Structure and Physiology of Aging Skin

Epidermis

Overall, the epidermis thins with advancing age. The stratum corneum loses its basket-weave pattern and becomes thin and compact, providing a less effective barrier. Keratinocytes exhibit some nuclear irregularity, especially in sun-exposed areas. Melanocyte density decreases by 10% to 20% each decade, but the exact decrease varies according to anatomic site. There is loss of cellular immunity with aging, as demonstrated by a nearly 50% decrease in the bone marrow-derived dendritic Langerhans’ cells.

Dermis

With aging, the dermis loses roughly 20% of its thickness secondary to the loss of proteoglycan as well as collagen. Collagen fibers become more cross-linked. Elastic fibers decrease and fragment, particularly in sun-exposed skin. In addition to decreased cellularity, the dermis becomes less vascular, as demonstrated by the loss of capillary loops. These vascular alterations may lead to pallor and contribute to decreased inflammation, a decreased rate of healing after injury, and delayed clearance of foreign materials.

Subcutaneous Fat

The underlying subcutaneous fat also undergoes atrophy with age. These changes predispose the elderly to the effects of trauma and cold. The loss of subcutaneous fat in the face contributes to what we interpret as an aged appearance.

Appendages

Age-related changes of the hair include most notably loss and graying of the hair. The loss of hair is determined by both androgenetic and involutional alopecia. The former begins before age 40 and is dependent upon male sex hormones and genetics. It is marked by bitemporal recession and hair loss on the vertex and frontal areas of the scalp in men, and by hair loss just posterior to the frontal rim in women, as the hair changes from thick to fine vellus-like hairs. Involutional alopecia, seen after the age of 40, is a process where there is a decrease in the hair shaft diameter and a decrease in the number of hair units, resulting in diffuse hair thinning. This phenomenon poses a cosmetic issue for some people. Therapy is available for androgenetic alopecia. Topical minoxidil, 2% solution 1.0 mL twice daily, has been shown to significantly increase hair counts. Oral finasteride at a dose of 1 mg daily has also been proven to increase hair counts and improve scalp coverage in men. There is also the option of scalp reduction and hair transplants to restore hair.

Hair graying, often synonymous with aging, is a hereditary loss of functional melanocytes in the hair bulb. By age 50, roughly half of all body hairs become gray.

Nail plate thickness and linear growth rate also decrease with age. Nails become thinner, more brittle, and may develop longitudinal ridges. This fragility may be improved with the application of lactic acid 12% lotion or a creamy moisturizer massaged daily into unpolished nails.

Sebaceous glands undergo hypertrophy with age, although sebum production decreases with age, partly due to a decline in androgen levels. Apocrine and eccrine sweat glands decrease in density as they accumulate lipofuscin, an aging pigment of unknown significance. These glandular changes contribute to decreased thermoregulatory abilities, making the elderly person more susceptible to hyperthermia.
Changes in Aging Skin from Physical Elements

In addition to the intrinsic process of senescence, there is the extrinsic component of skin aging that results from photodamage, smoking, and diet. The cutaneous changes of chronic ultraviolet (UV) damage manifest as wrinkling, solar lentigines (“liver spots”), telangiectasias, mottled pigmentation, and, most significantly, premalignant and malignant neoplasms. Therefore, it is very important to take appropriate measures to protect the skin from these environmental effects.

Sun Exposure

Dermatoheliosis

The sun is the most damaging physical element to the skin. The cumulative effects of chronic sun damage on the skin is called dermatoheliosis. Appropriate lifelong use of sunscreens, protective clothing (hats and long-sleeved shirts), and avoiding sunlight between 10 A.M. and 3 P.M. may significantly reduce the cutaneous effects of photoaging. It is now known that both ultraviolet A (320–400 nm) and ultraviolet B (290–320 nm) light are responsible for chronic damage from the sun. Therefore, the ideal photoprotectant agent shields against both UVB and UVA radiation and has a sun protection factor rating of 15 or more.

The epidermal changes that occur with chronic sun exposure range from benign hyperplasia to epidermal dysplasia and neoplasia. There is thinning of the epidermis with a predominance of atypical keratinocytes. Melanocyte hyperplasia and uneven melanin distribution lead to the blotchy pigmentation characteristic of photo-damage. With solar damage, there is deposition of abnormal clumps of elastin within the dermis, called elastosis. Actinically damaged skin, which is thicker than intrinsically aged skin, has smaller and fewer collagen fibers. Clinically, elastosis leads to wrinkling. Wrinkling is determined by solar damage as well as genetics. Facial movements play a role in those wrinkles formed at the nasolabial fold, forehead, and periorbital areas. Cosmetic treatment of aging skin is discussed in detail at the end of this chapter.

The American Academy of Dermatology consensus conference estimated that most of the UV-induced photoaging occurs within the first 20 years of life. Thus, early preventive measures play a key role in the defense against solar radiation. By age 70, the number of melanocytes per unit area decreases by 40%. Fair-skinned persons inherently have less photoprotectant melanin pigment, so they are far more susceptible to sun damage than others with greater pigmentation.

Cutis Rhomboidalis Nuchae

This condition is characterized by thickened, furrowed skin that forms a rhomboid pattern at the back of the neck. It is most often seen in men. Treatment is not necessary, unless it poses an aesthetic problem for the patient.

Nodular Elastoidosis with Cysts and Comedones (Favre–Racouchot Syndrome)

Cysts and comedones develop most commonly at the temporal and periorbital areas in patients with advanced actinic damage. Clinically, they appear as either a blackhead (filled with a greasy blue-black plug) or a whitehead with no apparent opening (milium). Like cutis rhomboidalis nuchae, this condition does not require medical intervention, unless it is cosmetically bothersome to the patient.

Colloid Milium

Clinically, colloid milium is an entity in which numerous, translucent papules, approximately 2 mm in diameter, form on the dorsal hands and face. Histologically, the dermis is expanded by discrete lobules of homogeneous, eosinophilic material called colloid, formed after years of overexposure to sunlight.

Senile/Solar Purpura (Bateman’s Purpura)

This term refers to the transient red-purple patches on the forearms and dorsal hands in older persons (Fig. 58.1). The elastotic dermis does not provide adequate structural support for the blood vessels. Therefore, even minor trauma to the aging skin can cause small vessels to rupture and form purpuric lesions.

Stellate Pseudoscars

These white, atrophic, irregularly shaped scars form after minor trauma. They are commonly found on the fore-