Wound healing is a complex and dynamic process used to restore the integrity of damaged skin or other tissues. The wound healing process can be broken down into 3 phases: debridement, repair, and maturation. The debridement phase begins immediately following wounding. It begins with vascular contraction to help mitigate blood loss. Once hemostasis has been achieved, blood vessels dilate to allow essential antibodies, white blood cells, and growth factors to reach the wounded tissues. The predominant cells at work here are the phagocytic cells, neutrophils and macrophages. Neutrophil infiltration is crucial within the first days after wounding because their ability to produce essential proteases and perform phagocytosis kills local bacteria and helps to degrade necrotic tissue. They start their debridement by releasing highly active antimicrobial substances like cationic peptides and eicosanoids, elastase, cathepsin G, proteinase 3 and an urolinas e-type plasminogen activator. Approximately 72 hours after wounding, macrophages enter the zone of injury and support the ongoing process by performing phagocytosis of pathogens and necrotic cell debris, and by secreting growth factors, chemokines and cytokines. Macrophages have many functions including host defense, the promotion and resolution of inflammation, the removal of necrotic or apoptotic cells and the support of cell proliferation and tissue restoration following injury. Besides their immunological functions as antigen-presenting cells and phagocytes during wound repair, macrophages play an integral role in a successful healing response through the synthesis of numerous growth factors, such as transforming growth factor (TGF-β, TGF-α), basic FGF, platelet derived growth factor and vascular endothelial growth factor, which promote cell proliferation. During the repair phase of wound healing, granulation tissue deposition provides a wound bed for re-epithelialization. It begins 3 to 10 days after wounding. The fundamental goals of the repair phase of wound healing are covering the wound surface, the formation of granulation tissue and restoring the vascular network. The phase is characterized by angiogenesis, granulation tissue formation, epithelialization and wound contraction. Granulation tissue basically consists of fibroblasts, collagen, and new blood vessels. The first step in new vessel formation is the binding of growth factors to their receptors on the endothelial cells of existing vessels, thereby activating intracellular signaling cascades. The endothelial cells proliferate and migrate. The newly built vascular sprouts form small tubular canals that interconnect with other advancing vascular loops. Near the end of this stage, fibroblasts migrate to the wound bed. They are either attracted from the edge of the wound or come from the bone marrow, and their recruitment is stimulated by macrophages. Some of these fibroblasts become specialized, i.e. myofibroblasts. Myofibroblasts are contractile cells that, over time, bring the edges of a wound together. Wound contraction is a biological means where the edges of an open wound are pulled together by forces resulting from the wound-healing process. The maturation phase is the last phase of wound healing and occurs from approximately day 21 and to up 1 year after injury. During the repair phase of wound healing, formation of granulation tissue ceases by apoptosis of the cells responsible for its production. This process is important because a mature wound is characterized as avascular and acellular. With wound maturation, the composition of the extracellular
matrix undergoes change. The type III collagen deposited during the repair phase is slowly degraded and replaced with stronger type I collagen. This type of collagen is oriented in small parallel bundles and is, therefore, different from the interlaced collagen in healthy dermis. The angiogenic processes also diminish; the wound blood flow declines and the acute wound metabolic activity slows down and finally stops.

Vacuum-assisted closure of wounds, or VAC, is a noninvasive, active, closed wound management system that exposes the wound bed to local, sub-atmospheric pressure in order to enhance wound healing. Also referred to as negative-pressure wound therapy, VAC has many benefits including the stimulation of granulation tissue formation, reducing interstitial edema and inflammatory cytokines, and improving circulation while maintaining a moist wound-healing environment. While the physiology behind the VAC system can be thought of as quite complex, applying the techniques while using the VAC on a clinical case is quite simple. The system works by first introducing a piece of 300-400nm, open cell foam into the wound or across the wound bed. A wound fluid drain tube with lateral perforations is then laid on top of the open cell foam. The entire area is then covered with a transparent, occlusive and adhesive membrane, which is firmly secured to the healthy skin around the wound margin. When the exposed end of the drain tube is connected to a vacuum source, fluid is drawn from the wound through the foam and into a reservoir container. The occlusive membrane prevents the ingress of air and allows a partial vacuum to form within the wound, reducing its volume and facilitating the removal of fluid. The foam ensures that the entire surface area of the wound is uniformly exposed to the negative pressure effect, which eliminates the theoretical possibility of localized areas of high pressure and resultant tissue necrosis.

The practice of exposing a wound to sub-atmospheric pressure for an extended period to promote debridement and healing was first described in 1993. The system was successfully used to treat 15 human patients with open fractures. They reported that the treatment resulted in "efficient cleaning and conditioning of the wound, with marked proliferation of granulation tissue." Over the next two and half decades, well over 900 peer-reviewed articles have been published describing the VAC system. The clinical and economic benefits of the VAC system are widely known - faster and more effective healing when compared to gauze sponges - leading to improved healing at an overall lower cost of care. In the early studies, negative pressure within the wound was achieved using a standard wall suction apparatus or surgical vacuum system. These systems were associated with problems in terms of the delivery, control and maintenance of the required levels of negative pressure. In 1995, a commercial system for promoting negative pressure wound therapy was introduced into the United States market by a company called Kinetic Concepts Incorporated or KCI. At the heart of this technology lies a microprocessor-controlled vacuum unit that can provide controlled levels of continuous or intermittent sub-atmospheric pressure ranging from 25 to 200 mmHg.

In the first series of experiments using the VAC system, blood flow measurements were obtained in the subcutaneous tissue and muscle surrounding wounds as they were exposed to increasing levels of negative pressure, applied both continuously and intermittently. The results indicated that while an increase in blood flow equivalent to four times the baseline value occurred with negative pressure values of 125 mmHg, blood flow was inhibited by the application of negative pressures of 400 mmHg and above. A negative pressure value of 125 mmHg was therefore selected for use in subsequent studies. The rate of granulation tissue production under negative pressure was determined by measuring the reduction in wound volume over time compared to control wounds dressed using saline.
soaked gauze sponges. Microbiological studies were also undertaken in which large numbers of microorganisms were introduced to various wounds and bacterial counts tallied while using the VAC system or controls. The results showed that the bacterial counts of vacuum-treated wounds were lower compared to wounds treated using conventional methods. Negative pressure wound therapy has also been shown to increase free graft and skin flap survival compared with control values.

VAC therapy has many biologic and mechanical mechanisms attributed to its success, one of the main being the removal of interstitial fluid which decreases localized edema and increases blood flow, which in turn decreases tissue bacterial levels. Another proven benefit of the application of sub-atmospheric pressure is mechanical deformation or stress within the tissue resulting in protein and matrix molecule synthesis and enhanced angiogenesis. Negative pressure wound therapy also stimulates the production of granulation tissue and has been shown to enhance the formation of epithelialization. I like to think of the VAC system as a granulation tissue making machine requiring less hospital visits and wound dressing changes.

There are many indications for utilizing the VAC system in animals including the treatment of large, open, contaminated wounds, dehisced surgical wounds with or without exposed orthopedic implants or bones, degloving injuries and skin avulsions, and chronic non-healing wounds. VAC wound care can also be used to prevent postoperative seroma or edema formation and enhance healing following a free graft procedure. The VAC system can reduce wound size and volume, enhance the acceptance following a free skin graft, improve patient response to antibiotic therapy, and reduce the duration of open wound management ultimately reducing hospitalization time and number of bandage changes. VAC is most efficacious during the debridement stage of wound healing, but can be applied to almost any wound after appropriate debridement. To eliminate a potential nidus for bacterial growth and prevent proliferation of granulation tissue over necrotic tissue, which could result in delayed wound healing and abscess formation, devitalized tissue should be removed before VAC therapy is initiated. VAC wound care is contraindicated when neoplasia is present at the wound site and should not be used as sole therapy for osteomyelitis. Severe hemorrhage may occur when VAC is used on wounds with exposed arteries or veins, as these vessel walls could erode and cause life threatening hemorrhage. VAC should also be used with caution in patients with coagulopathies.

The most common pressure setting used in VAC wound care is continuous pressure of –125 mm Hg. When VAC is used to diminish postoperative edema or seroma formation on incision lines, pressure can be reduced to –75 mm Hg. Application of intermittent negative pressure can improve blood flow and granulation tissue formation, but in humans has reportedly caused additional discomfort. For this reason, VAC usage in small animal patients is usually done under continuous negative pressure. Frequency of bandage changes is determined by wound characteristics, but VAC bandages are typically changed every 48 to 72 hours, with more frequent changes early in the process to allow appropriate debridement. An airtight seal is essential to maintain continuous negative pressure and prevent wound desiccation. Complications of VAC wound care are generally minor, but can include loss of an airtight seal and local dermatitis associated with removal of the adhesive drape.

References:


