

1

The Wound Healing Process

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The skin is the body's largest organ, primarily serving to protect the underlying tissues and organs from injury. It consists of two layers: the epidermis and the dermis. The outermost epidermal layer forms a physical, waterproof barrier against invading pathogenic microorganisms, harmful chemicals and foreign bodies. When the skin is broken, localised repair processes respond to the injury and initiate healing. In horses, traumatic skin wounds are common and often require extensive wound management. This presents a significant welfare concern and places a considerable financial burden on the global equine industry (Theoret and Schumacher 2017, p. 1). Similarly, a survey of horse owners in the United Kingdom revealed that certain management practices increased the risk of traumatic injuries and related wounds (Owen et al. 2012).

When an open wound is left untreated, as happens with animals in the wild, it naturally begins to heal. What follows is a remarkable sequence of continuous and well-defined phases that influence each other, occurring at the correct times and for the appropriate durations, aiming to close the wound and restore the area to good functioning health. Disruptions and prolonged adverse conditions that affect this process can cause delays in wound healing.

Wound Healing Physiology

The objectives of wound management are to

- achieve a functional and cosmetic outcome,
- minimise pain and distress,
- promote a rapid return to normal use,
- deliver healing at a reasonable cost (Knottenbelt 2003; Owen and Wenke 2007).

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Wound healing is a complex but predictable sequence of biological events that begins at the moment of injury and concludes with the remodelling and maturation of new tissue. In horses, this process is notably influenced by the wound's location, size and the relative mobility and vascularity of surrounding tissue.

Healing by second intention – when wounds are allowed to heal naturally without surgical closure – is common in equine practice. The time required for full resolution depends mainly on the capacity for wound contraction and epithelialisation. This process is influenced by local factors such as skin elasticity and regional blood supply. Wounds on the upper body or trunk, where there is greater skin laxity and vascular support, tend to contract substantially during healing. In contrast, wounds on the distal limbs typically contract to a lesser degree because of tight, immobile skin and limited subcutaneous tissue (Theoret and Schumacher 2017, p. 34).

These regional differences in healing capacity partly explain why distal limb wounds are considered particularly challenging. Adding to this is the equine species' relatively inefficient inflammatory response, which predisposes horses to wound chronicity and the formation of exuberant granulation tissue (Wilmink and van Weeren 2005).

A sound understanding of each stage of the wound healing process – along with their expected timelines – allows clinicians to intervene appropriately, manage expectations and optimise outcomes. While second intention healing is biologically remarkable, early, well-timed surgical reconstruction (e.g. primary closure or grafting) often results in a more cosmetic and functional result in less time and can ultimately be more cost-effective.

While financial constraints frequently lead to the use of dressings and bandages alone, this 'cheaper' option may become more costly if healing is delayed. In many cases, early interventions such as pinch grafting can significantly accelerate healing and reduce the risk of complications.

All complications and delays in healing stem from disruptions in the healing cascade. Understanding the typical sequence and timing of tissue repair provides a baseline for clinical decision-making and timely intervention.

Phases of Wound Healing

Wound healing occurs in overlapping phases: haemostasis and inflammation occur first, followed by tissue proliferation and contraction and then long-term collagen remodelling (Figure 1.1). Understanding this process helps guide the timing of wound management measures.

Haemostasis (Immediate – Minutes Post-injury)

Within minutes of injury, vasoconstriction reduces blood loss. Platelets adhere to exposed subendothelial collagen, forming a temporary plug. Platelet activation initiates a cascade of cytokine and chemokine release, triggering the early inflammatory response. The platelets release a variety of bioactive molecules, which attract neutrophils and macrophages to the wound site. Simultaneously, fibrinogen is converted to fibrin, stabilising the clot. Clot formation typically occurs within 10–15 minutes unless major vessels are involved.

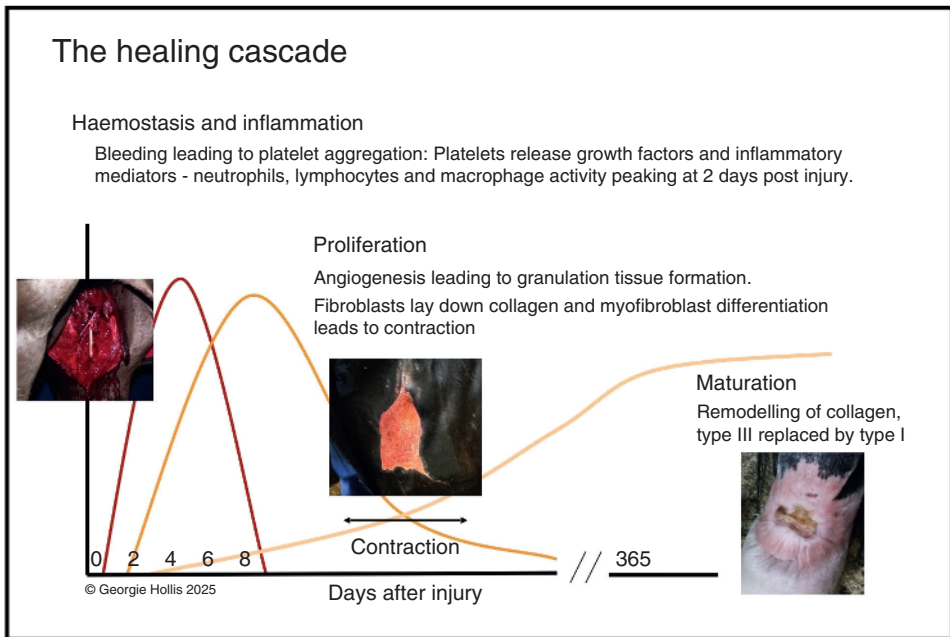


Figure 1.1 The wound healing cascade. *Source:* Reproduced with permission of Georgie Hollis.

Inflammation (Hours to ~4 Days Post-injury)

The inflammatory phase begins within hours and peaks around 48 hours post-injury. Vasodilation increases vascular permeability, allowing neutrophils to infiltrate within 6–12 hours. These cells neutralise bacteria and start clearing contaminants. Macrophages arrive later to phagocytose dead tissue and pathogens, and release proteases that prepare the wound for the next phase. Although wounds in this phase may appear sloughy or malodorous, these signs do not necessarily mean infection – they reflect critical immune activity.

Bacterial contamination occurs immediately and can progress to colonisation, critical colonisation and then infection if not addressed early. Human guidelines suggest that when bacterial numbers exceed $10^5/\text{g}$, healing will be delayed and infection may follow as bacteria proliferate further (Bowler 2003) (Figure 1.2). Decontamination within the first 6 hours is essential to prevent local or systemic infection.

Effective management during this phase includes

- Copious and early lavage with tissue-compatible solutions improves the ability to remove bacteria before they establish deeper colonisation. For every hour earlier a wound is lavaged, the rate of bacterial proliferation is halved (Owen and Wenke 2007).
- Sharp, mechanical or autolytic debridement.
- Use of topical agents to soften devitalised tissue.

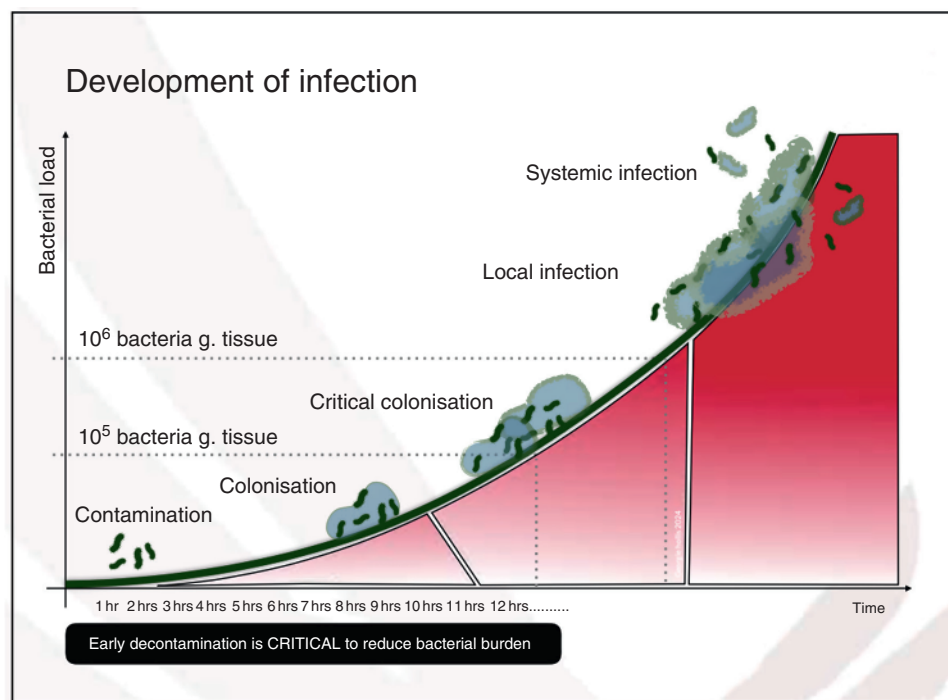


Figure 1.2 The development of infection over time. *Source:* Reproduced with permission of Georgie Hollis.

Prolonged inflammation often results from an inhibitor such as persistent necrotic tissue, exposure of bone or tendon, foreign bodies or excessive movement at the wound site (see Chapter 3, Factors that Delay Wound Healing). These factors prolong macrophage activity and protease release, hindering progression to proliferation.

Proliferation (Approximately Days 4–21 Post-injury)

Angiogenesis

Fibroblasts migrate into the wound within days of injury and begin to lay down layers of collagen to form a matrix for new capillary buds to grow into. This tissue is fragile, and when damaged by movement and repeated trauma, it stimulates macrophage activity that can revert the wound back to the inflammatory phase. This is why maintaining a moist environment to support optimal cell proliferation, minimising interference and immobilising wounds over joints is recommended during this phase.

As granulation tissue fills the wound bed, fibroblasts begin differentiating into myofibroblasts, which possess contractile properties that enable wound contraction. This may begin as early as 7 days post-injury but becomes most evident around 10–14 days once a healthy granulating wound bed has formed and the first signs of an epithelial margin appear.

Epithelialisation occurs concurrently with granulation. Basal epithelial cells from wound edges and adnexal structures, such as hair follicles, begin migrating over the granulation

tissue within 4–5 days post-injury. It is a slow process, with keratinocytes progressing centripetally at approximately 1–1.5 mm every 10 days under ideal conditions, but this may take longer on the equine limb (Knottenbelt 2003).

Maturation/Remodelling (3 Weeks to Months or Longer)

Once the wound bed is covered with epithelium and contraction is largely complete, the wound enters the remodelling phase. During proliferation, type III collagen is deposited in a haphazard manner. Remodelling can take up to 2 years, by which time collagen levels do not increase, but the existing fibres have rearranged into a more organised structure (Theoret and Schumacher 2017, p. 10). The wound has minimal tensile strength until type III collagen is gradually replaced by type I collagen, resulting in a more supple scar with improved tensile strength. At 1 year, the final tensile strength of the scar rarely reaches 80% of that of uninjured tissue (Knottenbelt 2003).

The Optimal Wound Healing Environment

It was Dr. George Winter, the British pioneer of the moist wound healing concept, who in 1962 published research from the University of London that changed the way wounds are managed in modern medicine. His research went against the conventional wisdom and practice of the time, demonstrating that wounds maintained in a moist environment would heal up to 50% faster than those left to dry and form scabs (Winter 1962). This revolutionised wound management, and subsequent findings confirm that maintaining a moist environment promotes healing while reducing the risk of scarring and complications.

Subsequent scientific evidence demonstrated that providing a moist wound environment offers advantages beyond reducing scarring, including supporting autolytic debridement during the inflammatory phase, encouraging angiogenesis, aiding cell function, wound contraction and maturation. In equine medicine, moist wound healing is now recognised as the preferred method after debridement and granulation tissue management, as it acts as a medium for cellular activity and growth factors.

Benefits of moist wound healing in horses also include

- better healing through an environment that promotes cell migration and tissue repair,
- reduced infection risk by discouraging bacterial growth,
- less scar tissue is formed by preventing scab formation,
- pain relief by protecting nerve endings,
- healthy tissue formation with nutrient-rich granulation and elastic epithelial layers,
- maintaining moist wounds enhances overall healing quality (Figure 1.3).

Supporting a moist wound environment through this proven principle has been one of the most significant advances in human wound management. Unfortunately, many over-the-counter products available to horse owners fail to support this, with their marketing leading to confusion, as claims of unproven science promise quick wound healing.

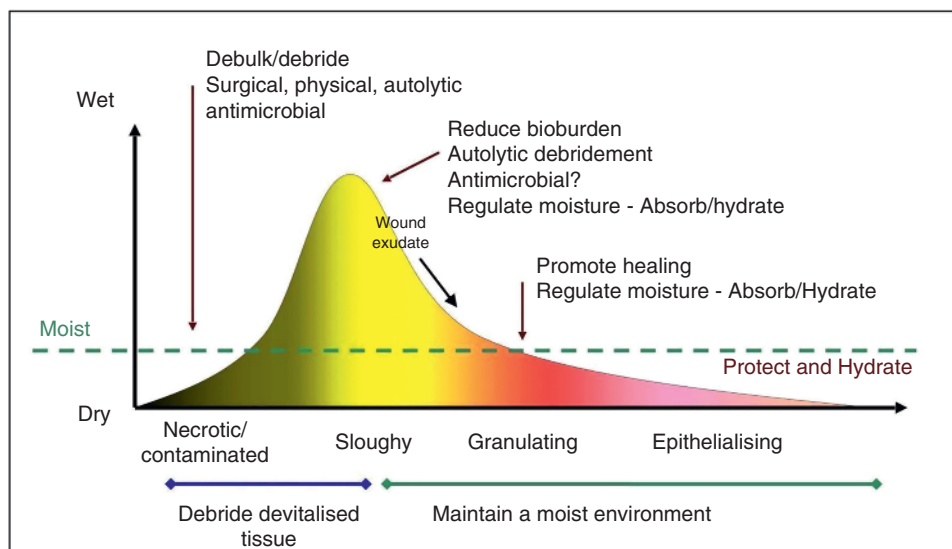


Figure 1.3 Wound exudate levels change throughout the healing process, peaking during the inflammatory phase and decreasing significantly during proliferation, which can risk tissue dehydration if the wound remains uncovered. Maintaining a moisture balance is essential for optimal cellular proliferation, achieved through dressings that provide hydration and protection. *Source:* Reproduced with permission of Georgie Hollis.

In Summary

Understanding the timeline and physiological basis of wound healing is essential for effective case management. Healing is a dynamic process that can either progress or regress depending on local conditions. Equine nurses and clinicians must remain vigilant, anticipate complications and make timely interventions to guide the wound towards a successful outcome – functionally, aesthetically and economically.

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