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Critical Overview of Wound Care Modalities

Keith W. Hartnett

University of North Dakota

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CRITICAL OVERVIEW OF WOUND CARE MODALITIES

by

Keith W. Hartnett
Bachelor of Science in Physical Therapy
University of North Dakota, 1993

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

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in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

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May
1994
This Independent Study, submitted by Keith W. Hartnett in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and hereby approved.

(Chairperson, Physical Therapy)

(Faculty Preceptor)

(Graduate School Advisor)

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Title Critical overview of Wound Care Modalities

Department Physical Therapy

Degree Master of Physical Therapy

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ABSTRACT

The purpose of this independent study is to explore Physical Therapy modalities used in adjunct to traditional wound management. There are many factors that influence the healing rate. A basic knowledge of the body's healing process is needed in order to understand the affects modalities have on the wound.

The key to wound management is the initial and constant assessment. The patient history, subjective information, and objective measure are critical to the etiology and progression of the wound, and will be discussed.

Modalities can accelerate wound healing. Mechanical and physiological effects of the different modalities will be explored. This study is not designed to promote one modality; rather it gives a general overview of the most widely used techniques. There is ongoing research with these modalities.
CHAPTER I
INTRODUCTION

Wound healing is a complex process which is constantly being studied. Although sophisticated technology is present in today's healthcare field, many of these new technologies lack the ability to increase the body's own natural process of wound healing. Wound management is not just a single process having only one discipline providing care. Wound management today uses a multi-disciplined organization of healthcare professionals with common goals: to prevent and heal the wounds of their patients.

Physical therapy (PT) has always had a traditional role in the rehabilitation of the patient's musculoskeletal disorders. Physical therapy is now being thought of as a primary provider in the area of prevention, identification, and alleviation of acute and chronic soft tissue trauma. Physical therapists assess, measure, and treat these traumas which differ in anatomical and physiological natures. Physical therapists have been educated in the fundamentals of the body's natural healing process. They can now apply their knowledge and skills of therapeutic modalities for the purpose of wound care. Modalities used include thermal, sound, and electrical agents.

The basis for this independent study is to discuss the use of modalities and their effects on the healing process of wounds. An overview of the body's healing process will be provided. The phases of wound healing will be
presented. The assessment, measurement, and documentation of the wounds will be covered. Finally treatment modalities an area of expertise for PTs, will be reviewed.

Debridement and hydrotherapy the more traditional treatments, will be discussed. Ultrasound and electrical stimulation are new treatment techniques which are being used in the clinic: a more in-depth look at these modalities will be given. The physical properties, physiological effects, clinical application guidelines, limitations, and some research evidence concerning the efficacy of these procedures will be presented.
CHAPTER II

WOUND HEALING PHYSIOLOGY

When damage to vascularized tissue occurs the body goes through a distinct healing process. There are three phases of this process, reaction, regeneration, and remodeling. Even though there are some basic time lines for each phase, the phases overlap considerably due to variability within each individual wound.

Reaction Phase

The reaction phase begins at the time of injury and continues to approximately day five. Inflammation is the name used synonymously with the reaction phase. It has been stated that "no inflammation no repair".\textsuperscript{1} The extent of the inflammatory response is dependent upon the severity of the injury. The cardinal signs are edema, heat, redness, and pain.\textsuperscript{2} The reaction phase is further divided into the hemostatic response, vascular response, and cellular response.

Hemostatic Response

The hemostatic response to injury includes coagulation within arterioles. Platelets deposit fibrin, which adhere to the vessel walls trapping red blood cells; a clot is formed and bleeding is decreased. Fibrin also occludes lymphatic channels preventing drainage from the wound, localizing inflammation, and reducing the spread of bacterial infection.
Vascular Response

The vascular response is initiated almost as early as platelet adherence. The response includes vasoconstriction, vasodilation, and arterial permeability.

This vascular response starts with a vasoconstriction which is mediated by norepinephrine. It lasts approximately five to ten minutes after the initial injury. When the wound is severe enough, secondary vasoconstriction occurs due to the release of serotonin from platelets. Subsequent vasodilation is caused by the release of histamines, bradykinins, or prostaglandins. Histamines are released by the mast cells and platelets at the site of the injury. The histamines produce a short lived increase in the permeability of the venules. Bradykinins are released from plasma proteins (globulins) by the plasma enzymes. Prostaglandins are produced by all cells in the body when the cell membrane is injured. Prostaglandins are short lived and act early in the vascular response, approximately 20 to 30 minutes after initial injury. Vasodilation enhances diapedesis as part of the cellular response allowing white blood cells into the extravascular space to begin wound healing.

Cellular Response

Activated platelets are known to release biologically active substances. These substances are platelet-derived and epidermal growth factors (PDGH, EGH), both of which facilitate cell migration and growth at the injured sites. The release of PDGH and EGH is an important factor in initiating migration of leukocytes into the extravascular space. Leukocytes line the walls of the post-capillary venules within two to nine minutes after
injury. Through diapedesis these leukocytes move into the extravascular space and begin phagocytizing the injured tissue and bacteria. This sets the stage for tissue healing. There are numerous leukocytes (polymorphonuclear cells, mononuclear cells) which are present during the initial inflammatory stage. The number and type of leukocytes vary at different stages in the inflammatory process. Neutrophils are prevalent early in inflammation due to the release of chemotactic agents at the site of injury. Their primary role is that of phagocytizing bacteria. Their digestive enzymes also act as attractions for more leukocytes. Once the neutrophils have successfully completed their task and disintegrate, their digestive enzymes are absorbed into the tissue. Phagocytosis also creates toxins which can harm healthy tissue surrounding the injured site if the inflammation were to last for long periods of time.

Just as the neutrophils are important to the wound healing process, macrophages are critical to the initiation of tissue repair. These macrophages come from monocytes and are considered to be the heavy duty bacteria fighters. The macrophages are attracted by the chemotactic agents released from the neutrophils. Their life in extravascular space is approximately two days.

The macrophages release angiogenesis factor (AGF) which initiates and propagates granulation tissue. It does this through affecting the blood oxygen-carbon dioxide exchange. The AGF from the macrophages in combination with PDGH and EGH released from platelets cause the fibroblasts to enter the wound within 24 hours post injury. This activity is a vital to the restoration of structural integrity of the wound.
Regeneration Phase

The regeneration phase of wound healing includes mesenchymal cell migration, mesenchymal cell proliferation, and neovascularization. These intermediate wound healing responses are controlled by the release of growth factors. Endocrine factors are secreted into the bloodstream; paracrine factors are released from cells adjacent to the injury; autocrine factors are secreted by the epithelial cells themselves.

First, a number of factors stimulate mesenchymal cell migration. These factors are fibronectin, lymphokines, and complement peptides, EGF, and collagen. Fibroblasts accumulate in the wound.

The second intermediate response is mesenchymal cell proliferation, which repopulates the injury site with fibroblasts. These fibroblasts are recruited to replace the extracellular matrix. Platelet-derived growth hormones (PDGH) and Transforming Growth Factor-B (TGF-B) are stimulants for this process. Other factors such as insulin from the pancreas and somatomedins function to stimulate mesenchymal cell proliferation.

The third intermediate response is neovascularization. This is sprouting of capillaries from small venules in the area of injury. Collagenase secreted by inflammatory cells in response to the degradation of the basement membrane is involved with the migration of the epithelial cells. Both acidic and basic Fibroblast Growth Factors (aFGF, bFGF) stimulate neovascularization. These sprouts initially grow as solid sprouts, but soon develop a curvature which later develops into a lumen. These sprouts grow from all sides of the wound. When a sprout comes into contact with another
sprout, they will join at their tips and blood begins to flow through the new loop. New sprouts will grow from the loop and the process repeats itself. As neovascularization matures the previously released growth factors will diminish.

Remodeling Phase

The initial phases of reaction and regeneration rid the wound of all foreign substances and bacteria. This phagocytosis creates a barrier to prevent further injury to viable tissue. During the final phase; the remodeling phase, granulation tissue formation begins with re-epithelialization at the edges of the wound. This process begins approximately three days post injury and last twenty days. Epithelialization occurs with help of the proteins fibronectin, and vitronectin. At 24 hours, epithelial cells from the basal levels begin to elongate and migrate from wound edges. New epithelial cells are also generated around hair follicles, sweat glands, and sebaceous glands. Once epithelialization starts, the layers are thicker near these edges and thinner over the rest of the wound. Some cells stop migrating; other cells leap frog over these cells in order to keep migration going. After the epithelial cells have joined together and vascularization has been established, the tissue shows a red granular appearance, hence the name granulation tissue.

The epithelialization process has laid down the ground substances for the connective tissue matrix. During epithelialization fibroplasia takes place with myofibroblasts proliferating the wound. The proliferation is modulated by PDGF, EGF, and FGF. Mesenchymal cells migrate up the strands of fibrin to start scar tissue formation. Collagen is synthesized in
large quantities about five days after the myofibroblasts proliferate the wound. Collagen provides structural integrity to the connective-tissue matrix. This newly formed matrix is unorganized and lacks in tensile strength. By day 21 the wound is 20 percent of its original strength. A repaired wound has only approximately 70 percent of its original strength. 4

During the final portion of the remodeling phase wound contraction take place. Myofibroblasts accumulate around the margins of the wound. The myofibroblasts contract in response to bradykinin, epinephrine, and prostaglandin. 24 Wound contraction is cell-mediated, with an active rate of (.06-.05mm/day), which is directly related to the number of cells within the collagen matrix. 27 This wound contraction force pulls the greatest amount on local tissue rather than distant tissue. Myofibroblast cells generate traction forces rather than contraction forces. 25 The connective tissue matrix contributes to wound contraction by its interactions between normal fibroblasts. 26 After initial maximal contraction the wound relaxes. 28

During scar remodeling, collagenase breaks down previously deposited collagen. Collagen fibers increase in diameter during this phase. 29 New collagen responds to stresses on the wound. A wound under stress heals more securely at an earlier time. The collagen synthesis rate may remain elevated for six months. The wound reaches 90 percent of its final strength approximately six weeks after the initial wound injury.

An abnormal response to wound healing is hyperactive collagen formation. Hypertrophic or keloid scars result. The hypertrophic scars resolve themselves within one to two years. However, keloid scars exist
indefinitely. These scars occur in areas of high tension on the skin. There are few treatment modalities that will decrease this hyperactive collagen formation.

Reaction, regeneration, and remodeling are three distinct phases of the wound healing process. These phases may overlap. The reaction phase is a complex process in which the wound is rid of all infectious and bacterial substances. During the regeneration phase mesenchymal cell migration, proliferation, and neovascularization are the first stages of epithelialization. The remodeling phase begins with granulation tissue formation and re-epithelialization. Fibroplasia, contraction, and scar formation are the final stages of wound healing. Basic knowledge of wound healing is necessary for understanding the wound's reaction to various outside factors. Assessment of the wound is an essential part of wound healing. Identifying the characteristics of the wound at each phase can direct wound management.
CHAPTER III
WOUND CLASSIFICATION

The key to wound management is the initial and constant assessment. The entire physiological makeup and function of the patient must be considered. The information gathering process incorporates the patient's history, subjective information, risk factors, and clinical measurements (type, severity, size, shape, depth) of the wound. Neurological and vascular screenings should be performed during wound assessment.

History and Subjective Assessment

History is the first step in the information gathering process. Even if this information has been gathered by other health care workers, new information may be gathered by restating the questions, a new interpretation of the patient's response, or by new developments in the patient's physiological problems.

There are multiple components important to the history of the patient. For example, the patient's occupation may impact the wound; venous pooling of blood during prolonged standing will delay wound healing. Knowledge of events leading to wound onset is necessary. A traumatic wound will receive different management than a wound secondary to disease. Does the patient have any allergies or secondary diagnoses? A personal history of diabetes, peripheral vascular disease, or cardiac disease may impact the healing
The patient's medications such as non-steroidal, steroidal, or immunosuppressive drugs may complicate the wound healing process. These drugs could affect the regeneration phase of wound healing by decreasing collagen synthesis leading to a decrease in the tensile strength of the tissue. Oral Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) decrease the tensile strength of connective tissue by causing vasoconstriction early in the inflammatory phase. Topical NSAIDs do not affect re-epithelialization of the wound.

Patients on corticosteroids demonstrate decreased wound healing due to an inhibition of collagen synthesis. This inhibition affects the tensile strength of the collagen. Steroids also affect the cells during the regeneration phase by inhibiting fibroblastic function.

Immunosuppressive drugs (Azanthipine and Prednisone) most often used with transplant patients have been associated with decreased tensile strength of wounds. Patients on immunosuppressive drugs may not respond to conventional therapies. A complete list of the patients' medications is essential in choosing a correct treatment modality.

The patient's social history affects their wound healing. Cigarette smoking is significant in preventing arterial ulcers from healing. Nicotine causes vasoconstriction of arteries, long term vessel wall damage, and arterial insufficiency. Alcohol can have similar effects.

Through subjective assessment, the patient is questioned about their symptoms, the degree, and location of these symptoms. For example, is the pain located in, around, or distant from the wound. Parathesia around the
wound could be secondary to peripheral neuropathy. These symptoms could lead to further objective testing. A decrease or increase of symptoms can be helpful in determining the type of ulceration involved.

Risk Factors

Risk factors affect the wound healing process. The risk factors of medications, allergies, medical history, occupation, previous wounds, and social history have previously been mentioned. Other risk factors are increased age, nutritional deficits, and multiple diagnoses. Many wound care patients are elderly. The affects of aging are shown in Table 3-1. With increased age cellular activity of the fibroblasts decreases. There is a decreased tensile strength of the connective tissue matrix. Once the elderly have developed wounds, they are more likely to redevelop chronic wounds because of their decrease in cellular activity. With aging there is diminished blood flow, which will not allow metabolites, bacteria, or other toxic substances to be carried away; this affects closure of the wound.

Nutritional care during wound healing plays a major role in maintaining the body's homeostasis and its resistance to infection. The energy demand during healing is double that of normal. Symptoms of possible nutritional deficits are shown in Table 3-2. Physical therapists are not trained in nutritional care. If any of these are present then the patient should be referred to a registered dietician.

As previously stated, multiple diagnoses can affect wound healing. Diagnosis which may alter management are spinal cord injuries, stroke, amputations, any patient with decreased sensory status, and decrease in cognitive awareness. With limited resources the physical therapist may not
Table 3-1. Effects of Aging on Wound Healing

<table>
<thead>
<tr>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Decrease wound virulence</td>
</tr>
<tr>
<td>* Delayed cell migration and proliferation</td>
</tr>
<tr>
<td>* Decreased epithelialization</td>
</tr>
<tr>
<td>* Delayed rate of capillary growth</td>
</tr>
<tr>
<td>* Delayed fibroblast collagen synthesis</td>
</tr>
<tr>
<td>* Decreased tensile strength</td>
</tr>
<tr>
<td>* Decreased wound contraction rate</td>
</tr>
</tbody>
</table>
Table 3-2. Symptoms of Malnutrition

* Muscle weakness
* Patient becomes easily fatigued
* Patient intolerance to cold temperatures
* Skin shows flaky dermatitis
* Excessive ankle edema
have the opportunity to influence these risk factors. Recognition of these risk factors presents the therapist with the chance to work with and refer the patient to other health care professionals.

Clinical Measurements

The objective test and measurements of the wound are the most important aspect of wound assessment. Wounds are put into three categories: The type of lesion, the depth of the tissues involved, and the progression of the destruction. The type of wound could be described as a pressure, abrasion, laceration, surgical, blister, hematoma, stasis, or shearing wound. The depth of the tissue involved is either that of partial or full thickness. Wound status and progression are identified by stages set by The International Association for Enterostomal Therapy.4

In stage I, the wound is limited to the epidermis and presents with erythema and edema. The epidermis, dermis and sub-cutaneous fat are involved with a stage II wound. The tissue shown is moist and pink. A small shallow crater appears in a stage III wound, and there may be necrotic tissue, undermining of deeper tissue, and the presence of infection with exudate. In stage IV, tissue destruction extends through the subcutaneous tissue and involves fascia, muscle and bone. Undermining and infection with exudate are present.

Wounds are also classified by their color. Red indicates healthy granulating tissue. Yellow indicates necrotic debris and exudate. Black is
the color of necrotic tissue or eschar; this eschar must be removed to accurately identify the wound. Wounds covered by necrotic tissue should be assessed for the percentage of total body surface area involved.

Other wound factors that should be assessed are edema, drainage, skin color, and temperature. The degree of edema gives information about the cause or contributing factors to the wound. If not corrected, edema will lead to tissue necrosis. Pain may be noted with the edema. Girth and volumetric measurements are means of assessing the edema. Drainage from the wound should be documented for its amount, color, odor, and consistency. Increased drainage indicates wound retrogression. The color and odor of the drainage will tell about the extent of the infection. Skin color points to wound etiology. Cyanotic or brownish skin indicates a decrease in venous return. Red skin is a sign of inflammation and arterial vasodilation. Redness from a pressure ulcer is due to the rupture of small capillaries in the area. Information about the oxyhemoglobin levels, can be obtained by looking at the fingernails, lips, and mouth membranes. Temperature of the wound will determine the severity and limits of the wound. Temperatures greater than $98^0F$ indicates inflammation and temperatures less than $95^0F$ indicate decreased circulation, possibly ischemia. Temperature baselines are needed in order to progress the response to therapy.

Determining the size, shape, and depth of a wound is often difficult to document. One objective way to measure the size of a wound is with use of a sterile acetate sheet. The sheet is placed over the wound and the margins of the wound are traced. The edges of the wound are then measured and documented. Topical dressings have a metric grid within the dressing; after
the wound is traced this backing can be peeled of and used as part of the documented record. More commonly, Polaroid snapshots are taken daily to provide a visual progress report of wound healing. Measuring wound margins does not give any information on undermining of healthy tissue. The clinician needs to investigate the extent of the undermining. One method for doing this is to place a sterile cotton swab in the wound and take measurements. Sequential drawings of the undermining will chart wound progression. Another technique uses Jeltrate. This is a alginate hydrocolloid paste that is put into the wound to provide a positive mold. This mold is then volumetrically measured.

**Vascular Tests**

Wound evaluation should include the assessment of the patient's vascular and neurological systems. These systems relate directly to wound development and progression. Patients with a arterial, venous, or neurotropic problems often demonstrate differing wound characteristics. Findings of vascular and neurological tests may lead to treatments secondary to that of the wound therapies.

Patient pulses should be constantly monitored for quality as well as presence. Pulses should be documented as normal, diminished, or absent. The pulse should also be compared to the uninvolved extremity. The most commonly monitored lower extremity pulses are the common iliac, femoral, popliteal, posterior tibial, and dorsalis pedal. Blood pressure should be monitored to get an idea of cardiovascular function. Auscultation can be performed over arteries.
### TABLE 3-3. Vascular Wound Characteristics

<table>
<thead>
<tr>
<th>Type</th>
<th>Cause</th>
<th>Location</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous</td>
<td>venous insufficency</td>
<td>distal 1/3 leg</td>
<td>* irregular shape</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>* pink-red base</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>* mild pain</td>
</tr>
<tr>
<td>Arterial</td>
<td>arterial insufficency</td>
<td>toes, feet,</td>
<td>* irregular shape</td>
</tr>
<tr>
<td></td>
<td></td>
<td>distal 1/3 leg</td>
<td>* pales base</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>* severe pain</td>
</tr>
<tr>
<td>Neurotropic</td>
<td>insensitivity</td>
<td>planter surface of foot</td>
<td>* circular shape</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>* painless</td>
</tr>
</tbody>
</table>
to assess if there is any turbulent flow or swishing sound (bruit) which would be an indication of a decrease in arterial supply, which would be addressed by a physician. Common arteries to be tested are in the neck, abdomen, and extremities.

Tests of peripheral artery circulation are the rubor of dependency, venous filling time, and claudication time. The rubor of dependency assesses skin color changes with extremity elevation and dependency. Venous filling time measures the time needed for that superficial veins to refill after emptying; anything greater than fifteen seconds denotes an arterial insufficiency. Claudication time assesses the time it takes for a severe pain to appear in the calf with walking.

Tests for peripheral venous circulation are the percussion test, Homans's sign, and Trendelenburg test. The percussion test assesses the viability of the great saphenous vein. One distal segment of the vein is palpated while percussion to the same vein 20-30 cm proximal in the leg is done. Percussions felt at the distal point show a decrease in valve competency. Homans's sign is positive for deep vein thrombophelbitis if the patient shows pain in the gastrocnemius with pressure on the muscle belly and dorsiflexion of the ankle. The Trendelenburg test assesses the competence of veins connecting to the great saphenous vein. The patient is supine with the leg elevated to ninety degrees to empty the venous blood. A tourniquet is applied around the proximal thigh, the patient stands, and the response filling time is observed. Normal time is approximately thirty seconds; a longer time indicates insufficiency.
Other tests can be performed. The Doppler ultrasound determines the lower extremity systolic pressure. A venograph or an arteriograph diagnoses deep vein thrombosis (DVT).

This chapter has shown that initial and constant assessments are essential for patients suffering from either chronic or new wounds. This chapter emphasizes the importance of the patient history and subjective information to the etiology or progression of the wound. Risk factors were discussed that could be an adjunct to decreased wound healing. Patients wounds would be classified according to symptoms. Some objective clinical guidelines were given to determine the size, shape, and depth of the wound. Neurological and vascular tests can provide additional information necessary to the overall diagnosis. All of this information is compiled to set reasonable goals and to formulate a treatment plan. Treatments are multidisciplinary and multifaceted. Specific physical therapy modalities to be discussed are debridement, topical agents, whirlpool, ultrasound, and electrical stimulation.
CHAPTER IV
TREATMENT MODALITIES

In the previous chapters, it has been shown how the body's own wound healing process works. Guidelines have been established to determine the type and classification of such wounds. Many times this "normal" wound healing process cannot take place without the appropriate selection and application of wound care products. This chapter will cover conservative management of wounds, including debridement and topical agents. The mechanical and physiological effects of traditional whirlpool, ultrasound and electrical stimulation will be explored.

Conservative Management of Wounds

In many wounds necrotic tissue impedes the formation of granulation tissue and prevents epithelial cells from migrating across the wound. Debridement of a wound is the removal of necrotic and dead tissue.

As previously stated, debridement takes place at the cellular level during the inflammatory or reaction phase. The leukocytes and polymorphonuclear cells are found during the initial response. Monocytes and neutrophils arrive later at the wound site to further debride the necrotic tissue. If large amounts of necrotic tissue are present, additional methods of debridement are necessary to facilitate this repair process.
There are two types of debridement interventions, selective and non-selective. Selective debridement removes only necrotic tissue. Non-selective removes both viable and non-viable tissue. Selective debridement can include: 1) enzymatic, which uses topical proteolytic enzymes to remove necrotic tissue; 2) autolytic and synthetic dressings; and 3) surgical excision. Non-selective debridement includes 1) dressings, 2) whirlpool, and 3) hydrogen peroxide.

The most common topical proteolytic enzymes are fibrolysin and deoxyribonuclease. These are very effective on necrotic tissue. However they are not as effective on heavy necrotic tissue which may be attached to the wound surface by undenatured collagen. Collagenase is the enzyme that can facilitate debridement of necrotic tissue. 39

Autolysis is the self-digestion of necrotic tissue by enzymes that are naturally found in tissue fluid. This type of phagocytic activity is though to be the most select form of debridement. Many wounds are covered with dressings providing a closed moist environment for the digestion of necrotic tissue. This environment also promotes granulation development and cell migration. 40

Surgical excision is the quickest way to selectively debride. This method however poses risk of infection and bleeding. The patient population this is used for is somewhat restrictive.

Non-selective forms of debridement consist of gauze dressings, whirlpool (which will be discussed later in this chapter), and hydrogen peroxide \((H_2O_2)\). 40 Gauze dressings (wet-wet, wet-dry) should only be used on necrotic tissue because viable skin and new epithelial growth can adhere
to the dressings. Hydrogen peroxide is a mechanical cleaner. Hydrogen peroxide has a foaming effervescent effect when it comes into contact with necrotic tissue and body fluids. When $\text{H}_2\text{O}_2$ comes in contact with necrotic tissue, it is decomposed into $\text{H}_2\text{O}$ and $\text{O}_2$. This effervescence can cause damage to new granulation tissue.\textsuperscript{41}

After wound debridement topical agents containing antibacterial agents are applied to prevent contamination and infections. Some agents will provide a closed moist environment to enhance the body's own healing process. Common agents are neosporin and silvadine. These agents retard the bacterial microorganisms in the wound without inhibiting reepithelialization.\textsuperscript{42,43} In addition, one of the components of neosporin is zinc. This had been found to increase epidermal healing.\textsuperscript{52} Silvadine (silver sulfadiazine) also enhances epidermal cell growth.\textsuperscript{52} Another product with zinc in it is the unna boot. This is rolled gauze impregnated with zinc oxide, calamine, and gelatin. The zinc oxide promotes reepithelialization; the boot helps with edema control.

**Whirlpool**

Whirlpool is one of the oldest methods that is used in physical therapy to treat dysfunction. The affects of whirlpool consists of debridement, heating or cooling, pain relief, increased blood flow, and relaxation. Antimicrobial agents can be added to the water to kill bacteria that may be present in wounds.

**Mechanical Effects**

As previously stated, whirlpool is a non-selective form of debridement. The turbulence from the whirlpool softens the eschar and necrotic tissue.
removing viscous exudate. Another method of debridement is to soak off dressings that become adhered to the wound.

**Physiological Effects**

The turbulence from the whirlpool causes mechanical stimulation to skin receptors. This causes an analgesic effect, closing the gates in the "gate theory" of pain modulation. The turbulence acts as a counterirritant affecting the large faster afferent sensory fibers, over riding the pain impulses.

The main effects of whirlpool are associated with the increase in tissue temperature, resulting in increased cellular metabolic rate, and blood vessel vasodilation. The chemical activity of cells and the metabolic rate increases two to three times for every 10°C rise in temperature. As the metabolic rate increases the inflammatory and repair process accelerates. The tissue temperature rise and increased metabolic rate cause vasodilation. Therefore, more nutrients, antibodies and leukocytes are available to promote tissue healing. Metabolites and cellular debris will be removed from the area by the increase blood flow.

**Whirlpool Additives**

Many antimicrobial agents are added to the whirlpool to counteract infection. The most common are povidone-iodine, sodium hydrochlorite (household bleach), and chloramine-T (chlorazene). Povidone-iodine has been shown to be an effective bactericide. It may however, retard epithelialization and decrease wound tensile strength. Sodium Hydrochlorite (household bleach) is a good agent to control sepsis. It is
cytotoxic to the fibroblast which adversely affects wound healing.

Chloramine-T is now a more widely use antimicrobial agent and it has a less irritating affect on fibroblasts.

Protocols for use of whirlpool during wound care are often dependent on the wound itself. Table 4-1 shows a brief condensed list of guidelines to follow.

Ultrasound

For more than forty years ultrasound (US) has been used as a modality for treatment of soft-tissue trauma. Today ultrasound is being used to promote wound healing. Ultrasound has two biophysical effects; thermal, or elevation of tissue temperature, and non-thermal, the change in physiological tissue not related to tissue temperature change. Ultrasound is available in 1.0, 2.0, and 3.0 MHz. Traditionally, 1.0 MHz is used for deeper tissue (greater than 3 cm from surface). The most commonly used frequency for wound care is 3.0 MHz, because often more superficial tissues is involved.

Thermal Effects

Ultrasound affects the tissues by causing a disruption of molecules. The molecules vibrate due to the sound waves, which in turn causes more molecules to vibrate. This increase in activity increases temperature, increases blood flow, increases cell migration, and increases removal of metabolites. These changes stimulate wound healing.

One of the main advantages of ultrasound is its tissue heating capabilities. A continuous wave is most often used to produce a rise in tissue temperature. For tissue with a healthy vascular system
Table 4-1 Whirlpool Guidelines for Wound Care

<table>
<thead>
<tr>
<th>Phase</th>
<th>Characteristics</th>
<th>Desired Effects</th>
<th>Temp (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5 days</td>
<td>Exudative</td>
<td>Vasodilation/Debridement</td>
<td>34.4</td>
</tr>
<tr>
<td>5-21 days</td>
<td>Fibroblastic</td>
<td>Vasodilation/Debridement</td>
<td>34.4</td>
</tr>
<tr>
<td></td>
<td>Proliferation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 days</td>
<td>Maturation</td>
<td>Vasodilation/Debridement</td>
<td>36.7 &amp; 38.8</td>
</tr>
</tbody>
</table>
this heating would be fine. However, if the wound is a stasis ulcer with decreased venous return, the increased temperature will cause tissue damage. In this instance, non-thermal ultrasound may be preferred. Non-thermal effects are mechanical and chemical changes which alter the makeup of the tissue.

Non-Thermal Effects

The non-thermal effects of ultrasound are physiological changes related to the mechanical deformation. A pulse wave is used for non-thermal effect of cavitation, acoustical streaming, and standing wave formation.

Cavitation is a vibrational effect of the ultrasound beam, causing micro-bubbles to form in blood, lymph, and tissue fluid. Bubbles will vibrate in response to the cycles of the ultrasound. This vibration of the bubbles can cause stimulation of tissue repair by altering cell permeability and the diffusion rate of metabolites. In contrast, exposure to a higher levels of therapeutic ultrasound can cause too much cell permeability, causing too many metabolites to be carried away from the wound site. This excessive exposure can cause cellular damage with resultant tissue damage.

Acoustical streaming is the movement of fluid along the edges of a cell. The microscopic bubbles have high velocity gradients next to the cell membrane. These gradients contain high viscous forces which increase cell permeability. This increase in permeability enhances sodium/calcium ion exchange across these membranes. Another significant effect is the release of serotonin from platlets, which is important in promoting cell migration to the wound site. Also, contained in the platlets are PDGH and EGH, both of which facilitate cell migration and growth.
Standing wave formation is the result of ultrasound waves reflected by a medium, i.e. soft tissue or bone. The combination of the initial wave and the reflected wave causes a resultant wave. Gas bubbles in the blood, lymph, or tissue fluids are trapped by this wave. The vibration of these gas bubbles can cause cavitation, which can be detrimental to tissue. Blood cell movement can be impeded or even stopped, due to the cells becoming packed together while the plasma continues to move. There is a decrease in gaseous exchange. Standing waves are avoided by constantly moving the sound head, thus ensuring the wave varies throughout the tissue.\(^4\)

**Physiological Effects**

Ultrasound produces physiological changes in tissues to accelerate the wound healing process. The cellular activities that are thought to be most affected by ultrasound are, 1) general protein and collagen synthesis by fibroblasts, 2) fibroblast motility, 3) fibroblast membrane permeability, 4) tensile strength and elasticity of scar tissue, and 5) modification of skin contraction.\(^5\) The following will show the affects of ultrasound during wound healing phases.

Ultrasound applied shortly after the injury can accelerate the inflammatory phases, in turn accelerating the wound healing.\(^5\) Ultrasound treatment during this phase have been shown to stimulate the release of histamines from mast cells by degranulation.\(^5\) These histamines produce a short lived increase in cell permeability of tissues, allowing the release of chemotactic agents attracting neutrophils and macrophages to the site of injury.\(^4\) Ultrasound treatments have been shown to increase sodium/calcium ion migration across the cell membranes into the mast cells and fibroblast.\(^4\)
Ultrasound also stimulates endothelial cells activity. Once released into the interstitial fluid PDGH and EGH are essential for stimulation and development of mesenchymal, fibroblast and endothelial cells.

During the regeneration phase fibroblasts begin to infiltrate the wound. When exposed to ultrasound fibroblasts increase the production of collagen, thus increasing the strength of the connective tissue. Shear stress from the acoustical streaming has been implicated in the increased production of collagen. The increased strength of the scar tissues is due to the increase in collagen content. The strength of the scar tissue is affected by ultrasound applied during the early inflammatory stage. Later, ultrasound increases the rate of contraction of the wound. During this late regeneration and remodeling phase fibroblasts turn into myofibroblasts, which resemble smooth muscle. These myofibroblast can be induced to contract when stimulated by ultrasound.

**Treatment Parameters**

Ultrasound has been shown to enhance the effects of wound healing. The treatment parameters vary with wounds. Decisions to be made about wound treatment include:

1. The nature and location of the wound.
2. Is the wound acute or chronic? Acute wounds receive pulsed waves, chronic wounds a continuous wave.
3. What is the depth of the wound? Superficial wounds use 3MHz US, deep wounds use 1 MHZ.
4. What is the size of the area to be treated? The wound should be divided into areas 1.5 times the size of the sound head with a treatment time of one to two minutes per area.
5. What medium should be used? An aqueous-gel base dressing is used of broken skin. Gels and cremes, are used on epithelialized skin.
6. What is the
intensity? Less than 0.3 W/CM$^2$ is used on acute wounds, 0.3 -1.2 W/CM$^2$ is used on chronic wounds. 7) What is the duration of treatment? A general guideline is one minute/CM$^2$ of wound area.

Ultrasound is an adjunct to a wound management. The ultrasound can be applied anytime during the wound healing phases. However, ultrasound works extremely well if it is started soon during the inflammatory phase. It can also have a detrimental effect if used incorrectly. The clinician must be proficient in the use of ultrasound.

**Electrical Stimulation**

For centuries electrical stimulation has been used to enhance wound healing.$^{55}$ The first documentation of electrical stimulation was in 1688. A charged gold leaf was used over small pox lesions. Healing was observed without a formation of a scar.$^{49}$ In the 1940s electrical potentials were found over open wound surfaces.$^{56}$ These potentials were positive for four days. Post four days, the potential became negative.

In the early 1960s direct current (DC) was most often associated with wound care.$^{56}$ Different histological responses were found beneath the anode versus the cathode.$^{57}$ Increases in wound tensile strength were reported under the cathode. Direct currents applied from the anode increased collagen synthesis and epithelialization.$^{58}$ Many clinics today prefer to use high voltage stimulation (HVS). High voltage stimulation treatment of a wound with the anode promotes healing, while use of the cathode provides bactericidal effects.$^{49,59}$

There are several types of therapeutic electrical stimulation. Those used for wound healing are galvanic current (DC), and high voltage pulsed
current (HVPC). Direct current is a continuous monophasic unidirectional flow of current. The voltage does not vary during the treatment, and the intensity is approximately 3-5 milliamperes (ma). High voltage pulsed current is an alternating monophasic bydirectional flow of current. The short duration voltage does vary during the treatment with an intensity of approximately 6 ma. These currents and their electrochemical and physiological effects will be discussed.

Electrochemical Changes

Direct current produces chemical changes at the tissue level, with a redistribution of sodium and chlorine ions. Antibacterial effect enhances enzymatic activity. Electrical stimulation also affects the stimulation of protein and DNA synthesis. High voltage pulsed current tends to penetrate into deeper tissue, because of the pulse current.

Physiological Changes

Diminished circulation inhibits wound healing and slows the inflammatory phase. This causes limited O₂ intake and decreased nutritional support to the wound and movement of metabolites away from the wound. The physiological effect of HVPC include increase circulation, edema reduction, and increase activity during wound recovery phases.

It has been shown that after a treatment with HVPC, the O₂ intake by cells has increased. This increase in O₂ aids the macrophages to release ACF, which propagates granulation tissue. Increase in blood flow was noted after a treatment of HVPC. The greatest increase in blood flow was measured under the cathode. The increased blood flow allows the leukocytes, neutrophils, and macrophages to be carried to the wound site to
begin phagocytosis. This increased blood flow is needed to carry away the metabolites and debri. This decreases microvessel leakage thus decreasing edema. Edema reduction may also be affected by cataphoresis, which is movement of interstitial fluid toward the cathode.

Wound Recovery Changes

Electrical stimulation affects the phases of wound recovery. HVPC has been shown to stimulate fibroblasts with a 20 minute treatment. Two hours after stimulation the fibroblastic rate of protein synthesis was increased by 160%. The fibroblasts migrate toward the cathode during stimulation. There is also a significant increase in DNA production. Epidermal cell migration is also influenced by HVPC. Epidermal cells undergoing migration have origins in the border strip of skin surround the wound.

Additional Effects of Electrical Stimulation

HVPC has been shown to inhibit hypertrophic scar formation. This is thought to be accomplished by reducing the number of mast cells allowed to proliferate the wound.

Wound healing is inhibited with the presence of infection. Electrical stimulation can inhibit or destroy bacteria present in wounds, thus enhancing wound healing. Cathodal direct current produces bactericidal effects on escherichia coli treatment. The continuous cathodal stimulation bombards the bacteria with electrons, continuously exciting the cell membranes and depleting the bacterial substrate. Another study showed migration of leukocytes and macrophages toward the cathode, while neutrophils migrated toward both poles. The significance is that phagocytic cells are drawn to the wound site to begin the wound repair process.
This chapter has shown there are a number of therapeutic modalities used in physical therapy. These have been shown to be an adjunct to the normal wound healing process. Many studies have demonstrated their outcomes on wound healing. These modalities can be used individually, or in combination with others. Many of these therapies are relatively new, and research is ongoing.
CHAPTER V
CONCLUSION

Understanding the fundamentals of wound healing is essential to anticipating and preventing adverse results. The inflammatory phase is the first response in wound healing. These vascular and cellular responses prepare the wound for the remaining healing processes.

The tissue healing and repair response consists of the regeneration and remodeling phases which includes granulation, re-epithelialization, fibroplasia, neovascularization, wound contraction, and scar formation. Although there is a basic repair process, many intrinsic and extrinsic factors complicate it.

To maximize recovery the multi-disciplined team must have a thorough knowledge of the patient's medical history including his or her nutritional status, medications, and treatments for unrelated medical problems. Objective tests determine the cause and status of the lesion. Correct treatment can thus be planned and initiated.

The conservative management of wound healing including debridement and topical agents was discussed. Normally debridement takes place at the cellular level. If large amounts of necrotic tissue are present, additional methods such as whirlpool are required to facilitate the repair process. These methods can compromise healthy tissue.
Various modalities can enhance wound healing. Ultrasonic therapy is one of those modalities. Ultrasound should be applied shortly after injury, during the inflammatory phase. It accelerates the inflammatory response and produces an increase in the regeneration and remodeling phases. It was shown to produce a wide range of effects on cells and tissue.

Throughout history, dating back to the torpedo fish in the 1600s, to the use of a charged gold leaf, electrical stimulation has been used in the treatment of wounds. The most commonly used stimulations are direct and high voltage pulsed current. Both the cathode and anode effect wound healing. Most phases of wound healing are affected.

This independent study reviewed wound repair physiology and selected physical therapy modalities used to accelerate repair processes. The author does not recommend one modality over another. There is ongoing research with these modalities. Many research studies offer differing treatment parameters based upon the affects desired in the research. As with most treatment, there can be tissue damage if the modalities are not used properly. Health professionals should examine the effects, benefits and consequences of each therapy administered. Care to the patient should be optimal.
REFERENCES


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