Dermatologic Cosmetic Surgery 1:

Skin Anatomy, Derm Conditions, Treatment Modalities

Art of Skin MD
Solana Beach, CA

ABCS Board Review

March 11, 2022

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Disclosure

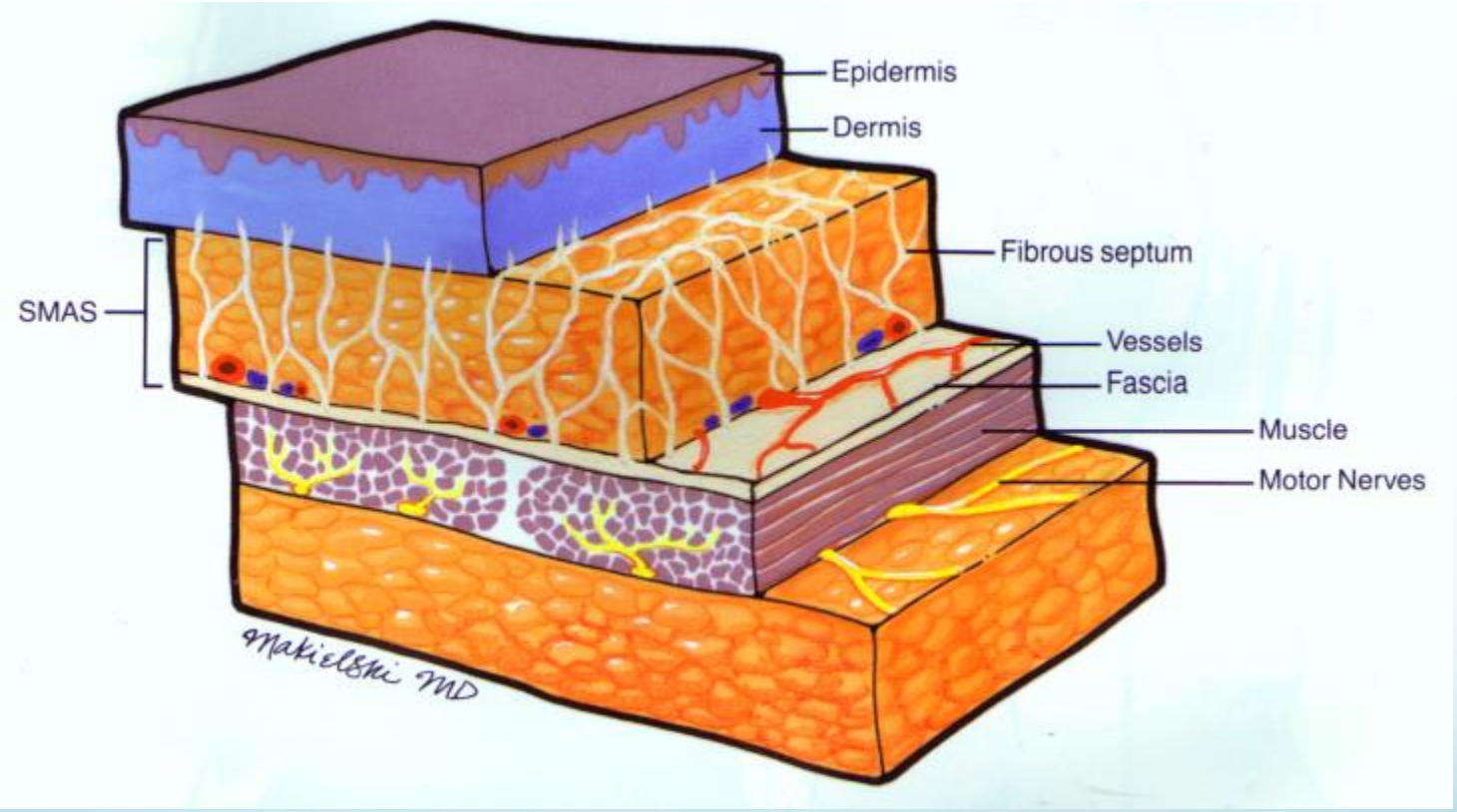
- Galderma: speaker, physician trainer, clinical investigator, advisory board member
- Allegan/Kythera: speaker, trainer, clinical investigator, advisory board member
- Merz: advisory board member, clinical investigator
- Lumenis: speaker, physician trainer
- Lutronic: speaker, consultant
- BTL: speaker, clinical investigator, advisory board member
- Alastin: speaker, clinical investigator
- Revision: Advisory Board, clinical investigator
- Elta MD: Advisory Board
- L'Oreal: clinical investigator, consultant

Topics for Discussion

- Skin Anatomy and Function
- Wound Healing
- Skin Aging and Classification
- Treatment Modalities for the Skin
- Common Skin Lesions for the Cosmetic Surgeon

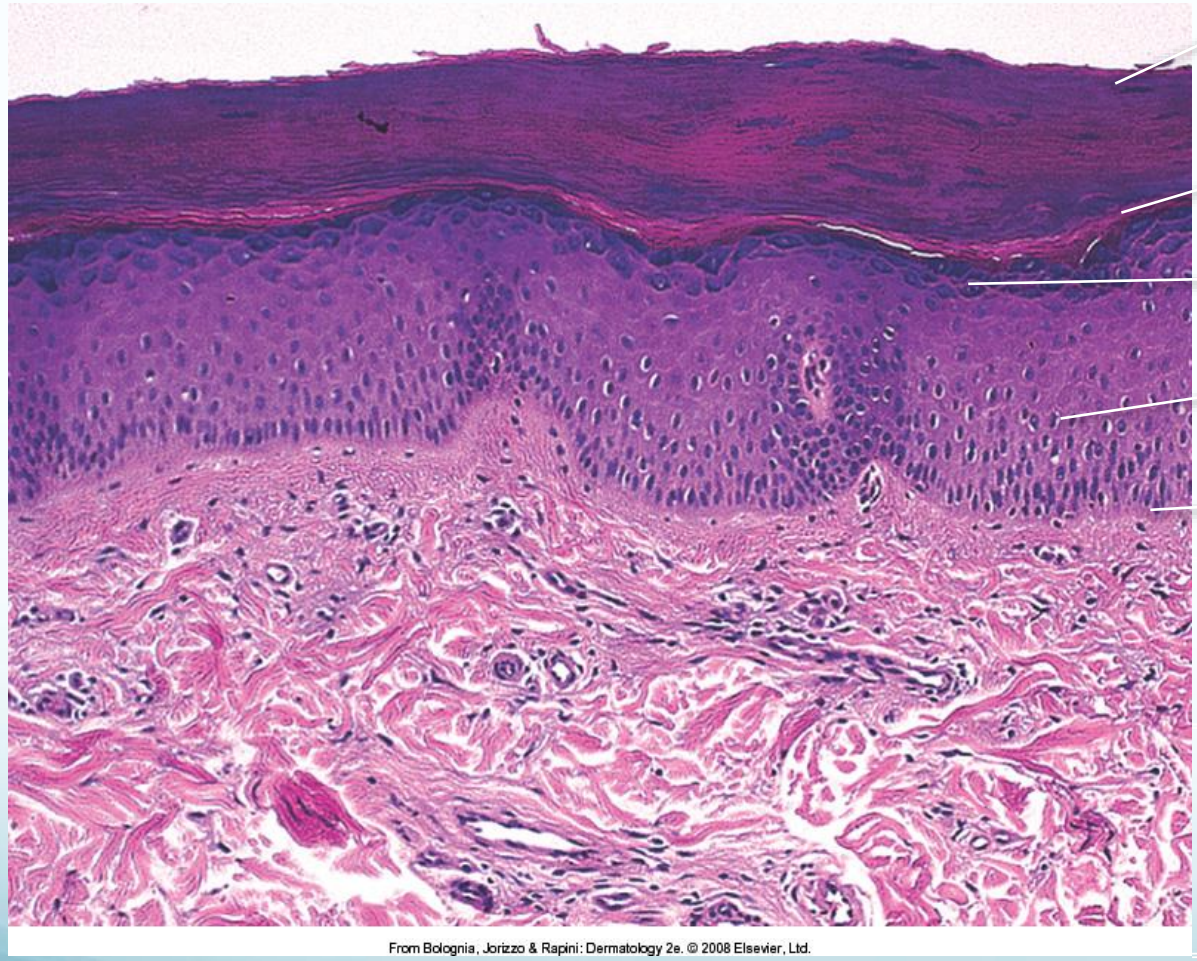
Skin Anatomy & Function

Structural Components of the Skin & Subcutaneous Tissue



Cross-section of the SMAS in the lower face

Epidermis & Dermis



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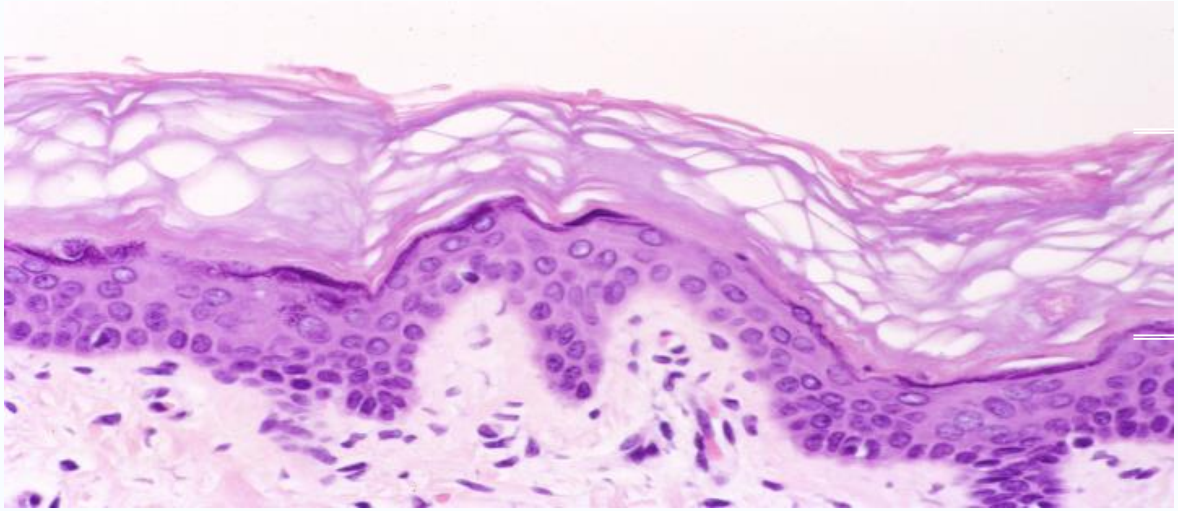
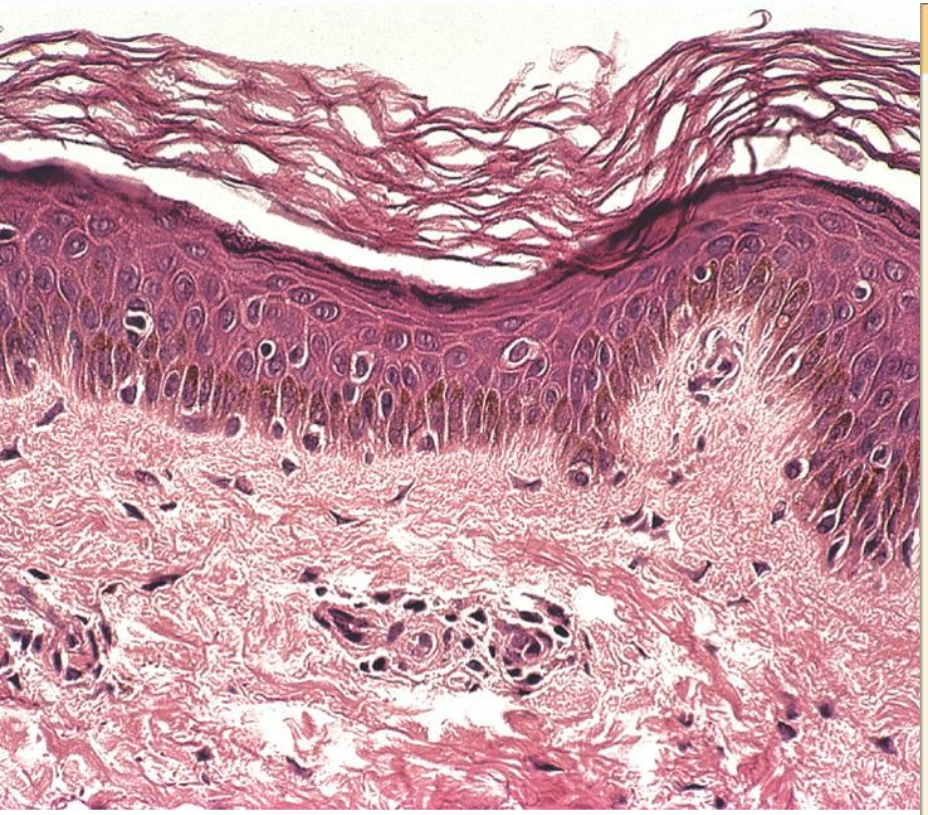
H&E preparation of palmar skin

Stratum Corneum



- Cornified, stratified squamous epithelium; no nuclei
  - Metabolically active
- Mechanical protection and barrier
  - Important for chemical peeling, laser resurfacing
- Average thickness 0.1mm (0.04 mm to 1.6 mm)



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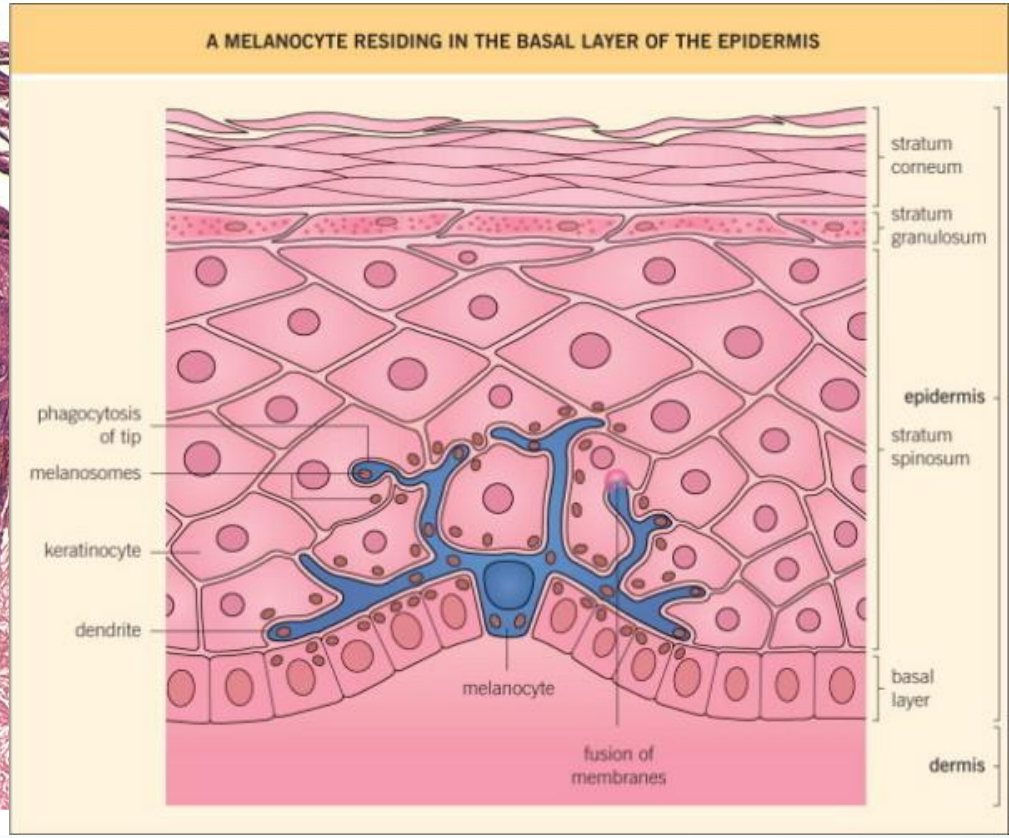
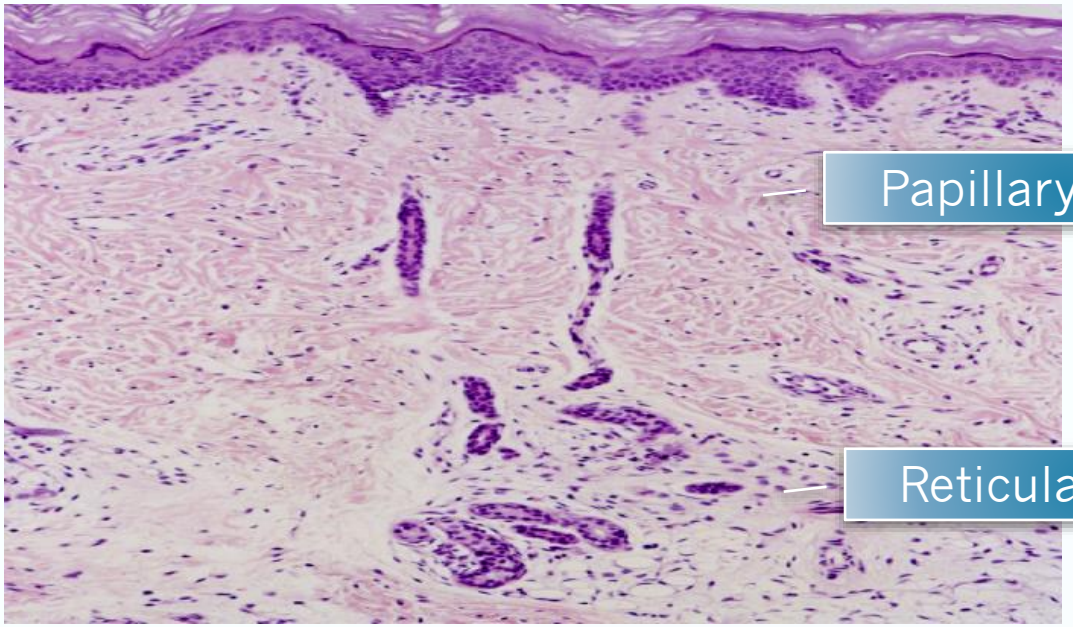


- Epidermal turnover every 30 days
  - Slower turnover when surface intact
  - Faster turnover with removal of stratum corneum

Cells of the Epidermis

- Keratinocytes: 80% of total epidermis
  - Provide structure
- Melanocytes
  - Basal layer of skin; protective pigmentation
    - Facultative melanogenesis: increase production and spread of melanosomes in response to UV exposure (protects DNA)
  - 1:4 to 1:10 per keratinocytes
- Langerhans Cells
  - Immune response/antigen processing to present to T cells
- Merkel Cells
  - Basal layer of epidermis
  - Mechanoreceptors for tactile sensitivity

Dermis



- Papillary (upper 1/3) dermis
  - Location of majority of skin appendages
- Reticular (lower 2/3) dermis—extends to fat
  - Skin destruction through deep dermis leads to scarring

Dermal Composition

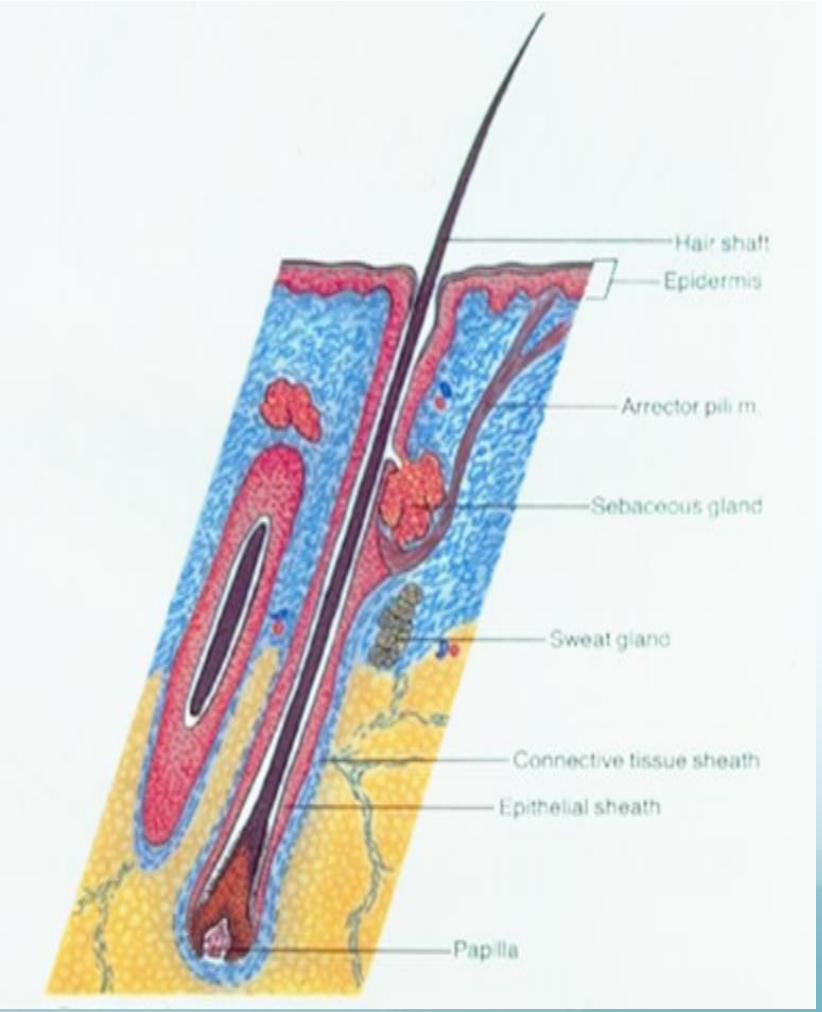
- Non-cellular connective tissue
  - Collagen (70% dry weight)
  - Elastic fibers (3% dry weight)
  - Ground substance (27% dry weight)
    - Mostly proteoglycans
- Nerves
- Blood vessels
- Lymphatic vessels
- Muscles (arrector pili muscle)
- Folliculo-sebaceous units
- Apocrine-eccrine glands

Hair Follicles

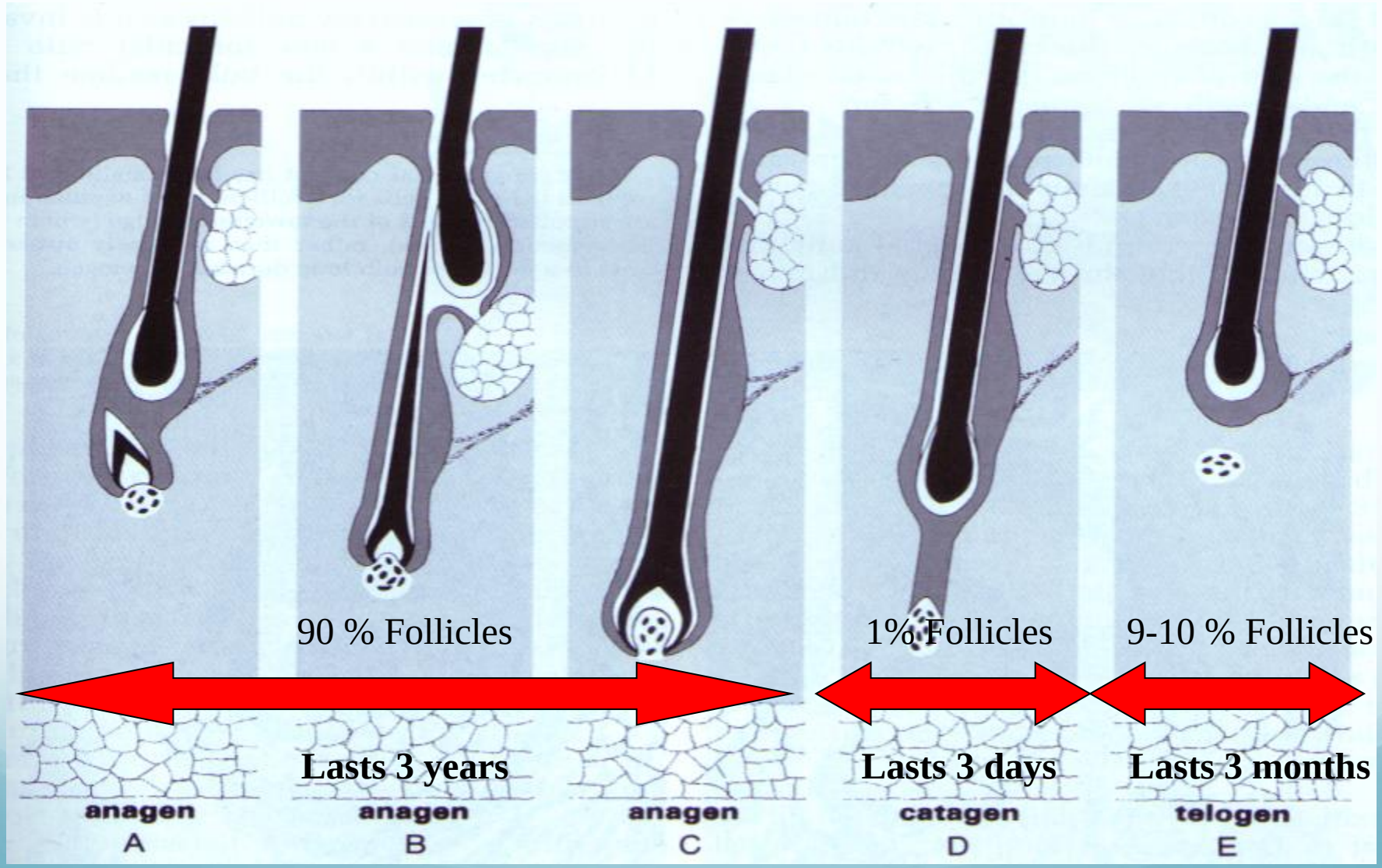
<u>Four Categories</u>:

Straight, Spiral, Helical, Wavy

1) Infundibulum - ostia to sebaceous ducts
2) Isthmus - sebaceous ducts to arrector-pili muscle
3) Lower segment - arrector-pili muscle to Adamson's fringe (keratogenous zone ends below) and bulb



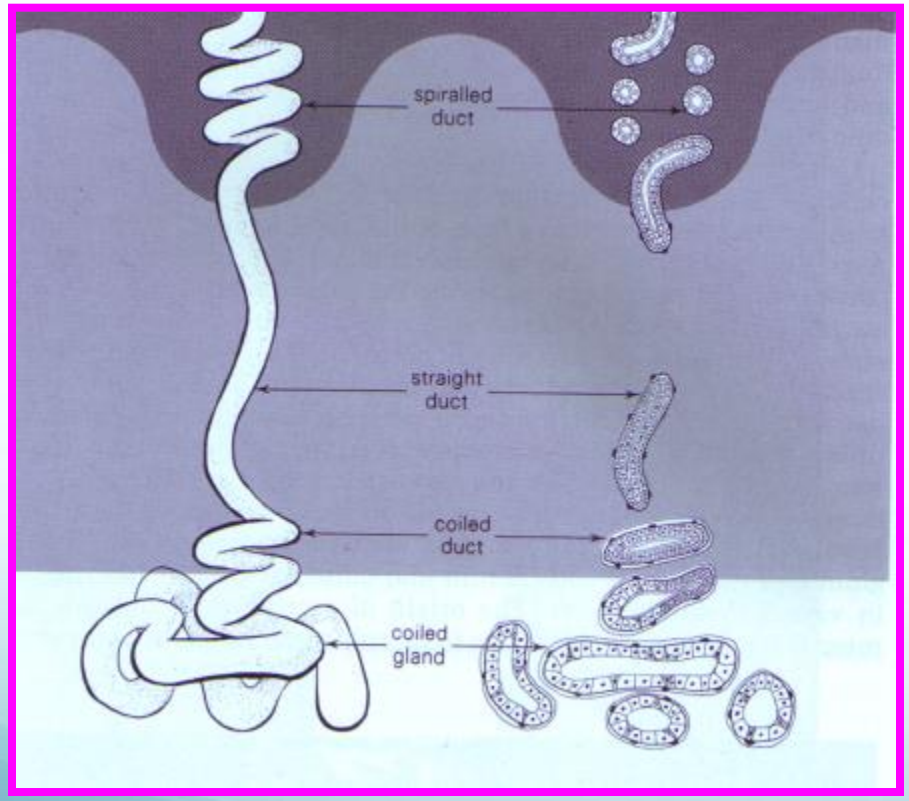
Trichoregulatory Cycle

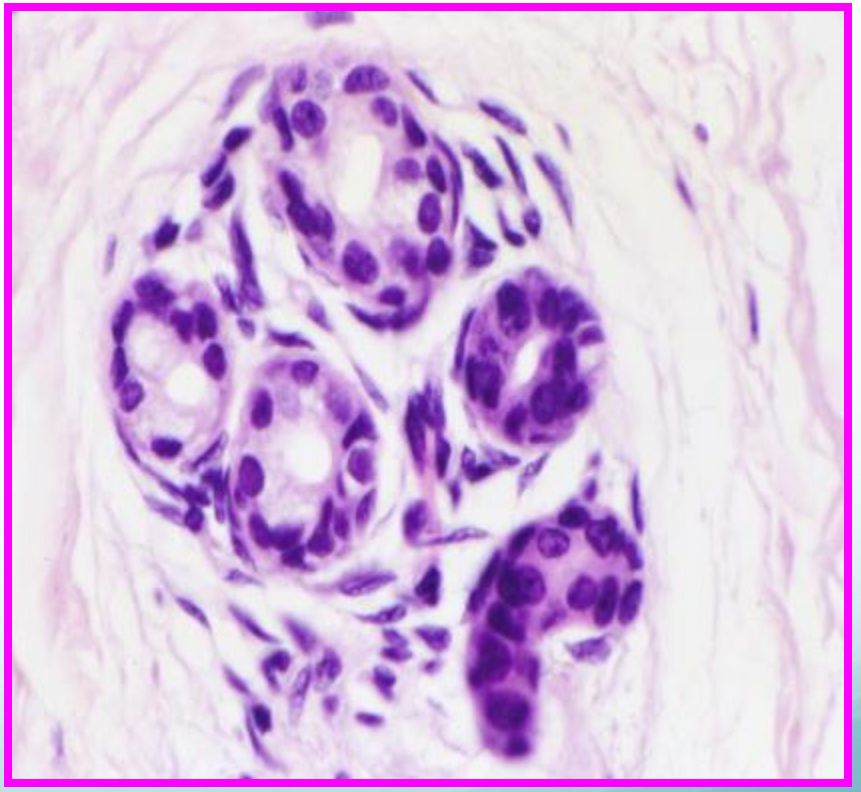


Eccrine Glands

- Only true sweat glands in humans
- Concentrated on palms, soles, axillae, forehead
- Composed of secretory and myoepithelial cells
- Serve as reservoir sites of slow re-generation after chemical peeling, dermabrasion and laser resurfacing procedures

Eccrine Glands



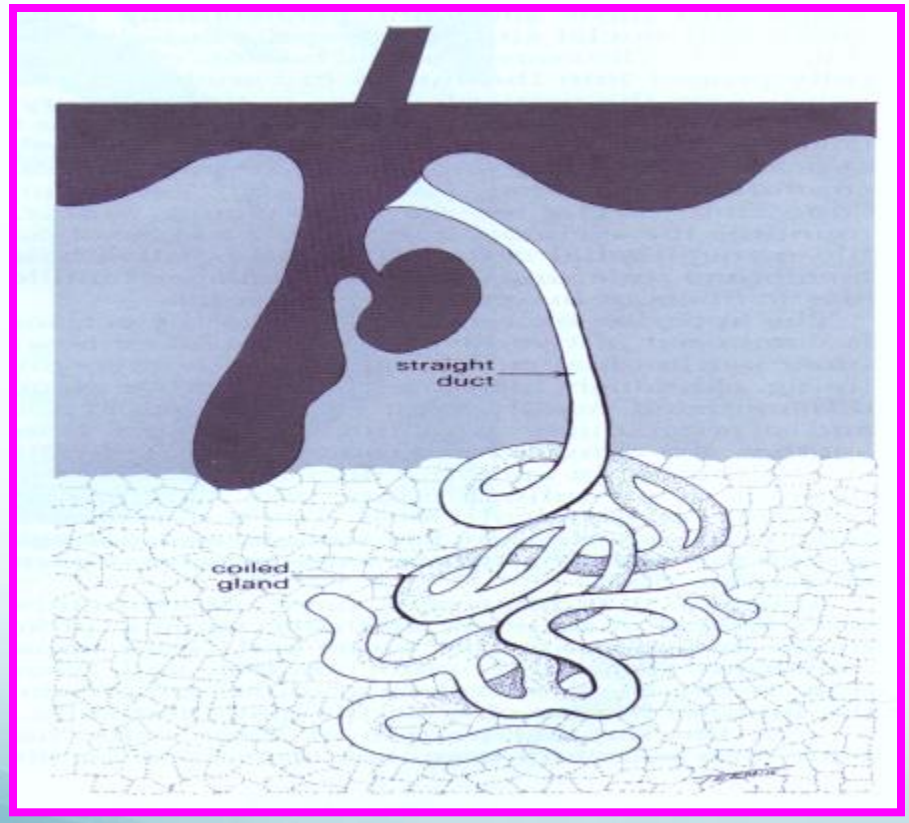


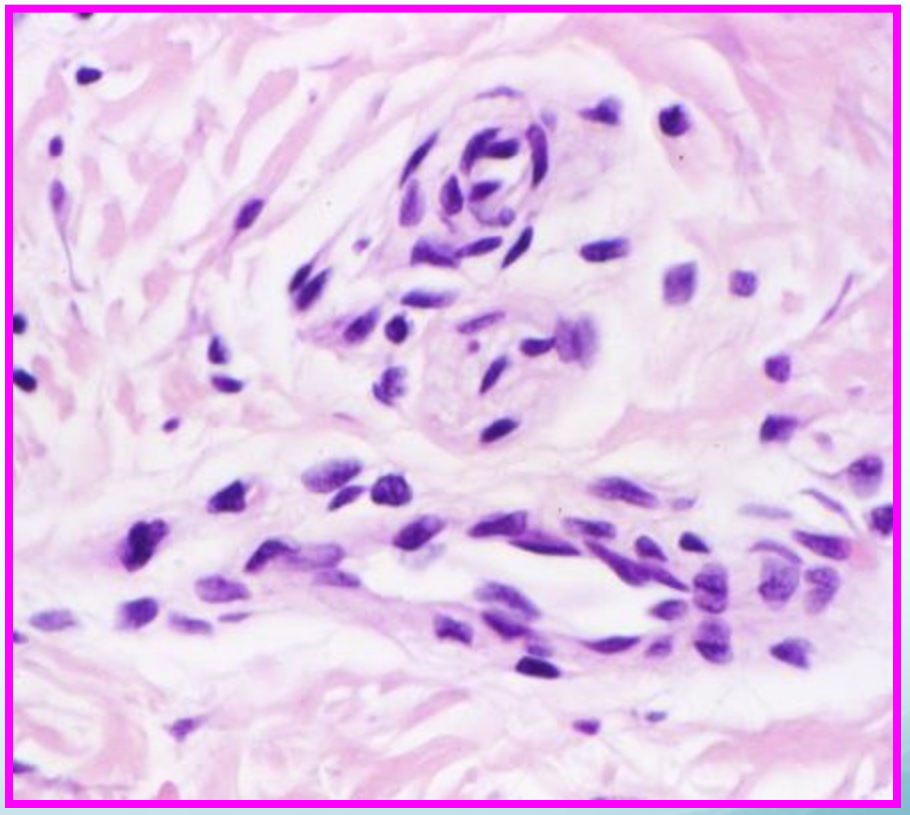
Apocrine Glands

- Most with no known function

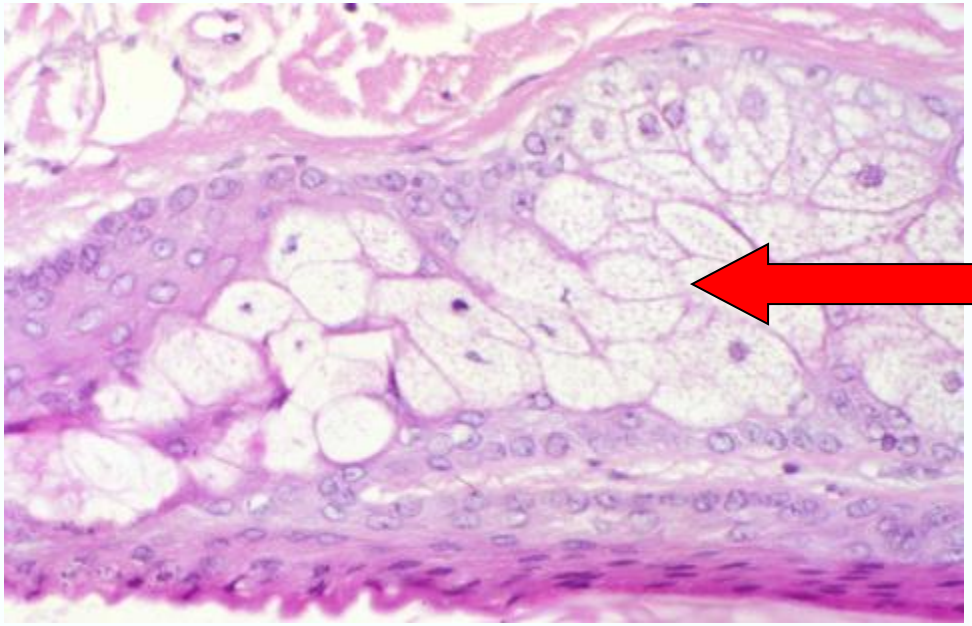
- Found in the external ear canal (ceruminous glands), eyelids (Moll's glands), axillae, areolae, peri-umbilical area, perineum, anus, mons pubis, labia minora, prepuce, and scrotum

Apocrine Glands





Basic Unit



Primary Fat Microlobule

Gynoid Distribution (Women)

Breasts
Buttocks
Hips
Anterior thighs
Inner aspect of knees
Lower abdomen
Pubis

Android Distribution (Men)

Nape of neck

Deltoid

Epigastric regions

Wound Healing

Wound Healing

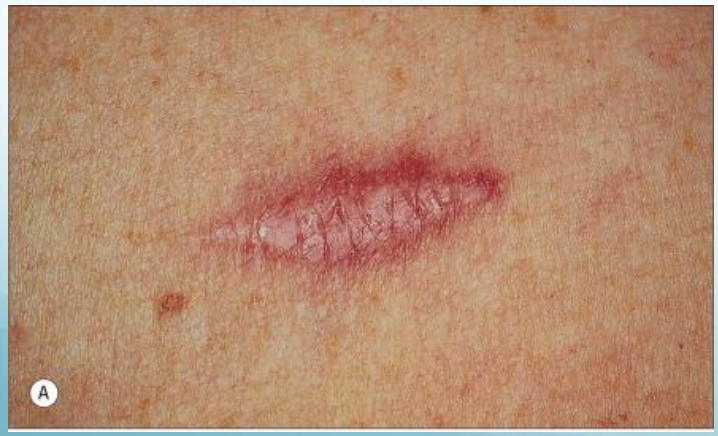
- Occurs in 4 phases:
  - **1. <u>Vascular phase</u>:**
    - Initial vasoconstriction→vasodilation; formation of hemostatic plug
  - **2. <u>Inflammatory phase</u>:**
    - Neutrophils (1st 24 hours) → monocytes & macrophages (tolerate low oxygen tension)
    - Secretion of various angiogenic, wound debridement, and collagen factors (histamine, serotonin, kinins, PGs, growth factors)
  - **3. <u>Proliferative phase (re-epithelialization)</u>:**
    - As early as 24 hours, **cells begin to migrate from wound edges and adnexa (fibronectin)**
    - Type III collagen formed first
    - **Occluded wounds facilitate keratinocyte migration; may allow moist environment for healing and decrease risk of infection**

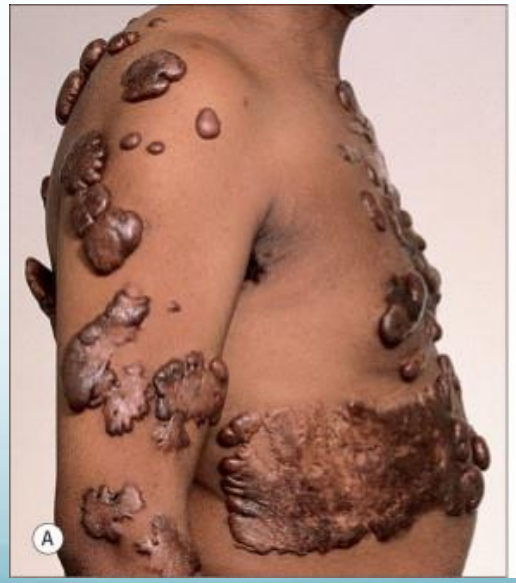
Wound Healing

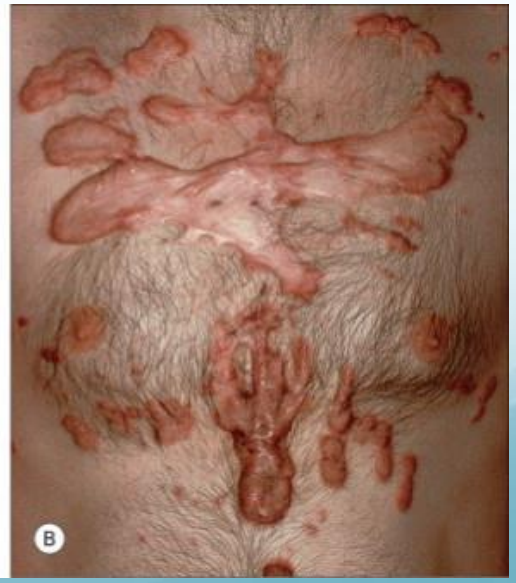
- Occurs in 4 phases:
  - **4. <u>Maturation phase (wound contraction/remodeling):</u>**
    - **Collagen III dissipates, collagen I forms**
    - **Wound strength—tensile strength increases with time:**
      - **10% original at 2 weeks**
      - **40% at 1 month**
      - **70-80% at 1 year**

Abnormal Wound Healing

- Hypertrophic Scar—thickened, does not extend beyond wound edges
- Keloid—thickened wound beyond wound margin
  - Risks: darker skin types (16% of African American population), adults, area of chest/upper trunk, ear lobes







Keloids

- Treatment:
  - Must continue to treat if symptomatic (enlarging, painful, or pruritic)
  - Treatment options:
    - **Intralesional steroids: most commonly used modality**
    - Intralesional 5-FU
    - **Surgical excision should be combined with other treatment**
      - Intralesional therapy, radiation, topical imiquimod
    - Silicone gel sheeting, pressure
    - Intralesional rapamycin, interferon-α-2b, verapamil
    - Pulsed dye laser, IPL, botulinum toxin type A

Scar Revision

- **May be given scenarios of how to treat**
- Early scar: e.g. 1 month or less
  - Consider occlusion if erosion/ulceration present
  - Silicone gel sheeting
- Redness in scar: consider pulsed dye laser
- Tethered scar: subcision +/- filler augmentation
- Burn scar, textural abnormalities (acne scar): consider laser resurfacing (fractional CO_2, Erbium:YAG)
- Contracted scar line: consider Z-plasty

Wound Infection

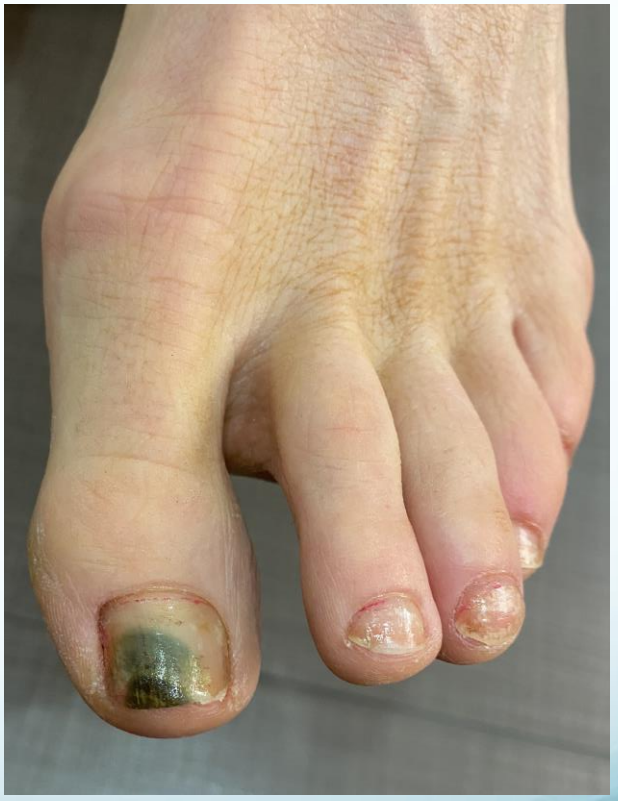
- Always obtain culture for bacteria with antibiotic sensitivities prior to treating
- Remember cardinal signs and symptoms of infection—scenarios are likely
  - Think patient safety first with these questions
- Specific microbes/infections to consider:
  - MRSA
  - Pseudomonas
  - Anaerobes—often occur below the waist
  - Mixed flora (necrotizing fasciitis)

Methicillin-resistant Staph aureus

- Community-acquired (CA-MRSA) most common subtype
  - Bacterial alteration of the penicillin-binding protein (PBP2a) leading to resistance to β-lactam antibiotics
  - Mutation: *mecA gene* produces the PBP2a protein
    - *mecA gene* contained within a mobile unit of the bacterial DNA called the *SCC (staphlococcal cassette chromosome)*
    - the cassette codes for virulence factors such as PVL (*Panton-Valentine leukocidin)*
- CA-MRSA most commonly causes furunculosis
- Rx: oral abx (doxy, TMP-SMX, azithromycin, vancomycin) and antimicrobial washes/ointment
  - Nares and perirectal area may have colonization

Pseudomonas aeruginosa

- Gram-negative bacteria; "water-loving"
- Grape-like odor
- Forms pigments that can discolor nails green (Pyocyanin)
- Common cause of hot tub folliculitis and otitis externa
  - Cover for *Pseudomonas* when suspected ear infections occur
  - Rx: fluoroquinolones



Necrotizing Fasciitis

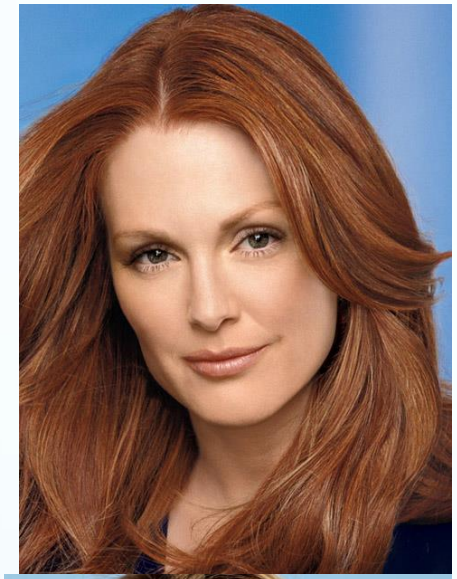
- Life-threatening, rapid destruction of subcutaneous tissue and fascia caused by
  - Type 1: polymicrobial—anaerobic and aerobic bacteria
    - Strep, Staph (including MRSA), *E. coli, Bacteroides, Clostridium*
  - Type 2: group A streptococcus—10% of cases
- Clinical features
  - Hot, red, shiny, painful skin, rapidly expanding, progressing from red/purple→blue/gray over 36 hours
  - Malodorous, bullae may form, death of tissue
  - Anesthesia due to death of cutaneous nerves
- Requires early recognition, surgical debridement, and early antibiotic coverage

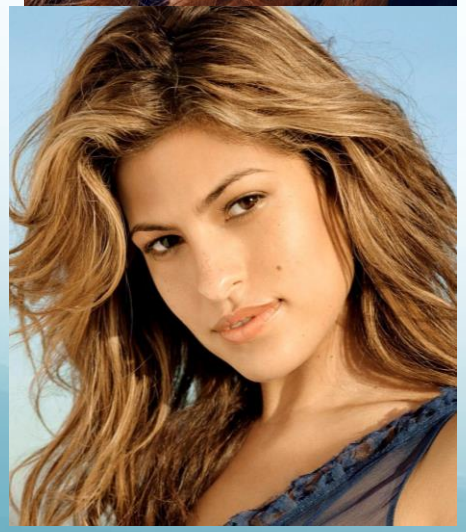
Skin Classification & Aging

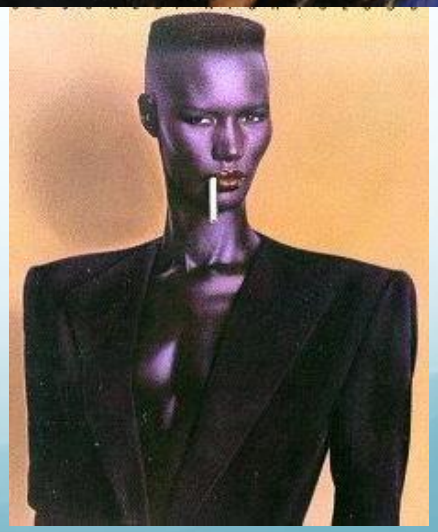
Fitzpatrick Skin Type Classification

Skin Type	Skin Color (example)	Tanning Response
I	White (Albino, Celtic—fair skinned, freckled, red or blond hair, blue eyes)	Always burns, never tans
II	White (Scandanavian—fair skin, less freckled, blue/hazel/green eyes)	Always burns, sometimes tans
III	White (Middle Europe—German, Italian, Mediterranean—fair skin brown hair, brown eyes)	Sometimes burns, usually tans
IV	Light Brown (Hispanic, Asian, light African American skin)	Rarely burns, tans easily
V	Brown (African American, Eastern Indian, Middle Eastern)	Very rarely burns, tans very easily
VI	Black (very dark African skin)	Never burns , deeply pigmented

Fitzpatrick Skin Type Classification



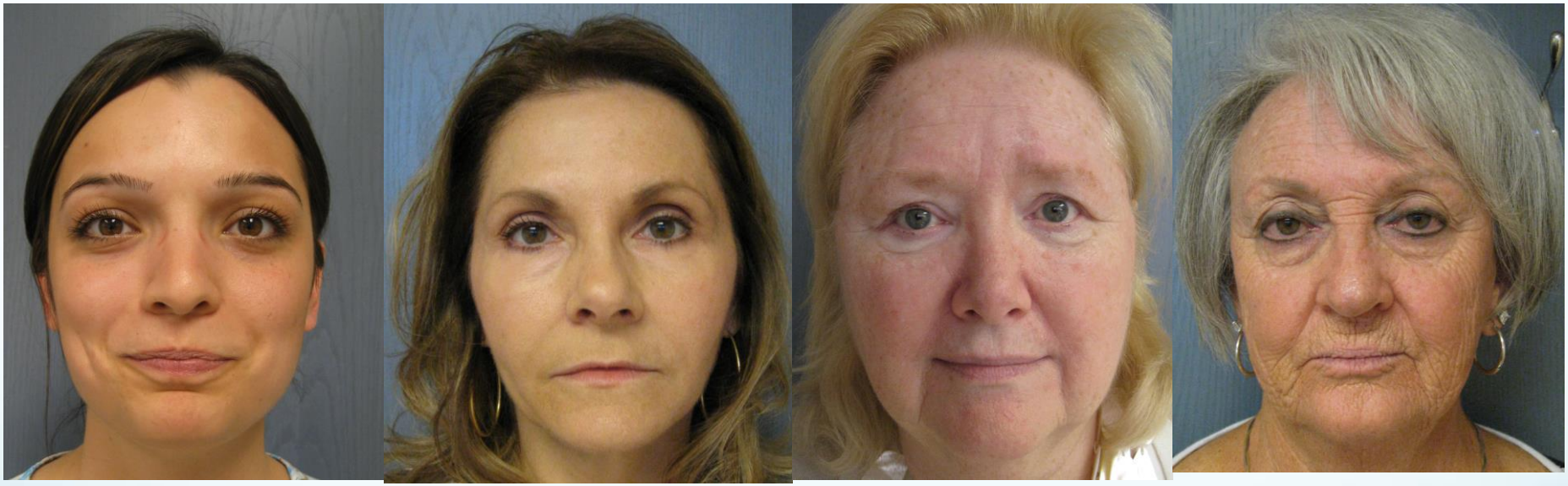




Glogau Classification for Photoaging

Glogau Class	Description
I	Age: 20-34 Early photoaging; mild pigment alteration; no keratoses; no/minimal wrinkling
II	Age: 35-49 Moderate photoaging, early solar lentigines; +/- keratoses; wrinkles with expression
III	Age: 50 Advanced photoaging with obvious dyschromia; visible actinic keratoses, static wrinkles
IV	Age: 60+ Severe photoaging with sallow color to skin; history of skin cancer(s); confluent/severe wrinkles

Glogau Classification of Photoaging



I II III IV

Three Factors Influence Skin Condition

Genetic Aging of the Skin

Cause	Effect
Decreased vascularity	Yellow skin (sallow)
Dermal thinning	Atrophy
Decreased dermal cellularity	Irregular texture
Loss of elastic fibers, decreased recoil	Fine lines, skin laxity

UV-related Changes of the Skin

Cause	Effect
Altered cell maturation	Actinic keratoses & skin cancer (epidermis), coarse texture (epidermis & dermis)
Melanocyte alteration	Solar lentigines, mottled pigmentation (melasma, poikiloderma of Civatte)
Decreased strength and number of collagen fibers	Irregular texture, senile purpura
Loss of elastic fibers	Fine lines, loss of tissue recoil

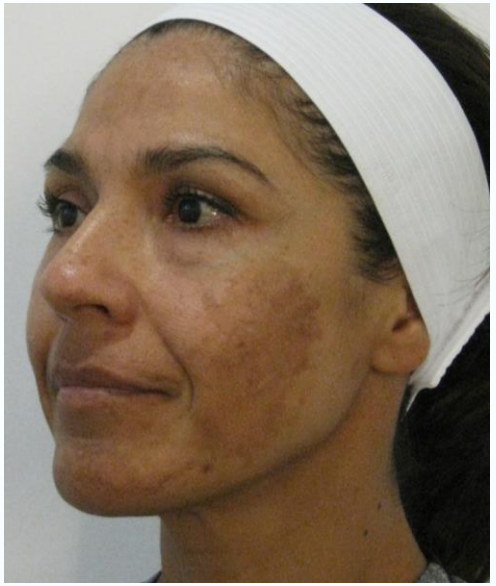
Treatment Modalities for the Skin

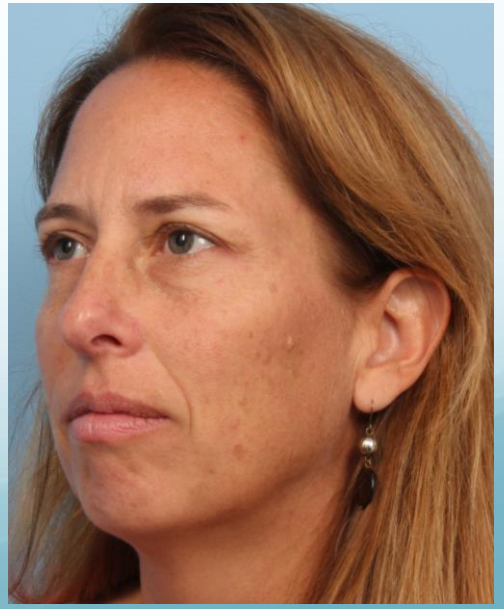
Acne Treatment

- Determine type: comedonal, inflammatory, cystic, ?scarring
- Topical treatment: BP/clinda (inflammatory/comedonal), retinoids (comedonal)
  - Isotretinoin (Accutane): nodulocystic, **abnormal wound healing** for 6-12 months after completion
- Aminolevulinic acid photodynamic therapy: ALA-PDT
  - Usually blue light source; PDL and IPL also used
- Acne scarring: treatment depends on type
  - Depressed/tethered/boxcar: subcision +/- fillers
  - Shallow scars: laser resurfacing, dermabrasion, deep chemical peels, RF microneedling, fillers
  - Icepick scars: difficult to treat, punch excision, TCA cross

Epidermal (Superficial) Aging-Pigmentation

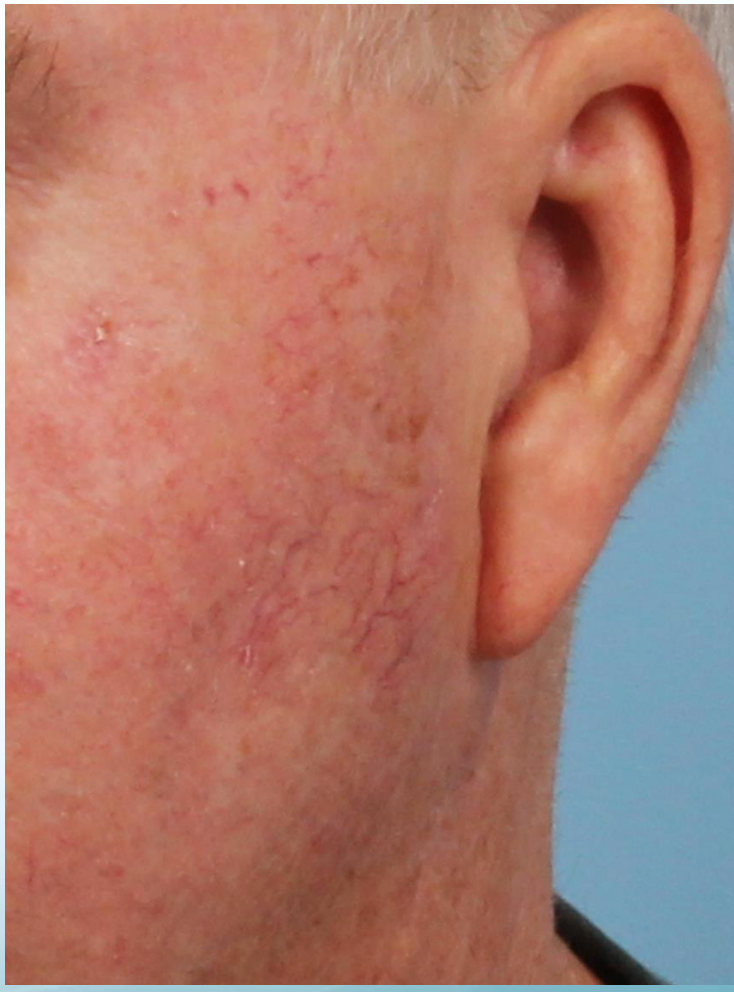
- Ex: ephelides, mottled pigmentation, melasma, solar lentigines, +/- fine wrinkling
- Topical therapy: sunblock (Zn oxide), retinoids, hydroquinone, vitamin C, ?AHAs, kojic acid, licorice root, tranexamic acid
- IPL: removes dyspigmentation, improves broken capillaries, +/- collagen production
- Q-switched or thulium lasers
- Light to medium depth peels





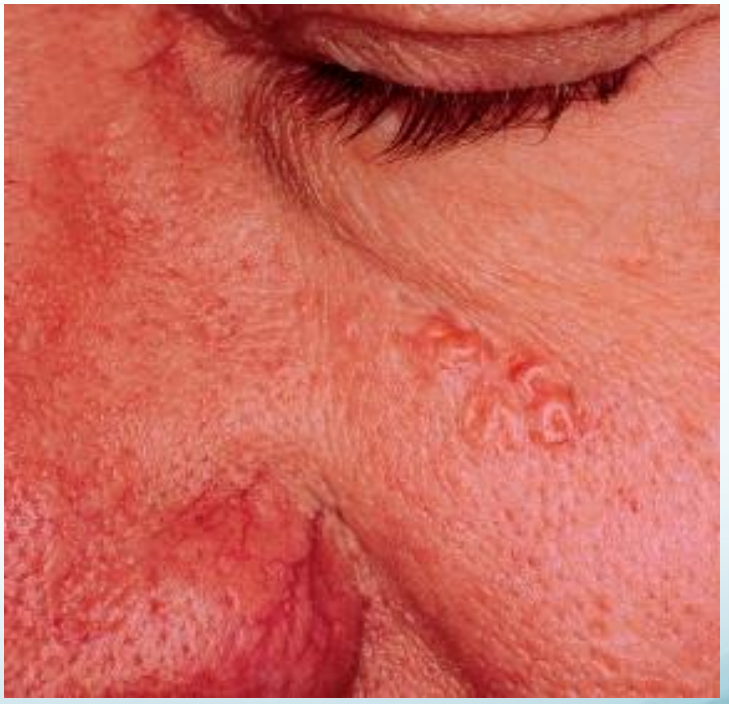
Superficial Aging: Vascularity

- Usually superficial dermis:
  - Broken capillaries, background erythema (rosacea)
- Treatment modalities
  - IPL, PDL, ALA-PDT
- Post-inflammatory erythema
  - Following laser procedures or acne
  - Time, PDL/IPL, short-term topical steroids (if procedure-related)



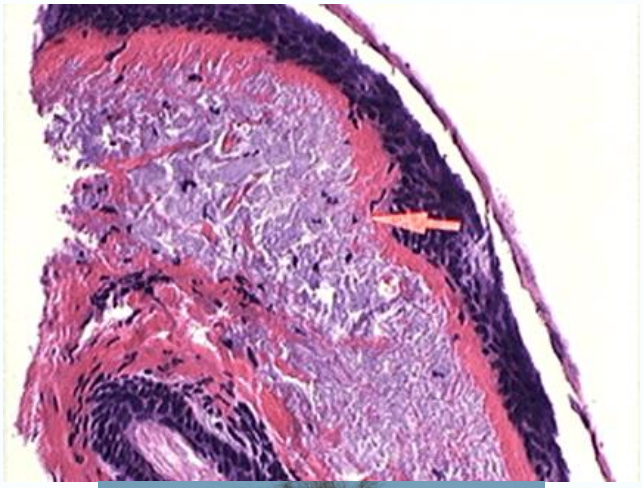
Superficial Aging: Pilosebaceous changes

- Sebaceous hyperplasia, widened pores
- Destructive modalities
  - Electrodessication, 100% TCA spot treatment, RF destruction, spot ablative laser
- ALA-PDT



Deep Dermal Changes

- Clinical Features
  - Rhytides, scars, loss of elasticity
- Treatment
  - Combined treatment modalities
  - Topical retinoids
  - RF/IR technology
  - Laser resurfacing, dermabrasion, chemical peels
  - Chemomodulators
  - ?Filler augmentation



Deep Tissue Atrophy

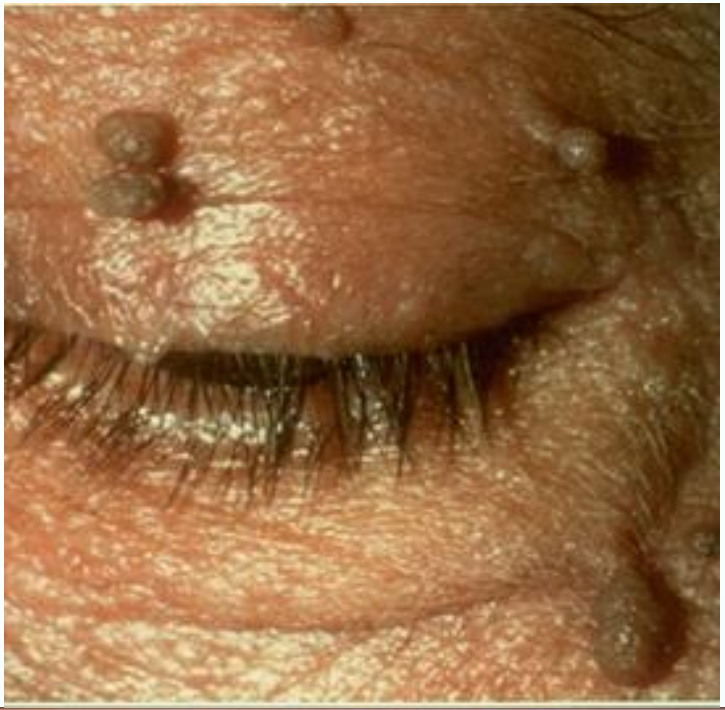
- Loss of soft tissue, muscle, bone mass resulting in...
  - Severe rhytides
  - Lipoatrophy
  - Muscle atrophy
  - Bony resorption
- Treatment
  - Soft tissue augmentation
  - Prostheses
  - Resurfacing/tissue excision

Common Skin Diagnoses for the Cosmetic Surgeon

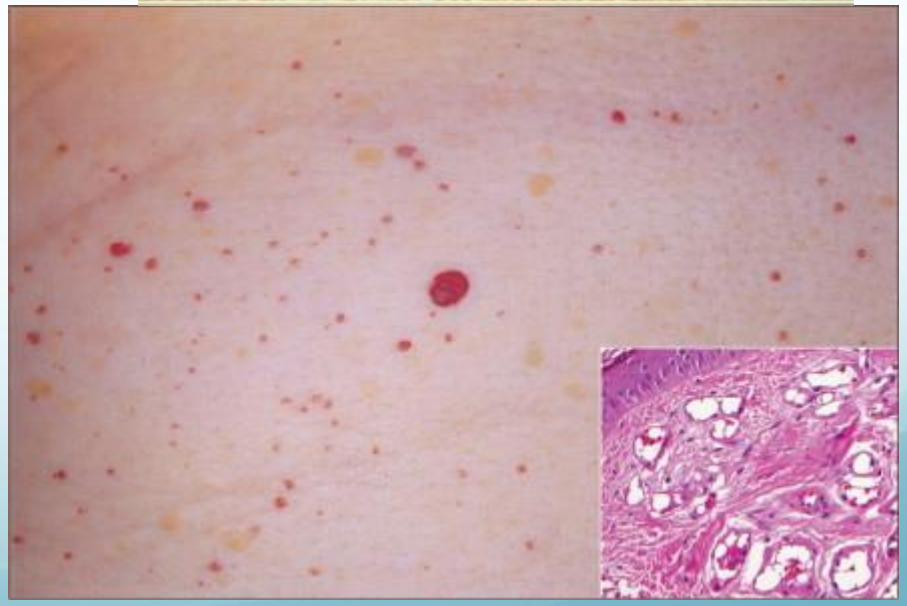
Skin Lesions

- Benign
  - Acrochordons (skin tags), seborrheic keratoses, cherry angiomas, sebaceous hyperplasia
- Malignant
  - BCC
  - Actinic keratoses → SCC
  - Melanoma
  - Consider patient safety
    - If enlarging, changing, symptomatic, or bleeding, **BIOPSY it!**

- Acrochordons (skin tags)
  - Body folds
  - Genetic, weight related, EGF-sensitive

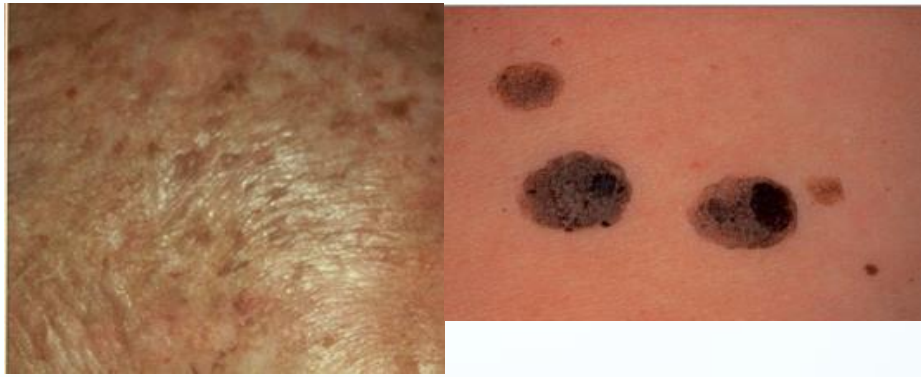


- Cherry angiomas
  - Benign vascular proliferations
  - Common over age 30, especially on trunk
  - Bleed when traumatized



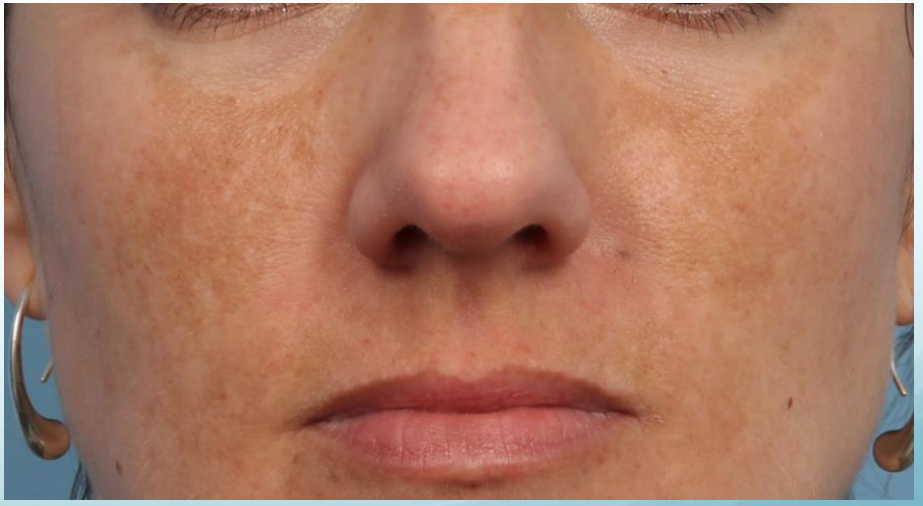
Benign Pigmentary Skin Lesions

- **Solar lentigines**
  - Epidermal pigmentation due to UV exposure



- **Seborrheic keratoses**
  - Benign epidermal growths
  - May be mistaken for melanoma

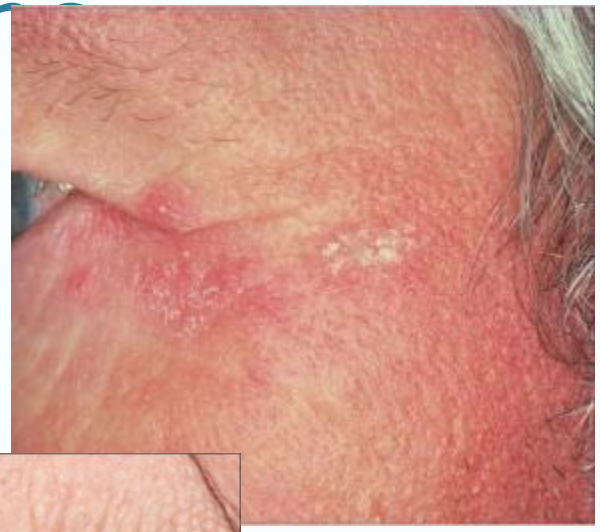
- **Melasma**
  - Epidermal or dermal pigmentation
  - Related to UV exposure
  - Hormonal component, females, pregnancy, heat/inflammation

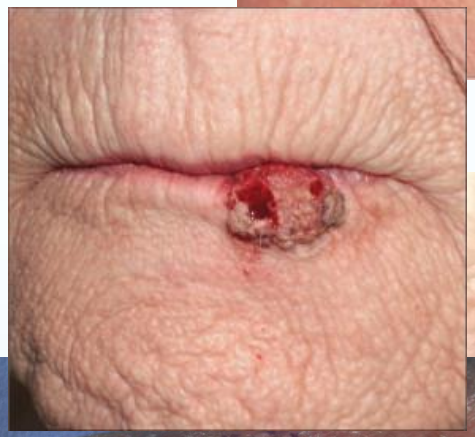


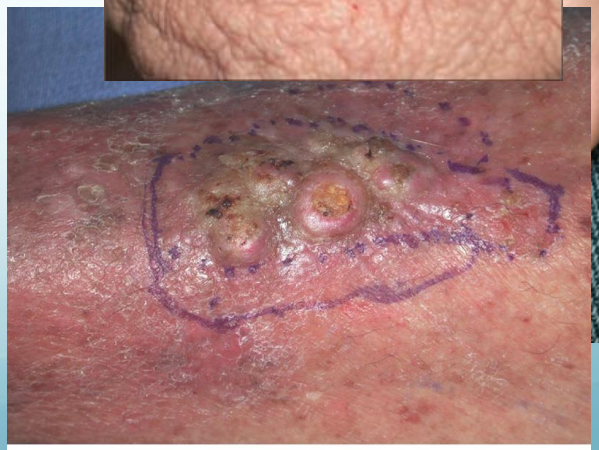
Pre-Malignant/Malignant Skin Lesions

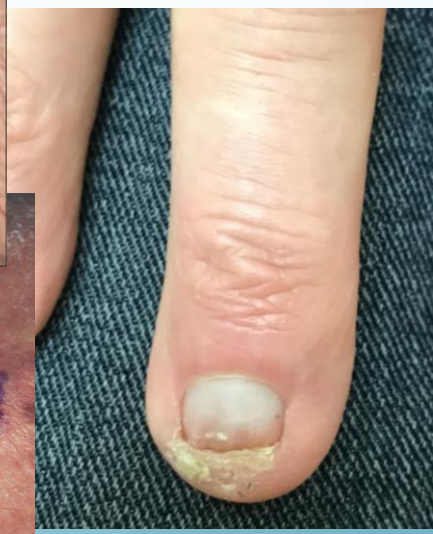
- Actinic keratoses
  - Related to UV; common in skin types I-II
  - premalignant
  - ~3% progress to SCC

- Squamous cell carcinoma
  - 2nd most common skin cancer
  - May metastasize; lip & ear high risk sites
  - May occur in chronic ulcers, burns, warts, or scars



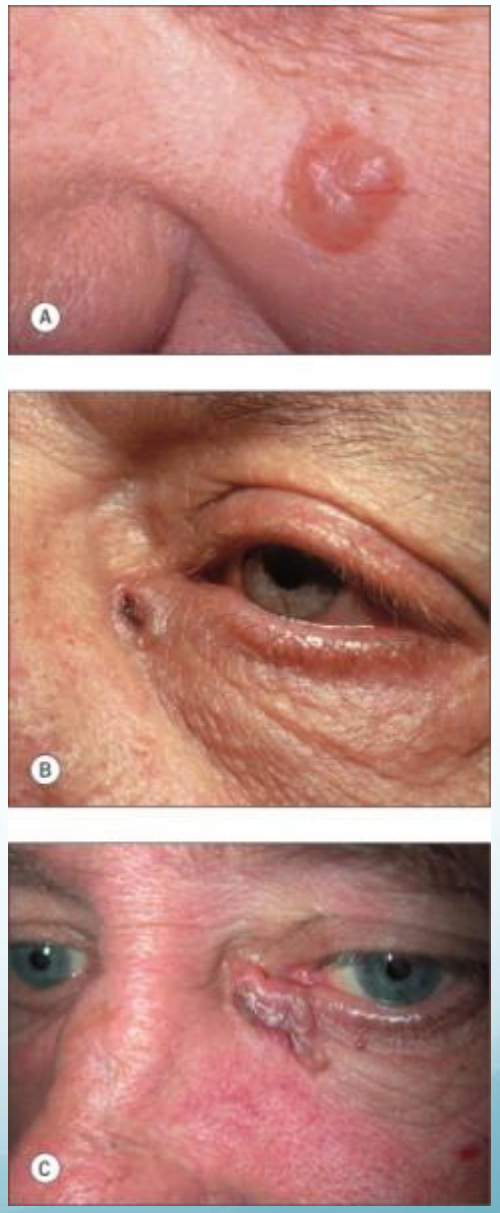






Malignant Neoplasms

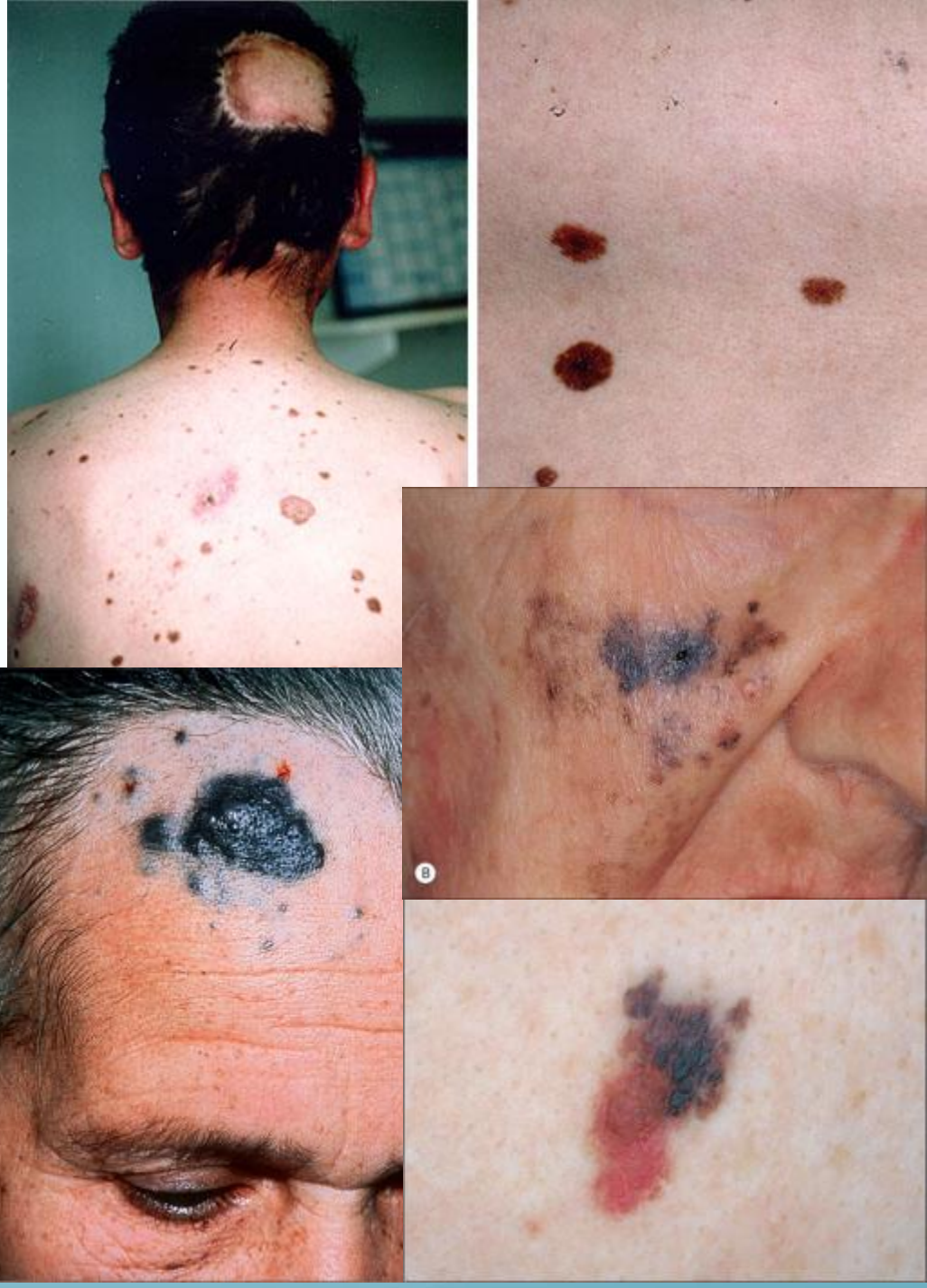
- Basal cell carcinoma
  - #1 most common skin CA
  - Related to intense, intermittent sun exposure
  - May bleed or ulcerate
  - Rarely metastasizes



Dysplastic Nevi & Melanoma

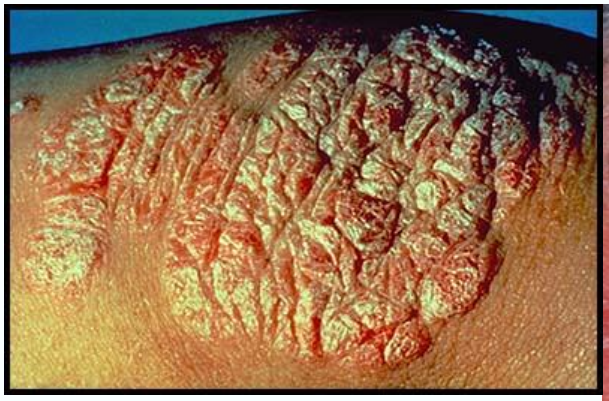
- Dysplastic Nevi
  - Familial trait
  - Worsened by UV
  - Major risk factor for melanoma

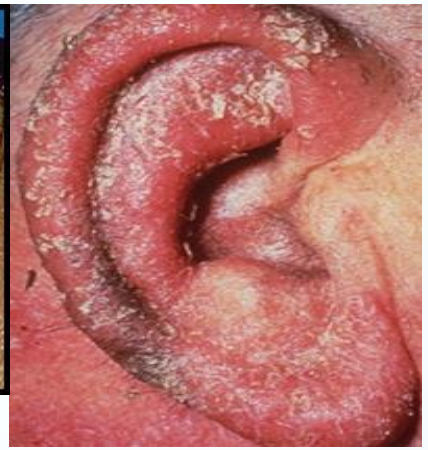
- Melanoma
  - 80% related to UV, strong genetic component
  - 2x risk with tanning bed
  - Shins, upper back
  - Prognosis related to tumor thickness, ulceration, mitotic rate
    - 1/5 cases lethal
  - ABCDEs

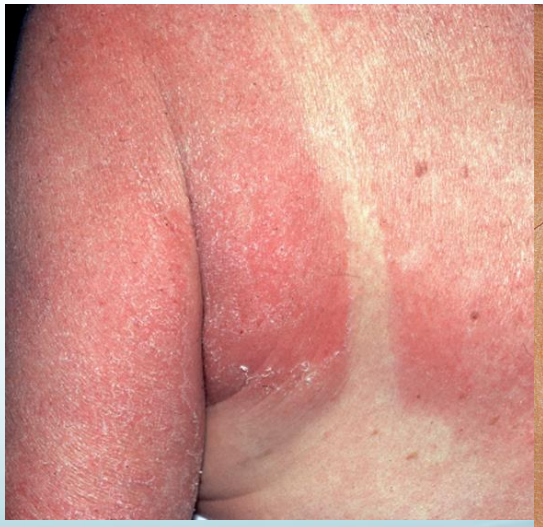


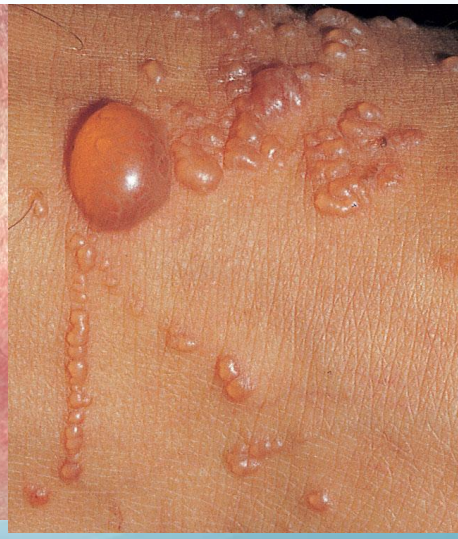
Inflammatory Skin Conditions

- **Acne vulgaris**
- **Psoriasis**
  - 1% population, genetic predisposition
  - Elbows, knees, scalp, arthritis
- **Seborrheic dermatitis** (inflammatory dandruff)
  - Scalp, eyebrows, NLFs, alar crease, ears
- **Photosensitizing or allergic contact dermatitis**
  - Areas of sun exposure or geometric shape









End of Section I:

Dermatologic Cosmetic Surgery