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WOUND TREATMENT PRINCIPLES - PART ONE

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Abstract: The skin is an organ of the body whose main function is to prevent the internal contact of the organism with chemical, physical agents and pathogenic microorganisms present in the external environment. However, as this tissue has a barrier function, it is vulnerable to injury. The repair of these injuries is called scarring, a process consisting of the replacement of injured tissue with fibrous connective tissue, with the aim of restoring the normal and primary function of the skin. This process is considered complex, but essential to maintain the integrity of tissues in the body. It occurs in the same way in all wounds, regardless of their cause, and can be divided into three phases: inflammation, proliferation and remodeling. Some factors influence the healing process, which may be intrinsic or extrinsic to the organism. The objective of this chapter is to detail each phase of healing, as well as the factors that can help or delay this process and the treatment plan according to the particularities of each case.
Keywords: Healing, wounds, skin, tissue repair.

INTRODUCTION

The skin is an organ that performs multiple functions essential to the proper functioning of the body, such as maintaining hydration, participating in the activation of vitamin D, excreting toxins and assisting in thermoregulation (TOTTOLI et al., 2020). However, one of its main functions is to serve as a protective barrier against agents from the external environment (KONDO; ISHIDA, 2010), and due to this, it is a vulnerable tissue to suffer various types of injuries (SINGER; CLARK, 1999).

When the skin suffers some trauma caused by the external environment or is affected by some clinical condition, damage to its structure can occur, giving rise to cutaneous wounds, which are defined as any interruption of the continuity solution of the body tissues, which may be to a greater or lesser extent. extension (LEITE et al., 2012).

The repair of these lesions, or continuity solutions, consists of the replacement of the injured tissue by a fibrous connective tissue with the objective of restoring the normal and primary function of the skin (HATANAKA; CURI, 2007, TAZIMA et al., 2008). This repair is called healing, a dynamic process to restore the integrity of the skin after an injury, which occurs through the harmonious combination of biochemical and physiological events that begin immediately after the installation of the injury (MANDELBAUM et al., 2003).

The healing process is divided into 3 distinct phases: inflammatory, proliferative, and remodeling. These three phases are not linear and independent, but superimposed (LI et al., 2007). Multiple factors can negatively or positively affect healing, interfering with one or more phases of this process and leading, respectively, to a delay or optimization in tissue repair (GUO; DIPIETRO, 2010). These factors can be divided into intrinsic, when associated with changes in the body itself, and extrinsic, when related to factors external to the body (STANLEY; CORNELL, 2018). These factors will be detailed throughout the chapter.

Therefore, knowledge of the physiological events involved in wound healing, as well as the factors that can negatively or positively influence this process, are of great importance for understanding the injury, the repair phase related, and with that, the best therapy for that moment (TAZIMA et al., 2008).

ANATOMICAL ASPECTS OF THE SKIN

The skin covers the entire body surface, consisting of an epithelial portion of ectodermal origin, the epidermis, and a

conjunctive portion of mesodermal origin, the dermis (JUNQUEIRA; CARNEIRO, 2013). Below the epidermis and dermis is the hypodermis, or subcutaneous tissue, which has the function of adhering the layers above (epidermis and dermis) to underlying organs and deeper structures, allowing the integument to move over them. It is composed of loose connective tissue and when infiltrated by many adipose cells it can be called adipose tissue (BANKS, 1993).

The epidermis consists of five layers known as the basal, spinous, granular, lucid and corneal, and is composed of a keratinized stratified squamous epithelium, which mainly houses keratinocytes; other cells present in the epidermis are melanocytes, Langerhans cells and Merkel cells (JUNQUEIRA; CARNEIRO, 2013). The epidermis is avascular, and receives the nutrition necessary for its functioning through body fluids that penetrate its deeper layers, and also by capillary permeation through the dermis (MACPHAIL, 2015).

The dermis is composed of a connective tissue that supports the epidermis, and connects it to the subcutaneous tissue or hypodermis; consists of two layers, the papillary, more superficial, and the reticular, deeper (BANKS, 1993). The subcutaneous tissue is composed of collagen, reticular and elastic fibers, fibroblasts, macrophages, lymphocytes, mast cells, blood and lymph vessels, nerves, hair follicles, sweat and sebaceous glands, ducts and smooth muscle fibers.

Due to the composition and characteristics described above, the skin performs multiple functions: it protects the body against dehydration by preventing water loss; acts as a barrier against external attacks; through its sensory nerve endings, it constantly receives information from the external environment that is sent to the central nervous system; participate in the body's thermoregulation and excretion of various toxins and metabolites; due to the melanin pigment produced by melanocytes, the skin has a protective function against ultraviolet rays; participates in the vitamin D3 activation cycle; and they also have immune system cells that act in the defense against the invasion of microorganisms (LUCAS, 2004, ALVES et al., 2016).

WOUND CLASSIFICATION

There are several types of wounds that can occur, and their knowledge, as well as the stage they are in, are essential for the best choice of treatment in each case and moment. Through history, anamnesis and physical examination, wounds can be classified in relation to their condition (PAVLETIC, 2018), according to depth, degree of contamination, and time of evolution (TAZIMA et al., 2008, SMANIOTTO et al., 2010, PAVLETIC, 2018).

As for depth, they are classified as the superficial or deep. When they do not affect the entire skin structure, affecting only its most superficial layer, the epidermis, they are classified as the superficial wounds, with abrasions belonging to this classification. When the wound reaches deeper layers of the skin, reaching the dermis and even the muscles and bones, it is classified as a deep wound, with incisions, avulsions, perforations and lacerations present in this group (TAZIMA et al., 2008).

According to the degree of contamination, they are classified as clean, clean-contaminated, contaminated and infected. Clean wounds are those performed with minimal trauma, within the aseptic conditions of a surgical unit, following Halsted's principles, and which do not involve the respiratory, oropharyngeal, gastrointestinal and genitourinary tracts, which are naturally contaminated systems. Clean-contaminated procedures are carried out under aseptic conditions, but which

involve the respiratory, oropharyngeal, gastrointestinal and genitourinary tracts, but without any breach of aseptic technique (HEAL et al., 2016). Contaminated wounds are of traumatic origin, which occurred within 4 to 6 hours. Also belonging to this group are surgical wounds in which there was a great breach of aseptic conditions or spillage of gastrointestinal/genito-urinary contents into the cavity, and those that occurred close to contaminated or inflamed areas, but without the presence of purulent content. Finally, infected wounds are of traumatic origin, which occurred more than 4-6 hours ago or which already show visual signs of infection, such as the presence of purulent exudate. They are also classified this way when there are perforations of viscera or organs of the genitourinary system, and when there are more than 100,000 living organisms per gram of tissue (VAN HENGEL et al., 2013, PAVLETIC, 2018).

According to the evolution, they are classified as acute or chronic. Acute wounds are those in which the healing process occurs in an orderly and expected manner, usually within a short period of time, up to 3 weeks. Chronic wounds are those in which their progression does not occur as expected, with a delay in healing. Wounds that take more than 3 months to heal are classified as chronic (SMANIOTTO et al., 2010).

WOUND HEALING

After tissue disruption, the repair process begins, in which a sequence of reactions occurs in order to rebuild the injured tissue. This series of reactions is known as healing, being a physiological and spontaneous process that occurs through three distinct phases: inflammatory, which begins immediately after the injury, and can last for 4 to 6 days; proliferation, starting on the 4th day after tissue injury, extending to the 14th day; and remodeling, starting on the 8th day and extending for more than 1 year (BROUGHTON et al., 2006).

INFLAMMATORY

The first event immediately after the injury is bleeding control, where the affected blood vessels perform vasoconstriction, around 5 to 10 minutes, through stimulation by catecholamines, serotonin, bradykinin and histamine (MACPHAIL, 2015). After that, the vascular endothelium together with the platelets close to the lesion site activate the coagulation cascade, generating the formation of a clot, composed of collagen, platelets, thrombin and fibronectin (CAMPOS et al., 2007). The clot formed serves as a framework for the concentration and storage of cytokines and inflammatory mediators, such as prostaglandins, prostacyclins, thromboxanes and leukotrienes that initiate the inflammatory response. Platelets also secrete important vasoactive substances such as histamine and serotonin and some factors such as epidermal growth factor (EGF), platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-beta). Due to all these factors, cytokines and vasoactive substances released, blood vessels that were previously constricted dilate to increase chemotaxis, with the aim of guiding defense cells to the site of injury (KONDO; ISHIDA, 2010, STANLEY; CORNELL, 2018).

Neutrophils are the first defense cells attracted to the lesion site, and their migration is stimulated by chemotactic substances such as the complement system, interleukin-1, tumor necrosis factor-alpha, PDGF, TGF-beta, bacteria and others. Its function is to clean the wound bed, releasing proteinases that degrade non-viable and necrotic tissue in the wound bed, phagocytosing bacteria and removing damaged cells and parts of the extracellular matrix that are denatured

or damaged. When their function has been performed, they go into apoptosis (PAVLETIC, 2018).

Monocytes are also attracted to the area of tissue injury by PDGF, elastin, fibronectin, thrombin, and TGF-beta, and subsequently transform into activated macrophages at the site of injury. They come into action after the programmed death of neutrophils and continue the process of cleaning the wound, removing bacteria, contaminants and cellular debris through phagocytosis. They also release proteases that aid in enzymatic wound debridement (KONDO; ISHIDA, 2010, STANLEY; CORNELL, 2018, PAVLETIC, 2018). Macrophages play an essential factor in the transition from the inflammatory to the proliferative phase, as they stimulate and direct in an organized way various growth factors, cells and components of the cellular matrix for the beginning of the second phase (LAWRENCE; DIEGELMANN, 1994).

PROLIFERATION

This phase is composed of three important events that follow the inflammatory phase period: fibroplasia, angiogenesis and re-epithelialization. At this moment, fibroblast proliferation and granulation tissue synthesis occur, which consists of new vessels to be formed, fibroblasts, macrophages, a loose arrangement of collagen, fibronectin and hyaluronic acid, followed by the formation of new capillaries and the proliferation of cells. epithelial cells (TAZIMA et al., 2008, ISAAC et al., 2010).

During fibroplasia, fibroblasts that were at the edge of the wound migrate to the wound site, where they proliferate and synthesize collagen by stimulation of PDGF, EGF and fibroblast growth factor (FGF), secreted by macrophages and platelets. In response to these factors, the fibroblasts begin to synthesize a new provisional extracellular matrix, which is looser, formed mainly by type III collagen, glycosaminoglycans and fibronectin, which together with the new capillaries that will be formed and other cells present in the bed of the wound, form granulation tissue. Finally, fibroblasts are transformed into myofibroblasts by the stimulation of TGF-beta produced by macrophages, and start wound contraction (BALBINO et al., 2005, BROUGHTON et al., 2006).

Myofibroblasts have intracellular actin and myosin fibers that bind with fibronectin and collagen fibrils that are present in the extracellular matrix. In turn, the collagen fibrils that cross the entire length of the matrix interconnect within the granulation tissue reaching the dermal layer at the edge of the wound. Through all this connection, myofibroblasts are able to apply a traction force through the cells and matrix, causing wound contraction (PAGANELA et al., 2009, PAVLETIC, 2018).

Mainly by the stimulation of vascular endothelial growth factor (VEGF) caused by keratinocytes, macrophages, fibroblasts, platelets and other endothelial cells present at the edge of the wound, new capillaries begin to be formed in the injured bed, initiating the process of angiogenesis. This development is essential for the synthesis, deposition and organization of a new extracellular matrix (KONDO; ISHIDA, 2010), being responsible not only for the nutrition of the tissue that requires a greater metabolic demand, but also for the increase in the supply of cells, such as macrophages, fibroblasts and stem cells to the wound site (TAZIMA et al., 2008).

Reepithelialization occurs by the proliferation of epithelial cells that were on the edge of the wound, occupying the injured area for protection as a barrier against fluid loss and bacterial invasion. These cells are stimulated by EGF and transforming growth factor alpha (TGF-alpha), produced by activated

macrophages and platelets. The main cells of reepithelialization are the keratinocytes, receiving stimulation from the EGF and TGF-alpha factors to proliferate and differentiate into a new epithelium. In addition, fibroblasts also act by synthesizing and secreting growth factors exclusively related to keratinocytes (KGF-1, KGF-2), and this way, they also end up stimulating these cells to proliferate (BROUGHTON et al., 2006, LAUREANO; RODRIGUES), 2011).

REFURBISHMENT

The main occurrence of this phase is the deposition of collagen in an organized and successful manner and the replacement of the granulation tissue by a more resistant matrix, composed of type I collagen. During this phase, the production of type III collagen ceases, increasing the production of type I collagen, which is stronger, by TGF-beta. This initial type I collagen is thinner than the one existing in skins that have not suffered any type of injury, and therefore, over months to years, this collagen is reabsorbed, synthesized and remodeled again, increasingly resistant, so that at the end of the process, this region that was injured can reach a resistance close to what it was before the tissue injury (KONDO; ISHIDA, 2010).

It is in the remodeling phase that the wound begins to gain strength and strength. The fastest gain in this strength occurs between 7 and 14 days after injury, as collagen accumulates in the wound bed. The wound gains only 20% of the final strength in the first 3 weeks after the injury. Subsequently, there is a slower increase in this strength over time, but it is never regained in its entirety, and only 80% of the original strength can be recovered after healing is complete (MACPHAIL, 2015).

It is important to note that each of these phases does not happen in isolation, and can often overlap with each other. It is common to find elements from the subsequent phase, or even from the previous phase, at a given moment (AMORIM et al., 2006).

FACTORS THAT INFLUENCE WOUND REPAIR

Even though animals have lower rates of complications in wound repair when compared to humans, in view of issues such as smoking and alcoholism, eventually some factors end up negatively impacting the healing process. These factors can be divided into intrinsic or extrinsic. The intrinsic ones are associated with changes in the body itself, such as low tissue perfusion, accumulation of fluid or infection in the wound bed, endocrinopathies, concomitant diseases, malnutrition and age. Extrinsic factors are related to factors external to the body, such as excessive administration of corticosteroids, and treatment with radiotherapy and chemotherapy (STANLEY; CORNELL, 2018).

INTRINSIC FACTORS

It is necessary that the wound has adequate tissue perfusion for its healing to occur successfully. Low tissue perfusion can happen due to systemic factors such as shock and hypotension, hypovolemia, pain and diabetes mellitus, which must be controlled and resolved in conjunction with wound care (WORTHLEY, 2000, STANLEY; CORNELL, 2018). These factors can alter and decrease the normal blood flow to the wound site, hindering the transport of nutrients and drugs, and impairing the migration of cells, such as those of the immune system, to the wound site, delaying the healing process. et al., 2008).

For a good evolution in the repair process, there can be no devitalized or necrotic tissues and foreign bodies in the wound bed, as it will be stagnant in the inflammatory phase, where the defense cells will constantly try

to clean the wound site (KIRSHEN). et al., 2006). Non-viable tissues and cellular debris must be removed in the early stages of the wound through debridement, which can be performed mechanically or in more severe cases, through surgical debridement (AYELLO; CUDDIGAN, 2004).

The accumulation of fluid in the wound, whether blood or inflammatory fluid, can exert excessive pressure on the wound bed, impairing tissue perfusion and possibly generating ischemia of adjacent tissues, with consequent necrosis. These fluids also serve as a medium for the proliferation of bacteria in the wound bed, in addition to increasing the dead space between tissues and hindering the migration of molecules and cells during the phases of inflammation and proliferation. To avoid the accumulation of these fluids, the wound can be left open, draining constantly in the chosen dressing, or the use of drainage techniques is chosen (STANLEY; CORNELL, 2018). The mode of drainage will mainly depend on the characteristics of the wound and which treatment method was chosen.

The formation of granulation tissue is essential for good healing, but its excessive growth beyond the wound edges prevents the healing sequence, as it blocks the proliferation of epithelial cells and consequent epithelialization (WALDRON; ZIMMERMAN-POPE, 2003). This is especially important in equids, as wounds that occur on the distal parts of the limbs, when compared to the upper part, place greater tension on the skin. This factor leads to a reduced epithelialization rate and consequent exuberant growth of granulation tissue (WILMINK; VAN WEEREN, 2004).

Another factor of great impact in the delay of wound healing is the presence of infection at the wound site. Skin wounds are susceptible to bacteria colonizing their tissues, as the primary protective barrier established by the intact skin has been lost (EDWARDS; HARDING, 2004). The colonization of the wound by microorganisms ends up stimulating the persistence of the inflammatory phase (JONES et al., 2004), which impairs the evolution of the repair process and prevents the wound from healing (BUCKNALL, 1980).

Some of the changes that are caused by infection at the wound site are the increased release of pro-inflammatory cytokines, caused by the release of endotoxins by bacteria, generating an exacerbated and harmful inflammatory response to the wound. There is also an increase in the production of reactive oxygen specimens by the release of cytotoxic enzymes, causing great tissue damage at the site of injury because they are capable of damaging nucleic acids, lipids, proteins and DNA. In addition, they lead to a greater consumption of complement system proteins, causing a decrease in chemotaxis and consequent decrease in the action of leukocytes and macrophages in the wound bed (ROBSON et al., 1990, VALKO et al., 2007, STANLEY; CORNELL, 2018).

And not only the inflammatory phase is affected, it can extend to the proliferative phase. Due to the release of endotoxins, proteases and other enzymes, keratinocyte migration is impeded, fibroblast proliferation slows down, collagen production becomes disorganized and granulation tissue becomes friable. In addition, components present in the bacterial wall can interfere with VEGF expression and consequent proliferation of endothelial cells, impairing the formation of new vessels and blood supply to the wound (POWER et al., 2001).

Malnutrition directly affects tissue repair. Malnourished animals with low plasma protein may have delayed healing generated by a reduction in wound strength (MACPHAIL, 2015), which is linked to decreased action of fibroplasia and collagen production. In

addition, glucose deficiency impairs the functioning of cells, affecting fibroblasts and defense cells, such as neutrophils, decreasing their action during the healing phases (PAVLETIC, 2018).

Still in relation to nutritional diseases, hepatic alterations resulting from inadequate diets can alter the course of wound hemostasis, by causing a deficiency of clotting factors that help in clot formation. This is a very important factor that helps to initiate the inflammatory phase (MACPHAIL, 2015).

Finally, older age can impair all stages of healing. The inflammatory phase becomes exacerbated as there is an increase in the stimulation of pro-inflammatory cytokines and the migration of inflammatory cells to the wound when the animal is aged. There is also a delay in epithelialization due to decreased migration and proliferation of epithelial cells, and reduced proliferation of fibroblasts, decreasing the rate of extracellular matrix production (ASHCROFT et al., 2002).

EXTRINSIC FACTORS

Immunosuppression resulting from the use of chemotherapy and radiotherapy, and the excessive administration of glucocorticoids negatively affect all phases of healing (STANLEY; CORNELL, 2018).

Corticosteroids reduce vascular permeability, inhibit macrophage migration and fibroblast proliferation, delay angiogenesis, and significantly decrease the synthesis and deposition of type I and III collagen, first delaying the inflammatory phase of the wound and thereby affecting subsequent phases. They also increase the chances of infection at the wound site (NUUTINEN et al., 2003, PAVLETIC, 2018).

Radiation therapy can inhibit wound healing depending on the dose and exposure time. Due to the principle of modifying DNA and RNA, it affects cell division, causing a decrease in epithelialization, destruction of fibroblasts, with a consequent reduction in collagen synthesis. There is also a decrease in the formation of new capillaries, leading to a restriction of blood supply (HOSGOOD, 2003, MACPHAIL, 2015).

Some chemotherapeutic agents interfere with the action of vitamin B6, B12, folic acid, ascorbic acid, zinc and iron metabolism. It is necessary to monitor the animal that makes prolonged use of these drugs, as it can interfere with the animal's nutrition (PAVLETIC, 2018).

PRINCIPLES IN WOUND TREATMENT

The choice of a wound care plan must take into account the extent of the wound and the degree of local contamination. In the evaluation of the extension, the area, the depth and the tissues that were affected must be considered, as well as the factors that can interfere in the healing process (REITER, 1995).

Wounds can be closed immediately after injury, characterizing treatment by first intention or primary closure; they can be left open, for closure from the inside out with the formation of granulation tissue, followed by epithelialization and contraction of the wound, called in this case, treatment by second intention; or the lesion can be treated initially openly until the formation of granulation tissue and wound disinfection, and be closed later by simple sutures or reconstructive surgeries, characterizing the treatment by third intention or tertiary closure (HOSGOOD, 2018).

TREATMENT BY FIRST INTENTION OR PRIMARY CLOSURE

It concerns the direct closure of the wound (PAVLETIC, 2018), mainly obtained by the use of sutures, however, skin staplers, adhesives or clips can be used (MURTHA et

al., 2006). This treatment is indicated in cases of clean and clean-contaminated wounds. It is not indicated for wounds with devitalized tissue, excess cellular debris, considerable tissue loss, visible tissue contamination or the presence of exudate (FAHIE, 2018). Another parameter to be considered is the time elapsed between the occurrence of the injury and its treatment, which must not exceed 6 hours (GOTTRUP, 1999, MACPHAIL, 2015). The success of the procedure depends on wide shaving around the wound, adequate antisepsis of the bed and adjacent regions and a good approximation of the wound edges, avoiding tension during closure (TOBIAS, 2010).

Treatment by first intention can still be applied in cases of wound occurrence for more than 6 hours or where there are local factors that increase the risk of wound contamination. This is only indicated if the wound has adequate characteristics for closure, such as absence of infection, appearance of tissue vitality, good bed antisepsis, successful debridement. It is noteworthy that in these cases the use of systemic antibiotics is mandatory until the wound is healed (FAHIE, 2018).

Closed wounds with the presence of contamination, necrotic tissue, excessive tension or inadequate abolition of dead space are very susceptible to dehiscence, additional tissue loss, and necrosis from increased tissue tension. When there is dehiscence, it is indicated that the wound receives appropriate treatment and is subsequently treated by second or third intention (MACPHAIL, 2015). When the wound presents infection but there is no dehiscence, the sutures must be removed to allow the drainage of the exudate and the cleaning of the bed, and afterwards the wound must also be treated in an open way (VERMEULEN et al., 2004).

Treatment by first intention provides simpler postoperative care, less extensive and labor-intensive dressings, faster healing process, less discomfort and pain, less scarring, greater protection of structures underlying the wound bed and more pleasant aesthetic results. Therefore, the use of closure by first intention brings greater benefits to the patient and must be preferred when the characteristics of the wound allow the use of this technique (FAHIE, 2018).

TREATMENT BY SECOND INTENTION OR SECONDARY CLOSURE

Treatment by second intention is indicated for contaminated wounds that do not allow primary closure and that are not extensive enough to warrant surgical intervention to close them. This treatment consists of letting the wound heal openly, through the formation of granulation tissue, epithelialization and contraction of the injured bed (HOSGOOD, 2018). It is indicated for traumatic wounds with considerable tissue loss, which occurred more than 6 to 8 hours, with the presence of visible contamination or infection, or for those that are at high risk of becoming infected, 2015). The second intention is performed in cases where treatment by first intention is contraindicated. Also suitable for this type of treatment are wounds or skin defects that cannot be closed properly using surgical methods, usually in areas of great tension, without loose skin to facilitate primary closure. These locations are found on limb extremities, tail and some regions of the trunk (PAVLETIC, 2018).

Closure by second intention allows for optimal drainage of the wound, whether of inflammatory or purulent content. However, it has the disadvantage of taking a longer time for total tissue repair of the injured bed to occur (WALDRON; ZIMMERMAN-POPE, 2003). In addition, they may still have

some failures to produce complete healing. The new epithelium being formed is fragile, wound contraction may occur excessively, epithelization may not be complete in certain cases, leaving granulation tissue exposed in the wound bed that can grow excessively). The preparation of the wound for the treatment by second intention is the same as already described, an adequate shaving, antisepsis and debridement must be performed. After bed treatment, the wound must be closed with dressings or bandages and have an adequate control to avoid the occurrence of possible infections and traumas (WALDRON; ZIMMERMAN-POPE, 2003).

In most cases, treatment by second intention is a practical and cost-effective method of wound closure, provided that proper care is taken in wound management. However, in cases where this healing can be prolonged due to systemic or local changes, the cost can increase considerably, due to the constant requirement of materials for the dressing and medicines (PAVLETIC, 2018).

TREATMENT BY THIRD INTENTION OR TERTIARY CLOSURE

Treatment by third intention is indicated for contaminated and very extensive wounds, which cannot be closed by first intention, precisely because they are contaminated, and which would take a long time to close by second intention. This treatment consists of leaving the wound open in cases of infection of the bed with the presence of non-viable tissues. After treatment, when there is no more evidence of infection and the wound is healthy, surgical occlusion is performed using sutures or tissue reconstruction techniques (BELDON, 2010). This procedure is also indicated in cases where wound epithelialization and contraction were not successful in healing by second intention, or when it was not possible to perform closure by first intention immediately (MACPHAIL, 2015). It is also used in cases of persistence of devitalized and necrotic tissues, requiring additional care with the wound and performing debridement for a few days; and when the presence of a moderate to severe persistent inflammatory response occurs (PAVLETIC, 2018). By treating the wound openly for a few days, it is possible to reduce bacterial contamination of the bed to improve its appearance before closure. During this time, granulation tissue will develop, providing a vascular substrate, which helps to control contamination and facilitate healing (HOSGOOD, 2018). For wound occlusion, resection or debridement of excess granulation tissue must be performed. Next, the skin margins must be resected, washed and the edges approached so that a good suture occurs, without the occurrence of large areas of tension (MACPHAIL, 2015). Very extensive wounds may require the use of reconstruction techniques, such as muscle, skin and myocutaneous flaps, or even the use of free skin grafts.

The use of drains must be considered when there are doubts about the degree of tissue contamination or the effectiveness of dead space reduction. Drains allow the removal of exudates and daily cleaning of the bed, improving the quality of the wound and increasing the speed of healing (WALDRON; ZIMMERMAN-POPE, 2003, MACPHAIL, 2015).

FINAL CONSIDERATIONS

The high incidence of wounds, both in veterinary medicine and in human medicine, shows us the relevance of this subject and the importance of study and deepening for the best approach to each patient.

Therefore, knowledge of the physiological events involved in each phase of the healing

process, as well as the factors that can influence this process, either negatively or positively, are of great importance for understanding the injury, the repair phase in which it takes place. finds, and with that, the best therapy indicated for each patient and wound in question.

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