

Wound Cleansing, Wound Irrigation, Wound Disinfection

George T. Rodeheaver, PhD;
Catherine R. Ratliff, PhD, GNP-BC, CWOCN, CFCN

Objectives

The reader will be challenged to:

- Differentiate between wound cleansing, wound irrigation, and wound debridement
- Identify nontoxic wound cleansing solutions
- Select the appropriate devices for irrigating wounds
- Analyze which antimicrobials are toxic to wounds (antiseptics) and which are safe for wounds (antibiotics).

Additional Resources:

Cochrane review: Pressure Ulcer Cleansing
http://www.cochrane.org/CD004983/WOUNDS_wound-cleansing-to-help-pressure-ulcers-heal

Cleansing solutions for wound treatment systematic review
http://journals.lww.com/jbisir/Abstract/2014/12100/The_effectiveness_of_cleansing_solutions_for_wound.12.aspx

Introduction

Wound cleansing is one of the most important components of an effective wound management protocol. Optimal wound healing cannot occur until all pro-inflammatory material and foreign bodies have been removed from the wound. In its broadest sense, wound cleansing can encompass aggressive debridement of all devitalized tissue, extensive use of fluids for cleansing, and selective use of topical antimicrobial agents to control bacterial contamination and colonization. Each of these steps is essential for obtaining a clean, vital wound that has the greatest potential for healing at an optimal rate. However, in its strictest meaning, wound cleansing is the use of fluids to gently remove loosely adherent contaminants and devitalized material from the wound surface. If the materials cannot be removed gently with fluids, more specific mechanical techniques are required. These mechanical techniques are termed debridement. For further information on debridement, refer to Chapter 6.

The benefits of obtaining a clean wound must be weighed against the trauma to the wound that results from the cleansing. Wound cleansing is a mechanical process that traumatizes the wound. The practitioner must always attempt to minimize wound trauma during wound cleansing. By definition, the wound is already a traumatic insult to the body's integrity, and any additional trauma inflicted in attempts to manage the wound will only delay the reparative process. If contaminants cannot be removed with gentle wound cleans-

ing, more specific debridement technique should be employed.

Wound trauma incurred during wound cleansing can be chemical, mechanical, or both. Mechanical trauma occurs when the mechanical forces of scrubbing and high-pressure irrigation must be absorbed by the delicate wound tissue. Chemical trauma occurs when the fluids used to cleanse wounds contain chemicals that are toxic to the wound tissue. A philosophy to consider is: "Don't do to a wound what you wouldn't do to your own eye." Few caregivers treat someone else's wound as sensitively as they would treat their own eyes. Yet the sensitive care required for your eye is exactly the quality of care required for wounds to optimize their potential for rapid resolution.

Wound Cleansing

Cleansing solutions. Wound cleansing is the process of using fluids to gently remove inflammatory contaminants from the wound surface. In the majority of cases, water or saline is sufficient for cleansing the wound surface. Because of the limited contact time between the wound and the cleansing solution, it is not essential that the solution be isotonic (0.9% sodium chloride). No differences have been noted in the rates of infection and healing between the use of tap water and sterile normal saline in the cleansing of acute and chronic wounds.^{1,2} Tap water can be used for cleansing if it comes from a properly treated drinking supply. An acceptable saline solution can be made by adding 1 teaspoon of salt to 1 quart of boiling water (8 teaspoons per gallon). Any water with known or suspected contaminants should not be used for this purpose.

When enhanced cleansing efficacy is needed, a commercial wound cleanser can be used. Commercial wound cleansers contain surface-active agents to improve removal of wound contaminants. Surface-active agents (surfactants) by the nature of their chemical structure and chemical charge help break the bonds of the foreign bodies to the wound surface. The strength of their chemical reactivity is directly proportional to their cleansing capacity and toxicity to cells. Therefore, cleansing capacity needs to be balanced against toxicity to wound healing cells.

Surfactants can be categorized according to

their chemical charge in solution (cationic, anionic, or nonionic). Most surfactants with charges and many nonionic surfactants have been shown to be toxic to cells, delay wound healing, and inhibit the wound's defenses against infection.³⁻⁷ ***Since the US Food and Drug Administration (FDA) does not critically scrutinize the safety and efficacy of wound cleansers, it is the responsibility of the practitioner to select a wound cleansing solution that has been documented by independent testing to be safe for use in open wounds.***

Documentation of safety is difficult, since standardized tests for wound cleansers have not been established. However, tests that directly compare wound cleansers under controlled conditions can provide useful information on relative safety. A study has ranked the relative toxicity of several commercial wound cleansers based on their relative toxicity to white blood cells.⁸ Polymorphonuclear leukocytes (PMNs) were isolated from rabbit blood, exposed for 30 minutes to increasing 1:10 dilutions of the test solutions, then assayed for viability (Trypan blue dye exclusion) and functionality (phagocytic efficiency). The extent of dilution required to provide viability and functionality similar to PMNs exposed to Hanks' Balanced Salt Solution alone was used as the basis of an index of toxicity. If a wound cleansing solution required a 1:1,000 dilution to eliminate its toxicity, then its toxicity index was 1,000. The results showed that there was a wide range in the relative toxicity indices (10 to 10,000) for the cleansers tested. In general, the relative toxicity indices for wound cleansers were 10 to 1,000, while those for skin cleansers were 10,000. Cleansers that are formulated to remove fecal contamination from intact skin (skin cleansers) are stronger and thus more toxic than cleansers that are meant to be used in wounds. Skin cleansers should never be used in wounds.

In a subsequent study, additional wound cleansers with and without the presence of an antiseptic were tested.⁹ The toxicity indices for the wound cleansers ranged from 10 to 1,000 without the presence of an antiseptic (Table 1). However, when an antiseptic was added to the wound cleanser, the toxicity index, in general, increased to 10,000. The benefit of adding the antiseptic to the wound cleanser has not been documented.

The relative toxicity values listed in Table 1 are

Table 1. Relative toxicity indices of nonantimicrobial and antimicrobial wound cleansers

Product (nonantimicrobial)	Manufacturer	Toxicity Index
Dermagran®	Derma Sciences, Inc	10
Shur-Clens®	Conva Tec®	10
Biolex™	Bard Medical Division, CR Bard Inc	100
Cara-Klenz™ Wound & Skin Cleanser	Carrington Laboratories Inc	100
Saf-Clens® Chronic Wound Cleanser	ConvaTec®	100
Clinswound™	Sage Laboratories, Inc	1,000
Constant-Clens™ Dermal Wound Cleanser	Sherwood Medical-Davis & Geck	1,000
Curaklense™ Wound Cleanser	Kendall Healthcare Products Co	1,000
Curasol™	Healthpoint Medical	1,000
Gentell Wound Cleanser™	Gentell	1,000
Sea-Clens® Wound Cleanser	Coloplast Sween Corp	1,000
Ultra-Klenz™ Wound Cleanser	Carrington Laboratories, Inc	1,000
Product (antimicrobial)	Manufacturer	Toxicity Index
Clinical Care® Dermal Wound Cleanser	Care-Tech® Laboratories, Inc	1,000
Dermal Wound Cleanser	Smith & Nephew United, Inc	10,000
MicroKlenz™ Antimicrobial Wound Cleanser	Carrington Laboratories, Inc	10,000
Puri-Clens™ Wound Deodorizer & Cleanser	Coloplast Sween Corp	10,000
Restore™	Hollister Inc	10,000
Royl-Derm™	Acme United Corp	10,000
SeptiCare™ Antimicrobial Wound Cleanser	Sage Laboratories, Inc	10,000

Reprinted with permission from *WOUNDS* 1997;9(1):15–20. Copyright ©1997, HMP Communications.

based on the results of in-vitro testing, and their clinical relevance has not been determined. In addition, the values of relative toxicity should not be used as a guide for diluting the commercial wound cleanser. The commercial wound cleanser should be used at its recommended strength. The toxicity index is only a guide to help in the selection of a commercial wound-cleansing agent. A similar in-vitro test of cleanser toxicity was conducted using fibroblasts.¹⁰ Monolayers of cultured fibroblasts were exposed to dilutions of the various cleansers for 15 minutes, and cell viability was determined by cell uptake of fluorescein diacetate. The results were similar to those reported in Table 1.

A third study utilized human fibroblasts, red blood cells, and white blood cells as test cells for several wound cleansers.¹¹ This study involved Constant-Clens™ (Kendall Healthcare, Mansfield, Mass), Shur-Clens®, Saf-Clens™ (ConvaTec, Princeton, NJ), Cara-Klenz™, and Ultra-Klenz™ (Carrington Laboratories, Irving, Tex). The relative results were somewhat differ-

ent than those shown in Table 1. Constant-Clens was found to be the most biocompatible cleanser tested, and Shur-Clens was found to be the least biocompatible. Analysis of the Shur-Clens tested indicated that it did not meet the manufacturer's specification on pH, which accounted for its unexpected toxicity. The relative results for the other cleansers were similar to those reported in Table 1.

In a more recent study, the toxicity index of several skin and wound cleansers was evaluated using both fibroblasts and keratinocytes.¹² In this study, the cell monolayers were exposed to the test solutions for 30 minutes and their viability determined by an MTS assay. The definition of toxicity index was identical to that of previous reports. Their results in general agreed with those in Table 1 but contained the results for several agents not included in Table 1. The reader is encouraged to review the article.

Scrubbing devices. Use of a scrubbing device, such as a cloth, sponge, or brush, can enhance the efficacy of wound cleansing solutions.

Whenever these devices are used, the user must realize that mechanical trauma is being imparted to the wound. It is essential to minimize this trauma by using nonabrasive devices and as little force as necessary to achieve appropriate cleansing. If the desired cleansing is not achieved with moderate force, other means of wound cleansing should be considered. One should not try to cleanse the wound by increasing the force applied to the scrubbing device.

Saline has minimal ability to reduce the frictional forces encountered by the wound tissue from a scrubbing device. The surfactant properties of commercial wound cleansers significantly reduce the coefficient of friction between a scrubbing device and the wound tissue.¹³

The coarseness of the scrubbing device should be as low as possible while still providing cleansing action. Wounds scrubbed with coarse sponges were shown to be significantly more susceptible to infection than less traumatized wounds scrubbed with a smoother sponge.¹³

Wound Irrigation

Wound cleansing can also be accomplished by irrigating the wound with fluid. The hydraulic forces generated by the stream of fluid act on the debris on the wound surface and flush it from the wound. In order to remove the wound debris, the force of the irrigation stream has to be greater than the adhesion forces holding the debris to the wound surface. Therefore, it would be logical to assume that increasing the pressure of the irrigation stream would increase the cleansing efficiency of the irrigation process.

Several studies have documented that increasing the pressure of the irrigating stream enhances removal of bacteria and soil from wounds.¹⁴⁻¹⁶ Pressures up to 25 pounds per square inch (psi) were more effective than lower pressures, especially the low pressure produced by a bulb syringe. The efficacy of bacterial removal at 15 psi was significantly greater than that achieved at 10 psi. However, increasing the irrigation pressure to 20 psi or 25 psi did not significantly improve upon the result obtained with 15 psi.

Irrigation devices that delivered fluid streams at pressures of 70 psi were developed for dental hygiene in the early 1960s. It was not long before surgeons suspected that these devices might pro-

vide benefit in cleansing contaminated orificial wounds¹⁷ and traumatic wounds in general.¹⁸⁻²⁰ The use of the mechanical irrigation device at various pressures from 10 psi to 70 psi was shown to be significantly more effective in removing bacteria and debris from wounds when compared to irrigation with a bulb syringe.²⁰⁻²² The use of 70 psi was also found to be more effective in removing wound debris than 25 psi or 50 psi.²⁰ Using quantitative tissue biopsies, irrigation at 50 psi was shown to more effectively remove bacterial contamination than gravity irrigation from a height of 60 cm to 65 cm or irrigation by bulb syringe.^{23,24}

Some results suggest that the pressure of the irrigating stream is the important component, not whether it is pulsatile or continuous. Experimental studies comparing pulsatile or continuous stream irrigation have not documented the superiority of the pulsatile stream.^{14,21,22} In addition, these high-pressure, pulsatile, irrigation devices are expensive, cumbersome, and difficult to keep sterile. A more practical and convenient way to produce pressurized irrigation is to deliver the irrigant from a syringe through a needle or catheter. It has been shown that delivery of saline from a 35-mL syringe through a 19-gauge needle delivers a stream of irrigant to the wound surface at 8 psi.²⁵ Plastic tubing or angiocatheters that do not have a point but have the same bore size as a 19-gauge needle would deliver the same pressure of irrigation fluid but would be safer than the needle due to the potential hazard of accidental needle sticks. When compared to irrigation with a bulb syringe, irrigation with a 35-mL syringe and 19-gauge needle resulted in significantly enhanced removal of bacteria and a significantly reduced incidence of wound infection. This experimental benefit has been confirmed in a human study.²⁶ Three hundred and thirty-five patients who presented to the emergency department with traumatic wounds of less than 24 hours duration were randomly assigned to wound cleansing by standard bulb syringe (controls, low pressure) or a 12-cc syringe and 22-gauge needle (experimental, 13 psi). Two hundred and seventy-seven patients (83%) returned for wound evaluation; of these, 117 were in the control group and 151 were in the experimental group. In the control group, 27.8% of the wounds were inflamed

and 6.9% were infected. In the experimental group, 16.8% of the wounds were inflamed and 1.3% were infected. ***There was a statistically significant decrease in both wound inflammation and wound infection for wounds cleansed with syringe and needle irrigation (13 psi) compared to wounds cleansed with bulb syringe (0.05 psi).***

Other combinations of syringes and catheter sizes can also be utilized. In general, as the size of the syringe increases, the pressure decreases, because the force applied to the plunger is distributed over a larger cross-sectional area. For example, with a 19-gauge needle, the pressures generated by 6-, 12-, and 35-mL syringes are 30 psi, 20 psi, and 8 psi, respectively. In contrast, increasing the size of the needle increases the pressure because there is greater flow. For example, with a 35-mL syringe, the pressures generated with a 25-, 21-, and 19-gauge needle are 4 psi, 6 psi, and 8 psi, respectively.

Delivery of saline at increased pressure has been made more convenient with the availability of devices that insert directly into an IV bag. These systems usually involve a syringe and tubing with a valve that allows for ejection of the fluid under pressure and then the refilling of the syringe by pulling back the plunger. This is a quick and easy way to achieve bedside wound irrigation.

Other convenient devices are tapered tips on squeezable bottles that when squeezed yield pressurized irrigation. The tips can be purchased separately and placed on the bottles of sterile saline. Some products are sold with the irrigation tips already in place. The user controls the exact pressure generated by these devices, and therefore, the pressures have not been reported.

Recently, irrigation of wounds has been made more convenient by the introduction of battery-powered, disposable, pulsatile, irrigation systems (Stryker® Instruments, Kalamazoo, Mich; Davol Inc, Cranston, RI; Zimmer, Inc, Dover, Ohio) to remove larger amounts of wound debris. These self-contained, sterile systems insert into an IV bag and pump the sterile saline through a choice of tips at elevated irrigation pressures. The fluid is delivered in a pulsatile stream through the tips. The tips are single orifice or multiple orifices and deliver different spray patterns from streams to showers. The spray tips also contain a suction cone and a vacuum line so that the irrigation

fluid can be contained and aspirated into a vacuum canister. The impact pressure generated by these new devices is currently being determined. Preliminary results indicate that the manufacturers are aware of the concern about exceeding 15 psi and have engineered their devices to deliver irrigation fluid below this level. Since these devices are relatively new, clinical reports of their performance are limited.²⁷⁻³⁰ However, in a recent in-vivo animal study, the benefits of pulsed lavage irrigation compared to bulb syringe in reducing wound bacterial levels were quantitated.³¹

These devices may be considered as an alternative to whirlpool therapy because they provide suction to remove the irrigation fluid and all of the loosened wound debris. For the patient with a chronic wound who is unresponsive, has cardiopulmonary compromise, or has venous insufficiency and is not a candidate for whirlpool therapy, pulsatile lavage with suction may be an effective alternative. In a comparison of pulsatile irrigation and whirlpool therapy, Haynes et al³² found pulsatile irrigation to be more effective. Wounds treated with pulsatile irrigation developed new healthy granulation tissue at a rate of 12.2% per week compared to 4.8% per week for similar wounds treated with whirlpool. In addition to being an alternative to whirlpool therapy, these pulsatile irrigation systems also minimize cross contamination because they are disposable, decrease treatment time because of their ease and convenience, and increase wound healing and decrease hospital stays because of efficacy of cleansing. These benefits suggest that disposable pulsatile irrigation systems may be cost effective. Additionally, these devices are versatile, since the pressure, tip configuration, and frequency of irrigation can be personalized to provide the best outcome for each patient and wound. Environmental contamination is possible with these devices, and infection control precautions should be used routinely.³³

Pressurized irrigation can also be accomplished by applying pressure to the IV bag of a standard irrigation set up.³⁴⁻³⁶ In this situation, a standard blood pressure cuff is wrapped around the IV bag and inflated to the desired pressure. Increasing or decreasing the pressure on the cuff will adjust the pressure of the exiting irrigation stream. However, applying 15 mmHg pressure to the blood

pressure cuff does not mean that the fluid exiting the tip is at 15 psi. Quantitative testing with standardized equipment (type of cuff, size of IV bag, design of irrigation tip, etc) would be required to establish the direct correlation between cuff pressure and irrigation pressure.

A pressurized canister is another concept for conveniently delivering pressurized saline to the wound. It is claimed that the saline is delivered in a 19-gauge stream at 8 psi. Data to support this claim are not available. A clinical study comparing this product to the bulb syringe procedure suggested that the pressurized canister was effective in cleansing debris and bacteria from the wound in less time and with less expense than the bulb syringe procedure.³⁷ The study demonstrated that the pressurized canister needed to be 6 inches from the wound, and the stream must contact the wound at a 45-degree angle to minimize splashing. Similar results were reported in another study comparing the pressurized canister to a 30-mL syringe with a 20-gauge IV catheter.³⁸

A common problem with all of these innovative techniques for delivering pressurized irrigation fluid is that the manufacturers do not know what impact pressure their system delivers to the wound surface. In the report by Singer et al,³⁴ the pressure the authors measured was within the system, not the exiting fluid impact pressure. The studies by Weller³⁷ and Chisholm et al³⁸ claim to deliver a pressure of 8 psi, but this value is unsubstantiated.

When using pressurized irrigation, the practitioner should always be concerned about splashing the irrigation fluid out of the wound. It is essential to wear protective clothing, gloves, and eye-wear during the irrigation procedure because of the significant risk of viral contamination from contact with such fluid. In addition, the use of splash shields on the irrigation device or over the wound is also recommended. These splash shields have been shown to significantly reduce environmental splatter.³⁹ For large cavity wounds, it may be practical to seal the wound with a transparent film dressing and irrigate through the film to prevent splashing.⁴⁰

Another complication of high-pressure irrigation is dispersion of fluid into the adjacent tissue or along tissue planes.^{17,41,42} The extent of this dispersion is related to the magnitude of the

pressure. Fluid dispersion into wound tissue was significantly greater for a 70-psi irrigation stream than for an 8-psi irrigation stream.⁴¹ When a single orifice tip was used to irrigate wounds in dogs, the irrigation fluid extensively penetrated the tissue, especially when the pressure was increased above 30 psi.⁴² When a multijet tip (shower head) was utilized, irrigating fluid was not forced into the surrounding tissue. The influence of pressure on tissue penetration was further clarified by a study that compared irrigation at 15 psi to irrigation at 20 psi.¹⁶ Following irrigation of partial-thickness wounds on the backs of rats with saline containing 1% aniline blue dye, full-thickness wound biopsies were excised, and the depth of penetration into the skin was quantitated. When the wound was exposed to 20 psi, the irrigation stream penetrated the entire thickness (100%) of skin. In contrast, irrigation with saline at 15 psi only resulted in superficial (10%–15%) penetration of the wound tissue. These results strongly suggest that soft tissue wounds should not be irrigated with fluids delivered at greater than 15 psi.

The efficacy of high-pressure irrigation in removing bacteria decreases with the age of the wound. For acute wounds treated in the emergency room, most bacteria are surface contaminants and are more easily removed than bacteria within the tissue. As the wound ages without appropriate control of wound bacteria, the bacteria invade the tissue and cannot be removed without antibiotics or surgical debridement. Daily irrigation of infected experimental wounds with high-pressure irrigation was ineffective in significantly reducing the mean level of bacteria within the wound tissue.⁴³ These results would suggest that irrigation alone will not reduce the level of bacteria within the tissue of chronic wounds. In this situation, surgical debridement or, alternately, topical antibiotics should be considered.

Despite the ability of Saxe et al⁴³ to obtain significant reduction in bacterial levels in their animal experiment, the benefit of irrigation has been reported in 1 study involving chronic wounds. Diekmann⁴⁴ used a dental irrigating device on its lowest setting (6 psi) to irrigate pressure ulcers on 8 patients twice a day for 2 weeks. Eight other patients with similar type pressure ulcers received standard care. Wounds treated with pressurized irrigation had a mean decrease in wound area of

51%, while the mean decrease in wound area of the control wounds was only 13%. The large standard deviation and small number of wounds in each sample made the difference not statistically significant. These results are encouraging and support the contention that clean wounds heal faster than unclean wounds.

Another form of wound irrigation is the whirlpool bath that contains a pump that generates pressurized streams of water in the bath through jets. The use of whirlpool is recommended for chronic wounds that contain thick exudate, slough, or necrotic tissue. Wound cleansing is enhanced in the whirlpool because of the extended time of contact between the wound and the fluid. This extended soaking time saturates and softens the wound debris and facilitates its removal. The aggressiveness of the irrigation process can be controlled by how close the wound is placed to a jet. The impact pressure generated by the jets has not been determined. The practitioner and the patient should know when maximal acceptable pressure has been achieved.

Using bacteria as a marker for cleansing action, 2 studies have demonstrated that whirlpool is an effective cleansing technique.^{45,46} These studies indicated that a 20- to 30-minute immersion with agitation followed by 30 seconds of rinsing at maximum force tolerated was the most effective cleansing technique. Feedar and Kloth⁴⁷ recommend whirlpool twice daily in conjunction with interim wound dressings to facilitate debridement of necrotic tissue. However, once the wound has been cleansed of foreign debris, the trauma to the newly exposed healing tissue outweighs the benefits of the whirlpool. Therefore, clean, granulating wounds should not be exposed to whirlpool therapy.

The Cochrane collaboration conducted a review of wound cleansing studies on wound cleansing for pressure ulcers and found no large scale randomized, controlled trial.⁴⁸ Only 1 study demonstrated a statistically significant difference in outcomes for wounds cleansed with saline spray containing aloe vera, silver chloride, and decyl glucoside (Vulnopus) compared to isotonic saline. No statistically significant improved healing rate was seen when water was compared to saline. One study compared different cleansing techniques, but no statistically significant change

in healing was seen with or without whirlpool.

Wound Disinfection

All chronic wounds are colonized with bacteria. Unless the patient is severely immunocompromised, these bacteria can be controlled with the host's resistance and best clinical practice based on standard, physiologically sound management procedures. The single most important parameter in reducing the level of bacterial colonization in the chronic wound is removal of all devitalized material. Bacteria thrive in devitalized tissue and exudate. Aggressive debridement and thorough cleansing are the physiologically sound procedures for disinfecting wounds. Bacteria do not normally survive in clean, healthy tissue.

A chronic wound that has been converted to a clean wound by physiologically sound procedures should show signs of healing within 2–4 weeks.^{49–51} If healing is not apparent, the treatment plan needs to be critically reviewed to ensure that pressure relief, adequate nutrition, acceptable blood supply, and proper wound dressings are being provided. If all of these factors have been evaluated and the wound is not healing, high levels of bacterial colonization or organisms could be establishing critical colonization in the wound tissue, and this bacterial damage may be inhibiting the healing process.

Although the influence of bacteria on healing is controversial, it seems obvious that viable bacteria in a wound would be competing with the wound healing cells for nutrients and oxygen. Bacteria would also be elaborating metabolic wastes, reactive enzymes, and toxins. All of these agents would be inflammatory, and prolonged inflammation retards wound healing.

Several well controlled clinical studies have documented that patients with pressure ulcers and venous ulcers with high bacterial levels ($> 1.0 \times 10^5$) do not heal.^{52–57} Other studies involving leg ulcers have reported that healing occurred despite high levels of bacteria.^{58–61} However, these studies did not report the rate of wound healing. Wound healing can occur in the presence of high levels of bacteria, but it is probable that healing would have occurred much faster if the level of bacterial critical contamination had been reduced.

In addition to the level of bacteria involved in

Table 2. Partial list of antiseptic agents that have been used in the false hope of killing bacteria without killing wound cells

Acetic acid
Alcohols
Aluminum salts
Boric acid
Chlorhexidine
Formaldehyde
Gentian violet
Hexachlorophene
Hydrogen peroxide
Hypochlorite
Iodine, povidone-iodine
Merthiolate
Permanganate
Silver nitrate

Reprinted with permission from *WOUNDS* 1997;9(1):15–20. Copyright ©1997, HMP Communications.

critical colonization and contamination, the species of bacteria present may also be important. Several of the bacterial studies identified specific species of organisms present in the nonhealing wounds and found a strong correlation with the gram-negative organisms, such as *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Escherichia coli*, and *Bacteroides* species.^{52–54} These studies indicated that *Proteus* species may be more deleterious to wound healing than the other gram-negative organisms. Clinical experience has documented that beta-hemolytic streptococci are always a significant concern in the wound regardless of their number. In addition, anaerobic organisms become an important part of the microbial population as the wound deteriorates. Synergy is known to occur between aerobic and anaerobic bacteria in chronic wounds, which increases their pathological effect.⁶²

When high levels of bacteria in the wound are suspected as the cause of nonhealing, a quantitative culture needs to be obtained. Quantitative cultures are different than standard swab cultures, since extreme effort is made to thoroughly cleanse the wound surface of contaminants and then obtain a specified volume, weight, or surface area of wound material for analysis. Quantitative cultures can be tissue biopsies, needle aspirates, or standardized quantitative swabs.⁶³ Tissue bi-

opsy—removal of a piece of tissue with a scalpel or punch biopsy and quantification of the number of organisms per gram of tissue—has been the gold standard with which other methods of monitoring tissue bacteria have been compared.⁶⁴ Needle aspiration utilizes a 22-gauge needle and 10-cc syringe inserted into the tissue to aspirate fluid that subsequently can be quantified in colony forming units per volume of fluid.⁶⁵ Quantitative swab culture was first described by Levine et al⁶⁶ and consists of cleansing the wound with saline followed by rotating the end of a cotton tip applicator over a 1 cm² surface area of the wound with sufficient pressure to express fluid from underlying tissue. Serial dilutions are made and spread on agar plates, and results are expressed as organisms per swab or by categorizing from scant to heavy bacterial growth.

Although tissue biopsy is the gold standard, there is excellent correlation between the results of tissue biopsies and quantitative swabs, even when semiquantitative results have been utilized.^{66,67} Any microbiology laboratory can perform a semiquantitative analysis of a swab obtained under controlled conditions. The important component is obtaining the sample according to a specified protocol.⁶⁸ It is important to remember that rather than trying to determine the exact number of organisms, you are determining if there is a high level ($> 1.0 \times 10^5$) of bacteria in the wound that may be responsible for impaired healing. In chronic wound specimens, Bill et al⁶⁹ have demonstrated a 79% correlation between quantitative swabs and tissue biopsies in identifying the wounds with greater than 10^5 organisms. In a subsequent study, the same group showed a 79% correlation between quantitative and semiquantitative swabs of chronic wounds.⁷⁰

To reduce a harmful level of increased bacterial burden, a short course of a topical antimicrobial agent should be considered if the wound is clean, further debridement and cleansing are not required, and nutritional status is adequate to support healing. The agents of choice are topical antiseptics or topical antibiotics. Do not use antiseptics in wounds to reduce bacteria in clean wound tissue. Unlike antibiotics that can selectively kill bacteria without harming tissue, antiseptics do not have a selective antibacterial mechanism and thus damage all cells upon contact. *Therefore, the*

repeated use of uncontrolled antiseptics in chronic wounds may cause such damage to the cells essential for wound repair that optimal wound healing is delayed.

The scientific literature is replete with documentation of the ability of antiseptics to rapidly kill high levels of bacteria. These favorable results are obtained by exposing bacteria suspended in fluid directly to the antiseptic solution. Thus, there is direct contact between the bacteria and the antiseptic, and the results are optimized. However, a test tube of fluid does not represent a chronic wound. When wound exudate, necrotic tissue, or blood is added to the test tube, the effectiveness of the antiseptics is significantly reduced, if not completely eliminated.

Antiseptics are used primarily as prophylactic agents for killing bacteria on the surface of tissue because of their inability to effectively penetrate tissue. In order to be effective as a therapeutic agent, an antiseptic has to penetrate into critically colonized tissue in an active form with sufficient concentration to provide antimicrobial activity. Antiseptics actively bind to many organic substrates present in the wound because of their chemical reactivity.⁷¹⁻⁷³ Thus, antiseptics, when used at clinically appropriate concentrations, might never reach the bacteria in the wound tissue with effective antimicrobial activity. In 1919, after extensive experimentation, Fleming⁷² summarized his results as follows: "This would seem to indicate clearly that it is impossible to sterilize a wound with an antiseptic, even if it were possible to keep the antiseptic solution in the wound for a long time without dilution..."

No controlled clinical study has been able to refute Fleming's conclusion that topical antiseptics offer little benefit in reducing the number of bacteria that reside within wound tissue. The benefit that most authors report when evaluating antiseptics as part of a wound management study is most likely due to another aspect of the wound management protocol, such as debridement. Most wound bacteria reside in necrotic tissue. When more aggressive debridement is instituted as part of a clinical study, the bacterial burden in the wound is reduced, and the wound improves. In uncontrolled studies, the improvement in the wound has been inappropriately ascribed to the utilization of antiseptics.

Numerous reports in the literature describe the benefits of different antiseptics (Table 2). However, when all of these reports were reviewed for scientific validity, none of them truly validated the ability of an antiseptic agent alone to decontaminate a pressure ulcer.⁷⁴

A more recent literature review from 2004 confirms that clinical results involving antiseptics are confusing.⁷⁵ Some recent reviews suggest that the history associated with uncontrolled antiseptic solutions should not prevent clinicians from considering antiseptics that have been reformulated in more effective and safer delivery systems.^{76,77} By controlling the release of the antiseptic, they may reduce toxicity but they also may reduce killing capacity. This is a complex issue, and each report should be read critically to determine safety versus efficacy.

Even the clinical standard, povidone-iodine, does not have well controlled studies to validate its efficacy. Several published studies indicate that the use of povidone-iodine decreases bacterial levels and promotes healing.⁷⁸⁻⁸² None of these studies were controlled by treating a similar group of patients the same way but with saline rather than povidone-iodine. When such a study was conducted, povidone-iodine was shown to be ineffective.⁸³ The use of povidone-iodine continues to be controversial. A recent analysis of the literature concluded that the "outcome supports the continued use of PVP-I with caution."⁸⁴

Another formulation of bound iodine is cadexomer iodine.⁸⁵ In this situation, the iodine is trapped in a 3-dimensional spherical microbead starch lattice. The iodine is at a concentration of 0.9% (w/w). Cadexomer iodine (CI) is available as a powder, ointment, gel, or paste. One key property of CI is its high absorption capacity: 1 g CI can absorb up to 7 mL of water or body fluid. As CI absorbs fluid, it slowly releases iodine. It would be difficult to differentiate the clinical benefits of absorption versus the benefits of iodine because of CI's ability to absorb wound exudate, bacteria, toxins, etc.

Most of the clinical efficacy studies involving CI have compared the product to standard care.⁸⁵ In most of those studies, CI was shown to be beneficial in controlling exudate, cleansing the wound, reducing bacterial levels, and improving healing. The benefits of CI's absorption proper-

ties versus the presence of iodine were not differentiated. When a clinical study was conducted comparing CI directly to another absorbing bead product without iodine, there was no significant difference in healing or control of bacteria.⁸⁶

Dakin's solution (0.5% sodium hypochlorite) is another antiseptic solution that is commonly used to treat chronic wounds. Alexis Carrel established the popularity of Dakin's solution in his miraculous treatment of open war wounds in World War I.⁸⁷ However, under those conditions, any agent would have proven beneficial. Despite its long history of clinical use, no controlled studies have documented its antimicrobial efficacy compared to standard practice. The clinical benefit of Dakin's solution is probably due to its ability to dissolve necrotic tissue.⁸⁸ Removal of necrotic tissue would be correlated with a reduction in the level of bacteria in the wound and an improvement in healing. In this situation, Dakin's solution is acting as a chemical debriding agent and as such should be discontinued when the necrotic tissue has been removed. Dakin's solution should never be used to pack a clean wound.

Acetic acid is another agent that has a long history of clinical use. The activity of acetic acid is probably due only to its physiologically unacceptable low pH.⁸⁹ Because *Pseudomonas* species are extremely sensitive to acidic environments, topical acetic acid (5%) has been shown to be of benefit in 2 uncontrolled trials where *Pseudomonas* infections were present.^{90,91}

Hydrogen peroxide is another agent that has an undocumented reputation as an effective antiseptic agent. Hydrogen peroxide has little antimicrobial activity, but it is very effective in dissolving blood clots. Therefore, under the right condition where blood clots or hematomas are present, hydrogen peroxide acts as an effective chemical debriding agent, not as an antiseptic. The American Medical Association reviewed the literature on hydrogen peroxide and concluded that it had little bactericidal effect in tissue but that its effervescence might provide some mechanical benefit in loosening debris and necrotic tissue in the wound.⁹²

Silver in the form of 0.5% silver nitrate solution was used extensively in the care of burn wounds⁹³ in the late 1960s and early 1970s. For maximum effectiveness, the dressings had to be saturated ev-

ery 2 hours, a process that is time consuming, and silver stained everything black. These issues were resolved with the introduction of silver sulfadiazine cream.⁹⁴ This cream still needs to be manually removed and reapplied twice daily.

Adding silver to dressings has revolutionized the wound care market. Regardless of how the silver is added, by nanocrystalline silver metal or complexes of silver salts, the active agent is the silver cation. Almost every type of dressing previously available is now available with silver. The challenge to the clinician is to determine if all of the *in vitro* results characterizing the various dressings have any relevance to clinical benefit. After evaluating numerous parameters of dressing performance including silver content, rate of silver release, and antibacterial activity, Parsons et al⁹⁵ concluded that the dressing should be selected on standard clinical parameters rather than on a dressing's silver content or release kinetics. However, in order to obtain effective antimicrobial kill, Warriner and Burrell⁹⁶ reported that a minimum concentration of 30–40 mg/L of Ag⁺ had to be generated in the local wound fluid.

As with other antiseptics delivered by controlled-release vehicles, the true benefit of the presence of silver has to be evaluated in human wounds. Recent reviews have indicated that clinical evidence of benefit for silver-containing dressings is lacking.^{97–99} (See Chapter 8) Even when benefit is claimed, the clinician has to read the report carefully to document that the claimed benefit did not actually come from the dressing itself. ***Do not forget that these new dressings with silver were FDA approved as devices not drugs. As devices, dressings cannot make a therapeutic claim for a released active ingredient; otherwise, they would be drug-delivery vehicles.***

Conclusion

Although no scientifically valid documentation exists, practitioners continue to use antiseptics in wounds because of tradition. This tradition must stop. Antiseptics are toxic chemicals that, when used in clean wounds, do more harm than good. The volume of literature that documents the extreme toxicity of these agents is overwhelming. It includes *in-vitro* tests^{100–102} as well as *in vivo* tests in animals^{100,103–106} and humans.^{107,108}

Accepting the fact that traditional concen-

trations of antiseptic solutions are too toxic for wound care, some practitioners have assumed that diluting the antiseptic will dilute its toxicity to wound healing cells while maintaining its toxicity to bacteria. Certain reports in the literature support this contention by finding a “magic” dilution of antiseptic that kills bacteria but not wound healing cells.^{100,109} These reports are misleading, because the antiseptic agents were tested in test tubes with saline that contained no wound materials, such as exudate or tissue.¹¹⁰ Although the basis for diluting antiseptics appears dubious, the process is fully encouraged. If everyone that continues to use antiseptics would dilute them 1:1,000 or 1:10,000, they would see a significant improvement in wound healing because they significantly reduced the toxicity of the topical antiseptic agent they were using.

Another way of controlling the toxicity of antiseptic solutions is to control the dose of antiseptic exposed to the wound by using controlled delivery vehicles or dressings instead of solutions.⁷⁷ The clinician has to be aware that by reducing the level of antiseptic present in the wound, the antiseptic will be less effective in killing the bacteria. Because antiseptics do not have a specific mechanism for killing bacteria, it stands to reason that an antiseptic level sufficient to kill bacteria should also kill important wound healing cells.

When an antimicrobial agent is deemed necessary to reduce bacterial levels within the wound, a topical antibiotic should be utilized. The use of topical antibiotic therapy has been the mainstay of burn care for the past 2 decades. Some topical antibiotics that have been used for chronic wounds are listed in Table 3. None of these agents are commonly used systemically in clinical practice. Clinically utilized systemic antibiotics should not be used topically on chronic wounds because of the risk for selecting out resistant strains of bacteria. Although Bendy et al⁵² documented the success of topical gentamicin in reducing bacterial levels and promoting healing in pressure ulcers, there are other topical antibiotics that will do a similar job without developing strains of bacteria that are resistant to clinically essential systemic antibiotics.

The use of topical antibiotics in chronic wounds has not been reported often in the literature. Despite the limited number of studies

Table 3. Topical antibiotics that have been utilized to control bacteria in chronic wounds

Mafenide acetate
Metronidazole
Mupirocin
Nitrofurazone
Polysporin
Silver sulfadiazine

that have been reported, the results have been impressive. The use of silver sulfadiazine cream in heavily contaminated pressure ulcers resulted in reduction of bacterial levels to less than 10^5 organisms per gram of tissue in all treated ulcers within 3 weeks.⁸³ In another study, 10 patients with putrid-smelling ulcers and positive cultures for anaerobic organisms were treated twice daily with metronidazole gel.¹¹¹ After 5 days of treatment, all odor was eliminated, and repeat cultures were negative for anaerobic organisms. A review of antimicrobial treatments for diabetic foot ulcers concluded that the “evidence is too weak to recommend any particular antimicrobial agent.”¹¹²

Topical antibiotics can be effective when used against sensitive organisms. When in doubt about the sensitivity of the organisms in the wound to the antibiotic being used, consult your micro-

Take-Home Messages for Practice

- Effective wound cleansing is essential for effective wound healing.
- The benefits of wound cleansing must always be balanced against the harm inflicted upon the wound.
- Select biocompatible wound cleansers and utilize them in a nontraumatic manner.
- When irrigating wounds, keep the irrigation pressure below 15 psi.
- Do not use antiseptic agents in clean wounds.
- For nonhealing, clean wounds that contain high levels of bacteria, consider a 2-week trial of topical antibiotic.
- Newer delivery systems that provide controlled release of antiseptics may provide benefit in wounds, but this has to be proven in well controlled trials.

biologist. Well defined tests for determining the sensitivity of wound organisms to topical antibiotic preparations exist.^{113,114} In general, topical antibiotics should not be used for more than 2 weeks, and patients must be monitored for any signs of reaction to the antibiotic.

Controlling wound colonization or critical colonization with topical antimicrobials is a complex issue.⁶² The primary concern with the use of topical antibiotics is the selection or development of more resistant bacteria. This concern is so predominant that the authors of the European Wound Management Association (EWMA) Position Document on Management of Wound Infection chose not to discuss the use of topical antibiotics.¹¹⁵ This concern can be minimized with short exposure to high doses of effective antibiotics approved for topical use. Even the use of antiseptics is not devoid of resistance selection or development.¹¹⁶ This is especially concerning now that controlled-release dressings are reducing the dose of antiseptic present in the wound. Low levels of antimicrobial agents are the primary causation of selection or development of bacterial resistance.

Self-Assessment Questions

- For a pressure ulcer containing adherent necrotic tissue, slough, and viscous exudate, what is the best wound care protocol?
 - Scrub wound vigorously to remove debris
 - Pack with gauze soaked in Dakin's solution and change every 8 hours
 - Debridement in conjunction with frequent wound irrigation
 - Cover with occlusive dressing for several days
- For a clean pressure ulcer containing moderate exudate, which is the least desirable treatment?
 - Irrigation with isotonic saline
 - Irrigation with a skin cleanser
 - Irrigation with a nontoxic wound cleanser
 - Irrigation with a wound cleanser containing an antiseptic
- Which of the following devices is the most appropriate for irrigating a wound with viscous exudate?
 - Saline from the bottle (0 psi)
 - Bulb syringe (2 psi)
 - 30-cc syringe with a 19-gauge catheter (8 psi)
 - Water Pik at high setting (> 15 psi)
- For a clean pressure ulcer with high bacterial levels, which is the best topical agent to use for a short time?
 - Iodophor solution
 - Dakin's solution
 - Hydrogen peroxide solution
 - Silver sulfadiazine cream

Answers: 1-C, 2-B, 3-C, 4-D

References

- Beam JW. Wound cleansing: water or saline? *J Athl Train.* 2006;41(2):196-197.
- Valente JH, Forti RJ, Freundlich LF, Zandieh SO, Crain EF. Wound irrigation in children: saline solution or tap water? *Ann Emerg Med.* 2003;41(5):609-616.
- Rydberg B, Zederfeldt B. Influence of cationic detergents on tensile strength of healing skin wounds in the rat. *Acta Chir Scand.* 1968;134(5):317-320.
- Betley FR. The toxicity of soaps and detergents. *Br J Dermatol.* 1968;80(10):635-642.
- Custer J, Edlich RF, Prusak M, Madden J, Panek P, Wangenstein OH. Studies in the management of the contaminated wound. V. An assessment of the effectiveness of pHisoHex and Betadine surgical scrub solutions. *Am J Surg.* 1971;121(5):572-575.
- Edlich RF, Schmolka IR, Prusak MS, Edgerton MT. The molecular basis for toxicity of surfactants in surgical wounds. 1. EO:PO block polymers. *J Surg Res.* 1973;14(4):277-284.
- Bryant CA, Rodeheaver GT, Reem EM, Nichter LS, Kenney JG, Edlich RF. Search for a nontoxic surgical scrub solution for periorbital lacerations. *Ann Emerg Med.* 1984;13(5):317-321.
- Foresman PA, Payne DS, Becker D, Lewis D, Rodeheaver GT. A relative toxicity index for wound cleansers. *WOUNDS.* 1993;5(5):226-231.
- Hellewell TB, Major DA, Foresman PA, Rodeheaver GT. A cytotoxicity evaluation of antimicrobial and non-antimicrobial wound cleansers. *WOUNDS.* 1997;9(1):15-20.
- Burkey JL, Weinberg C, Brenden RA. Differential methodologies for the evaluation of skin and wound cleansers. *WOUNDS.* 1993;5(6):284-291.
- Wright RW Jr, Orr R. Fibroblast cytotoxicity and blood cell integrity following exposure to dermal wound cleansers. *Ostomy Wound Manage.* 1993;39(7):33-40.
- Wilson JR, Mills JG, Prather ID, Dimitrijevic SD. A toxicity index of skin and wound cleansers used on in vitro fibroblasts and keratinocytes. *Adv Skin Wound Care.* 2005;18(7):373-378.
- Rodeheaver GT, Smith SL, Thacker JG, Edgerton MT, Edlich RF. Mechanical cleansing of contaminated

- wounds with a surfactant. *Am J Surg.* 1975;129(3):241–245.
14. Madden J, Edlich RF, Schauerhamer R, Prusak M, Born-er J, Wangenstein OH. Application of principles of fluid dynamics to surgical wound irrigation. *Curr Topics Surg Res.* 1971;3:85–93.
 15. Rodeheaver GT, Pettry D, Thacker JG, Edgerton MT, Edlich RF. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstet.* 1975;141(3):357–362.
 16. Foresman PA, Etheridge CA, Thacker JG, Rodeheaver GT. Influence of a Pulsatile Irrigation System on Bacterial Removal from and Tissue Injury to Contaminated Wounds (unpublished research report). Charlottesville, Va: University of Virginia Health Sciences Center; 1989.
 17. Bhaskar SN, Cutright DE, Gross A. Effect of water lavage on infected wounds in the rat. *J Periodontol.* 1969;40(11):671–672.
 18. Gross A, Bhaskar SN, Cutright DE, Beasley JD 3rd, Perez B. The effect of pulsating water jet lavage on experimental contaminated wounds. *J Oral Surg.* 1971;29(3):187–190.
 19. Gross A, Cutright DE, Bhaskar SN. Effectiveness of pulsating water jet lavage in treatment of contaminated crushed wounds. *Am J Surg.* 1972;124(3):373–377.
 20. Grower MF, Bhaskar SN, Horan MJ, Cutright DE. Effect of water lavage on removal of tissue fragments from crush wounds. *Oral Surg Oral Med Oral Pathol.* 1972;33(6):1031–1036.
 21. Green VA, Carlson HC, Briggs RL, Stewart JL. A comparison of the efficacy of pulsed mechanical lavage with that of rubber-bulb syringe irrigation in removal of debris from avulsive wounds. *Oral Surg Oral Med Oral Pathol.* 1971;32(1):158–164.
 22. Stewart JL, Carlson HC, Briggs RL, Green VA. The bacteria-removal efficiency of mechanical lavage and rubber-bulb syringe irrigation in contaminated avulsive wounds. *Oral Surg Oral Med Oral Pathol.* 1971;31(6):842–848.
 23. Hamer ML, Robson MD, Krizek TJ, Southwick WO. Quantitative bacterial analysis of comparative wound irrigations. *Ann Surg.* 1975;181(6):819–822.
 24. Brown LL, Shelton HT, Bornside GH, Cohn I Jr. Evaluation of wound irrigation by pulsatile jet and conventional methods. *Ann Surg.* 1978;187(2):170–173.
 25. Stevenson TR, Thacker JG, Rodeheaver GT, Bacchetta C, Edgerton MT, Edlich RF. Cleansing the traumatic wound by high pressure syringe irrigation. *JACEP.* 1976;5(1):17–21.
 26. Longmire AW, Broom LA, Burch J. Wound infection following high-pressure syringe and needle irrigation. *Am J Emerg Med.* 1987;5(2):179–181.
 27. Loehne H. Pulsatile lavage with concurrent suction. In: Sussman C, Bates-Jensen BM, eds. *Wound Care: A Collaborative Practice Manual for Physical Therapists and Nurses.* Gaithersburg, Md: Aspen Publishers; 1998:389–403.
 28. Cicione M. Making waves. Case Review. 1998;July/August:26–29.
 29. Ho C, Burke DT, Kim HJ. Healing with hydrotherapy. *Adv Directors Rehabil.* 1998;7(5):45–49.
 30. Morgan D, Hoelscher J. Pulsed lavage: promoting comfort and healing in home care. *Ostomy Wound Manage.* 2000;46(4):44–49.
 31. Svoboda SJ, Bice TG, Gooden HA, Brooks DE, Thomas DB, Wenke JC. Comparison of bulb syringe and pulsed lavage irrigation with use of a bioluminescent musculoskeletal wound model. *J Bone Joint Surg Am.* 2006;88(10):2167–2174.
 32. Haynes LJ, Brown MH, Handley BC, et al. Comparison of Pulsavac and sterile whirlpool regarding the promotion of tissue granulation. *Phys Ther.* 1994;74(5 Suppl):S4.
 33. Maragakis LL, Cosgrove SE, Song X, et al. An outbreak of multidrug-resistant *Acinetobacter baumannii* associated with pulsatile lavage wound treatment. *JAMA.* 2004;292(24):3006–3011.
 34. Singer AJ, Hollander JE, Subramanian S, Malhotra AK, Villez PA. Pressure dynamics of various irrigation techniques commonly used in the emergency department. *Ann Emerg Med.* 1994;24(1):36–40.
 35. Vadodaria SJ, Parekh DB. An irrigation system for large-wound toileting. *Ann Plast Surg.* 1990;25(2):152–153.
 36. Leslie LF, Faulkner BC, Woods JA, et al. Wound cleansing by irrigation for implant surgery. *J Long Term Eff Med Implants.* 1995;5(2):111–128.
 37. Weller K. In search of efficacy and efficiency. An alternative to conventional wound cleansing modalities. *Ostomy Wound Manage.* 1991;37:23–28.
 38. Chisholm CD, Cordell WH, Rogers K, Woods JR. Comparison of a new pressurized saline canister versus syringe irrigation for laceration cleansing in the emergency department. *Ann Emerg Med.* 1992;2(11):1364–1367.
 39. Pigman EC, Karch DB, Scott JL. Splatter during jet irrigation cleansing of a wound model: a comparison of three inexpensive devices. *Ann Emerg Med.* 1993;22(10):1563–1567.
 40. Chernofsky MA, Murphy RX Jr, Jennings JF. A barrier technique for pulsed irrigation of cavitary wounds. *Plast Reconstr Surg.* 1993;91(2):365–366.
 41. Wheeler CB, Rodeheaver GT, Thacker JG, Edgerton MT, Edlich RF. Side-effects of high pressure irrigation. *Surg Gynecol Obstet.* 1976;143(5):775–778.
 42. Carlson HC, Briggs RL, Green VA, Stewart JL. Effect of pressure and tip modification on the dispersion of fluid throughout cells and tissues during the irrigation of experimental wounds. *Oral Surg Oral Med Oral Pathol.* 1971;32(2):347–355.
 43. Saxe A, Goldstein E, Dixon S, Ostrup R. Pulsatile lavage in the management of postoperative wound infections. *Am Surg.* 1980;46(7):391–397.
 44. Diekmann JM. Use of a dental irrigating device in the treatment of decubitus ulcers. *Nurs Res.* 1984;33(5):303–305.
 45. Niederhuber SS, Stribley RF, Koepke GH. Reduction of skin bacterial load with use of the therapeutic whirlpool. *Phys Ther.* 1975;55(5):482–486.
 46. Bohannon RW. Whirlpool versus whirlpool rinse for removal of bacteria from a venous stasis ulcer. *Phys Ther.* 1982;62(3):304–308.
 47. Feedar JA, Kloth LC. Conservative management of chronic wounds. In: Kloth LC, McCulloch JM, Feedar JA, eds. *Wound Healing: Alternatives in Management.* Philadelphia, Pa: FA Davis; 1990.
 48. Moore ZE, Cowman S. Wound cleansing for pressure ul-

5 Rodeheaver and Ratliff

- cers. *Cochrane Database Syst Rev.* 2005;(4):CD004983.
49. Robson MC, Phillips LG, Thomason A, et al. Recombinant human platelet-derived growth factor-BB for the treatment of chronic pressure ulcers. *Ann Plast Surg.* 1992;29(3):193–201.
 50. Robson MC, Phillips LG, Thomason A, Robson LE, Pierce GF. Platelet-derived growth factor BB for the treatment of chronic pressure ulcers. *Lancet.* 1992;339(8784):23–25.
 51. van Rijswijk L. Full-thickness pressure ulcers: patient and wound healing characteristics. *Decubitus.* 1993;6(1):16–21.
 52. Bendy RH Jr, Nuccio PA, Wolfe E, et al. Relationship of quantitative wound bacterial counts to healing of decubiti: effect of topical gentamicin. *Antimicrobial Agents Chemother (Bethesda).* 1964;10:147–155.
 53. Lookingbill DP, Miller SH, Knowles RC. Bacteriology of chronic leg ulcers. *Arch Dermatol.* 1978;114(12):1765–1768.
 54. Daltrey DC, Rhodes B, Chatwood JG. Investigation into the microbial flora of healing and non-healing decubitus ulcers. *J Clin Pathol.* 1981;34(7):701–705.
 55. Sapico FL, Ginunas VJ, Thornhill-Joynes M, et al. Quantitative microbiology of pressure sores in different stages of healing. *Diagn Microbiol Infect Dis.* 1986;5(1):31–38.
 56. Lyman IR, Tenery JH, Basson RP. Correlation between decrease in bacterial load and rate of wound healing. *Surg Gynecol Obstet.* 1970;130(4):616–621.
 57. Margraf HW, Covey TH Jr. A trial of silver-zinc-allantoinite in the treatment of leg ulcers. *Arch Surg.* 1977;112(6):699–704.
 58. Gilchrist B, Reed C. The bacteriology of chronic venous ulcers treated with occlusive hydrocolloid dressings. *Br J Dermatol.* 1989;121(3):337–344.
 59. Alper JC, Welch EA, Ginsberg M, Bogaars H, Maguire P. Moist wound healing under a vapor permeable membrane. *J Am Acad Dermatol.* 1983;8(3):347–353.
 60. van Rijswijk L, Brown D, Friedman S, et al. Multicenter clinical evaluation of a hydrocolloid dressing for leg ulcers. *Cutis.* 1985;35(2):173–176.
 61. Eriksson G, Eklund AE, Kallings LO. The clinical significance of bacterial growth in venous leg ulcers. *Scand J Infect Dis.* 1984;16(2):175–180.
 62. Bowler PG, Duerden BI, Armstrong DG. Wound microbiology and associated approaches to wound management. *Clin Microbiol Rev.* 2001;14(2):244–269.
 63. Stotts NA. Determination of bacterial burden in wounds. *Adv Wound Care.* 1995;8(4 Suppl):46–52.
 64. Robson MC, Heggors JP. Bacterial quantification of open wounds. *Mil Med.* 1969;134(1):19–24.
 65. Lee PC, Turnidge J, McDonald PJ. Fine-needle aspiration biopsy in diagnosis of soft tissue infections. *J Clin Microbiol.* 1985;22(1):80–83.
 66. Levine NS, Lindberg RB, Mason AD Jr, Pruitt BA Jr. The quantitative swab culture and smear: a quick, simple method for determining the number of viable aerobic bacteria on open wounds. *J Trauma.* 1976;16(2):89–94.
 67. Thomson P, Taddonio T, Tait M, et al. Correlation between swab and biopsy for the quantification of burn wound microflora. International Congress on Burn Injuries Program and Abstract Book. 1990;8:381.
 68. Cuzzell JZ. The right way to culture a wound. *Am J Nurs.* 1993;93(1):48–50.
 69. Bill TJ, Ratliff CR, Donovan AM, Knox LK, Morgan RF, Rodeheaver GT. Quantitative swab culture versus tissue biopsy: a comparison in chronic wounds. *Ostomy Wound Manage.* 2001;47(1):34–37.
 70. Ratliff CR, Rodeheaver GT. Correlation of semi-quantitative swab cultures to quantitative swab cultures from chronic wounds. *WOUNDS.* 2002;14(9):329–333.
 71. Zamora JL, Price MF, Chuang P, Gentry LO. Inhibition of povidone-iodine's bactericidal activity by common organic substances: an experimental study. *Surgery.* 1985;98(1):25–29.
 72. Fleming A. The action of chemical and physiological antiseptics in a septic wound. *Br J Surg.* 1919;7:99–129.
 73. Lacey RW. Antibacterial activity of povidone towards nonsporing bacteria. *J Appl Bacteriol.* 1979;46(3):443–449.
 74. Morgan JE. Topical therapy of pressure ulcers. *Surg Gynecol Obstet.* 1975;141(6):945–947.
 75. Cooper R. A review of the evidence for the use of topical antimicrobial agents in wound care. Available at: <http://www.worldwidewounds.com/2004/february/Cooper/Topical-Antimicrobial-Agents.html>. Accessed February 13, 2007.
 76. Drosou A, Falabella A, Kirsner R. Antiseptics on wounds: an area of controversy. *WOUNDS.* 2003;15(5):149–166.
 77. White RJ, Cutting K, Kingsley A. Topical antimicrobials in the control of wound bioburden. *Ostomy Wound Manage.* 2006;52(8):26–58.
 78. Connell JF Jr, Rousselot LM. Povidone-iodine. Extensive surgical evaluation of a new antiseptic agent. *Am J Surg.* 1964;108:849–855.
 79. Gilgore A. The use of povidone-iodine in the treatment of infected cutaneous ulcers. *Curr Ther Res.* 1978;24(7):843–848.
 80. Lee BY, Trainor FS, Thoden WR. Topical application of povidone-iodine in the management of decubitus and stasis ulcers. *J Am Geriatr Soc.* 1979;27(7):302–306.
 81. Sugarman B. Infection and pressure sores. *Arch Phys Med Rehabil.* 1985;66(3):177–179.
 82. Michael J. Topical use of PVP-I (Betadine) preparations in patients with spinal cord injury. *Drugs Exp Clin Res.* 1985;11(2):107–109.
 83. Kucan JO, Robson MC, Heggors JP, Ko F. Comparison of silver sulfadiazine, povidone-iodine and physiologic saline in the treatment of chronic pressure ulcers. *J Am Geriatr Soc.* 1981;29(5):232–235.
 84. Banwell H. What is the evidence for tissue regeneration impairment when using a formulation of PVP-I antiseptic on open wounds? *Dermatology.* 2006;212(Suppl 1):66–76.
 85. Sundberg J, Meller R. A retrospective review of the use of cadexomer iodine in the treatment of chronic wounds. *WOUNDS.* 1997;9(3):68–86.
 86. Moss C, Taylor AEM, Shuster S. Comparison of cadexomer iodine and dextranomer for chronic venous ulcers. *Clin Exp Dermatol.* 1987;12(6):413–418.
 87. Carrel A, Dehelly G. The Treatment of Infected Wounds. New York, NY: Hoeber; 1917.
 88. Taylor HD, Austin JH. The solvent action of antiseptics on necrotic tissue. *J Exp Med.* 1918;27(1):155–164.

89. Leveen HH, Falk G, Borek B, et al. Chemical acidification of wounds. An adjuvant to healing and the unfavorable action of alkalinity and ammonia. *Ann Surg.* 1973;178(6):745–753.
90. Phillips I, Lobo AZ, Fernandes R, Gundara NS. Acetic acid in the treatment of superficial wounds infected by *Pseudomonas aeruginosa*. *Lancet.* 1968;1(7532):11–13.
91. Milner SM. Acetic acid to treat *Pseudomonas aeruginosa* in superficial wounds and burns. *Lancet.* 1992;340(8810):61.
92. AMA Drug Evaluation. 10th ed. Chicago, Ill: American Medical Association; 1994:620–621.
93. Moyer CA, Brentano L, Gravens DL, Margraf HW, Monafó WW Jr. Treatment of large human burns with 0.5 per cent silver nitrate solution. *Arch Surg.* 1965;90(6):812–867.
94. Fox CL Jr. Silver sulfadiazine—a new topical therapy for *Pseudomonas* in burns. Therapy of *Pseudomonas* infection in burns. *Arch Surg.* 1968;96(2):184–188.
95. Parsons D, Bowler PG, Myles V, Jones S. Silver antimicrobial dressings in wound management: a comparison of antibacterial, physical, and chemical characteristics. *WOUNDS.* 2005;17(8):222–232.
96. Warriner R, Burrell R. Infection and the chronic wound: a focus on silver. *Adv Skin Wound Care.* 2005;18(Suppl 1):2–12.
97. Bolton L. Are silver products safe and effective for chronic wound management? *J Wound Ostomy Continence Nurs.* 2006;33(5):469–477.
98. Tomaselli N. The role of topical silver preparations in wound healing. *J Wound Ostomy Continence Nurs.* 2006;33(4):367–380.
99. Bergin SM, Wraight P. Silver based wound dressings and topical agents for treating diabetic foot ulcers. *Cochrane Database Syst Rev.* 2006;(1):CD005082.
100. Lineaweaver W, Howard R, Soucy D, et al. Topical antimicrobial toxicity. *Arch Surg.* 1985;120(3):267–270.
101. Cooper ML, Laxer JA, Hansbrough JF. The cytotoxic effects of commonly used topical antimicrobial agents on human fibroblasts and keratinocytes. *J Trauma.* 1991;31(6):775–784.
102. Teepe RG, Koebrugge EJ, Lowik CW, et al. Cytotoxic effects of topical antimicrobial and antiseptic agents on human keratinocytes in vitro. *J Trauma.* 1993;35(1):8–19.
103. Branemark PI, Ekholm R. Tissue injury caused by wound disinfectants. *J Bone Joint Surg Am.* 1967;49(1):48–62.
104. Brennan SS, Leaper DJ. The effect of antiseptics on the healing wound: a study using the ear chamber. *Br J Surg.* 1985;72(10):780–782.
105. Cotter JL, Fader RC, Lilley C, Herndon DN. Chemical parameters, antimicrobial activities, and tissue toxicity of 0.1 and 0.5% sodium hypochlorite solutions. *Antimicrob Agents Chemother.* 1985;28(1):118–122.
106. Brennan SS, Foster ME, Leaper DJ. Antiseptic toxicity in wounds healing by secondary intention. *J Hosp Infect.* 1986;8(3):263–267.
107. Becker GD. Identification and management of the patient at high risk for wound infection. *Head Neck Surg.* 1986;8(3):205–210.
108. Viljanto J. Disinfection of surgical wounds without inhibition of normal wound healing. *Arch Surg.* 1980;115(3):253–256.
109. Hegggers JP, Sazy JA, Stenberg BD, et al. Bactericidal and wound-healing properties of sodium hypochlorite solutions: the 1991 Lindberg Award. *J Burn Care Rehab.* 1991;12(5):420–424.
110. Rodeheaver G. Commentary. *Diabetes Spectrum.* 1992;5(6):349–350.
111. Witkowski JA, Parish LC. Topical metronidazole gel. The bacteriology of decubitus ulcers. *Int J Dermatol.* 1991;30(9):660–661.
112. Nelson EA, O'Meara S, Golder S, Dalton J, Craig D, Iglesias C; DASIDU Steering Group. Systematic review of antimicrobial treatments for diabetic foot ulcers. *Diabet Med.* 2006;23(4):348–359.
113. Rodeheaver GT, Gentry S, Saffer L, Edlich RF. Topical antimicrobial cream sensitivity testing. *Surg Gynecol Obstet.* 1980;151(6):747–752.
114. Nathan P, Law EJ, Murphy DF, MacMillan BG. A laboratory method for selection of topical antimicrobial agents to treat infected burn wounds. *Burns.* 1978;4:177–187.
115. European Wound Management Association (EWMA). Position Document: Management of Wound Infection. London, UK: MEP Ltd; 2006.
116. Gilbert P. Avoiding the resistance pitfall in infection control: does the use of antiseptic products contribute to the spread of antibiotic resistance? *Ostomy Wound Manage.* 2006;52(10A Suppl):1S–7S.

Wound irrigation is the steady flow of a solution across an open wound surface to achieve wound hydration, to remove deeper debris, and to assist with the visual examination. The irrigation solution is meant to remove cellular debris and surface pathogens contained in wound exudates or residue from topically applied wound care products. Compared to swabbing or bathing, wound irrigation is considered to be the most consistently effective method of wound cleansing. [1]. Normal wound healing is characterized by 3 interrelated phases: inflammatory, proliferative or fibroplastic, and remodeling. Wound cleansing, wound irrigation, wound disinfection. Krasner D, Kane D. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals. 2nd. Title. Procedure: Wound Cleansing. Practice Level Background. Indications / Precautions/ Contra-indications. Wound cleansing and irrigation are defined as the application of fluid to a wound to remove exudate, slough, necrotic debris, bacterial contaminants and dressing residue without adversely impacting cellular activity vital to the wound healing process, or inoculating the underlying tissue with bacteria. Pressures between 8 and 15 psi are sufficient to cleanse or irrigate a wound. Wound cleansing is conducted with at least 100ml of sterile normal saline (NS) or sterile water container. (2007). Wound cleansing, wound irrigation and wound disinfection. In: Krasner D, Rodeheaver. G, Sibbald R., (Eds.)