MEDIHONEY®
Wound and Burn Dressing

Advanced Wound Care

Make Progress with Wound Debridement

A discussion on necrotic tissue, the importance of removing necrotic tissue from the wound environment, methods of debridement, and the role of MediHoney® dressings.

Necrotic Tissue and Necrotic Burden

Necrotic or avascular tissue is the result of an inadequate blood supply to the tissue in the wound area. It contains dead cells and debris that is a consequence of the dying cells.[1-3]

There are different types of necrotic tissue including eschar and slough. Avascular tissue exposed to the air will form a hard black crust known as **eschar**. If kept moist, avascular tissue will appear brown, yellow or gray and soft, flimsy or stringy. This tissue is called **slough**. Slough is fibrinous tissue consisting of **fibrin**, bacteria, intact leucocytes, cell debris, and serous exudates. After eschar is debrided, slough may be present as the wound is not completely clean. Thereafter, if a moist wound environment is not maintained, continued exposure to air may dessicate remaining slough, causing eschar to reform.[4-5]

Necrotic burden is the combination of necrotic tissue, excess exudate and high levels of bacteria present in dead tissue that accumulate in chronic wounds. Necrotic burden creates an altered cellular environment (elevated pH, proteases, biofilm, free radicals) which causes a cascading effect that can prolong the inflammatory phase, obstruct wound contraction and impede the reepithelialization process.[2]

Causes of Necrotic Burden

SKIN FAILURE

Skin failure happens when skin and underlying tissue die because of hypoperfusion, often concurrent with severe dysfunction or failure of other organ systems. Determining skin failure is currently done by gross examination of muscle mass, subcutaneous tissue thickness, wound granulation, and tissue necrosis. In addition, stratifying skin failure according to the patient's medical condition can be useful in planning interventions and setting treatment goals.

Skin failure can be typified as acute, chronic and end-stage.

Acute skin failure occurs when skin and underlying tissue die because of hypoperfusion concurrent with a critical illness.

Chronic skin failure is an event in which skin and underlying tissue die because of hypoperfusion concurrent with a chronic disease state. It typically happens more steadily over time, and in older individuals. Multiple co-morbidities combined with other age internal organs can manifest in the external organ of skin.

End-stage skin failure occurs when skin and underlying tissue die because of hypoperfusion concurrent with the end of life.

LACK OF BLOOD FLOW OR DECREASED TISSUE PERFUSION

Lack of blood flow or decreased tissue perfusion may be caused by occlusion, vasoconstriction, venous hypertension, hypotension, dehydration, medications, radiation, smoking, and inability to transport oxygen. Oxygen fuels the cellular functions essential to the repair process, making it critical to wound healing. Lack of blood flow causes a decrease in oxygen, slowing or stalling the healing process.

ELEVATED PROTEASE ACTIVITY

Elevated protease activity may occur in chronic wounds and may inhibit healing by degrading extracellular matrix proteins, growth factors, their receptors and protease inhibitors.[2] Protease activity can potentially be reduced by lowering the pH of a wound. This also may result in increased oxygen release, enhanced destruction of abnormal wound collagen, and increased macrophage and fibroblast activity.[6]

FREE RADICALS

Non-healing wounds typically display increased reactive oxygen species (ROS). ROS are deleterious in excess amounts due to their high reactivity, which causes oxidative stress. This is associated with reperfusion injury, one of the primary factors in chronic wound development. In the chronic wound environment, ROS attack DNA, which can lead to cell apoptosis.[7] Tight regulation of ROS production and detoxification is crucial for the repair process in wounds.

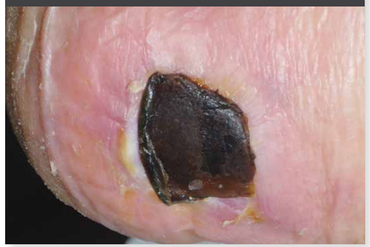
INFECTION AND BIOFILM

An infection is the presence of replicating microorganisms invading wound tissue and causing damage to the tissue and the host. Biofilms are created by colonies of bacteria attached to a substrate.[8] These colonies produce an extra-polymeric substance that holds the bacteria together and protects them. Biofilms and their causative organisms are not visible to the naked eye, and conventional swabbing is often inconclusive in identifying them.[8] Biofilms may repopulate in toxic wound environments in which cells break down and chronic inflammation occurs. This is exacerbated by the release of planktonic bacteria from the biofilm, which stimulates an inflammatory response.[9] Persister cells can repopulate the biofilm despite antibiotic susceptibility and therapy.

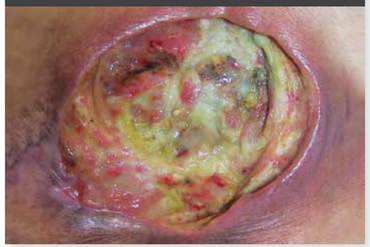
FOREIGN BODIES

Finally, external factors such as foreign bodies can complicate wound management. Debris (projectiles, splinters, glass and shrapnel), and even fragments of dressing and suture material, must be removed as they can interfere with healing.

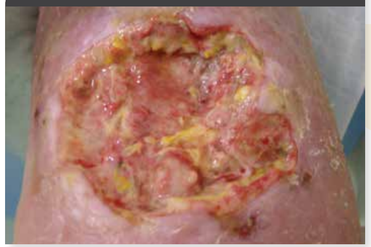
ESCHAR



SLOUGH



FIBRINOUS TISSUE



Importance of Optimizing and Controlling the Wound Bed Environment

A wound management plan should include a thorough wound assessment and selection of appropriate products to address the specific needs of the wound. Setting goal- oriented strategies to gain control over the wound environment will help get the wound back on track towards healing. Appropriate goals such as maintaining the physiologic wound environment (e.g., debridement, cleansing, prevention/management of infection)[1-3, 10] and providing systemic support (e.g., edema reduction, nutrition, hydration) are the foundation to the process.

When necrotic tissue is present, there are a number of related factors that could be the root cause of delayed healing:

- **Non-resolving inflammation**
- **Bacterial infection or 10^5 organisms per gram of wound tissue**
- **Elevated levels of proteases**
- **Impaired perfusion, decreased tissue oxygenation and/or oxidative stress**

Removal of necrotic tissue is therefore fundamental to allowing the wound to progress. Proper debridement removes and reduces the barriers that impede the healing process and provides an environment that stimulates the growth of healthy tissue needed for wound healing.[9]

Defining Stalled Wounds

- **If a Pressure Ulcer does not reduce in size at least 39% in two weeks it may not heal in a timely fashion.[11]**
- **If a Venous Leg Ulcer does not show at least 30% reduction in 4 weeks, it is probable that it will not be healed at 6 months.[12]**
- **9% of patients with Diabetic Foot Ulcers with <53% closure in 4 weeks, go on to close by Week 12.[13]**

Goal

Prepare the wound bed and promote moist wound healing.

Time Management[10]

Proper wound bed preparation is essential to promoting the wound healing process. Utilizing the TIME Management protocol will help to keep that wound on track.

Tissue management

Infection and inflammation control

Moisture balance

Epithelial (edge) advancement

Overview

Debridement is the process of removing devitalized or necrotic (dead) tissue from a wound or wound bed to expose healthy tissue, allowing the healthy tissue to granulate and advance the wound through the healing process. Debridement is indicated for any wound, acute or chronic, when necrotic tissue, foreign bodies or infected tissue is present. Necrotic tissue or foreign bodies within a wound can lead to a negative cascading effect causing the wound to become chronic. Because this effect can lead to more serious consequences, including infections or limb amputation, it becomes imperative that an effective wound management strategy be structured and implemented.

Primary purposes for debridement are to:

- Control and remove infectious materials, bioburden and/or biofilm[1-3]
- Interrupt the cycle of the chronic wound so that protease and cytokine levels more closely resemble those of the acute wound[1-3]
- Facilitate visualization of the wound edges and base for accurate assessment[1-3]

Debridement

Progress

Types of Debridement

The standard methods of debridement are autolytic, mechanical, enzymatic, sharp and biologic. The method of debridement used often depends on the amount of necrotic tissue present in the wound bed, the extent of the wound, and the patient's medical history and overall condition. Clinicians sometimes use more than one debridement method to achieve the most successful removal of necrotic tissue.

Autolytic debridement uses the body's own enzymes and moisture to re-hydrate, soften and finally liquefy eschar and slough. During autolysis, enzymes present in wound fluid have the effect of liquefying non-viable tissue. Clinicians foster autolytic debridement by utilizing moist wound dressings. By maintaining a moist wound environment, the body is able to use its own processes to eliminate necrotic tissue. Autolytic debridement can be achieved with the use of occlusive or semi-occlusive dressings which maintain wound fluid in contact with the necrotic tissue. It is virtually painless for the patient and safe, yet is generally slower than other forms of debridement. It can be used on its own, after surgical debridement, or in conjunction with enzymatic or mechanical debridement.

Mechanical debridement is a process in which a tool is used on the necrotic tissue to rip, pull, push, cut or abrade away devitalized tissue from the healthy tissue. Mechanical debridement is often non-selective and may remove or cause damage to healthy tissue as well as necrotic tissue. Examples of mechanical debridement include wet-to-dry dressings, wound irrigation, pulsatile lavage, whirlpool, contact ultrasound and scrubbing the surface with gauze. Wet-to-dry dressings do not provide a moist wound healing environment and are not optimal for wound care once the wound is free of necrotic tissue.

Sharp debridement, a form of mechanical debridement, is the removal of devitalized tissue by a skilled clinician, typically using a scalpel, scissors, curette or other sharp instrument. Clinicians use conservative sharp debridement to remove loosely adherent nonviable tissue at the bedside or in a clinical setting. Surgical debridement is done by a physician usually in the operating room, under anesthesia, with instruments and/or a laser when the tissue removal needs are extensive, or when the patient has a serious infection associated with the wound. Although sharp debridement is fast, it is non-selective and can be very painful to the patient.

Enzymatic debridement, or chemical debridement, makes use of certain enzymes and other compounds to dissolve necrotic tissue. It requires a prolonged period of enzyme activity, and a moist wound environment with appropriate pH and temperature. Enzymes are inactivated by metals in some wound care products (e.g. silver, zinc).[4] The enzyme used in the U.S., collagenase, works by dissolving the collagen "anchors" that secure the avascular tissue to the wound bed. Collagenase has been shown to be most active within a pH range of 6 to 8.[14]

Biologic debridement uses maggots grown from the sterilized eggs of *Lucilia sericata*. The larvae are placed in the wound bed, where it is theorized that they secrete proteolytic enzymes that break down necrotic tissue, which they then ingest. This is considered an option when the patient is not a surgical candidate and has not responded to other methods of debridement.

Continual vs. intermittent debridement

It has been found that rates of healing increase when wounds are debrided more frequently.[15] With the increased knowledge of biofilms and their ability to repopulate, as well as the damaging effects of elevated proteases (MMP's) of the chronic wound, more focus has been placed on continual debridement vs. single or intermittent debridement.

Continual debridement provides a consistent action of removing necrotic tissue from the wound bed over a period of time, unlike single or intermittent methods. The ability to provide continual and consistent removal of necrotic tissue helps to create an optimal environment for healing and allows for less disruption to the wound bed.

The Role of MediHoney

The overall goal for wound bed preparation is to remove factors that may delay healing.[2] These factors in a stalled or chronic wound include necrotic tissue and altered levels and composition of wound exudates.

MediHoney dressings, containing Active *Leptospermum* Honey (ALH), helps to address factors that may cause delayed healing in chronic wounds. As demonstrated in multiple RCTs[16, 22-30] and hundreds of clinical papers, ALH aids and supports autolytic debridement due to its key properties. The high sugar content of MediHoney aids in the increased flow of fluid to support the continual cleansing of the wound environment, helping to remove devitalized or necrotic tissue through an osmotic effect.[17]

1 AUTOLYTIC DEBRIDEMENT

During autolysis, the body breaks down tissue or cells. A moist environment, supported by MediHoney dressings, aids the body's process of bringing wound fluid to the surface with endogenous enzyme, thus loosening and liquefying necrotic tissue.

2 HIGH OSMOLARITY

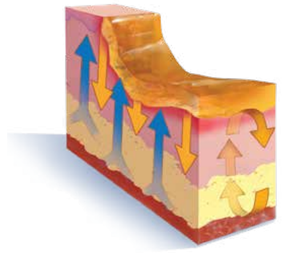
The high sugar content of honey facilitates movement of fluid from an area of higher concentration, across a membrane, to an area of lower concentration. Osmotic potential draws fluid through the wound, to the surface, helping to liquefy non-viable tissue.

3 LOW pH

The failure of a chronic wound to heal has been correlated with alkaline pH levels ranging from 7.15 to 8.94.[18] MediHoney has a low pH of 3.5–4.5. Maintaining more acidic pH levels within the wound environment can help to keep a wound on track towards healing.

Key Properties

HIGH OSMOLARITY



MediHoney aids and supports autolytic debridement

LOW pH LEVEL

MediHoney has a low pH of 3.5-4.5

Evidence Supporting the Use of MEDIHONEY®

Debridement of lower extremity wounds with Active *Leptospermum* Honey[19]

Becky Strilko RN, BSN, CWOCN, APN; Chris Barkauskas RN, BA, CWOCN, APN;
Andrea McIntosh, RN, BSN, CWOCN, APN, Silver Cross Hospital, Joliet, IL

CLINICAL ISSUE

Topical products containing papain, used for removal of necrotic tissue, control of inflammation, reduction of wound odor, and rehydration of the skin, are no longer approved for use in the United States. With the loss of papain-urea based debridement agents, there was a need to find a safe and cost-effective alternative for debridement of non-viable tissue and for wound bed preparation.

INCLUSION CRITERIA

Five patients were asked to participate in an open label pilot study. Selection criteria was limited to lower extremity ulcers with a minimum of 50% devitalized tissue. Active *Leptospermum* Honey (ALH), MediHoney, has experienced increased attention in the literature as an effective agent to promote autolytic debridement.[17, 20] Many patients in the outpatient setting are not candidates for surgical debridement.

OUTCOME

To determine the efficacy of this product for debridement of devitalized tissue as an adjunct to a successful wound treatment program.

TREATMENT PROTOCOL

In each case an ALH calcium alginate dressing was applied to non-viable tissue and covered with an absorbent cover dressing. Dressings were changed as needed based on wound exudate. Wound photography was performed on a weekly basis to document progression of debridement.

RESULTS

Dressings with ALH demonstrated the ability to promote debridement of necrotic tissue for five patients with lower extremity non-healing wounds. Several patients who were unable to tolerate hydrogel therapy reported improved dressing tolerance with the use of ALH. Improvement in all wounds was documented. There was a decreased percentage of non-viable tissue, increased percentage of granulation tissue, and increased dressing tolerance. The investigators confirmed that ALH dressings were an effective, first-line choice for promoting autolytic debridement and wound bed preparation for these cases.

CONCLUSION

Debridement is a vital part of the successful management of chronic wounds. ALH's ability to effectively promote autolytic debridement was observed in this group of five patients with differing wound types. Additional improved dressing tolerance were noted. The dressings were easy to use and well received by caregivers and patients.

CASE 1 - VENOUS ULCER

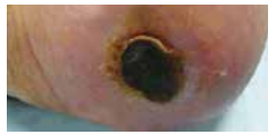
A 63 year-old female with a history of venous ulcer disease and Sjögren's syndrome presented with three full thickness, non-healing ulcers of the right posterior leg. She reported being "terrified" of surgical intervention and steadfastly refused sharp debridement. Persistent, consistent "bad" pain was reported as 4-6 on a 0-10 visual analog scale. Medications included hydrochlorothiazide and propoxyphene napsylate as needed for pain. Previous treatments with triple antibiotic ointment and papain-urea-chlorophyllin complex ointment were ineffective. Initially, the wounds measured 1.0 x 1.0 cm, 5.0 x 2.0 x 0.2 cm, and 1.5 x 1.5 cm with scant exudates and 100% eschar. Hydrogel therapy was prescribed to promote autolytic debridement. Increased pain was noted with hydrogel therapy (6-8 on a 0-10 visual analog scale). ALH calcium alginate was initiated on Day 0, covered with an absorbent cover dressing and changed one to two times per week. Gradual wound improvement was noted; the patient reported, "The dressings are natural and I did not need surgical debridement."

Day 0, ALH initiated

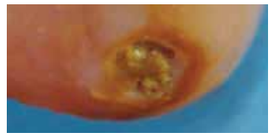
Day 116

CASE 2 - PRESSURE ULCER

An 89 year-old male with a history of coronary artery bypass graft, congestive heart failure, atrial fibrillation, chronic renal failure, and gout presented to the wound care center with an unstageable pressure ulcer on the left heel. The patient was taking the following medications: amlodipine besylate, levothyroxine sodium, niacininaide, valsartan, finasteride, allopurinol, furosemide, simvastatin, terazosin hydrochloride, and clonidine hydrochloride. Previous treatment with hydrogel therapy to promote debridement was ineffective. ALH calcium alginate dressings were initiated on Day 0, covered with an absorbent cover dressing and changed one to two times per week. Within one week progress toward debridement was noted.



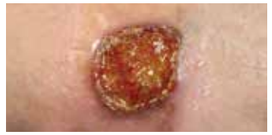
Day 0, ALH initiated



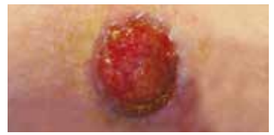
Day 8

CASE 3 - TRAUMATIC WOUND

A 53 year-old female with a history of hypertension, hyperlipidemia and bipolar disorder sustained a traumatic wound on the left anterior tibial region in a motor vehicle accident in May of 2008. Prior topical antimicrobial therapy (silver sulfadiazine) was ineffective for the promotion of wound management. Her medications included valproic acid, bupropion hydrochloride, a multivitamin, and zinc supplement. She presented to the wound care center on Day 0 with a full thickness wound with 90% slough and 10% granular tissue with a moderate amount of serosanguinous exudates. ALH calcium alginate was initiated, covered with compression bandaging and changed once per week. Within one week slough was eradicated, exudates decreased, and the patient reported the dressings were "comfortable". A skin graft was performed for final wound closure.



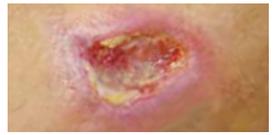
Day 7



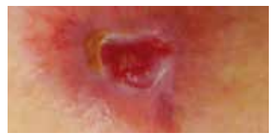
Day 14

CASE 4 - TRAUMATIC WOUND

A 60 year-old healthy male taking no medications sustained two full thickness wounds on the right patella from a motorcycle accident. Initial surgical debridement was performed. He presented to the wound center two weeks later on Day 14 with two full thickness wounds with 100% granulation tissue. Hydrogel therapy was prescribed to promote moist wound environment. One week later the base of each wound was covered with 90% slough and moderate amounts of wound exudates were noted. ALH calcium alginate dressings were initiated, covered with an absorbent cover dressing and changed every other day. The patient reported wound closure by week 6.



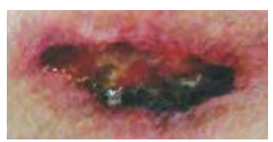
Day 36



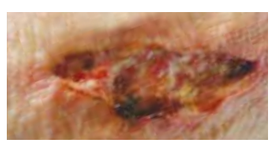
Day 65

CASE 5 - TRAUMATIC WOUND

A 77 year-old female with hypertension and COPD sustained an injury to her right lateral leg on Day 0. The patient was taking the following medications: fluticasonepropionate/salmetrol inhalation powder, multivitamin, tiotropium bromide, albuterol sulfate, furosemide, diltiazem hydrochloride, prednisone, and travoprost ophthalmic solution. She presented to the wound center on Day 37 with a 5 week old full-thickness wound surrounded by erythema and edema. The wound measured 6.0 x 2.5 x 0.3 cm with minimal exudates. The base of the wound was completely covered with slough (50%) and eschar (50%). Previous treatments were topical antimicrobial therapy, including silver sulfadiazine x 1 week and silver wound gel x 1 week. ALH calcium alginate dressings were initiated on Day 43, covered with an absorbent conforming gauze and secured with an adherent cohesive bandage once weekly. Within two weeks slough, eschar, and erythema were decreased.



Day 37, ALH initiated



Day 57

Evidence Supporting the Use of MEDIHONEY® (continued)

Use of Active *Leptospermum* Honey on difficult to heal wounds of various etiologies[21]

Nancy Chaiken, ANP-C, CWOCN, Swedish Covenant Hospital, Chicago, IL

PURPOSE/RATIONALE

Since ALH has multiple properties that address many common, underlying causes of non-healing wounds, it was chosen for use on several wound types to evaluate its therapeutic effects.

INCLUSION CRITERIA

The dressings were used on two patients with chronic non-healing wounds of varying etiologies, including rheumatoid ulceration and a stage IV sacral pressure ulcer. Patients also had multiple co-morbidities. The presence of one or more wounds was causing great pain and discomfort for each patient.

TREATMENT PROTOCOL

Patients were selected to receive ALH dressings based on the ability of ALH to aid in promoting autolytic debridement. ALH dressings were applied, covered with an absorbent cover dressing, and changed based on the amount of exudate.

RESULTS

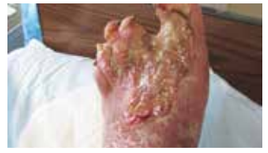
In each case significant wound improvement was noted as demonstrated by decreased slough. ALH dressings were easy to use, economical, effective, and well tolerated by each patient, subsequently improving life quality.

CONCLUSION

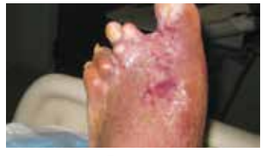
Choosing appropriate dressings and treatment modalities for individuals with chronic, non-healing wounds is challenging due to many underlying, causative factors. The use of dressings with ALH simplified the decision process. Patients in this study, with multiple co-morbidities and various wound types saw a reduction in slough.

CASE 1 - RHEUMATOID ARTHRITIS

A 53 year-old male with a history of rheumatoid arthritis, morbid obesity, myocardial injury, and hepatitis C was admitted to the hospital with a new diagnosis of esophageal cancer. He was referred for an evaluation for a foot wound that he had for 2 ½ years. Prior treatments of silver calcium alginate dressings and compression bandaging were ineffective. The patient was evaluated by rheumatology, however he refused systemic therapy for the rheumatoid ulcer; chemotherapy for esophageal cancer was in progress. ALH paste was initiated on Day 0, covered with an absorbent calcium alginate dressing, and secured with conforming gauze bandage. Compression bandaging was refused for edema management. Closure was achieved by Day 120, despite continual chemotherapy for esophageal cancer.


Day 0


Day 22


Day 120

CASE 2 - PRESSURE ULCER

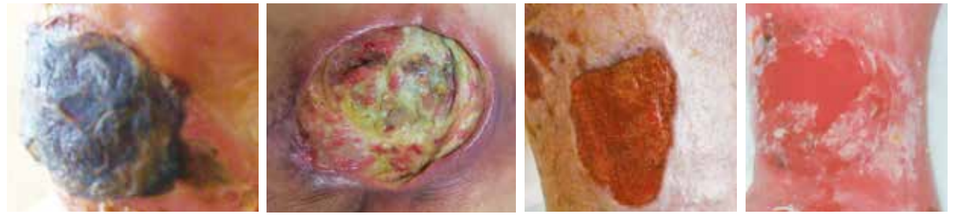
A 56 year-old female with a history of abdominal compartment syndrome, cirrhosis of the liver, acute pancreatitis, congestive heart failure, malnutrition and hepatic encephalopathy, developed a sacral pressure ulcer after an episode of ischemia. Initially the ulcer presented as deep tissue injury which then evolved to a stage IV pressure ulcer. The patient was not a candidate for surgical debridement and progress with alternative debridement methods was slow. On Day 0 ALH paste was initiated, and covered with an absorbent calcium alginate dressing daily. Minimal sharp debridement was performed as needed to remove loosened necrotic slough tissue. Wound closure, with only a small scab visible, was achieved by Day 122.


Day 0


Day 67


Day 122

MediHoney® Dressing Selection Guide for Superficial, Partial and Full Thickness Wounds and Burns



TYPE OF WOUND	Eschar		Sloughy		Granulating	Epithelializing
OBJECTIVE	Soften and Remove Eschar		Remove Slough		Promote Granulation	Maintain Moist Environment
EXUDATE	Light	Moderate	Light to Moderate	Heavy	Light to Moderate	Light
MediHoney Dressing (Primary Dressing)	Gel Paste HCS	Gel Calcium Alginate	Gel Paste HCS	Calcium Alginate	Gel HCS	HCS
Xtrasorb® Dressing (Secondary Dressing)	Foam HCS	Classic	Foam HCS Classic	Classic	Foam HCS	N/A

References: 1. Steed D, et al. Guidelines for the treatment of diabetic ulcers. Wound Rep Reg. 2006; (14) 680-692. 2. Enoch, S, Harding, K, Wound Bed Preparation: The Science Behind the Removal of Barriers to Healing, Wounds, 2003:15(7). 3. Landsman, Adam. Diabetic Foot Ulcers: Debridment and Reduction of Bioburdens. Podiatry Management. 2015; 105-110. 4. Vowden K and Vowden P. Wound Bed Preparation. World Wide Wounds: http://www.worldwidewounds.com/2002/april/Vowden/Wound-Bed-Preparation.html. 2002. 5. Langemo, Diane, Brown, Gregory, Skin Fails Too: Acute, Chronic, and End stage Skin Failure, Advances in Skin and Wound Care, 19(4). 6. Greener B, Hughes AA, Bannister NP, et al. Proteases and pH in chronic wounds. J Wound Care 2005; 14 (2) 59-61. 7. Telgenhoff, D, Shroot, B, Cellular senescence mechanisms in chronic wound healing, Cell Death and Differentiation, 2005:12, p. 695-698. 8. Wolcott R. Disrupting the biofilm matrix improves wound healing outcomes. Journal of Wound Care. 2015; 24 (8): 366-371. 9. Cooper, R. Biofilms and wounds: much ado about nothing? WOUNDS UK, 2010:6(4) 84-90. 10. European Wound Management Association (EWMA). Wound Bed preparation in practice. London: MEP Ltd, 2004. 11. van Rijswijk L and Polansky M. Predictors of time to healing deep pressure ulcers. Ostomy Wound Management. 1994; (8) 40: 40-51. 12. Cardinal M, et al. Early healing rates and wound area measurements are reliable predictors of later complete wound closure. Wound Rep Reg. 2008; (16): 19–22. 13. Sheehan P, et al. Percent Change in Wound Area of Diabetic Foot Ulcers Over a 4-Week Period Is a Robust Predictor of Complete Healing in a 12-Week Prospective Trial. DIABETES CARE. 2003; (26) 6: 1879-1882. 14. Rao DB, Sane PG, Georgiev EL. Collagenase in the treatment of dermal and decubitus ulcers. J Am Geriatr Soc 1975; 23 (1):22–30. 15. Saap LJ, Falanga V. Debridement performance index and its correlation with complete closure of diabetic foot ulcers. Wound Repair Regen. 2002;10(6):354-9. 16. Kamaratos AV, Tzirogiannis KN, Iraklianou SA, Panoutsopoulos GI, Kanellos IE, Melidonis AI. Manuka honey-impregnated dressings in the treatment of neuropathic diabetic foot ulcers. Int Wound J. 2012 ; 9: 1-7. 17. Amaya R. Safety and efficacy of active Leptospermum honey in neonatal and paediatric wound debridement. J Wound Care 2015; 24(3):97-103. 18. Gethin GT. The significance of surface pH in chronic wounds. Wounds UK. 2007; (3) 3: 52-56. 19. In house data: Strilko B, Barauskas C, McIntosh A. A safe and effective alternative for debridement of lower extremity wounds: Active Leptospermum honey dressings. Proceedings of Symposium on Advanced Wound Care and Wound Healing Society Meeting. April 2010, Orlando, FL. 20. Molan P, Rhodes. Honey: A Biologic Wound Dressing. WOUNDS 2015; 27 (6): 141-51. 21. In house data: Chaiken N. The use of Active Leptopermum Honey on difficult to heal wounds of various etiologies. Proceedings of symposium on advanced wound care, Orlando, FL 2010. 22. Gethin G, Cowman S. Bacteriological changes in sloughy venous leg ulcers treated with manuka honey or hydrogel: an RCT. J Wound Care 2008; 17(6):241-247. 23. Johnson D, Badre S, Pascoe E, et al. Antibacterial honey for the prevention of peritoneal-dialysis-related infections (Honey Pot): A randomized trial. Lancet Inf Dis. 2014 ;(1):23. 24. Johnson D, van Eps C, Mudge W, et al. Randomized, controlled trial of topical exit-site application of honey (Medihoney™) versus Mupirocin for the prevention of catheter-associated infections in hemodialysis. J Am Soc Nephrol 2005; 16(5):1456-62. 25. Johnson D, Clark C, Isbel N, et al. The Honeypot Study Protocol: A randomized controlled trial of exit-site application of Medihoney Antibacterial Wound Gel for the prevention of catheter-associated infections in peritoneal dialysis patients. Perito Dialy Int 2009; 29(3):303-309. 26. Jull A, Walker N, Parag V, et al. Randomized clinical trial of honey-impregnated dressings for venous leg ulcers. Br J Surg 2008; 95(2):175-182. 27. Lee VS, Humphreys IM, Purcell PL, Davis GE. Manuka honey sinus irrigation for the treatment of chronic rhinosinusitis: a randomized controlled trial. Int Forum Allergy Rhinol 2016; doi 10.1002/alr.21898 [epub ahead of print]. 28. Robson V, Yorke J, Sen R, et al. Randomised controlled feasibility trial on the use of medical grade honey following microvascular free tissue transfer to reduce the incidence of wound infection. Br J Oral Maxillofac Surg 2012; 50(4):321-7. 29. Robson V, Dodd S, Thomas S. Standardized antibacterial honey (Medihoney™) with standard therapy in wound care: randomized clinical trial. Journal of Advanced Nursing 2009; 65(3):565-75. 30. Thamboo A, Thamboo A, Philpott C, Javer A. Single-blind study of manuka honey in allergic fungal rhino sinusitis. J Otolaryngol Head Neck Surg. 2011; 40(3):238-243.z

A Guideline for Care – MediHoney® Dressing Application and Removal

- Wash hands thoroughly
- Apply gloves
- Assess the wound. Look for signs of healing. Also look for any signs of increased redness, pain, swelling, or heat within or around the wound*
- Cleanse the wound and skin around the wound with sterile saline, sterile water, or other safe wound cleansers
- Dry the skin around the wound by patting gently with gauze
- Protect the skin around the wound to avoid maceration. Apply a skin protectant barrier wipe or barrier ointment as necessary. (An initial increase in exudates may occur)
- Choose a MediHoney dressing that is appropriate for the amount of drainage:
  - MediHoney Paste or MediHoney Gel for lightly to moderately exudating wounds that are hard to dress
  - MediHoney HCS for non-draining to lightly exudating wounds that are superficial to partial thickness wounds
  - MediHoney Calcium Alginate dressing for moderate to heavily exudating wounds
- Apply the appropriate MediHoney dressing to fit the wound. The MediHoney Calcium Alginate and HCS Non-adhesive dresings can be cut to fit within the wound edges.
- Apply an absorbent cover dressing to manage exudate (Xtrasorb® super absorbent dressings are recommended)
- Dressing change: Remove the dressing gently, if the dressing is difficult to remove, moisten with saline or water

** The healthcare provider should be notified if the wound worsens. Report increased redness, pain, swelling, or heat on or around the wound.*

Contraindications

- On third degree burns
- With patients that have a known sensitivity to honey or any other component parts specific to each dressing (please see package insert for more information)
- To control heavy bleeding

Precautions

- If the dressing is not easily removed, soak with sterile saline or water until it is removed without difficulty.
- Due to the dressing's low pH, some patients may notice a slight transient stinging. If stinging does not stop or persists and cannot be managed with an analgesic, remove dressing, cleanse area, and discontinue the use of MediHoney dressing.
- During initial use of the dressing (depending on wound exudate levels, interstitial fluid, and edema surrounding the wound), the dressings high osmotic potential may contribute to increased exudate, which could lead to maceration if the excess moisture is not managed appropriately. Manage additional moisture by adding an absorptive cover dressing and/or adjusting the frequency of dressing change. Protect the peri-wound skin by applying a skin barrier protectant to the surrounding skin.
- During the healing process it is common for non-viable tissue to be removed from the wound resulting in an initial increase in wound size. Although an initial increase in wound size may be attributed to the normal removal of non-viable tissue, consult a healthcare professional if the wound continues to grow larger after the first few dressing changes.

MediHoney
Wound and Burn Dressing

PREPARE

MediHoney Wound and Burn Dressing

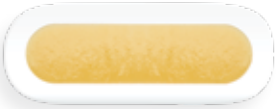
	Reference	Description	Packaging Unit/Case	HCPCS
Gel	31805	0.5 oz tube	10/box, 4 boxes/case	A4649
	31815	1.5 oz tube	1/box, 12 boxes/case	A4649
	31840	14 oz tube	1/jar, 6 tubs/case	------
Paste	31505	0.5 oz tube	10/box, 4 boxes/case	A4649
	31515	1.5 oz tube	1/box, 12 boxes/case	A4649
	31535	3.5 oz tube	1/box, 12 boxes/case	A4649
Hydrogel Sheet *Non-Adhesive*	31620	2.4 in x 2.4 in	10/box, 5 boxes/case	**A6242**
	31640	4.3 in x 4.3 in	10/box, 5 boxes/case	**A6243**
Adhesive	31720	2.8 in x 2.8 in (4.3 in x 4.3 in with adhesive border)	10/box, 5 boxes/case	**A6245**
	31740	4.5 in x 4.5 in (6 in x 6 in with adhesive border)	10/box, 5 boxes/case	**A6246**
HCS *Surgical*	31738	1.75 in x 6.5 in (3 in x 8 in with adhesive border)	10/box, 5 boxes/case	A4649
Fenestrated (Non-Adhesive)	31618	1.8 in x 1.8 in	10/box, 5 boxes/case	A4649
Non-Adhesive	31622	2.4 in x 2.4 in	10/box, 5 boxes/case	A4649
	31644	4.33 in x 4.33 in	10/box, 5 boxes/case	A4649
	31612	8 in x 12 in	2/box, 5 boxes/case	A4649
Adhesive	31722	2.8 in x 2.8 in (4.3in x 4.3in with adhesive border)	10/box, 5 boxes/case	A4649
	31744	4.5 in x 4.5 in (6 in x 6 in with adhesive border)	10/box, 5 boxes/case	A4649
Calcium Alginate	31012	0.75 in x 12 in	5/box, 4 boxes/case	A4649
	31022	2 in x 2 in	10/box, 10 boxes/case	A4649
	31045	4 in x 5 in	10/box, 5 boxes/case	A4649


Gel


Paste


Hydrogel Sheet *(Non-Adhesive)*


Hydrogel Sheet *(Adhesive)*


HCS *(Surgical)*


HCS -*Fenestrated (Non-Adhesive)*


HCS (*Non-Adhesive)*


HCS *(Adhesive)*


Calcium Alginate

Availability of these products might vary from a given country or region to another, as a result of specific local regulatory approval or clearance requirements for sale in such country or region.

- Non contractual document. The manufacturer reserves the right, without prior notice, to modify the products in order to improve their quality.
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- Consult product labels and inserts for any indication, contraindications, hazards, warnings, precautions, and instructions for use.

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