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In the Field Equine Wound Management Part I

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Introduction

Horses are flight creatures and this basic instinct puts them at a greater risk for traumatic wound injury. It is a common scenario for an equine veterinarian to be called out, usually in the dark, to the middle of a muddy field to assess a horse with one or several wounds. How the horse is treated in these initial stages has a huge impact on how the wound will go on to heal, the prognosis for the horse and the associated financial implications. There are many decisions that the attending veterinarian faces. A thorough knowledge of anatomy, the principles of wound healing, the products the veterinarian is using and potential complications is required so that the animal can get the best treatment and the owner the best advice.

The basics of anatomy, how the immune system responds, how wounds heal, and simple surgical techniques and principles form the building blocks of good wound care and management.

One of the first known commentators was Cato, the Roman agricultural writer (c 200 BC) who recommended the use of olive oil dregs, lupine extract and good wine for scab in sheep. Columella (c 70 AD) thought that it was better to get rid of suppuration with the surgeon's knife, rather than with medication, and then to wash the wound with warm ox urine and bind it up with linen bandages soaked in liquid pitch and oil. Even at this early time it was obviously appreciated that infected wounds would not heal without first removing infected tissue!

Principles of wound healing

By definition, this is "the restoration of the normal anatomic continuity to a disrupted area of tissue." The aim of wound management is to ensure rapid healing with optimal function and cosmetic outcome.

The Four Stages of Wound Healing:

1. Haemostasis

This begins immediately following an injury. Vasoconstriction facilitates homeostasis. Blood vessels constrict and platelets form a stable clot. Under the influence of ADP (adenosine diphosphate) leaking from damaged tissues, the platelets adhere to the exposed type 1 collagen. They are activated and secrete adhesive glycoproteins, leading to platelet aggregation. They also secrete factors that 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 haemostatic plug.

Finally, platelets also secrete growth factors such as platelet-derived growth factor, which is recognised as one of the first factors in initiating the subsequent healing steps. These growth factors recruit neutrophils and monocytes (initiating the next phase of wound healing), stimulate epithelial cells and recruit fibroblasts. Haemostasis occurs within minutes of the initial injury unless the patient has underlying clotting disorders.

2. Inflammation

Clinically, inflammation presents as erythema, swelling and warmth often associated with pain. Blood vessels become leaky, releasing plasma and neutrophils into the surrounding tissue. Neutrophils phagocytose debris and microorganisms, providing the first line of defence against infection. As they digest bacteria and debris, neutrophils die, releasing intracellular enzymes into the surrounding matrix, further digesting tissue. As fibrin is broken down as part of this clean up, the degradation products attract the next cells involved such as fibroblasts and epithelial cells. They are aided by local mast cells.

Cells communicate through soluble proteins called cytokines and growth factors, which are released by cells and bind to a receptor on a target cell. Once a cytokine binds to a target cell it stimulates the cell to move. Growth factors, on the other hand, stimulate the target cell to either divide and produce more cells or synthesise and release substances such as collagen, which is required to form the extracellular matrix. The extracellular matrix (ECM) plays an active role in wound healing by interacting with the cells through receptors called integrins, leading to platelet activation, epithelial migration and fibroblast movement.

Circulating monocytes differentiate into macrophages after they exit the blood vessels and come in contact with the ECM. Macrophages are able to phagocytose bacteria, providing a second line of defence. Macrophages secrete extracellular enzymes to degrade necrotic tissue at the wound site. These enzymes belong to a family of substances called matrix metalloproteases (MMP's).

About 20 different types of MMP's are secreted by many different cells including neutrophils, macrophages, epithelial cells and fibroblasts, under the influence of inflammatory cytokines such as tumour necrosis factor-alpha and interleukin 1 and 6. MMP's act on all components of the ECM and are responsible for removing devitalised tissue, repairing lost or damaged tissue and remodelling. MMP's are balanced by tissue inhibitors of metalloproteases (TIMP's), which are released locally by cells and inactivate MMP's by reversibly binding to them. Uncontrolled MMP's can degrade newly formed tissue or destroy growth factors.

Macrophages secrete a variety of cytokines and growth factors such as fibroblast growth factor, epidermal growth factor, transforming growth factor-beta and interleukin 1 which appear to direct the next stage.

Inflammation, the body's response to trauma, can be confused with infection. However, inflammation is a normal response to tissue injury.

3. Proliferation

Fibroblasts are the cells that build the framework for reconstruction of a wound. They secrete the collagen framework on which further dermal regeneration occurs. Specialised myofibroblasts are responsible for wound contraction. Angiogenesis occurs, regenerating the outer layers of capillaries, and the endothelial cells produce the lining. The keratinocytes are responsible for epithelialisation. In the final stage of epithelialisation, contracture occurs as the keratinocytes differentiate to form the protective outer layer or stratum corneum.

In a healing wound, the cells under the influence of growth factors divide to produce new cells, which migrate to where they are needed under the influence of cytokines. There is a balance between the MMP's and TIMP's so that there is a net production of new tissue. In chronic wounds, in contrast, where healing is stalled, cell division and

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migration are suppressed, there are high levels of inflammatory cytokines and MMP's, and low levels of TIMP's and growth factors. Cells are often senescent and unresponsive to the growth factors. This lack of response is characteristic of a chronic inflammatory state. It may be caused by an increased bacterial burden, the presence of devitalised tissue, chronic ischaemia or repetitive trauma.

Capillary migration occurs to provide blood supply. Epithelial cells migrate across wounds to re- establish skin continuity.

Wound healing rates in horses vary in relation to the wound severity and its anatomic site. For example, there is a greater tendency of exuberant granulation tissue growth and excessive scarring on the lower limbs of horses compared to similar wounds on the body. Myofibroblasts are less well orientated in the limb when compared to trunk wounds and this is suggested as a cause for the decreased contraction efficiency of wound beds in limb wounds. The relatively poor blood supply to the limbs may play a significant role through decreased oxygen supply and decreased distal limb temperature. These factors are thought to contribute to an imbalance in the growth factors required for effective healing. In particular, the prolonged presence of tissue growth factor β1 (TGF-β1) is blamed, in part at least, for the formation of exuberant granulation tissue.

4. Maturation

New reconstruction tissues are organised, providing wound strength over time. The healing process involves remodelling and realignment of the collagen tissue to produce greater tensile strength. Cell and capillary density decrease. The main cells involved in this process are fibroblasts. Remodelling can take up to 2 years after the initial injury. This explains why closed wounds can quickly breakdown if attention is not paid to the initial causative factors.

The ten major factors that will affect wound healing are:

- Infection
- Altered tissue pH
- Foreign bodies
- Poor oxygen supply
- Necrotic tissue
- Poor health status
- Movement
- Iatrogenic interference
- Poor blood supply
- Tumour transformation

<u>Emergency wound assessment</u>

Arriving on the scene

Take control of the situation. Ensure that the horse is adequately restrained. Assess bleeding. If there is significant ongoing blood loss, the priority is to stop the haemorrhage. The application of a pressure bandage with plenty of layers will usually suffice initially. The horse can then be moved to a cleaner, safe environment where a full clinical examination and assessment of the wound can be performed.

Clinical examination

It is easy to become distracted by the wound, but it is vital to check that no other body systems are involved, e.g. head trauma and neurological signs, mucous membrane colour and heart rate (cardiovascular status) if there has been extensive blood loss (hypovolaemia). The horse can then be safely sedated. Assess the degree of lameness. If the horse is acutely lame, radiography may be required. There may be synovial structure involvement requiring synoviocentesis and through-through distension to confirm. Beware the small wound and very lame horse!

History

Obtain a full history from the owner/yard manager. When was the horse last seen normal? How old is the wound? Does the owner know how the animal sustained the injury? Tetanus cover of the horse? Tetanus prophylaxis is indicated with any open contaminated wound. In vaccinated animals with wounds less than 12 hours old, booster vaccination results in rapid antibody production well before toxin is produced. In many instances with tetanus infection, the infection does not trigger a big enough immune response for antibody production, so re-vaccination at the time of injury is always wise.

It is best to minimise the use of analgesics upon initial assessment until fractures and/or synovial involvement can be ascertained.

Wound Assessment

The wound should be assessed carefully and fully. "TIME SPENT IN THE PREPARATION OF A WOUND IS NEVER WASTED" – Prof B Edwards. With the horse sedated, clip the hair from around the wound. Protect the wound itself by packing with 'Intrasite' gel or sterile 'KY' jelly. With a sterile gloved finger, the wound can be digitally palpated to discern which structures are involved. Good anatomical knowledge is vital. Prior to any digital palpation or cleaning of the wound, swabs for bacteriology should be taken.

Location of the wound

Is the wound within the region of the thorax or mediastinum (punctured pleura?), abdomen (contaminated peritoneal cavity? involvement of intestine/abdominal organ?) tendons, ligaments, muscle bellies and/or synovial structures?

Depth and direction of the wound

It may be useful to use a sterile probe and even to consider the use of radiography/ultrasonography with the probe in place.

Severity of the wound

Extent of contamination/ infection, degree of damage to tissue, loss of regional blood supply, all need consideration. Is there any collateral circulation if arteries have been severed?

Once the above has been assessed, further decisions can be made including, if and how the wound should be closed, are further imaging modalities required (radiography, ultrasonography), can this be done under sedation (with or without nerve blocks, regional anaesthesia, local infiltration) or is anaesthesia required? General anaesthesia can sometimes be a big advantage, for example large heavily contaminated wounds and multiple lacerations of the hind limbs (ensuring that there are no long bone fractures prior to induction). Can the work-up/treatment be continued on farm or does the horse require hospitalisation/referral? What dressings and bandaging will be required? Are antimicrobials necessary? Is there synovial structure and hence synovial sepsis involvement? What is the financial status of the owner, what are the expectations of the owner for the patients return to athletic ability?

<u>Managing the wound</u>

Wound cleaning

The wound should initially be flushed out. CARE SHOULD BE TAKEN WITH ALL DISENFECTANTS/ ANTISEPTIC SOLUTIONS as all of

these solutions are CYTOTOXIC and can do more harm than good, resulting in greater degrees of tissue die-back resulting in delayed healing. Limit these solutions to contact with the surrounding intact skin only. If in doubt of what to use, lavage the wound with sterile NaCl 0.9%. If not available, the benefits of lavaging with clean tap water by far outweigh the disadvantages of a non-sterile solution and not lavaging at all.

By inserting an 18-gauge needle into the fluid bag, the correct pressure will be produced for wound lavage. In acute wounds of less than 3 hours duration, lavage will effectively reduce the number of bacteria that reside on the wound surface. These bacteria attach to the wound surface by an electrical charge; therefore, a pressure of at least 7psi is required to remove them. Pressures of greater than 15 psi become detrimental, as these will then start to drive bacteria and foreign material into the tissues.

The fluid jet should be positioned at an oblique angle to the wound, the fluid should either be squeezed from the fluid bag through the 18-gauge needle, or a 60ml syringe, and 18-gauge needle should be used. This technique has been shown to be effective at removing 80% of adhered bacteria. Cleaning will only remove surface bacteria and has no effect on bacteria within tissue; therefore, chronically infected wounds will require more aggressive management, such as debridement and systemic and local antimicrobial therapy.

	Povidone iodine	Chlorhexidine
Strength	10%	2%
Dilution factor	0.1-0.2% (10-20ml per 1000ml) by diluting PI, more free iodine becomes available	0.05% (25ml in 975ml) >0.05% inhibits healing <0.05% results in significant *staph aureus* survival
Broad spectrum?	Yes	Yes
Fungi and candida?	Yes	No
Known resistance?	No	Yes- *Proteus* and *Pseudomonas*
Inactivated?	Yes- organic material, blood and high numbers of neutrophils	No
Residual activity	No	Yes
Cytotoxicity	Yes	Yes, especially ocular Narrow margin of dilution safety
Rinsing with Alcohol/water	Makes no difference	Removes residual antiseptic effects

Fig 2. Antiseptics povidone iodine versus chlorhexidine

Studies have shown that with a pressure of 14psi, povidone iodine and chlorhexidine resulted in almost complete removal of all adherent bacteria to bone. They had a 19-fold decrease in the numbers of bacteria compared with saline alone. HOWEVER, BEWARE CYTOTOXITY.

The addition of antimicrobials to lavage solutions has been shown to greatly reduce the number of bacteria adhered to a wound. In human studies, the application of penicillin to wounds prior to final closure, reduced wound infection by 75%.

HOWEVER, remember, even with these high kill rates, a small population of commensal bacteria will always remain in protected hair follicles, sebaceous glands and crevices within the lipid coat of the superficial epithelium.

Many studies have been done to determine the resident equine epithelial skin flora. Bacteria found include (in no particular order):

Corynebacterium, Streptomyces, *Streptococcus* spp., *Helcococcus ovis*, *Staphylococcus* spp., *Pseudomonas aeroginosa, Serratia, Enterococcus* spp., *Bacillus* spp., *Micrococcus* spp., and non-haemolytic *Staphylococcus*.

How well the wound is cleaned and debrided at the beginning will have an exponential effect on the overall result. Unhealthy tendon and ligament stumps are often preserved in the mistaken belief that they are required to act as a skeleton for repair. Any contaminated tissue left *in situ* will inhibit healing. On occasions, it is possible to minimise loss of healthy tissue by use of a curette but it should be remembered that this technique is not as complete as tissue resection. There are occasions when it is necessary to be as sparing of tissue as possible (e.g. skin flaps on the distal limb) and it may be advantageous to give tissues the benefit of the doubt before deciding that it is devitalised. Skin viability can often be predicted within a few hours of injury by its gross appearance (hydration, bleeding, temperature, sensitivity) but other soft tissues can be more difficult to assess.

Closing the wound

As well as ensuring that the horse is adequately sedated, the use of regional anaesthesia and analgesia can be very useful. In the authors' opinion, peripheral nerve blocks tend to work better than local infiltration and damage the healing tissue less as the local anaesthetic is cytotoxic and impairs healing. For example, it may be prudent to place a low six-point nerve block when suturing a hind limb dorsal fetlock wound.

If using local infiltration, a good tip is to insert the needle through the damaged edge of tissue rather than healthy intact surrounding skin as this seems to be less painful. For fractious horses, attaching the needle to an extension set, or using a butterfly catheter needle, may also help.

Sedation is best achieved with a combination of an alpha-2-agonist and the mixed opioid butorphanol. Acepromazine may also be included for the incredibly fractious patient. Romifidine and detomidine are the most commonly used alpha 2's, each with their theoretical positives and negatives.

Equine wound management part II will continue to cover closure of wounds, drains, dressings, debridement, the use of topical agents and skin grafting.

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QUESTION 1

In the correct order, the 4 stages of wound healing are:
a. haemostasis, inflammation, proliferation, maturation
b. inflammation, proliferation, haemostasis, maturation
c. proliferation, inflammation, maturation, haemostasis
d. haemostasis, maturation, proliferation, inflammation
e. maturation, proliferation, haemostasis, inflammation

QUESTION 2

Thrombin initiates the formation of:
a. fibrinogen from fibrin
b. fibrin from fibrinogen
c. prothrombin
d. factor X
e. adenosine diphosphate

QUESTION 3

Once cytokines bind to a target cell, they stimulate:
a. cells to divide
b. cells to produce more cells
c. cells to synthesis collagen
d. cells to move
e. cells to lyse

QUESTION 4

Tissue inhibitors of metalloproteases inactivate:
a. Matrix metalloproteases
b. The extracellular matrix
c. Macrophages
d. Monocytes
e. Neutrophils

QUESTION 5

The cells that build the framework for reconstruction of a wound are:
a. neutrophils
b. monocytes
c. macrophages
d. osteoclasts
e. fibroblasts

QUESTION 6

Specialised myofibroblasts are responsible for
a. wound contraction
b. granulation tissue formation
c. angiogenesis
d. epithelialisation
e. muscle regeneration

QUESTION 7

Cells involved in chronic wound healing often fail to respond to growth factors due to:
a. increased bacterial burden
b. presence of devitalised tissue
c. chronic ischaemia
d. repetitive trauma
e. all of the above

QUESTION 8

Tetanus prophylaxis is indicated:
a. only if the horse has never been vaccinated
b. in all open contaminated wounds
c. only if a wound is considered infected
d. in chronic non-healing wounds only
e. in wounds involving the distal limbs only

QUESTION 9

When lavaging a wound, the pressure in psi should be kept below:
a. 2
b. 4
c. 5
d. 10
e. 15

QUESTION 10

To create the correct psi for wound lavage a 60ml syringe should be attached to a:
a. 12 gauge needle
b. 14 gauge needle
c. 16 gauge needle
d. 18 gauge needle
e. 20 gauge needle

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