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INSIGHTS INTO TISSUE REPAIR: A SYSTEMATIC OVERVIEW OF WOUND HEALING

Rutuja Rangnath Khandade*, Dr. Sachin R. Hangargekar, Dr. Visveshwar Dharashive Aarti Ramdas Mekale, Rayan Sadik Sayyad, Madhav Marotirao Kabade

Shivlingeshwar College of Pharmacy, Almala.

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***Corresponding Author**

Rutuja Rangnath Khandade

Shivlingeshwar College of Pharmacy, Almala.

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ABSTRACT

Wound healing is a natural and complex biological process by which the body repairs damaged skin and restores its normal structure and function after injury. A wound results from disruption of the skin or underlying tissues due to physical, chemical, thermal, infectious, or pathological causes. The primary aim of wound healing is to re-establish the protective barrier of the skin and maintain tissue homeostasis. This process involves a coordinated interaction between cells, extracellular matrix components, cytokines, and growth factors. Healing occurs through four overlapping phases: hemostasis, inflammation, proliferation, and remodeling. Hemostasis prevents blood loss, inflammation removes debris and pathogens, and the proliferative phase promotes granulation tissue formation, angiogenesis, collagen deposition, epithelialization, and wound contraction. During remodeling, collagen is reorganized and the wound matures into a stronger, stable scar. Depending on wound characteristics and management, healing may occur by primary, secondary, or tertiary intention. Successful wound healing depends on local factors such as perfusion, oxygenation, moisture, and infection control, as well as systemic factors including nutrition, age, underlying diseases, and medications. Accurate assessment and appropriate management are essential for optimal healing outcomes.

KEYWORDS: Wound healing; Tissue repair; Inflammation; Angiogenesis; Collagen synthesis; Growth factors; Chronic wounds; Wound dressings.

1.0 INTRODUCTION

Wound healing is a complex and dynamic biological process that restores the integrity and function of damaged tissues. A wound is defined as a break or disruption in the normal anatomical structure and function of the skin or underlying tissues caused by physical, chemical, thermal, or pathological factors. The primary goal of wound healing is to re-establish the barrier function of the skin and maintain homeostasis.

The wound healing process involves a highly coordinated interaction between cells, extracellular matrix components, cytokines, and growth factors. It progresses through four overlapping phases: hemostasis, inflammation, proliferation, and remodeling. Each phase is regulated by specific cellular events—beginning with clot formation, followed by immune cell activation, collagen deposition, angiogenesis, and finally scar maturation.

Effective wound healing depends on both local factors (such as infection, oxygenation, moisture) and systemic factors (such as age, nutrition, diseases like diabetes, and medications). Any disturbance in these factors can lead to delayed healing, chronic wounds, or abnormal scar formation. Understanding the mechanisms of wound healing is essential in developing advanced therapeutic strategies, including biomaterials, growth factor therapies, stem-cell-based treatments, and novel drug delivery systems that enhance tissue regeneration. Due to its clinical significance, wound healing remains a major area of research in medicine, pharmacy, and biomedical sciences.

Wound healing is a complex, dynamic, and biological process through which the body restores the continuity, structure, and function of damaged tissue. It involves a coordinated cascade of cellular, molecular, and biochemical events that begin immediately after injury and continue until the tissue is fully repaired.

2.0 CLASSIFICATION OF WOUND HEALING

Wound healing can be classified based on several criteria.

A. Based on Mechanism / Type of Tissue Repair

1. Regeneration (Restorative Healing)

Damaged tissue is replaced with the same type of cells, restoring normal architecture.
Example: healing of superficial epidermal wounds.

2. Repair (Scar Formation)
Damaged tissue is replaced with fibrous connective tissue, resulting in scar formation.
Example: deeper dermal injuries.

B. Based on the Healing Process / Intention
1. Healing by Primary Intention (First Intention)
Occurs in clean, incised wounds with edges approximated (sutured).
Minimal tissue loss and minimal scarring.

2. Healing by Secondary Intention (Second Intention)
Occurs in open wounds with larger tissue loss.
Characterized by granulation tissue formation, wound contraction, and larger scar.

3. Healing by Tertiary Intention (Delayed Primary Closure)
Initially left open due to contamination or infection and later closed.
Used in complicated or dirty wounds.

C. Based on Depth of Tissue Damage
1. **Superficial Wounds**: involving epidermis only
2. **Partial-Thickness Wounds**: involving epidermis + part of dermis
3. **Full-Thickness Wounds**: involving epidermis, dermis, and possibly subcutaneous tissue or deeper structures.

D. Based on Etiology (Cause of Wound)
1. **Mechanical wounds**: cuts, abrasions, lacerations
2. **Thermal wounds**: burns, frostbite
3. **Chemical wounds**: acids, alkalis
4. **Radiation wounds**: radiation exposure
5. **Infectious wounds**: caused by bacteria, fungi, viruses
6. **Chronic wounds**: diabetic ulcers, pressure ulcers, venous ulcers

E. Based on Duration
1. Acute wound
Heal within a predictable, normal timeframe.
Example: surgical incisions, minor cuts.

2. Chronic Wounds
Fail to progress through normal stages of healing.
Example: diabetic foot ulcer, venous ulcer

F. Based on Cleanliness (Used clinically)
1. Clean wounds
2. Clean-contaminated wounds
3. Contaminated wounds
4. Infected or dirty wounds

3.0 Anatomy of Wound Healing

The skin is the largest organ of the body and serves as the first line of defense against physical, chemical, and microbial insults. Understanding its structure is essential to appreciating how wounds occur and how the healing process takes place. The skin consists of three major layers—epidermis, dermis, and subcutaneous tissue—each playing a unique role in wound repair.

Wound healing anatomy involves all layers of the skin (epidermis, dermis, subcutis) and underlying vessels, nerves, and connective tissue, working through four overlapping stages: hemostasis, inflammation, proliferation, and remodeling. Each stage has characteristic tissues and cells that appear in and around the wound as it progresses from an open defect to a scar.

Skin layers involved
The epidermis (outer layer) provides the barrier and is rebuilt by keratinocyte migration and proliferation during re‑epithelialization. The dermis (middle layer) supplies collagen, elastin, vessels, and appendages (hair follicles, glands) and is the main site of granulation tissue and scar formation. The subcutis (hypodermis) contains fat and larger blood vessels, helping with cushioning, vascular supply, and providing fibroblasts and immune cells to the wound bed.

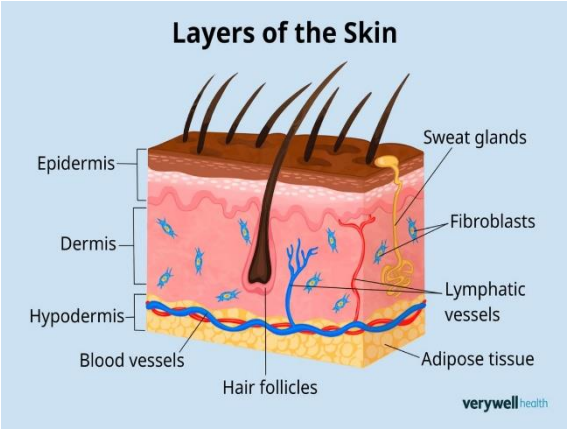
Stage 1: Hemostasis
Immediately after injury, damaged vessels constrict and platelets aggregate to form a fibrin clot that stops bleeding and fills the wound gap. This clot acts as a temporary extracellular matrix and scaffold for incoming inflammatory cells and later migrating keratinocytes and fibroblasts.

Stage 2: Inflammation
Neutrophils are first to enter the wound, clearing bacteria and debris; later, monocytes arrive and differentiate into macrophages.
Macrophages coordinate healing by releasing cytokines and growth factors that recruit fibroblasts, endothelial cells, and keratinocytes, bridging inflammation to the growth phase.

Stage 3: Proliferation and granulation
Fibroblasts in the dermis produce collagen and extracellular matrix, forming granulation tissue rich in small blood vessels (angiogenesis), which fills the wound from the base up. In parallel, keratinocytes at the wound edge and from appendages migrate over this granulation tissue (epithelialization), while myofibroblasts contract the wound, reducing its size.

Stage 4: Remodeling and scar
over weeks to months, type III collagen in the granulation tissue is gradually replaced and reorganized into predominantly type I collagen along tension lines. Vascularity decreases, cellularity falls, and the tissue becomes a scar that is mechanically strong but usually

reaches only about 80% of the tensile strength of uninjured skin.



3.1 Epidermis and dermis work together in wound healing

The epidermis mainly restores the surface barrier by re-epithelialization, while the dermis rebuilds the structural "support layer" with collagen, vessels, and extracellular matrix. Superficial wounds (only epidermis) heal mostly by rapid keratinocyte migration; deeper wounds (through dermis) require full granulation tissue and scar formation.

Epidermis in wound healing

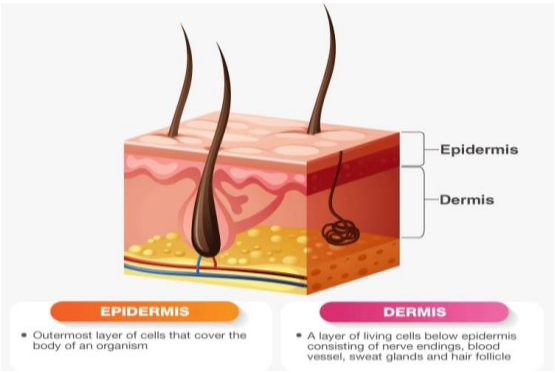
The epidermis is a stratified squamous epithelium composed mostly of keratinocytes, with a basal (stem cell) layer attached to the basement membrane. After injury, basal keratinocytes at wound edges and from appendages (hair follicles, glands) change shape, migrate over the wound bed, proliferate, and then re-form the layered epidermis and a new basement membrane (re-epithelialization).

Dermis in wound healing

The dermis is connective tissue rich in fibroblasts, collagen, elastin, blood vessels, nerves, and appendage roots (follicles, glands). Following injury, dermal fibroblasts and recruited cells form granulation tissue (new capillaries, collagen, ECM), later remodeled into a collagen-rich scar that restores tensile strength but usually not full original architecture.

Interaction between epidermis and dermis

Dermal fibroblasts and inflammatory cells release growth factors (for example TGF-β, PDGF, EGF family) that stimulate keratinocyte proliferation and migration, guiding re-epithelialization. In turn, keratinocytes secrete cytokines and growth factors that influence fibroblasts, angiogenesis, and immune cells, so successful healing depends on coordinated epidermal–dermal cross-talk rather than either layer acting alone.



4.0 Physiology of Wound Healing

Wound healing is a complex, dynamic, and highly coordinated physiological process that restores tissue integrity after injury. It involves interactions among cells, extracellular matrix (ECM), cytokines, chemokines, and growth factors. The healing process proceeds through four overlapping phases.

1. Hemostasis
2. Inflammation
3. Proliferation
4. Maturation / Remodeling

Each phase has distinct physiological events but overlaps with the others to ensure continuous repair.

4.1 Hemostasias Phase (Immediate: Seconds to Minutes)

This is the first response to injury and aims to prevent blood loss.

- Key Physiological Events

Vasoconstriction: Occurs immediately to reduce bleeding.
Platelet activation: Platelets adhere to exposed collagen and release.
Platelet-derived growth factor (PDGF)
Transforming growth factor-β (TGF-β)
Vascular endothelial growth factor (VEGF)

- Clot formation

Fibrin mesh forms via the coagulation cascade.
Acts as a temporary matrix for cell migration.

4.2 Inflammatory Phase (Hours to 3 Days)

This phase activates the immune system to remove debris, pathogens, and dead cells.

Key Physiological Events

Vasodilation: Mediated by histamine, nitric oxide → increases blood flow.

- **Neutrophil infiltration (first 24–48 hrs)**

Phagocytosis of bacteria and debris
Release of reactive oxygen species (ROS)

- **Macrophage activation (after 48 hrs)**

Most important cell in this phase
Secretes cytokines (IL-1, TNF-α)
Produces growth factors (PDGF, VEGF, FGFs)
Orchestrates transition to the proliferative phase

4.3 Proliferative Phase (3 Days to 3 Weeks)
This phase is characterized by new tissue formation.
Major Components.

a. Fibroblast Proliferation and ECM Formation
Fibroblasts migrate into the wound.
Synthesize collagen (mainly type III), proteoglycans, and fibronectin.
ECM provides structure for tissue regeneration.

b. Angiogenesis
Formation of new blood vessels driven by VEGF and FGF-2.
Ensures oxygen and nutrient supply.

c. Granulation Tissue Formation
Composed of new capillaries, fibroblasts, macrophages, and loose ECM.
Appears red and moist.

d. Epithelialization
migrate from wound edges and appendages.
Re-forms the epidermal barrier.

e. Wound Contraction
Mediated by myofibroblasts.
Reduces wound size significantly.

4.4 Maturation / Remodeling Phase (3 Weeks to Months–Years)
This is the final phase where tissue strength and stability increase.
Key Physiological Events
- Collagen remodeling

Type III collagen replaced by type I.
Collagen fibers reorganize along tension lines.

- Reduction in vascularity

Granulation tissue regresses.

- Scar maturation

Scar becomes flatter, softer, and stronger.

- Tensile Strength

At 3 weeks: ~20% of normal
At 3 months: ~70%
Never reaches 100%

5.0 Types of Wound Healing Process
Wound healing occurs through different pathways depending on the extent of tissue loss, degree of contamination, and management of wound edges. The three main types of wound healing are.

5.1 Healing by Primary Intention (Primary Union)
This type occurs when the wound edges are clean, closely approximated, and sutured or otherwise mechanically closed.
Characteristics.
- ✓ Minimal tissue loss
- ✓ Minimal inflammation
- ✓ Rapid healing
- ✓ Small, linear scar
- ✓ Low risk of infection
- ✓ Physiological Process:
- ✓ Clot forms immediately
- ✓ Limited granulation tissue formation
- ✓ Epithelial repair occurs quickly
- ✓ Collagen deposition and scar formation are minimal

Examples
Surgical incisions
Clean cuts

5.2 Healing by Secondary Intention (Secondary Union)
Occurs in wounds with extensive tissue loss, gaping wound edges, or infected wounds left open to heal naturally.
Characteristics
- ✓ Large tissue defect
- ✓ More inflammation
- ✓ Abundant granulation tissue
- ✓ Significant wound contraction
- ✓ Larger, more prominent scar
- ✓ Higher chance of infection

Physiological Process
- ✓ Extensive granulation tissue fills the defect
- ✓ Myofibroblasts cause wound contraction
- ✓ Slow epithelialization from wound edges

Examples
Pressure ulcers
Traumatic injuries
Large open wounds

5.3 Healing by Tertiary Intention (Delayed Primary Closure)
This is a combination of primary and secondary intention. The wound is left open initially (usually due to contamination) and closed later after the risk of infection decreases.

Characteristics
- ✓ Moderate tissue loss
- ✓ Initially high inflammation
- ✓ Granulation tissue forms first
- ✓ Closure performed after several days
- ✓ Results in intermediate scar formation

Physiological Process
- ✓ Wound is kept open for drainage
- ✓ Once clean, edges are approximated
- ✓ Epithelialization and collagen remodeling occur after closure

Examples
Contaminated traumatic wounds
Animal bites

Surgical wounds with high infection risk

6.0 Assessment of Wound Healing

Assessment of wound healing is the systematic evaluation of the wound to monitor its progress, identify complications, and guide appropriate treatment. Proper assessment ensures timely healing, prevents infection, and helps determine the effectiveness of therapeutic interventions.

Wound assessment involves evaluating local wound characteristics, systemic factors, and objective parameters that indicate healing status.

6.1 Visual and Clinical Assessment

6.1.1. Wound Size

Measured in length × width × depth.
Decreasing size over time indicates healing.
Tools used: ruler, digital planimetry, wound tracings.

6.1.2. Wound Bed Appearance

Granulation tissue (healthy red tissue) indicates good healing.
Slough (yellow/white) indicates delayed healing.
Necrotic tissue (black/brown) suggests poor perfusion or infection.

6.1.3. Wound Edges (Margins)

Advancing epithelial edge → positive sign.
Rolled edges (epibole) → chronic non-healing.

6.1.4. Exudate (Drainage)

Type: serous, sanguineous, purulent
Amount: none, scant, moderate, heavy
Color and odor help detect infection.

6.1.5. Signs of Infection

Redness, warmth, swelling.
Pain, fever.
Purulent discharge
Delayed healing or tissue breakdown.

6.2 Tissue Perfusion and Oxygenation

Proper blood supply is essential for wound healing.

Assessment Methods

Capillary refill time
Ankle-Brachial Index (ABI) for arterial ulcers
Transcutaneous oxygen measurement ($TcPO_2$)
Doppler ultrasound.
Reduced perfusion indicates delayed healing.

6.3 Measurement of Pain

Pain evaluation helps identify complications such as:
Infection
Ischemia
Neuropathic changes
Pain is assessed using:
Numeric Rating Scale (NRS)
Visual Analogue Scale (VAS)

6.4 Microbiological Assessment

Used when infection is suspected.
Methods.
Wound swab (least invasive)
Tissue biopsy (gold standard)
Quantitative culture
Indicates microbial burden and guides antibiotic therapy.

6.5 Histological and Cellular Assessment

Provides detailed information on cellular activity.
Key Indicators
Amount of collagen deposition
Angiogenesis
Inflammatory cell infiltration
Epithelial regeneration
Used mostly in research settings.

6.6 Biochemical and Molecular Markers

Modern wound assessment includes measurement of:
Matrix metalloproteinases (MMPs): high levels = poor healing
Growth factors (VEGF, PDGF, TGF-β): indicate healing activity
Cytokines (IL-1, TNF-α): elevated in chronic inflammation
Oxidative stress markers.
Useful for advanced wound care studies.

6.7 Scoring Systems and Standardized Tools

Several validated tools help assess wound healing progress:

- PUSH Tool (Pressure Ulcer Scale for Healing)

Measures ulcer size, exudate, and tissue type.

- Bates-Jensen Wound Assessment Tool (BWAT)

Assesses 13 parameters including depth, edges, necrotic tissue, and color.

- Wagner Classification (for diabetic foot ulcers)

Grades from 0 (intact skin) to 5 (extensive gangrene).

6.8 Photographic Documentation

Digital photographs taken periodically
Provides a visual timeline of healing
Useful for monitoring chronic wounds and treatment outcomes

6.9 Patient-Related Assessment

Systemic factors influencing wound healing include.
Nutrition (protein, vitamins A & C, zinc)
Diabetes control
Medications (steroids, chemotherapy)
Mobility and pressure relief
Comorbid diseases
Regular systemic assessment ensures holistic wound care.

7.0 Management and Treatment Strategies of Wound Healing

Effective management of wound healing involves a combination of local wound care, systemic support, infection control, and the use of advanced therapeutic

technologies. The choice of treatment is based on the type of wound, depth, presence of infection, and underlying patient factors.

Management strategies generally follow the TIME framework.
T – Tissue management
I – Infection/Inflammation control
M – Moisture balance
E – Edge advancement

7.1 Initial Wound Assessment and Stabilization
Identify wound type, cause, depth, and contamination level
Control bleeding
Irrigate with normal saline to remove debris
Assess for foreign bodies
Determine need for debridement or closure

7.2 Wound Cleansing and Debridement
Debridement removes devitalized tissue, reduces bacterial load, and promotes granulation.

Types of Debridement
1. Surgical/Sharp Debridement: Fastest, used for infected or necrotic wounds.
2. Mechanical Debridement: Wet-to-dry dressings, wound irrigation.
3. Autolytic Debridement: Uses the body's enzymes; facilitated by hydrogel or hydrocolloid dressings.
4. Enzymatic Debridement: Collagenase and papain-based ointments
5. Biological Debridement (Maggot Therapy): Sterile larvae digest necrotic tissue.

7.3 Infection Prevention and Control
- Local Infection Control

Antimicrobial dressings (silver, iodine, PHMB)
Topical antibiotics (e.g., mupirocin, fusidic acid)
- Systemic Antibiotics

Used only when there is spreading infection, cellulitis, osteomyelitis, or systemic involvement.
- Biofilm Management

Frequent debridement
Use of anti-biofilm agents (hypochlorous acid, silver dressings)

7.4 Moisture Balance and Appropriate Dressings
Maintaining a moist environment accelerates healing.

Types of Dressings
1. Hydrocolloid dressings – promote autolytic debridement
2. Hydrogels – hydrate dry wounds
3. Foam dressings – absorb moderate to heavy exudate
4. Alginate dressings – for highly exudative wounds
5. Collagen dressings – stimulate granulation
6. Antimicrobial dressings – silver, iodine, honey
7. Film dressings – for superficial or partial-thickness wounds
8. Negative Pressure Wound Therapy (NPWT)

7.5 Advanced Therapies
- Negative Pressure Wound Therapy (NPWT)

Uses vacuum-assisted closure
Improves perfusion, reduces edema, accelerates granulation
Useful for diabetic ulcers, pressure ulcers, trauma wounds
- Growth Factor Therapy

PDGF (becaplermin gel) approved for diabetic foot ulcers
VEGF, FGF, TGF-β used in research and advanced care
- Skin Substitutes and Tissue Engineering

Bioengineered skin: Apligraf®, Dermagraft®
Autologous skin grafts
Stem cell[6] therapies
Platelet-rich plasma (PRP)
- Hyperbaric Oxygen Therapy (HBOT)

Increases oxygen delivery to tissues
Useful for ischemic, infected, and diabetic wounds
- Laser and Phototherapy

Low-level laser therapy (LLLT) promotes epithelialization and collagen synthesis

7.6 Surgical Management
Used for complex wounds or wounds not healed by conservative methods includes:
Skin grafting
Flap surgery
Surgical closure (primary or delayed closure)
Removal of necrotic tissue or infected bone (e.g., in diabetic foot)

7.7 Systemic Factors and Patient Optimization
Wound healing depends on overall patient health.

Key Interventions
- Nutrition Support: High protein intake, Vitamins A, C, E., Zinc, copper, arginine, omega-3
- Glycemic Control: Essential in diabetic wound healing, Prevents infection and improves collagen deposition
- Hydration and Circulation Improvement: Adequate fluid intake, Treatment of anemia and vascular diseases, Smoking cessation
- Pain Management: NSAIDs or opioids if necessary, Ensures patient comfort and compliance
- Pressure Relief: Offloading (for diabetic foot ulcers), Position changes (for pressure ulcers)

7.8 Monitoring and Follow-Up
Regular evaluation ensures timely progress.
Monitoring Tools
PUSH tool
BWAT (Bates-Jensen)
Photographic documentation
Wound size measurement
Signs of infection and granulation

8.0 CONCLUSION

Wound healing is a complex, dynamic, and highly coordinated biological process essential for maintaining the integrity and function of the skin—the body's primary protective barrier. Understanding the anatomy of the skin provides the foundation for appreciating how wounds occur and how different tissues contribute to repair. The classification and types of wound healing—such as primary, secondary, and tertiary intention—offer insight into the patterns and pathways through which various wounds restore continuity.

Effective wound management requires accurate and systematic assessment, including evaluation of wound size, depth, tissue type, exudate, perfusion, infection status, and patient-related factors. Assessment frameworks and standardized tools help clinicians monitor progress, predict healing outcomes, and adjust treatment plans accordingly.

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