ANATOMY AND PHYSIOLOGY OF WOUND HEALING
Objectives

- Describe Key Components of Skin Anatomy and Physiology
- Understand the Main Functions of the Skin to Promote Homeostasis
- Recognize Differences Between Acute and Chronic Wound healing
- Understand the Phases of Wound Healing
- Describe Normal and Normal Scar Tissue
- Recognize Various Types of Wound Closure
The short version: The skin is the largest organ on the body, it makes up about 1/6th of total body weight. It is a major part of the passive immune system and it acts as a protective wrap to keep harmful environmental agents out (micro-organisms) and allows our interaction with the world. The functions of our skin are four-fold: Protection, Sensation, Thermoregulation, and Metabolic functions (Vitamin D is synthesized in the epidermis and subcutaneous fat is a major energy store). The skin is the body’s largest organ. About six pounds of skin cover eighteen square feet on an average adult. The top layer of skin is called the epidermis. It protects the underlying skin layers from the outside environment and contains cells that make keratin, a substance that waterproofs and strengthens the skin. The epidermis also has cells that contain melanin, the dark pigment that gives skin its color. Other cells in the epidermis allow us to feel the sensation of touch and provide the body with immunity against foreign invaders like germs and bacteria. The very bottom layer of the skin is the hypodermis. It contains the fat cells, or adipose tissue, that insulate the body and help it conserve heat. The layer between the epidermis and the hypodermis is the dermis. It contains the cells that give skin strength, support, and flexibility. As a person ages, the cells in the dermis lose their strength and flexibility, causing the skin to lose its youthful appearance. Located in the dermis are sensory receptors. They allow the body to receive stimulation from the outside environment and experience pressure, pain, and temperature. Small blood vessels provide the skin with nutrients, and remove its waste products. Sebaceous glands produce the oil in the skin, which keeps it from drying out. The oil from the sebaceous glands also helps to soften hair and kill bacteria that get in the skin’s pores. These oil glands are all over the body, except on the palms of the hands and the soles of the feet.
Skin

- hair shaft
- sweat pore
- dermal papilla
- sensory nerve ending
- for touch
- stratum corneum
- pigment layer
- stratum granulosum
- stratum spinosum
- stratum basale
- arrector pili muscle
- sebaceous gland
- hair follicle
- papilla of hair
- nerve fiber
- blood and lymph vessels
- sweat gland
- pacinian corpuscle
- epidermis
- dermis
- subcutis (hypodermis)
- vein
- artery
Layers of the Skin

Epidermis

- Five Layers of cells
- Superficial to deep

Functional components
• Made up of tough, flattened cells of the protein keratin
• Cells provide barrier to injury, contaminants, light, retain water
• Keratinocytes secrete protein keratin
• Melanocytes produce melanin (pigment)
• Basal and prickle cells regenerate epidermis, produce Vit D
• Langerhans cells are a component of the immune system
Stratum Germinativum is a single cell layer
Provides germinal cells necessary for the regeneration of the epidermis
Contains melanocytes which are responsible for the pigment of the skin.
Epidermis

The epidermis has an irregular shape, resembling downward fingerlike projections called rete ridges or rete pegs

- The significance of this anatomical structure is that the dermis has upward projections
- The upward and downward projections fit together, very much like a waffle iron
- The protuberances connect, anchoring the epidermis to the dermis
- This bond also helps to prevent the epidermis from sliding back and forth across the dermis with normal movement and skin manipulation

In healthy young skin the two layers of skin move as one. This is not the case in elderly skin over the age of 60.
As the skin ages the rete ridges begin to flatten between the dermal-epidermal junction. Such epidermal/dermal flattening typical appears by the sixth decade of life. With this anchoring now diminished, there is an increase potential for the epidermis to detach from the dermis, leading to tearing of the uppermost layers of the skin, especially in the older adult population- which leads to skin tears bruising ecchymosis and an increased susceptibility to damage from pressure, friction and shear. From Advances in kin and wound care Vol 20(6) June 2007 pp 315-321. Preventing and treating skin Tears Fleck, Cynthia A. MBA, BSN, RN, ET/WOCN,CWS, DNC, DAPWCA, FCCWS
Functions of the Skin

- Protection from
  - Fluid and electrolyte loss
  - Mechanical injury
  - Ultraviolet injury
- Fluid and electrolyte balance
- Metabolic
- Synthesis
- Sensation/touch
- Communication
- Cosmesis
Aging Skin

Decrease
- Dermal thickness
- Fatty layers
- Collagen and elastin fibers
- Size of rete ridges
- Sensation and metabolism
- Subcutaneous tissue
- Sweat glands

Increase
- Time for epidermal regeneration
- Damage to skin from sun
Gerontological Changes

With these changes

- Oxygen-carbon dioxide exchange decreases
- Tissue turnover slows
- Increase occurrence of ecchymosis
- Inflammatory response decreases
- Tissue regeneration is slower
Threats to Skin Integrity

- Pressure, friction, shear
- Moisture
- Malnutrition, dehydration
- Medications (topical and systemic)
- Cognition impairments
- Immobility
- Comorbidities
- Exogenous, endogenous and iatrogenic factors

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Why Do Wounds Happen?

- Trauma
- Scalds and burns physical and chemical
- Animal or insects bites
- Pressure
- Connective tissue disorders
- Malignancy
- Immunodeficiency
- Vascular compromise, arterial, venous or
- Metabolic disease including diabetes
- Nutritional deficiencies
- Psychosocial disorders
- Adverse effects of medications
Skin Assessment

Thorough skin assessment is paramount

Prevention is key

- Address all notifiable risk factors
- Early intervention
Acute vs Chronic Wound Healing

- Acute or normal wound healing allows for repair of skin function and integrity in a timely manner, lasting from 7-10 days to 2-3 weeks.

- Chronic wound healing is a disruption of the normal healing pathway due to underlying disease processes, infection, medications, and varying causes that may last months to years.
Acute Wound Healing

Acute wounds

- Injury that occurs to the integument
- Injury triggers a series of cascade/events to initiate hemostasis and protection of internal organs
- In a healthy, uncompromised individual, acute wounds close/resurface spontaneously without complications through the phases of wound healing=can be 2-3 weeks
Chronic Wound Healing

**Chronic Wounds**

- When the phases of wound healing are delayed a chronic wound results

- Lazarus, 1992 “fail to progress through a normal orderly and timely sequence of repair or wounds that pass through the repair process without restoring anatomic and functional results”

- Clinically, wounds that do not heal within 6 weeks
Acute & Chronic Wound Differences

Acute Wounds
- Low necrotic burden
- Low bacterial burden

Chronic Wounds
- High necrotic burden
- High bacterial burden

*MMPs are a family of protein degrading enzymes*
Hemostasis

Inflammatory phase

Early

Phagocytosis and removal of foreign bodies

Late

Granulation tissue formation

Neutrophils

Macrophages

Fibroblast proliferation

Collagen synthesis

Extracellular matrix remodelling

Angiogenesis

Epithelialisation

Growth factors

Proliferative phase

Epithelialisation and remodelling

Days after wounding (log scale)

0.1 0.3 1 3 10 30 100 300

Days

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Haemostasis is secured by platelet aggregation and clot formation. The inflammatory phase begins with the arrival of phagocytic neutrophils and, later, macrophages at the wound site; they are important sources of and substrates for growth factors. The proliferative phase is characterised by the formation of new blood vessels (angiogenesis), synthesis of extracellular matrix components such as collagen, granulation tissue formation, and re-epithelialization. The extracellular matrix is continually remodelled during the final phase; an avascular scar is the end result of the healing process. Once the source of damage to a house has been removed and before work can start, utility workers must come in and cap damaged gas or water lines. So too in wound healing damaged blood vessels must be sealed. In wound healing the platelet is the cell which acts as the utility worker sealing off the damaged blood vessels. The blood vessels themselves constrict in response to injury but this spasm ultimately relaxes. The platelets secrete vasoconstrictive substances to aid in this process but their prime role is to form a stable clot sealing the damaged vessel. Under the influence of ADP (adenosine diphosphate) leaking from damaged tissues the platelets aggregate and adhere to the exposed collagen. They also secrete factors which interact with and stimulate the intrinsic clotting cascade through the production of thrombin, which in turn initiates the formation of fibrin from fibrinogen. The fibrin mesh strengthens the platelet aggregate into a stable hemostatic plug. Finally platelets also secrete cytokines such as platelet-derived growth factor (PDGF), which is recognized as one of the first factors secreted in initiating subsequent steps. Hemostasis occurs within minutes of the initial injury unless there are underlying clotting disorders.
**Proliferative Phase**

- Epidermal cells begin to migrate across the wound bed
- Fibroblast proliferation occurs and collagen is laid down forming the wound matrix
- Formation of capillary buds and loops in the collagen matrix yield granulation tissue
- This leads to resurfacing of the defect which lasts from 4-14 days
Platelets

- Platelet aggregation
- Activates factor XII (Hageman) and clot formation begins
- Platelets release growth factors such as PDGF, TGF-alpha and TGF-beta
Growth Factors

- PDGF stimulates neutrophils, macrophages and fibroblasts, inducing cell migration, proliferation and angiogenesis
- TGF-alpha is closely related to epidermal growth factor and controls epithelial cell proliferation and migration
- TGF-beta plays a role in cell differentiation, immune function and regulates wound maturation and strength
Fibrin Clot

- Fibrin clot is the foundation for the next stage of wound healing.
- The matrix allows for the migration of other cells into the wounded area to begin the inflammatory process.
- To avoid extended clotting, fibrinolysis occurs as well to allow continued migration of the cells.
Inflammatory Phase

- Occurs within first 72 hours after injury
- Neutrophils, mast cells and macrophages begin to migrate into the site of injury
- Capillaries dilate and allow fluid into the site
- These lead to the typical signs of inflammation: redness, swelling, pain and warmth
Mast Cells

- Mast cells release histamine and serotonin which attract the neutrophils and macrophages to the injured area.
- Heparin is also released and is an anticoagulant that maintains hemostatic balance.
- Histamine also causes venous permeability leading to the redness and edema noted during the inflammatory process.
Neutrophils

- One of the most abundant lymphocytes making up approximately 60% of the total white blood cell count
- One of the first cells to arrive and they reach their highest number in the first 24-48 hours
- Responsible for killing bacteria and removing debris from the wound bed, thus aiding in controlling infection
Macrophages

- Macrophages differentiate from monocytes (blood) and arrive in the wound within 24-72 hours
- Essential for wound healing as they mediate all activities during this phase
- The macrophage excretes collagenases, fibronectin (fibroblasts) and keratinocytes which aid in debridement followed by collagen synthesis
Lymphocytes

- T-lymphocytes (thymus derived) play a significant role as the defense cells mediating many mechanisms of the immune system responding to infected or malignant cells.
  - There are three types of T lymphocytes: killer, helper and suppressor.

- B-Lymphocytes are white blood cells that originate from the bone marrow and are activated by T-lymphocytes, secreting antibodies to inactivate antigens in the bloodstream; “memory cells”
Fibroblasts/Myofibroblasts

- Fibroblasts produce collagen and proteoglycans
- Necrosis, foreign bodies and bacteria delay the development of fibroblasts and capillaries
- Myofibroblasts differentiate from fibroblasts and bind tightly to each other, as well as to the wound edge, causing contraction
Mitotic activity starts within 24-72 hours and the cells begin migrating across the wound bed.

These cells require a smooth moist environment to migrate across the wound surface.

If a crater or recess occurs the tissue may stop migrating or roll and form a closed wound edge called epiboly.
Maturation Phase

- Begins day 7 and can last up to 2 years
- Fibroblasts leave the wound and collagen continues to remodel and becomes more organized
- Results in a scar that is basically avascular and its tensile strength is now about 80% of the original tissue
Types of Wound Closure

- Primary Intention
- Delayed Primary Closure
- Secondary Intention
Primary Intention

- Drawing the wound edges together to achieve closure (i.e., surgical wounds)
  - Wound closure usually occurs in 3-7 days
  - Sutures, staples, surgical glues, zippers
- Decreases the risk of infection and allows for better scarring/cosmesis
Secondary Intention

- Wound is left open due to infection or large tissue loss and closes by contraction.
- Wound edges are drawn together by contractile forces of the myofibroblasts acting at the wound edge.
- Wound closure can take weeks to months.
Delayed Primary Closure

- To relieve tissue tension and to establish free drainage
- May be necessary due to infection to decrease the risk of abscess or dehiscence
- To prepare a wound bed for skin grafting
  - Allows wound to fill in large defect with granulation tissue; grafts require base of granulation tissue for successful closure
Skin Grafts

Types include:
- Partial or split thickness
- Full thickness

Sources include:
- Autografts
- Allograft
- Xenograft
Flaps

- Skin flap
- Free flap
- Muscle flap
- Musculocutaneous flap
Partial-Thickness Skin Loss

- Involves destruction of the epidermis and can extend into but not through the dermis
- It is superficial and is usually an abrasion, blister or shallow crater
- Regeneration of tissue is full and usually without scarring
Full-Thickness Skin Loss

- Involves loss of all layers of the skin with necrosis
- Extensive damage to underlying structures such as muscle, tendon and bone
- Presents as a deep crater which may include tunneling and undermining
- Will never remodel back to original tissues due to significant tissue loss and results in significant scarring
  - Dermis cannot regenerate
Summary

- The physiology of wound healing is influenced by endogenous and exogenous factors.
- Wound closure can be facilitated by external means through proper product utilization and/or surgical correction.
- Understanding the anatomy and physiology of the skin will enhance your understanding of proper treatment options.
After all, the world is full of amazing discoveries