many of their wonderful images. Dr. Leonard Swinyer took many outstanding photographs during his years in private practice. He gave his collection to the Dermatology Departments at the University of Utah and the Oregon Health Sciences Center. We thank all of them for their generosity.

This wide array of altruistic physicians and many others individually recognized in the atlas have allowed us to demonstrate entities rarely seen in the United States. More important, we had the full spectrum of patient ages, skin types, morphologies, classic examples, and disease subtypes available to us in choosing the best 3000+ images that make up this volume.

Charlotta Kryhl, Laura Schmidt, and Manchu Mohan from Elsevier assumed primary roles in the compilation of the book in a most expert and responsive manner.

No words of thanks could be complete without acknowledging the personal sacrifices of our families in allowing us the opportunity to pursue our professional dreams. Those include all of the individuals listed in the dedication.

Finally, to all of our past patients who generously allowed us to take their photographs, we hope this work helps fulfill your desire to aid future sufferers of skin disease recover more rapidly through earlier recognition and diagnosis of their conditions.

CONTENTS

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Structure and Function

1

The diagnosis of skin disease is based on color, morphology, and distribution of cutaneous lesions. The structure of the skin and associated appendages relates directly to these characteristics.

Folliculitis presents with papules or pustules. Follicular accentuation is characteristic of any eruption in darker-skinned races. In patients with miliaria, involvement of the sweat gland ostia results in erythematous papules, pustules, or superficial vesicles in areas of heavy sweating. The vesicles of miliaria crystallina are irregular in shape because the stratum corneum fails to impede the spread of the blister in random directions. This is in stark contrast to spongiotic and subepidermal blisters, which are distinctly round—as in acute dyshidrotic eczema or bullous pemphigoid.

The color of a cutaneous eruption relates to various pigments. Brown pigments include melanin, lipofuscin, and hemosiderin. Brown pigments located deeper in the dermis impart a blue hue because of diffraction of light. This is evident in blue nevi as a result of deep melanin and as a result of lipofuscin present in the sweat within nodular hidradenomas. Red pigment relates to oxygenated hemoglobin and blue to deoxygenated hemoglobin. Dilatation or proliferation of blood vessels and the rapidity of blood flow produce various shades of red and blue. Yellow pigments relate to lipid deposition or carotenoids dissolved in the cytoplasm of epithelial cells and histiocytes. In granulomatous disease, diascopy removes the visible appearance of oxygenated hemoglobin, allowing the observer to see the apple jelly yellow appearance of carotenoids within the cytoplasm. This section of the atlas will focus on the structure of the skin and how that structure translates to clinical manifestations of disease.

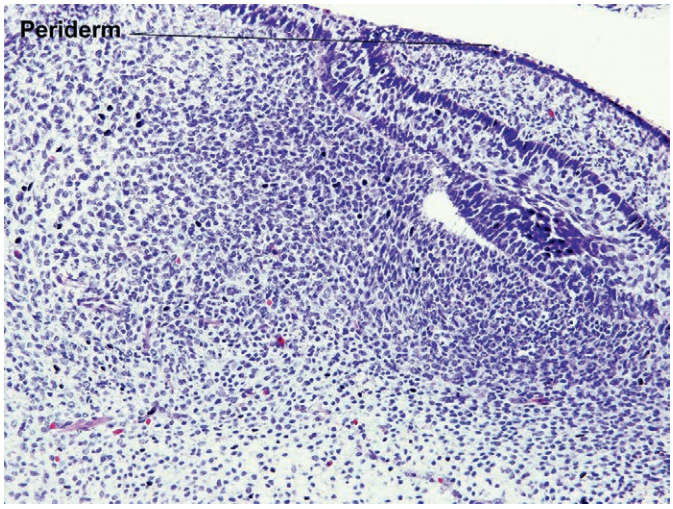

Fig. 1.1 In early fetal life, a cuboidal periderm is present rather than an epidermis. Fetal skin, H&E × 40.

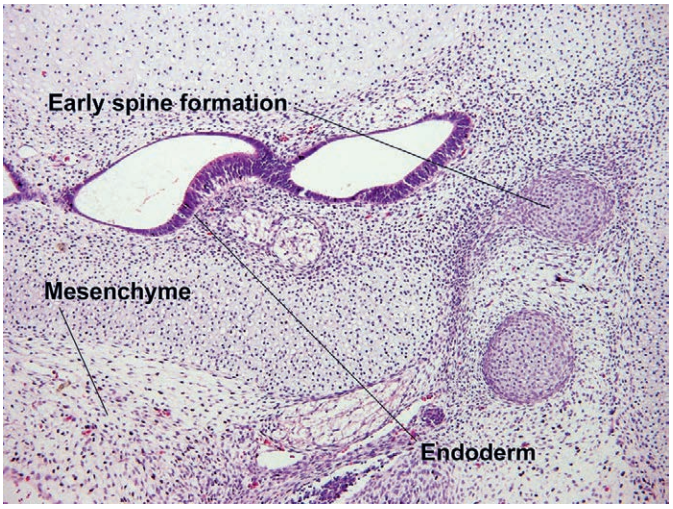

Fig. 1.2 In early fetal life, the spine is composed of cartilage, and mesenchyme is present rather than a dermis. Mesenchyme heals without scar formation. Once dermis forms, scars will occur after injury. Fetal skin, H&E × 40.

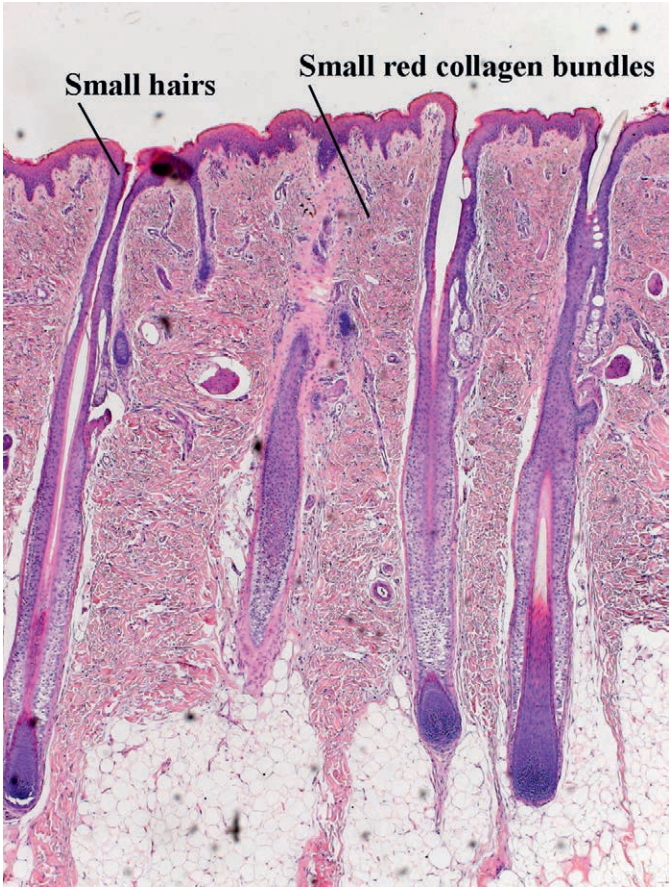

Fig. 1.3 Skin in young children is characterized by small adnexal structures and fine dermal collagen bundles that stain deep red in contrast to the thick, pink collagen bundles of an adult. Many plump fibroblasts are present in the dermis, actively synthesizing collagen. Childhood skin, H&E × 20.

Fig. 1.4 Facial skin is characterized by prominent sebaceous follicles, often containing *Demodex* mites. Facial skin, H&E × 40.

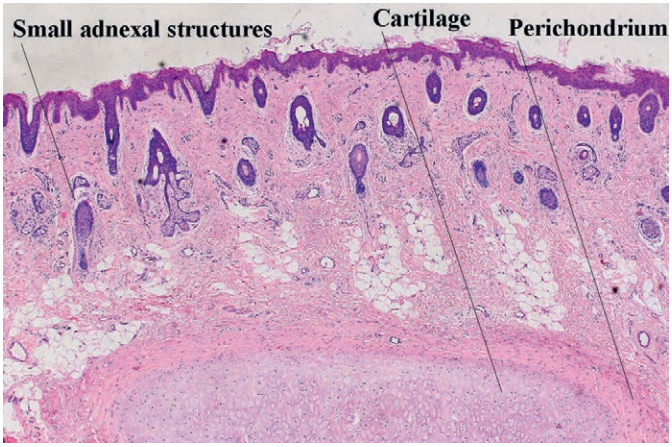

Fig. 1.5 Skin of the ear demonstrates small adnexal structures with an elastic cartilage surrounded by a red perichondrium. Ear skin, H&E × 20.

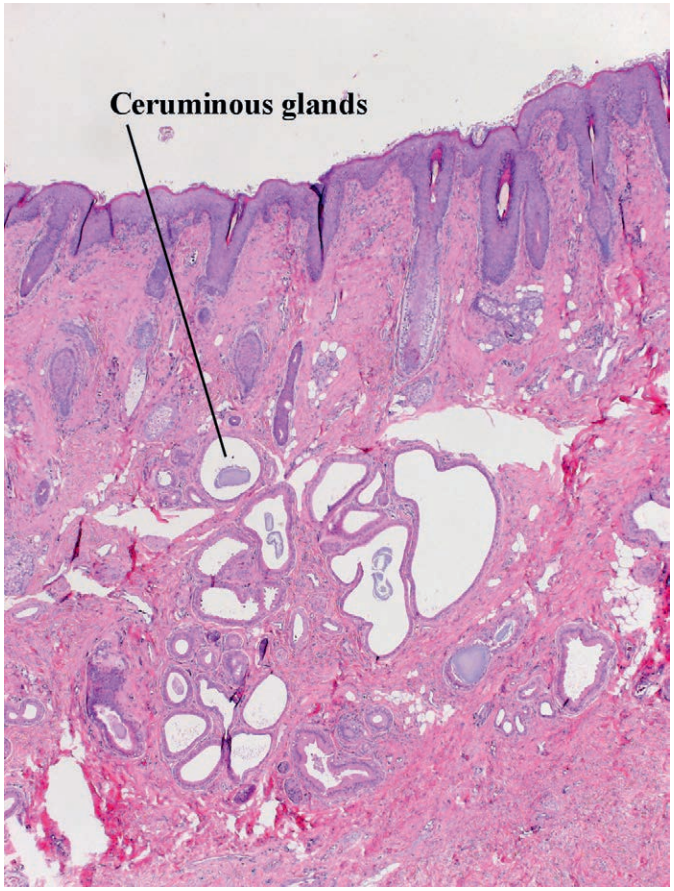

Fig. 1.6 The structure of the ear canal is similar to other parts of the ear, except for the presence of ceruminous glands, which represent modified apocrine glands. Ear canal skin, H&E × 20.

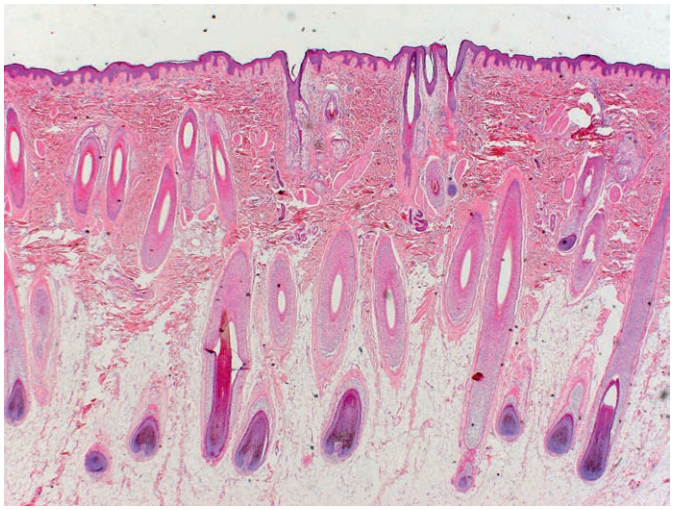

Fig. 1.7 Scalp skin demonstrates many terminal hair follicles. The inferior segment of each follicle sits within the subcutaneous fat. Scalp skin, H&E × 40.

Fig. 1.8 Axillary skin is rugose and demonstrates large apocrine glands. Axillary skin, H&E × 40.

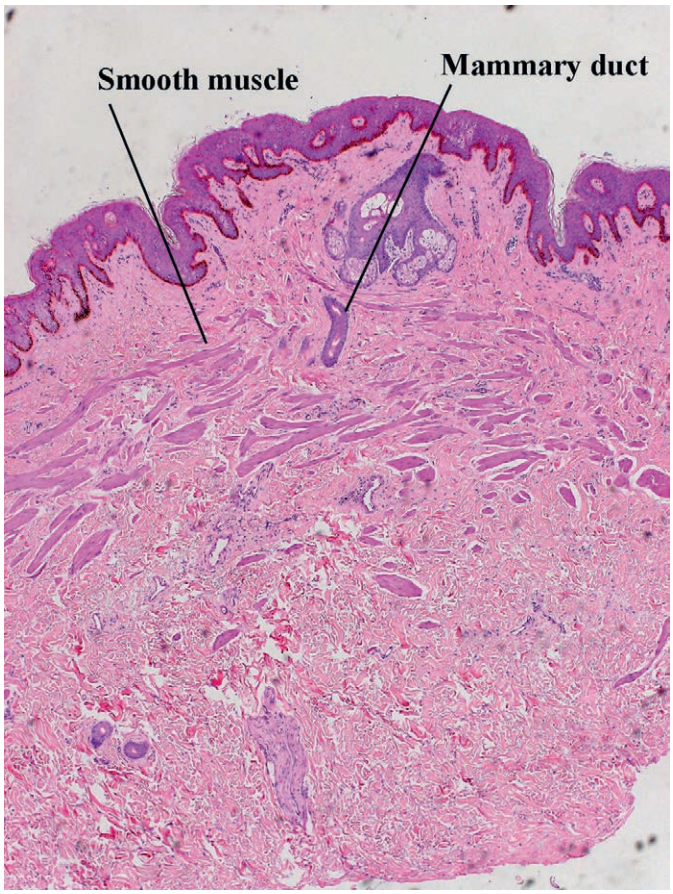

Fig. 1.9 Breast skin demonstrates numerous smooth muscle bundles. Breast skin, H&E × 20.

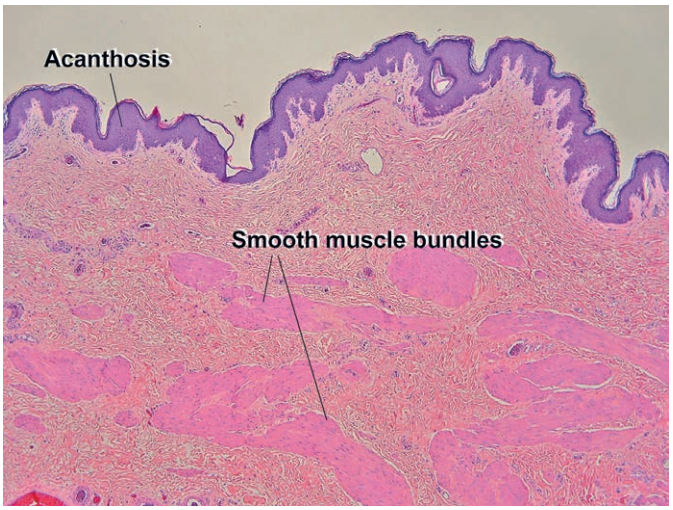

Fig. 1.10 Nipple skin demonstrates smaller smooth muscle bundles. The mammary duct resembles a large sweat duct. Breast skin, H&E × 20.

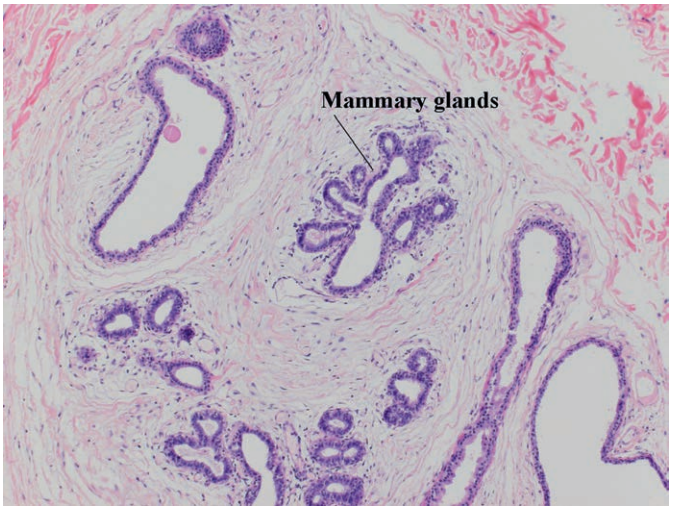

Fig. 1.11 The secretory portion of mammary glands demonstrates columnar epithelium forming complex lumens. Breast skin, H&E × 100.

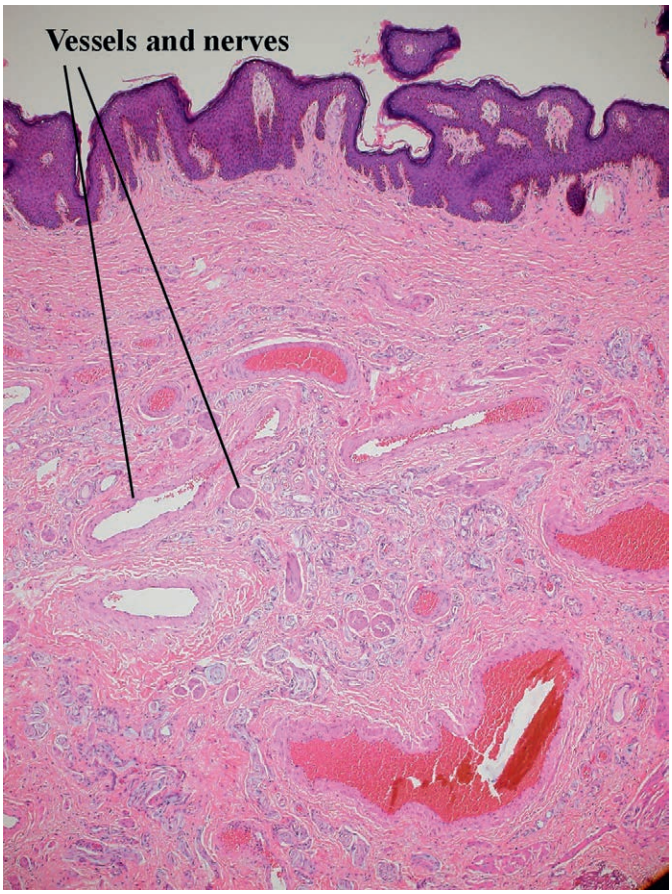

Fig. 1.12 Prepuce demonstrates a rugose appearance with many smooth muscle fascicles and high vascularity. Prepuce, H&E × 20.

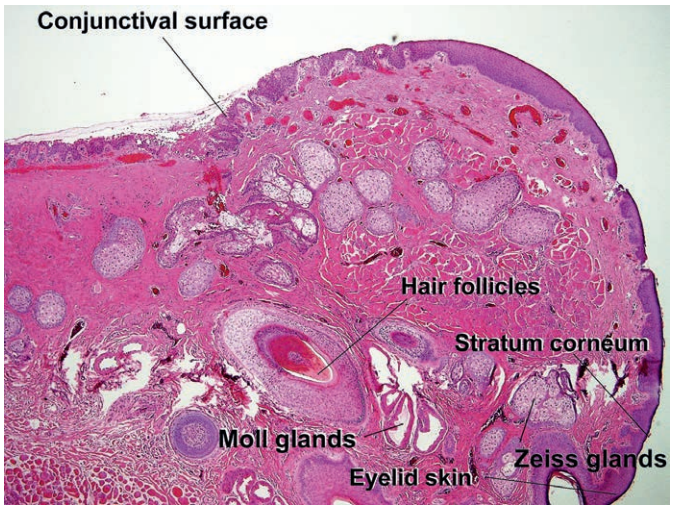

Fig. 1.13 Eyelid anatomy, below the conjunctiva; the densely fibrous tarsal plate contains sebaceous glands (meibomian glands), H&E × 100.

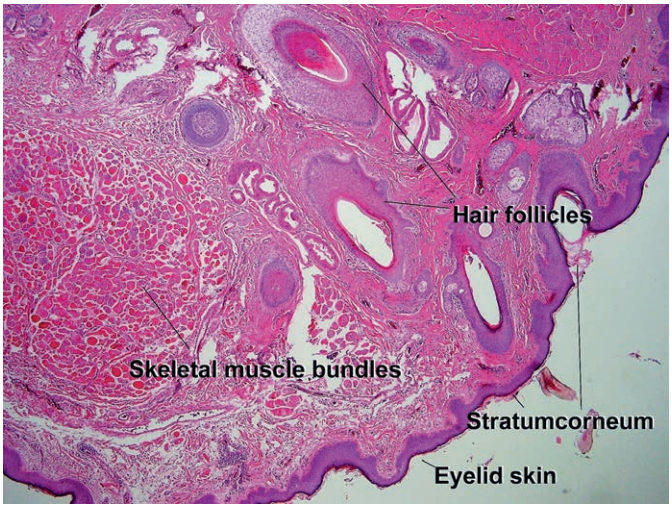

Fig. 1.14 The lid margin; on the cutaneous surface of the lid, a layer of striated muscle is present below the epidermis, H&E × 10.

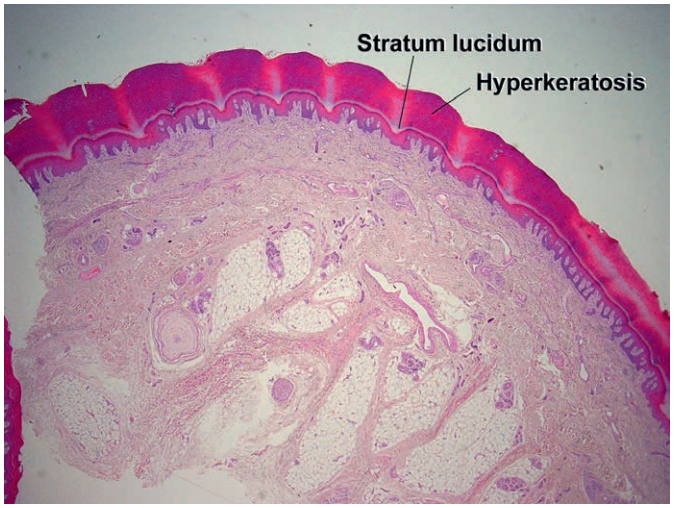

Fig. 1.15 Volar skin demonstrates a thick stratum corneum and lack of hair follicles, low power, H&E × 40.

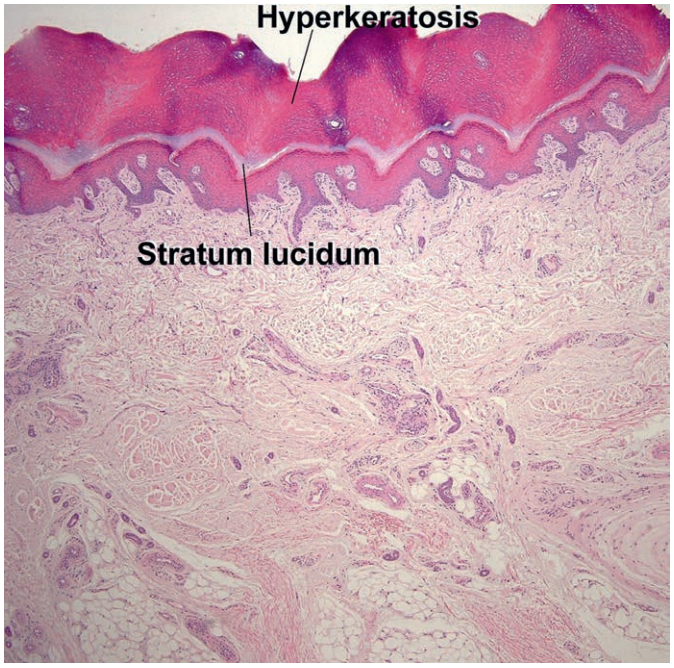

Fig. 1.16 Volar skin demonstrating a thick corneum and dermis, H&E × 100.

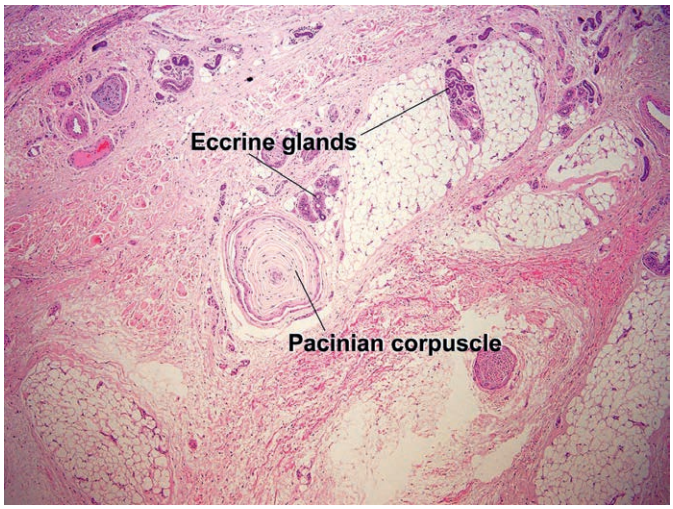

Fig. 1.17 Volar skin, with deep tissue demonstrating Pacinian corpuscles, H&E × 100.

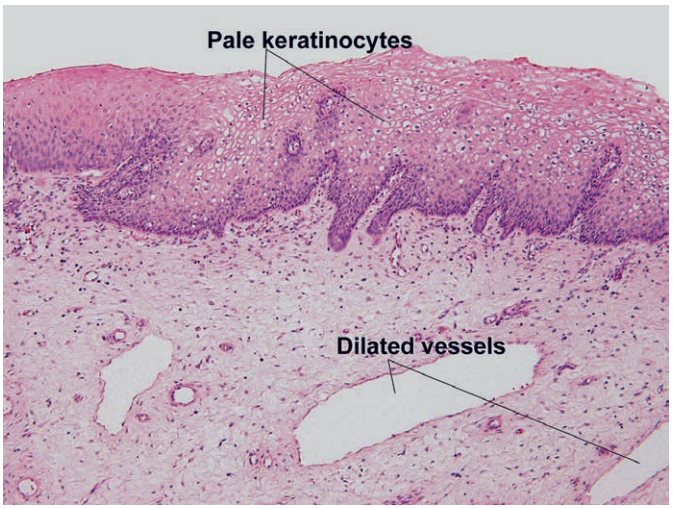

Fig. 1.18 Mucosal surface demonstrating nonkeratinizing epithelium and submucosa, H&E × 200.

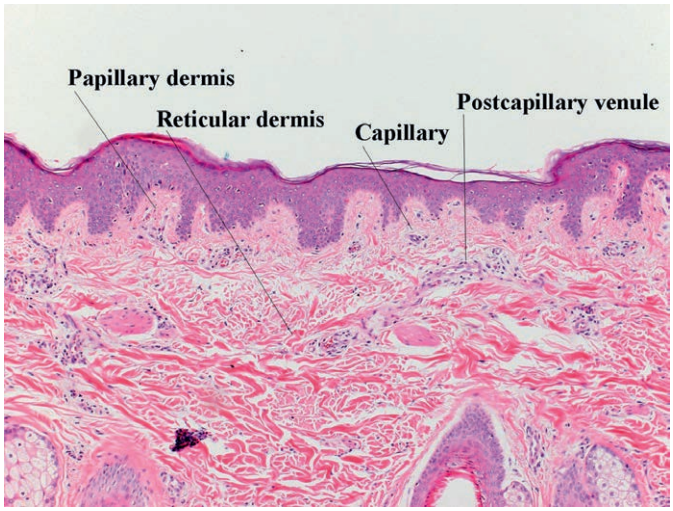

Fig. 1.19 Below the epidermis, the papillary dermis is composed of fine, nonbundled collagen. Capillaries are present within the papillary dermis, and the postcapillary venule sits at the junction of the papillary and reticular dermis. H&E × 40.

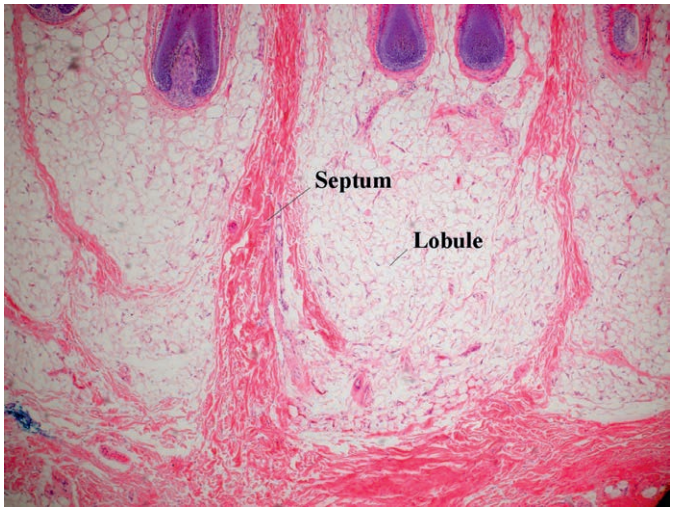

Fig. 1.20 The lobules of the subcutaneous fat are separated by fibrous septae. H&E × 40.

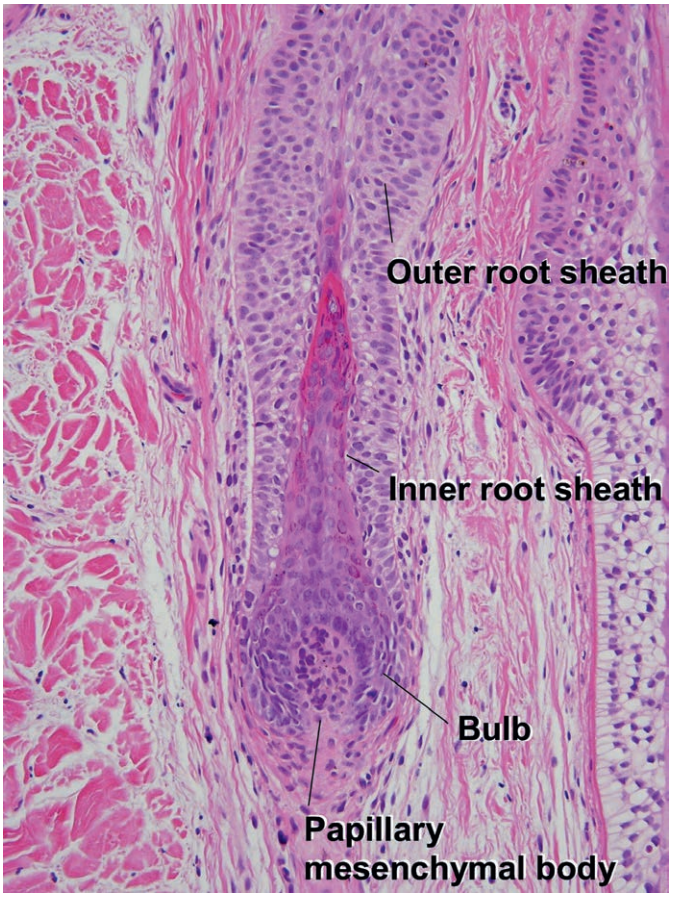

Fig. 1.21 Hair anatomy, vertical, H&E × 200.

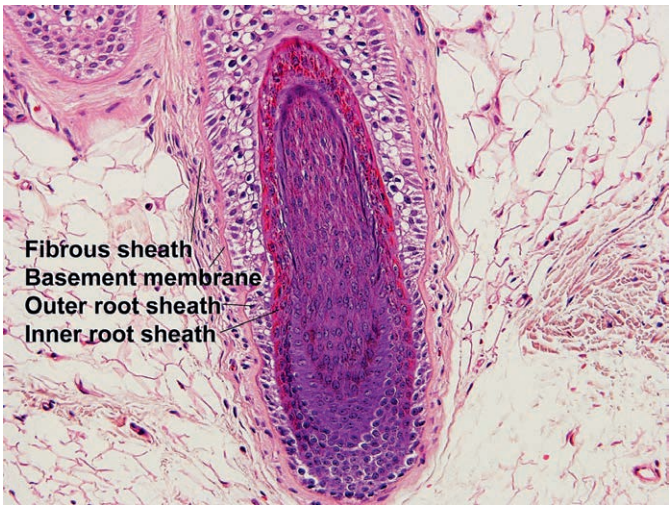

Fig. 1.22 In the inferior segment, the hair bulb gives rise to the inner and outer root sheath, H&E × 200.

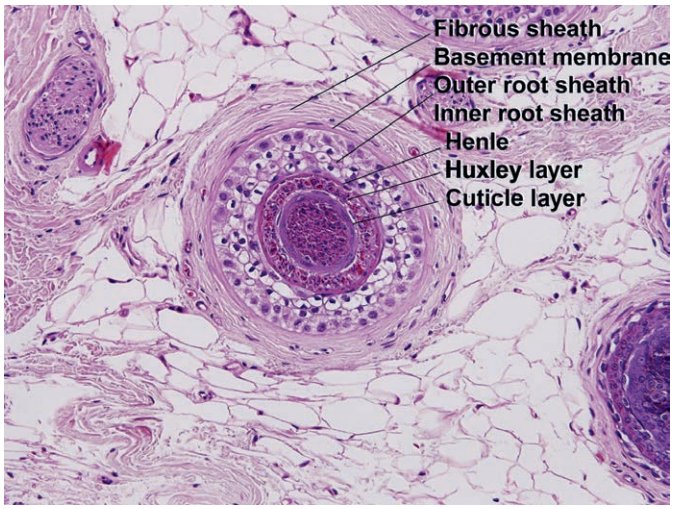

Fig. 1.23 Hair anatomy, transverse, H&E × 200.

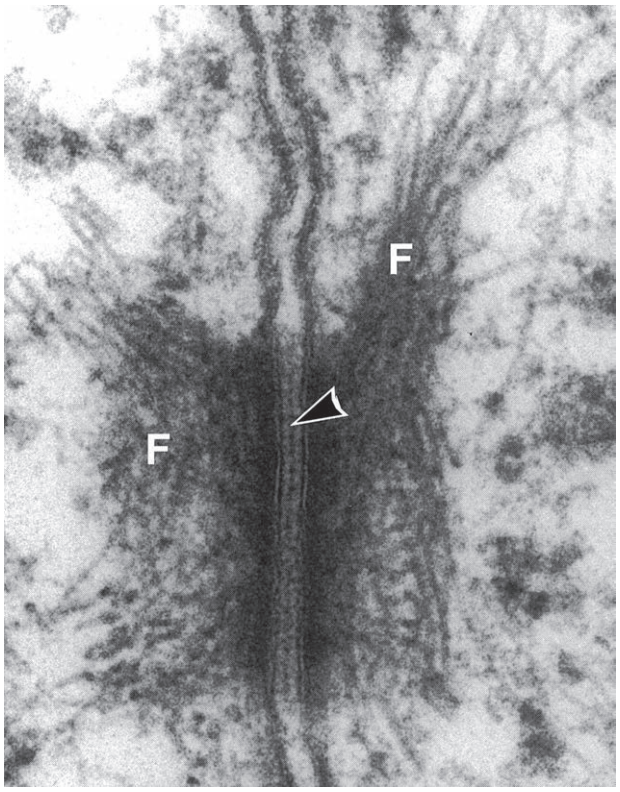

Fig. 1.24 Desmosome: A classic desmosome showing the following features: (1) Uniform gap of 20 to 30 nm between the apposed trilaminar plasma membranes with an intermediate line (*arrow*) in this gap. (2) Sharply delineated dense plaques into which tonofibrils (F) converge. *Courtesy Sunita Bhuta, MD.*

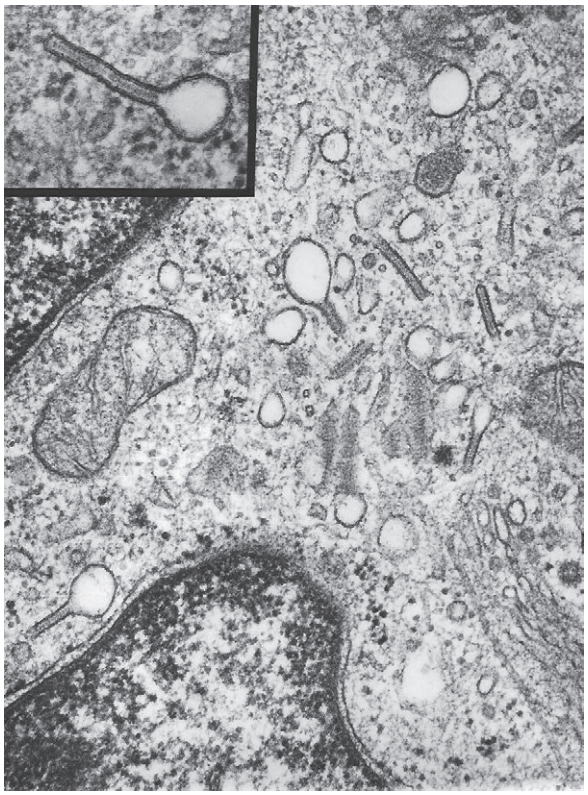

Fig. 1.25 Langerhans cell with Birbeck granules. This electromicrograph shows characteristic racket-shaped profiles of the granules in the cytoplasm (*inset* with higher magnification of the Birbeck granule). *Courtesy Sunita Bhuta, MD.*

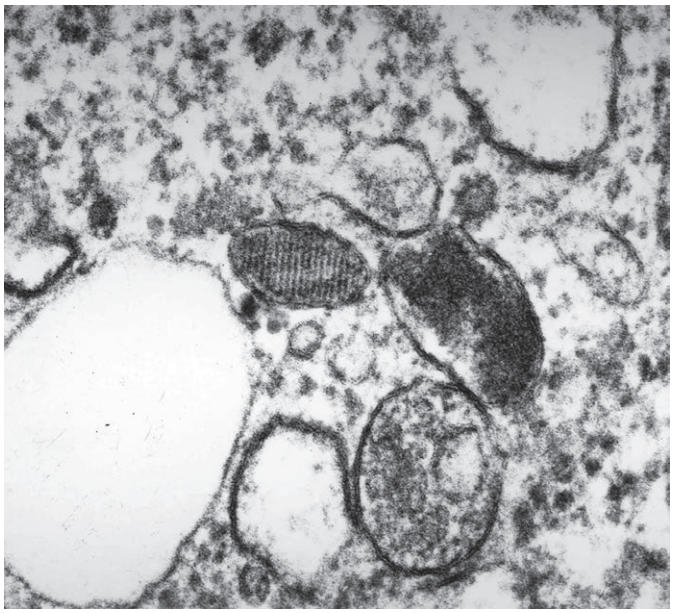

Fig. 1.26 Premelanosome: Solitary melanosome with characteristic internal striated structure. *Courtesy Sunita Bhuta, MD.*

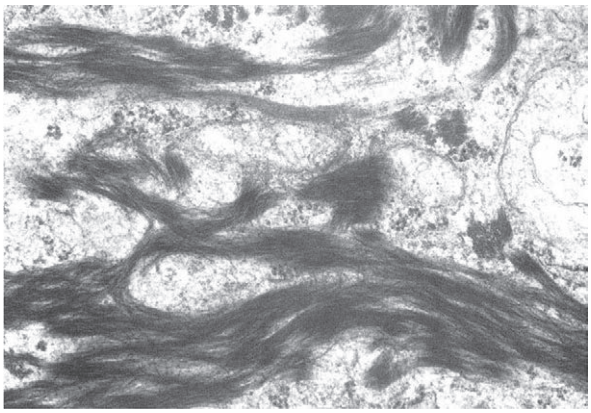

Fig. 1.27 Tonofibrils: Tonofibrils (intermediate filaments) lying free in the cytoplasm of a squamous cell. *Courtesy Sunita Bhuta, MD.*

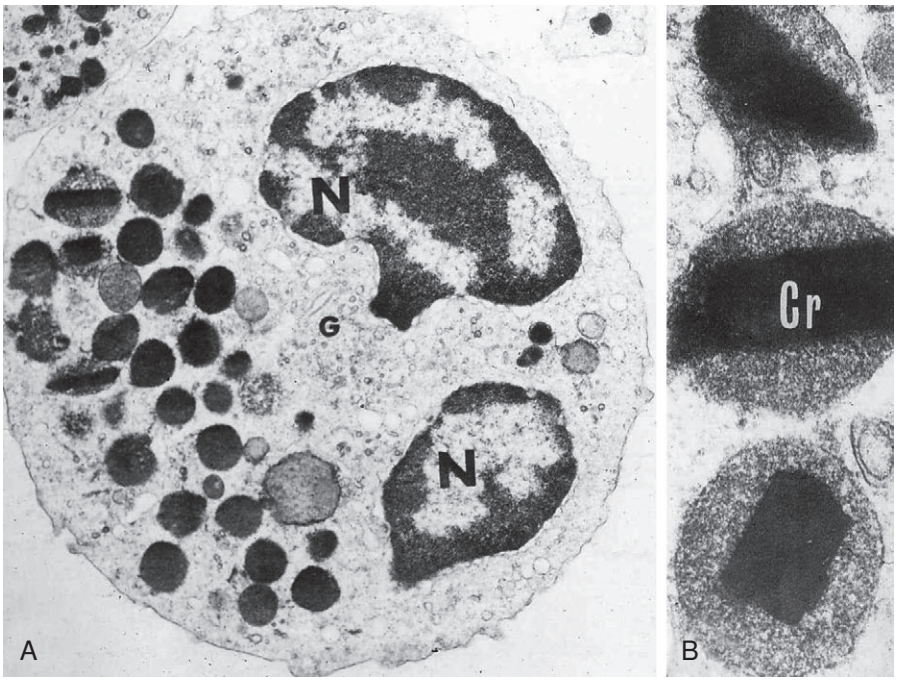

Fig. 1.28 Eosinophil: (A) Binucleate (N) with intracytoplasmic specific granules. (B) Specific granules have a finely granular matrix and a crystalline core. *Courtesy Sunita Bhuta, MD.*

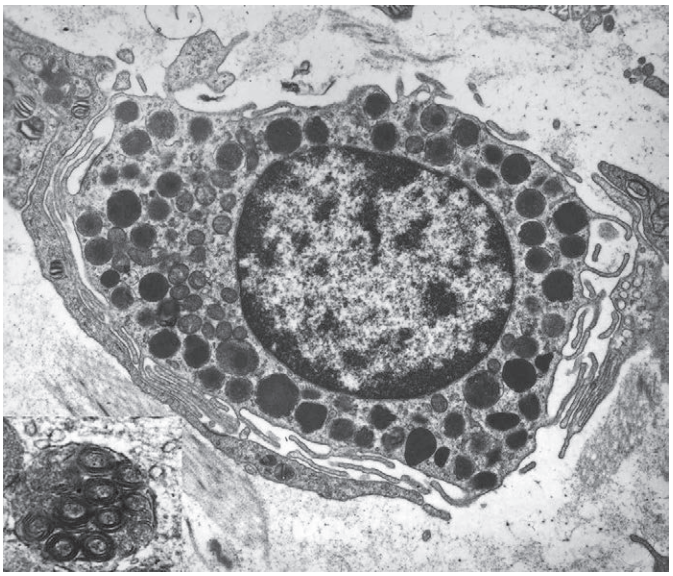

Fig. 1.29 Mast cell: Mast cell with numerous electron-dense granules. The inset shows internal structure of granules with membranous whorls (scrolls). *Courtesy Sunita Bhuta, MD.*

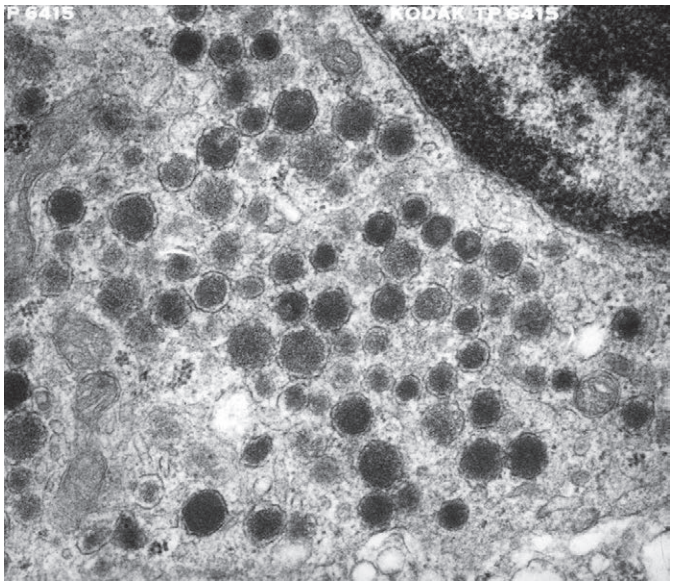

Fig. 1.30 Merkel cell: Merkel cell with intracytoplasmic membrane bound, electron-dense granules with a halo (neurosecretory granules). *Courtesy Sunita Bhuta, MD.*

Fig. 1.31 Skin tension lines relate to creases visible in older skin. They relate to passive skin tension, as well as active contraction of superficial muscular aponeurotic system muscle. Optimal surgical results can be achieved by aligning incisions along these lines.

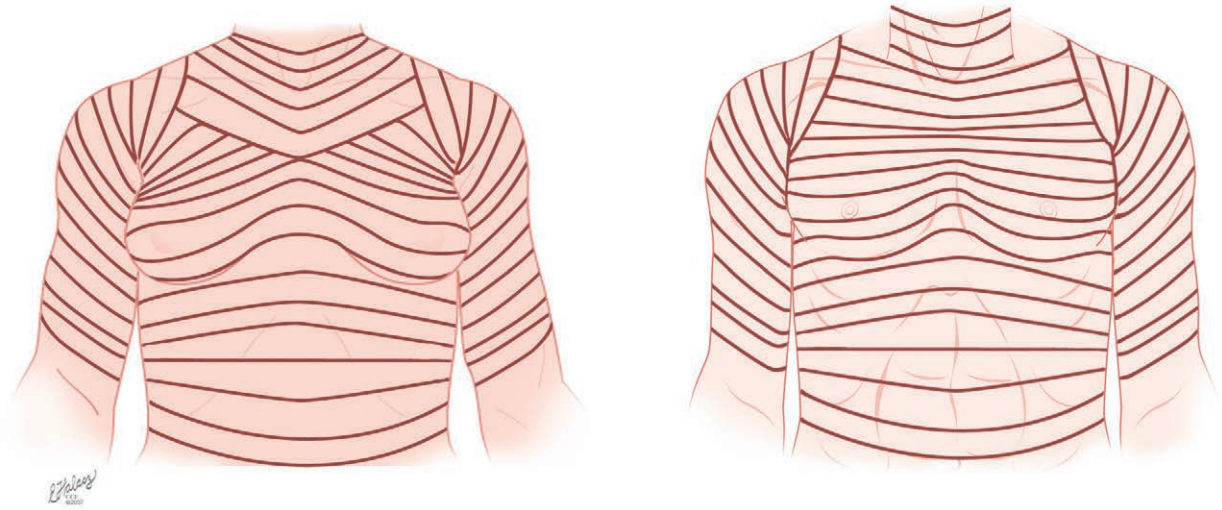

Fig. 1.32 Skin tension lines on the trunk.

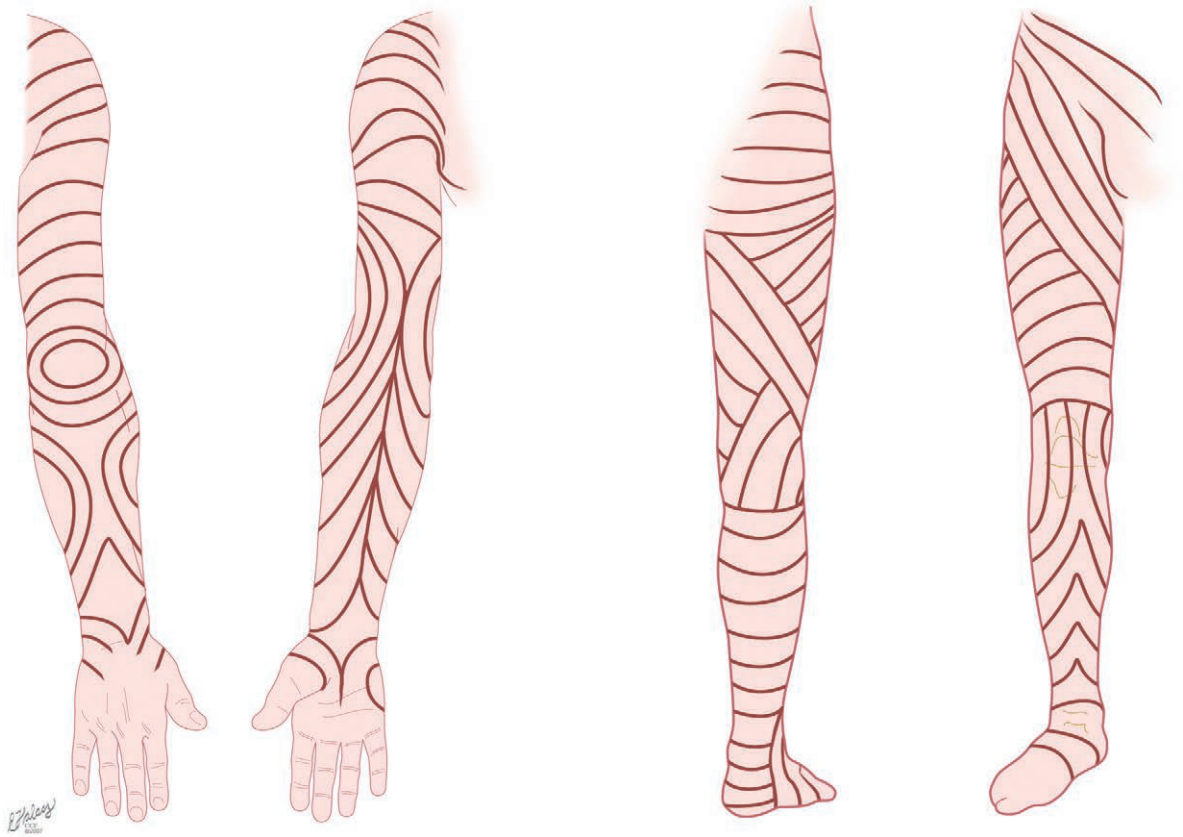

Fig. 1.33 Skin tension lines on the extremities.

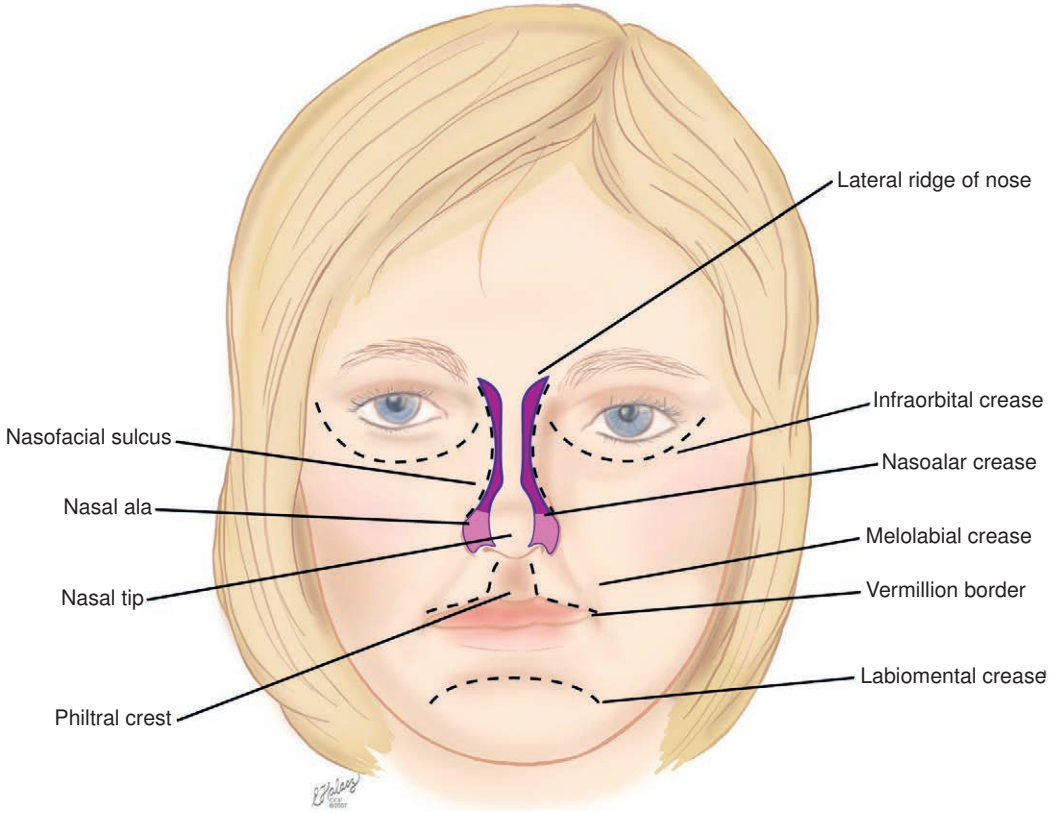

Fig. 1.34 Major anatomic landmarks.

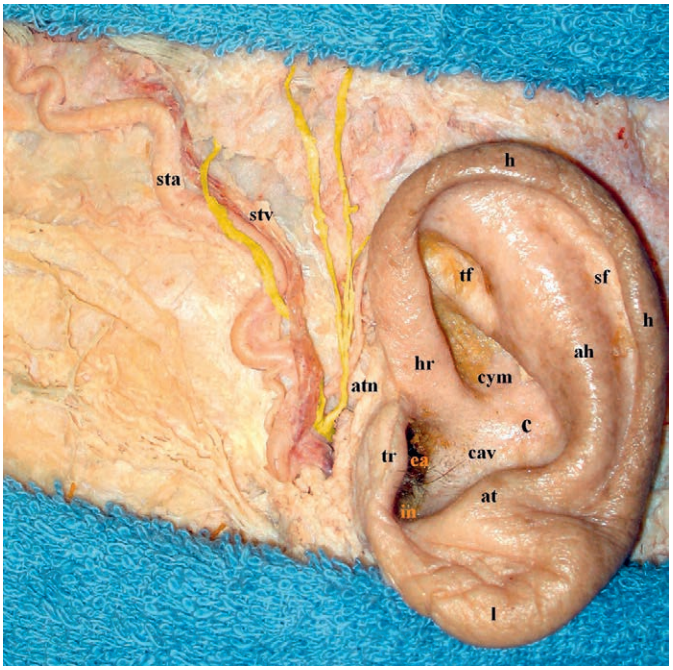

Fig. 1.35 Anatomy of the ear, superficial temporal artery, and auriculotemporal nerve. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

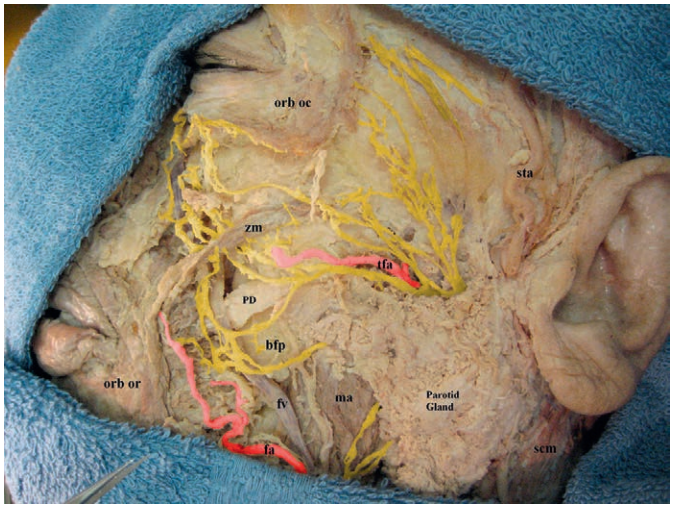

Fig. 1.36 Anatomy of the parotid gland and related structures. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

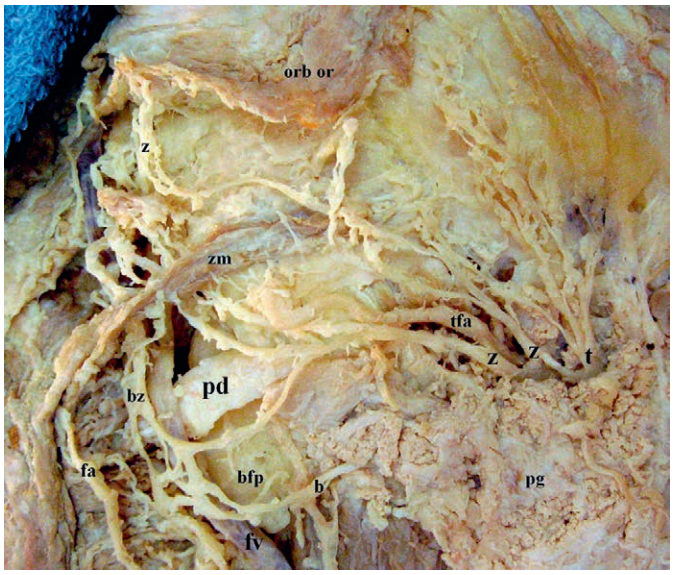

Fig. 1.37 Anatomy of the parotid duct and facial nerve. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

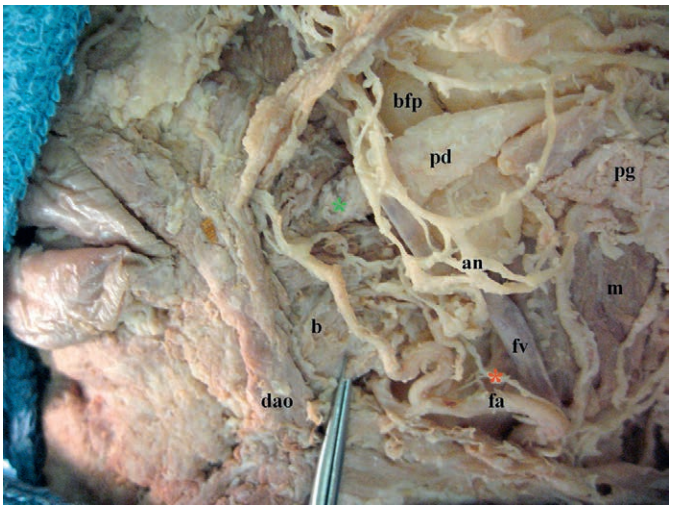

Fig. 1.38 Parotid duct as it pierces the buccinators muscle. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.39 Superficial muscular aponeurotic system. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

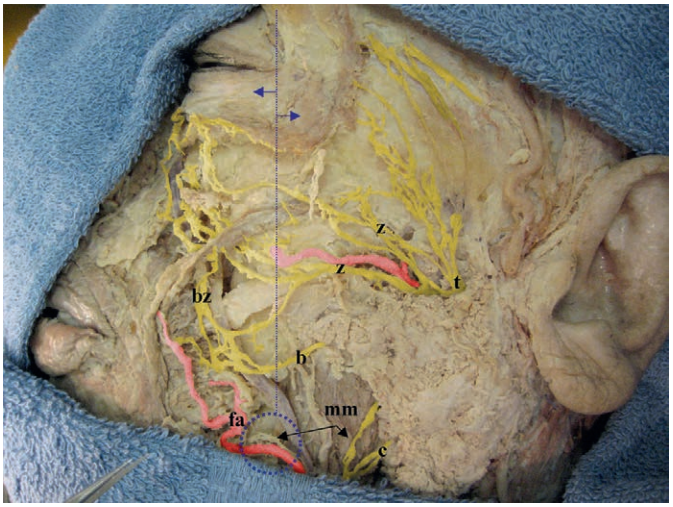

Fig. 1.40 Anatomy of the facial nerve. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

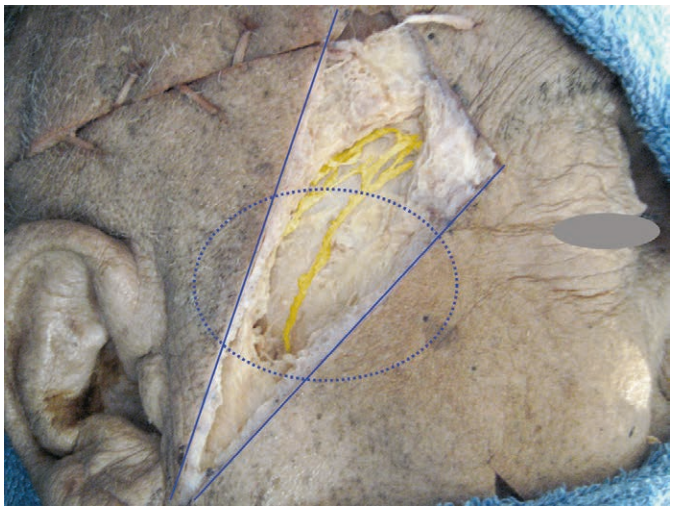

Fig. 1.41 The facial nerve: Danger zone for dermatologic surgery. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

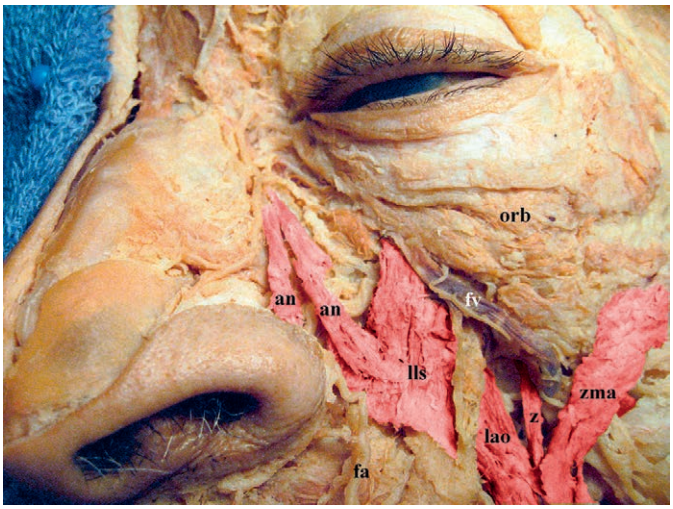

Fig. 1.42 Muscles of facial expression. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

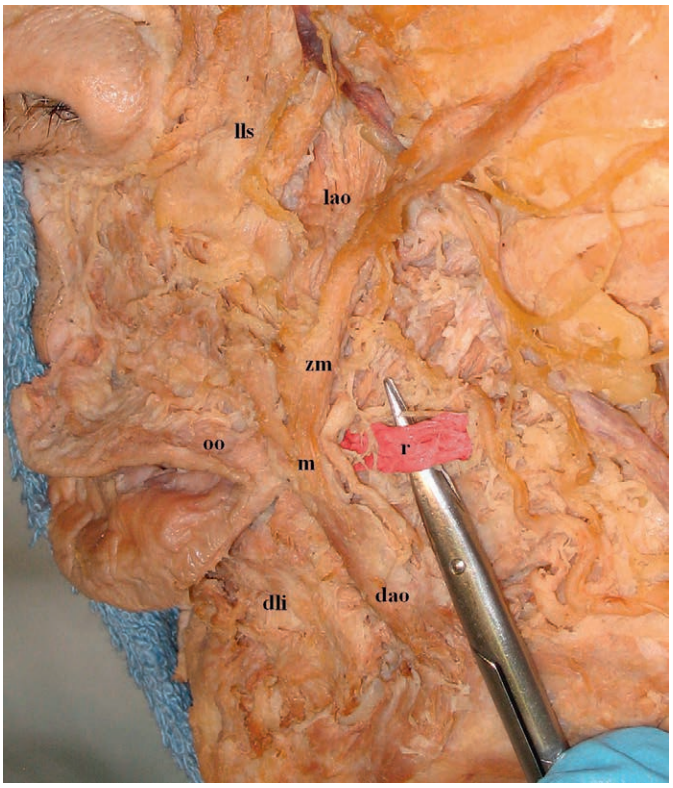

Fig. 1.43 Modiolus, elevators, and depressors. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

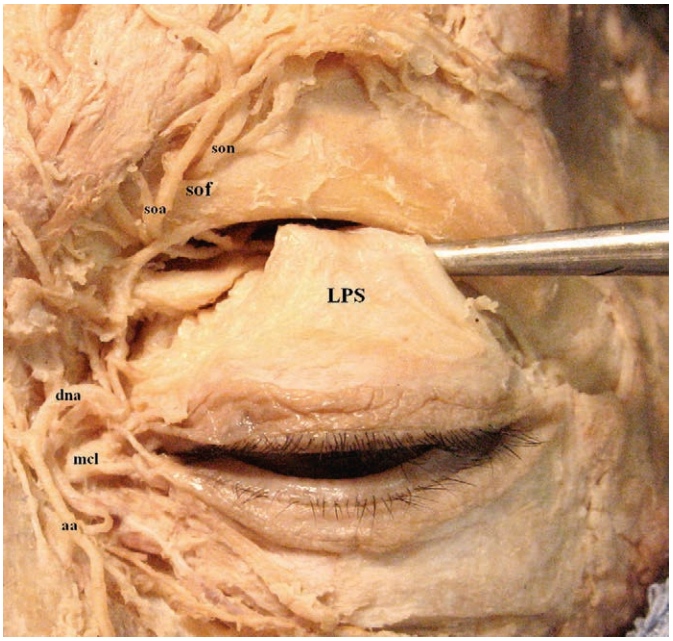

Fig. 1.45 Levator palpebrae superioris. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.44 Innervation of the facial skin. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.46 Medial forehead: Supraorbital and supratrochlear neurovascular structures. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

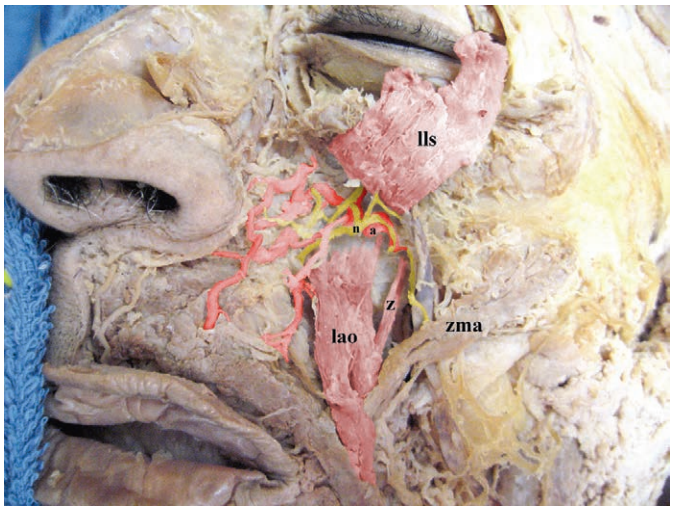

Fig. 1.47 Infraorbital foramen and related structures. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.48 Mental foramen and related structures. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

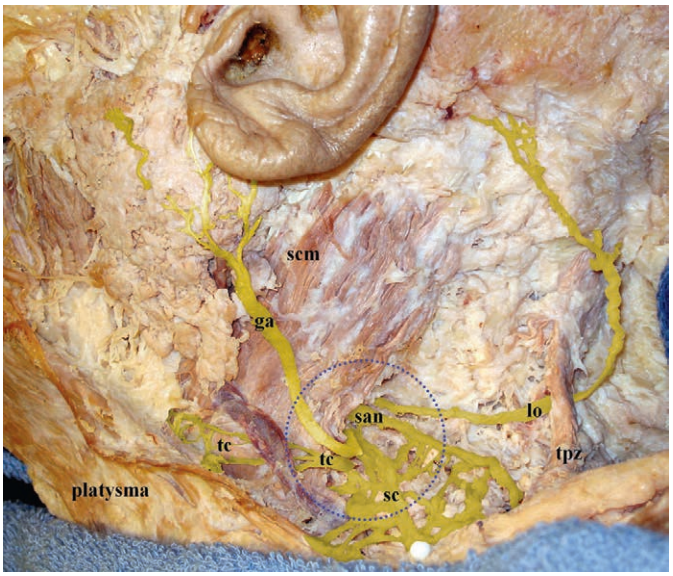

Fig. 1.49 Anatomy of the posterior triangle of the neck (Erb point). *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

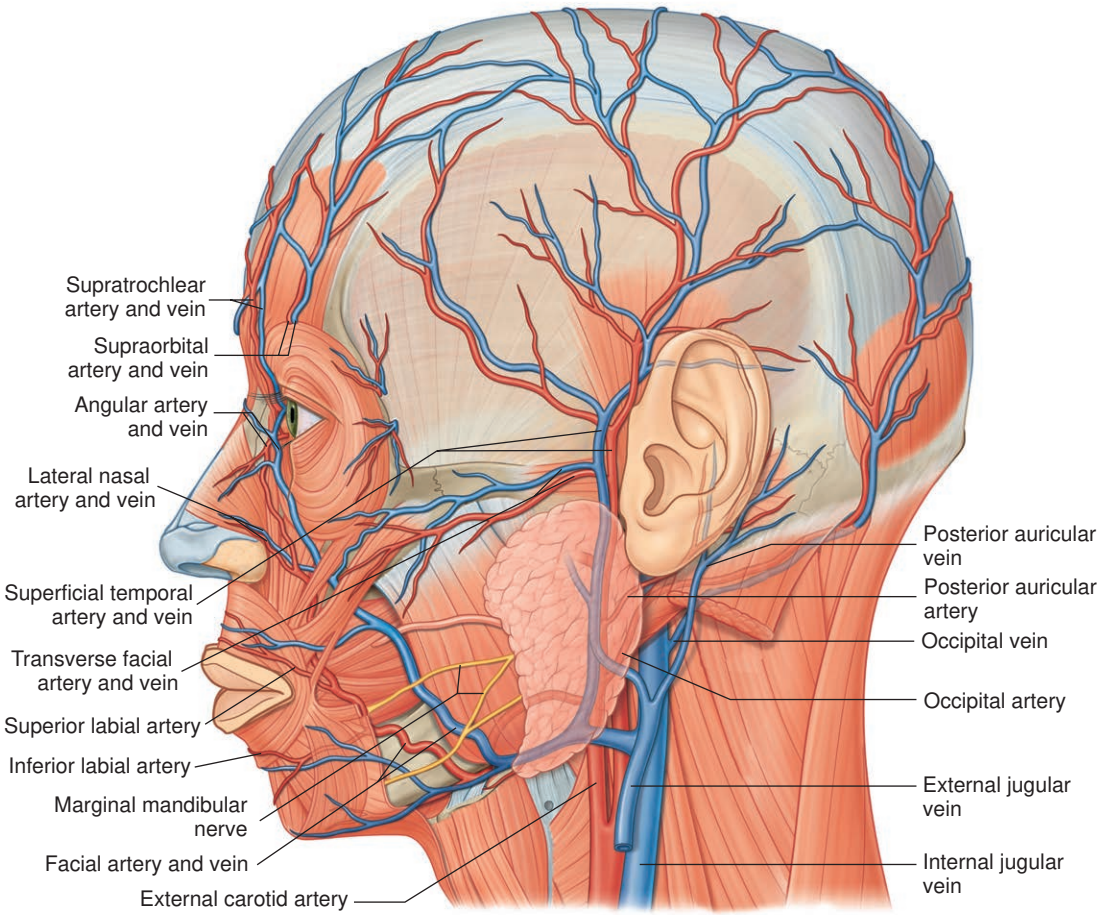

Fig. 1.50 Arterial and venous supply of the face.

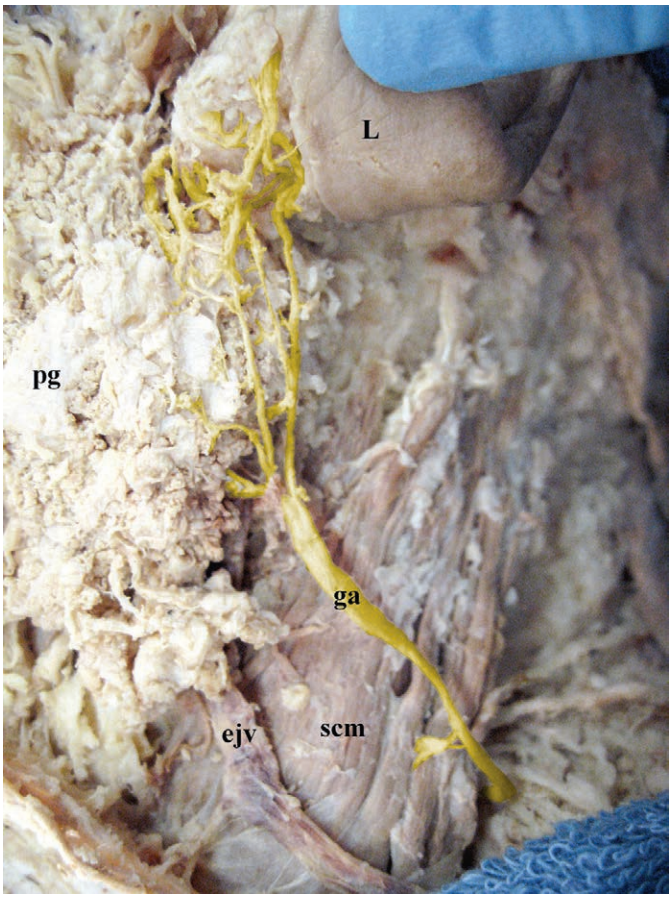

Fig. 1.51 Great auricular nerve and external jugular vein. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.52 Facial artery with angular artery. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Fig. 1.53 Inferior labial artery and related structures of the lower lip and chin. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

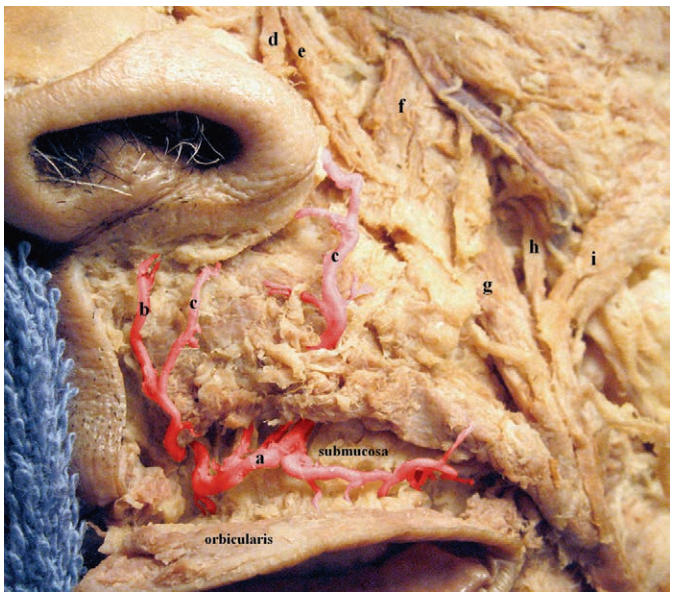

Fig. 1.54 Superior labial artery and related structures of the upper lip and cheek. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

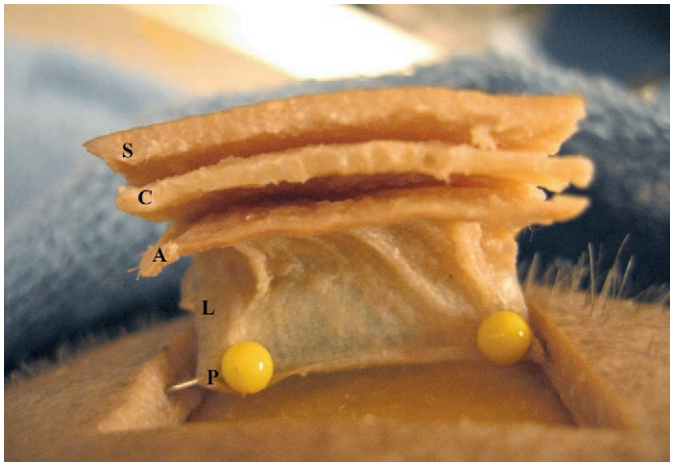

Fig. 1.55 Layers of the scalp. *Courtesy Joseph F. Greco, MD, and Christopher Skvarka, MD.*

Cutaneous Signs and Diagnosis

2

The astute clinician uses the appearance of the eruption and its accompanying symptoms to arrive at an accurate diagnosis. A symptom, such as itch or pain, is something that the patient reports. In contrast, a sign is elicited by the physician during examination. Pain is a symptom; tenderness to palpation is a sign.

This chapter will focus on the morphologic patterns skin lesions may have primarily or acquire over the course of time. The latter are called *secondary characteristics* and are not as helpful diagnostically as those present at the onset of the condition. Examples of each primary and secondary lesion are included, as well as other findings that are utilized to narrow the differential diagnosis such as the configuration, grouping, and color.

Finally, examples throughout will emphasize that the astute clinician should utilize all observable findings, inducing those of the hair, nails, and mucous membranes.

Fig. 2.1 Nevus of Ota (macule). *Courtesy Steven Binnick, MD.*

Fig. 2.2 Axillary freckling neurofibromatosis (macules).

Fig. 2.3 Voriconazole-induced lentigines (macules). *Courtesy Jennifer Huang, MD.*

Fig. 2.4 Vitiligo (patch).

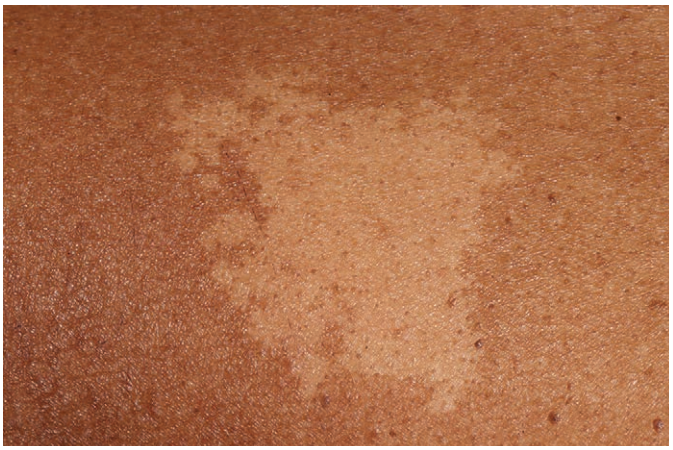

Fig. 2.5 Nevus depigmentosus (patch).

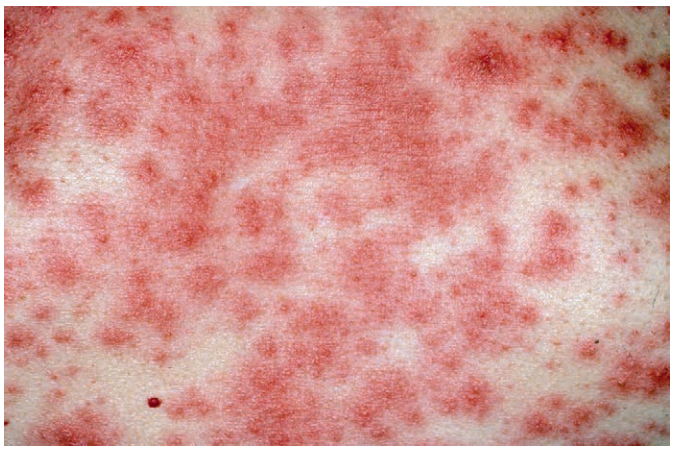

Fig. 2.6 Drug eruption (macules, some small papules present surmounting the erythema).

Fig. 2.7 Drug eruption (morbilliform appearance).

Fig. 2.8 Sarcoidosis (papules).

Fig. 2.9 Eruptive xanthomas (papules). Yellow color is uncommon in the skin and helps to narrow the differential diagnosis.

Fig. 2.10 Darier disease (papules).

Fig. 2.11 Verruca vulgaris (scaly papule).

Fig. 2.12 Hypertrophic lupus erythematosus (scaly papules and plaques). *Courtesy Steven Binnick, MD.*

Fig. 2.13 Oral hairy leukoplakia (plaques).

Fig. 2.14 Psoriasis (plaques). *Courtesy Steven Binnick, MD.*

Fig. 2.15 Xanthomas in homozygous familial hypercholesterolemia (plaques of yellow color).

Fig. 2.16 Squamous cell carcinoma (eroded nodule). *Courtesy Dr. Yi-Shuan Sheen.*

Fig. 2.17 Acute myelogenous leukemia (nodules).

Fig. 2.18 Erythema nodosum (subcutaneous nodules).

Fig. 2.19 Pyogenic granuloma (tumor). *Courtesy Curt Samlaska, MD.*

Fig. 2.20 Melanoma (tumor). *Courtesy Chris Miller, MD.*

Fig. 2.21 Basal cell carcinoma (tumor).

Fig. 2.22 Neurofibromatosis (macular hyperpigmentation, papules, tumors).

Fig. 2.23 Keloids (plaques and tumor).

Fig. 2.24 Acute urticaria (wheal). *Courtesy Curt Samlaska, MD.*

Fig. 2.25 Urticaria (wheal). *Courtesy Dr. Rui Carlos Taveres Bello.*

Fig. 2.28 Bullous pemphigoid (vesicles and bullae).

Fig. 2.26 Dyshidrosis (vesicles).

Fig. 2.29 Bullous pemphigoid (bulla and erosions on an erythematous base). *Courtesy Kaohsiung Chang Gang Memorial Hospital, Taiwan*.

Fig. 2.27 Bullous pemphigoid (vesicles and bullae).

Fig. 2.30 Piroxicam hypersensitivity (vesicles and bullae, some with hemorrhage).

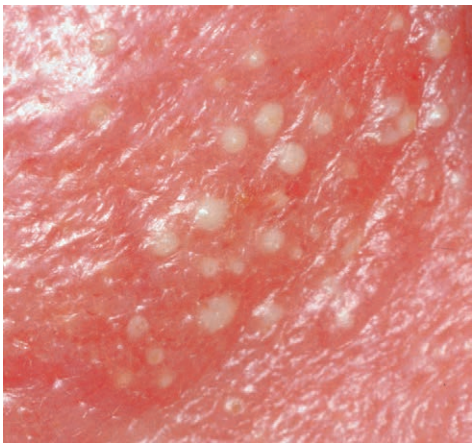

Fig. 2.31 Staphylococcal folliculitis (pustules).

Fig. 2.32 Plantar pustulosis (pustules).

Fig. 2.33 Autoinoculation vaccinia (umbilicated pustules).

Fig. 2.34 Staphylococcal folliculitis and impetigo (pustules and crusts).

Fig. 2.35 Hailey-Hailey disease (crusts).

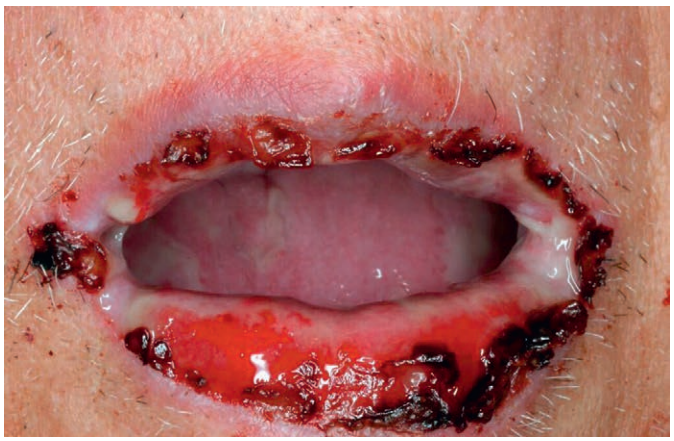

Fig. 2.36 Paraneoplastic pemphigus (hemorrhagic crusts). *Courtesy Department of Dermatology, Keio University School of Medicine, Tokyo, Japan.*

Fig. 2.37 Chronic hand dermatitis from cement (scale).

Fig. 2.40 Congenital ichthyosiform erythroderma (scale with desquamation). *Courtesy Scott Norton, MD.*

Fig. 2.38 X-linked ichthyosis (scale).

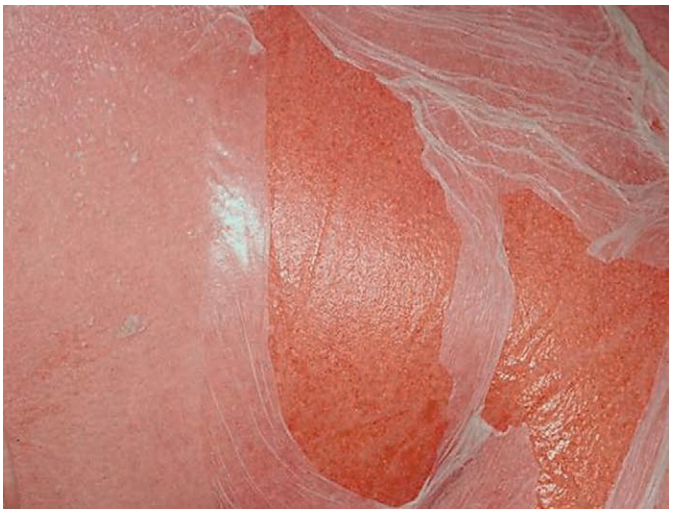

Fig. 2.41 Toxic epidermal necrolysis (sheets of desquamation).

Fig. 2.39 Fogo selvagem (scale). *Courtesy Dermatology Division, University of Campinas, Brazil.*

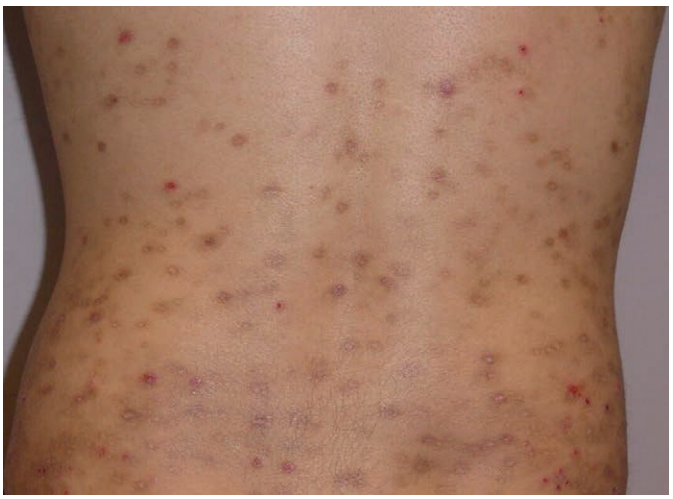

Fig. 2.42 Morgellons disease (excoriations). *Courtesy Scott Norton, MD.*

Fig. 2.43 Chronic pruritus (linear excoriations).

Fig. 2.44 Perianal fissure (fissure).

Fig. 2.45 Bullous pemphigoid (erosion).

Fig. 2.46 Isotretinoin skin fragility in a college crew team member (erosions).

Fig. 2.47 Basal cell carcinoma (ulcer). *Courtesy Steven Binnick, MD.*

Fig. 2.48 Pyoderma gangrenosum (ulcer).

Fig. 2.49 Basal cell carcinoma (ulcer).

Fig. 2.50 Epidermolysis bullosa (atrophy). *Courtesy Scott Norton, MD.*

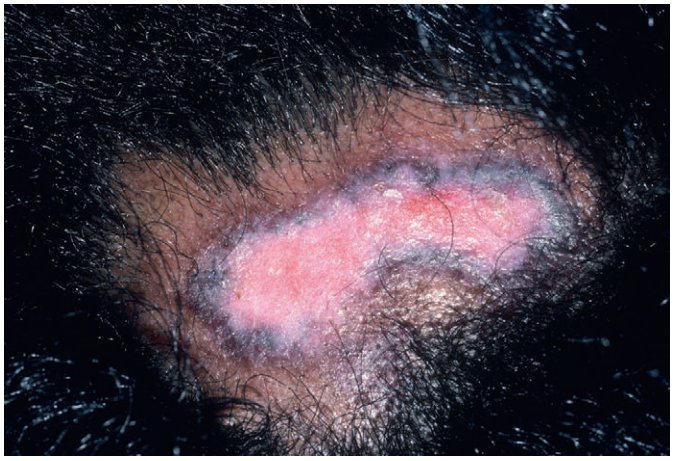

Fig. 2.51 Discoid lupus erythematosus (scarring alopecia). *Courtesy Steven Binnick, MD.*

Fig. 2.52 Rounded scars and edema from skin popping.

Fig. 2.53 Scarring after intentional trauma. *Courtesy Scott Norton, MD.*

Fig. 2.54 Keloid.

Fig. 2.55 Mycosis fungoides (poikiloderma). *Courtesy Steven Binnick, MD.*

Fig. 2.56 Limited scleroderma, CREST (gangrene).

Fig. 2.57 Atrophoderma (atrophy). *Courtesy Steven Binnick, MD.*

Fig. 2.58 Reeve sign (the reenactment of the cause of the condition during examination). Washboard nail dystrophy.

Fig. 2.59 Lichen simplex chronicus (lichenification).

Fig. 2.60 Lichen planus (linear, koebnerization).

Fig. 2.61 Granuloma annulare (annular).

Fig. 2.62 Tinea corporis (annular).

Fig. 2.63 Erythema ab igne (retiform).

Fig. 2.64 Cutaneous larva migrans (serpiginous).

Fig. 2.65 Herpes simplex (grouped).

Fig. 2.66 Linear epidermal nevus (blaschkoid).

Fig. 2.67 Systemic lupus erythematosus (photosensitivity pattern sparing nasolabial folds, under nose, under mouth). *Courtesy Steven Binnick, MD.*

Fig. 2.68 Melanoma (color variegation).

Fig. 2.69 Green nails from *Pseudomonas* infection (unusual color helps with the diagnosis).

Fig. 2.70 Pachyonychia congenita (oral).

Fig. 2.71 Kaposi sarcoma (conjunctiva).

Fig. 2.72 Nail groove from mucous cyst (nail).

Fig. 2.73 Alopecia areata (hair). *Courtesy Scott Norton, MD.*

Dermatoses Resulting from Physical Factors

3

This chapter catalogues many examples of how the skin reacts to the external environment from which it protects the body. Exposure to physical factors such as heat, cold, moisture, ultraviolet light, radiation, mechanical trauma and imbedded foreign bodies can manifest as uniquely patterned skin findings.

Of particular note are the many examples of photodistributed eruptions, symmetrically involving the face, chest, dorsal hands and forearms, as this pattern can be an important physical exam clue to one of many specific photosensitive conditions. Those who spend significant amounts of time outdoors are particularly susceptible to acute and chronic sun damage and may also sustain temperature-related (cold or hot) injuries at acral sites.

The distribution of mechanical injuries and foreign body reactions, however, is usually asymmetric. In this chapter, appreciate the geometric shapes and unnatural configurations that can be produced on the skin as a result of physical abuse, radiotherapy or phytophotodermatitis. The patient history, and at times tissue analysis, can help to confirm the diagnosis that was suspected after a thorough physical examination.

This portion of the atlas features examples of dermatoses and skin lesions resulting from physical factors.

Fig. 3.1 Thermal burn from child abuse. *Courtesy Paul Honig, MD.*

Fig. 3.2 Hot water burn. *Courtesy Steven Binnick, MD.*

Fig. 3.3 Hot water bottle injury. *Courtesy Steven Binnick, MD.*

Fig. 3.4 Burn scar.

Fig. 3.5 Electrical burn from biting electrical cord. *Courtesy Paul Honig, MD.*

Fig. 3.6 Hot oil burn. *Courtesy Steven Binnick, MD.*

Fig. 3.7 Miliaria crystallina. *Courtesy Steven Binnick, MD.*

Fig. 3.8 Miliaria rubra.

Fig. 3.9 Miliaria rubra.

Fig. 3.10 Miliaria pustulosa secondary to childbirth. *Courtesy Curt Samlaska, MD.*

Fig. 3.11 Erythema ab igne. *Courtesy Paul Honig, MD.*

Fig. 3.12 Erythema ab igne. *Courtesy Curt Samlaska, MD.*

Fig. 3.13 Pernio. *Courtesy Steven Binnick, MD.*

Fig. 3.14 Pernio.

Fig. 3.15 Pernio. *Courtesy Steven Binnick, MD.*

Fig. 3.16 Frostbite.

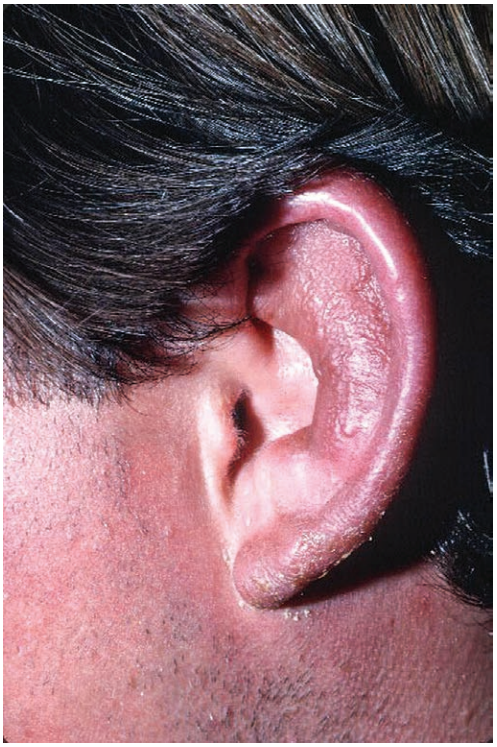

Fig. 3.17 Frostbite. *Courtesy The University of Utah and Oregon Health Sciences University Leonard Swinyer MD image collection.*

Fig. 3.18 Tropical immersion foot. *Courtesy Shyam Verma, MBBS, DVD.*

Fig. 3.19 Tropical immersion foot. *Courtesy Steven Binnick, MD.*

Fig. 3.20 Sunburn. *Courtesy Steven Binnick, MD.*

Fig. 3.21 Cutis rhomboidalis nuchae.

Fig. 3.22 Poikiloderma of Civatte.

Fig. 3.23 Favre-Racouchot.

Fig. 3.24 Favre-Racouchot with solar elastotic nodules.

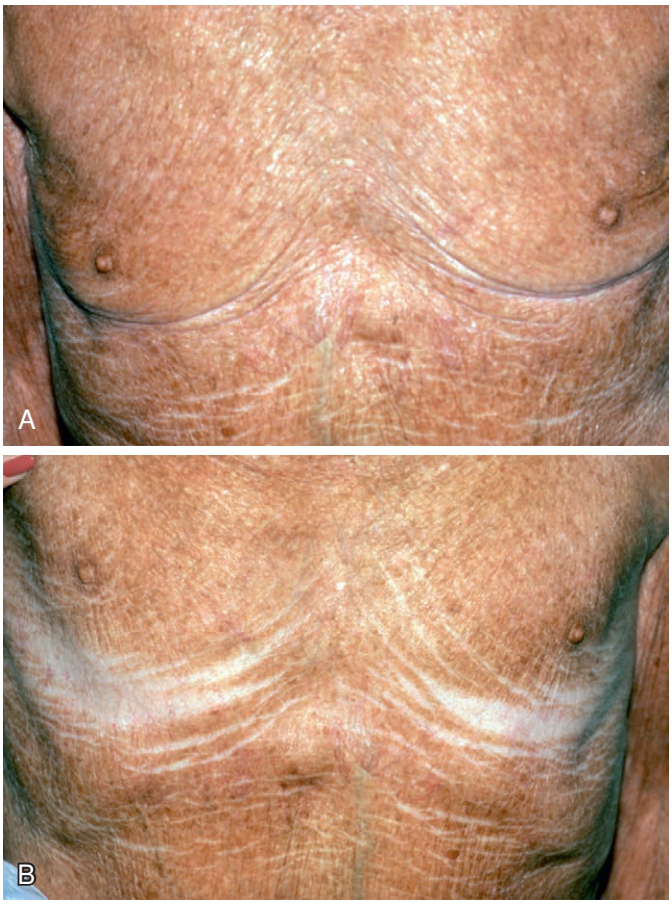

Fig. 3.25 (A) Chronic solar damage. (B) With upward traction, normal non–sun-exposed skin is revealed.

Fig. 3.27 Solar elastosis of the forehead.

Fig. 3.26 Stellate pseudoscars.

Fig. 3.28 Weathering nodules.

Fig. 3.29 Colloid milium. *Courtesy Ken Greer, MD.*

Fig. 3.30 Photo onycholysis secondary to doxycycline. *Courtesy Lindsay Ackerman, MD.*

Fig. 3.31 Phytophotodermatitis.

Fig. 3.32 Two friends who made limeade in the sun to sell in the summer.

Fig. 3.33 Hyperpigmentation of the hand and cheek after phytophotodermatitis. *Courtesy Paul Honig, MD.*

Fig. 3.34 Polymorphous light eruption, papular type.

Fig. 3.35 Polymorphous light eruption, papular type.

Fig. 3.36 Polymorphous light eruption, plaque type.

Fig. 3.37 Polymorphous light eruption, juvenile spring eruption type.

Fig. 3.38 Actinic prurigo. *Courtesy Steven Binnick, MD.*

Fig. 3.39 Actinic prurigo. *Courtesy Campbell Stewart, MD.*

Fig. 3.40 Solar urticaria.

Fig. 3.41 Hydroa vacciniforme. *Courtesy National Taiwan University Hospital.*

Fig. 3.42 Chronic actinic dermatitis.

Fig. 3.43 Chronic actinic dermatitis. *Courtesy Kaohsiung Chang Gang Memorial Hospital, Taiwan.*

Fig. 3.44 Chronic actinic dermatitis. *Courtesy The University of Utah and Oregon Health Sciences University Leonard Swinyer MD image collection.*

Fig. 3.45 Chronic radiation dermatitis.

Fig. 3.46 Chronic radiation dermatitis. *Courtesy Steven Binnick, MD.*

Fig. 3.47 (A) Fluoroscopy-induced radiodermatitis. (B) Close-up of *A*.

Fig. 3.49 Soft corn. *Courtesy Steven Binnick, MD.*

Fig. 3.48 Hard corn. *Courtesy Steven Binnick, MD.*

Fig. 3.50 Fire coral cuts. *Courtesy Steven Binnick, MD.*

Fig. 3.51 Decubitus ulcer. *Courtesy Steven Binnick, MD.*

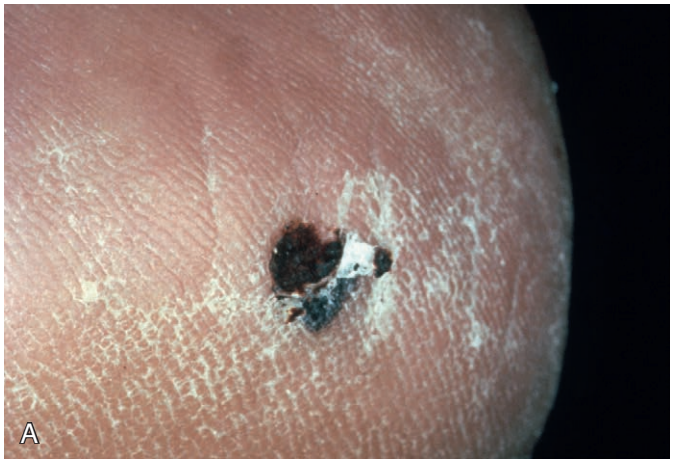

Fig. 3.53 (A) Black heel. (B) Immediately after scraping off blood from black heel.

Fig. 3.52 Sclerosing lymphangitis.

Fig. 3.54 Subcutaneous emphysema. *Courtesy Curt Samlaska, MD.*

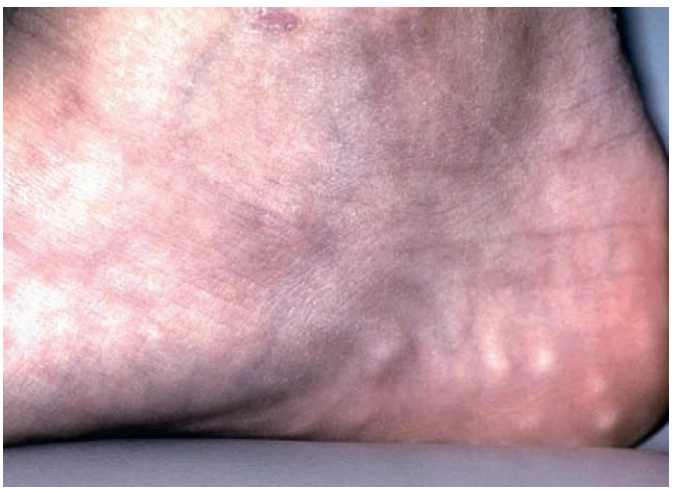

Fig. 3.55 Piezogenic papules. *Courtesy Paul Honig, MD.*

Fig. 3.56 Ulcerations and bacterial superinfection secondary to skin popping.

Fig. 3.57 Cotton granuloma.

Fig. 3.58 Hair granuloma in a barber.

Fig. 3.59 Red tattoo reaction. *Courtesy Rui Tavares Bello, MD.*

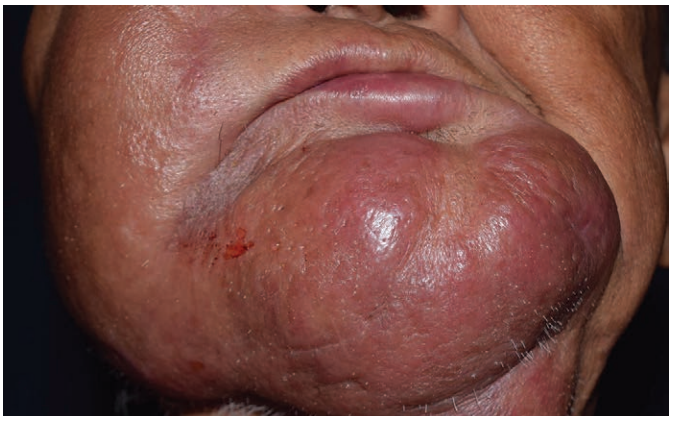

Fig. 3.60 Silicone granuloma. *Courtesy National Cheng Kung University, Taiwan.*

Fig. 3.61 Mercury granuloma from thermometer.

Fig. 3.62 Silica granuloma.

Fig. 3.63 Carbon stain, gunshot wound.

Fig. 3.64 Zyplast granuloma, test site.

Fig. 3.65 Facial swelling reaction to hyaluronic acid filler.

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Pruritus and Neurocutaneous Diseases

4

Pruritus often produces distinctive skin lesions, characterized by angulated borders. Endogenous diseases (in common parlance, an "inside job") tend to produce lesions that are rounded in character, whereas exogenous caustic agents, scratching, and other forms of external trauma tend to produce angulated, linear, or geometric shapes (signs of an "outside job").

Pruritus can also result in the isomorphic (Koebner) phenomenon, where lesions of an endogenous disease localize in areas of trauma. These lesions often demonstrate a hybrid morphology, suggesting both endogenous origin and external trauma.

Lichenification results from chronic scratching or rubbing and is characterized by hyperkeratosis and papillary dermal fibrosis. The resulting clinical morphology includes slight induration and accentuation of skin markings. Excoriation results in eosinophilic necrosis of the granular layer. The corneum can remain intact, but more severe excoriation results in loss of the corneum and viable epidermis. More pronounced trauma can result in ulceration, in which the injury extends to the level of the dermis.

Broad areas of lichenification are characteristic of lichen simplex chronicus. Discrete papules with focal evidence of excoriation are typical of prurigo nodularis and arthropod bites, including those caused by bedbugs. This portion of the atlas will guide you through the clinical manifestations related to pruritic disorders.

Fig. 4.2 Acquired perforating disease in renal failure.

Fig. 4.1 Excoriations secondary to pruritus of Hodgkin disease.

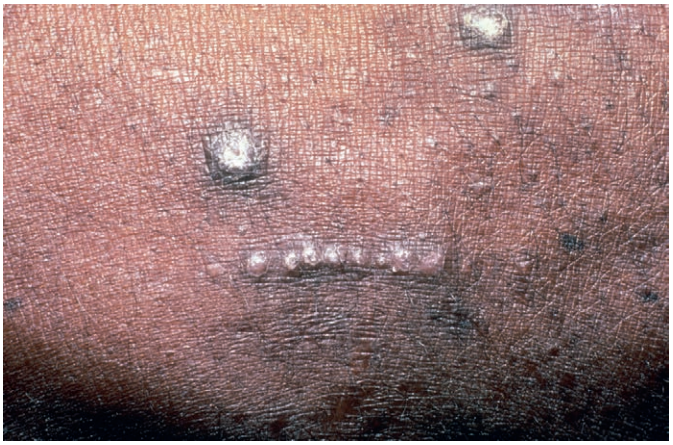

Fig. 4.3 Acquired perforating disease in renal failure.

Fig. 4.4 Acquired perforating disease in renal failure. *Courtesy Steven Binnick, MD.*

Fig. 4.5 Prurigo nodularis in a patient with chronic pruritus.

Fig. 4.6 Jaundice.

Fig. 4.7 Palmar xanthomas in hepatic cholestasis.

Fig. 4.8 Lichen simplex chronicus in Alagille disease.

Fig. 4.9 Hyperpigmentation in primary biliary cirrhosis.

Fig. 4.10 Winter itch.

Fig. 4.11 Lichen simplex chronicus in pruritus ani.

Fig. 4.12 Lichen simplex chronicus of the scrotum.

Fig. 4.13 Lichen simplex chronicus of the vulva.

Fig. 4.14 Prurigo pigmentosa.

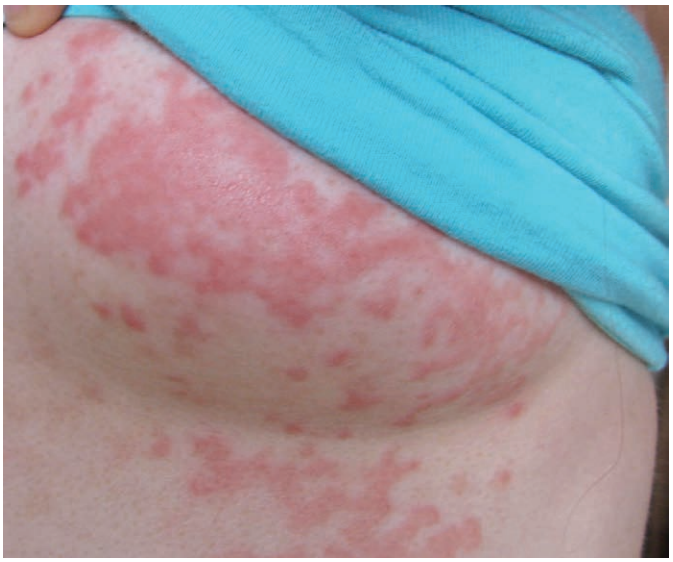

Fig. 4.15 Prurigo pigmentosa. *Courtesy Stephen D. Hess, MD, PhD.*

Fig. 4.16 Prurigo pigmentosa. *Courtesy Department of Dermatology, Keio University School of Medicine, Tokyo, Japan.*

Fig. 4.17 A and B. Papuloerythroderma of Ofuji showing the deck chair sign. *Courtesy Dr. Rui Carlos Taveres Bello*.

Fig. 4.18 Lichen simplex chronicus.

Fig. 4.19 Lichen simplex chronicus.

Fig. 4.20 Lichen simplex chronicus.

Fig. 4.21 Lichen simplex chronicus. *Courtesy Steven Binnick, MD.*

Fig. 4.22 Lichen simplex chronicus with dyspigmentation and early nodule formation.