



Newfoundland Labrador

Skin and Wound Care Manual





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July 2008

An initiative undertaken by the Skin and Wound Care Specialists of the Regional Health Care Authorities

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Section 1.0

INTRODUCTION & BACKGROUND

Skin and Wound Care is a major health care concern that affects many individuals with different types of wounds and consumes vast resources. Wounds have varying effects on the quality of life of those affected, their families and caregivers. Providing skin and wound care is a major common consideration in the day to day caring of patients with wounds whether in acute, long term or community based environment.

The Provincial Skin and Wound Care Manual will:

- Provide a full understanding of the wound healing process and how this affects patients general state of health.
- Identify risk factors affecting the wound healing and delaying process.
- Focus and apply the wound care principles based on evidence best practices.
- Identify/adapt strategies/measures in preventing wound re-occurrences.
- Increase knowledge on building technical skills about wound assessment and documentation.
- Familiarize with the current, innovative wound care technology.
- Select and apply the appropriate products pertinent to all types of wounds.
- Promote ongoing wound care education programs.
- Gain self-empowerment.

For the past two decades, many changes have occurred in the art of science on how wounds are managed. There has been great advancement in wound technology, research and development of sound policies and standards of care based on research and clinical evidence to achieve positive outcomes in wound healing.

Effective skin and wound management is built upon on how the clinician understands the processes of wound healing and is able to incorporate the knowledge and theory into practice. The clinician should also have the ability to identify and interpret when the wounds are failing to heal.

Successful wound management greatly depends on the collaboration and the integration of an intermultidisciplinary health care team approach. Having the coordinated approach can improve positive outcome through education, research, standardization and positively create a consistent delivery of evidence-based practices in wound care.

This manual is intended not only for the clinician involved in skin and wound care and other health care professionals, but for anyone who has the interest and passion in wound caring.

This manual will be an invaluable resource to all involved in skin and wound care.

For purposes of this Provincial Skin and Wound Care Manual, the term "clinician" is used to describe any health professional who is involved with providing wound care to a client.

Section 2.0

WOUND HEALING

In Section 2, the reader is introduced to the topics of skin and wound healing. The normal phases of wound healing are described. Factors that may interfere with the wound healing process are also discussed.

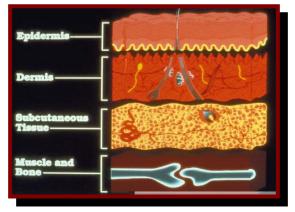
Wound healing is a complex series of events that begin when an individual develops a wound. Regardless of the nature of a wound, the same healing steps occur. A wound moves through a series of phases as it heals and the clinician's role is to support the wound healing process through proper assessment and treatment.

2.1 SKIN

2.1.1 Anatomy & Physiology of Skin

Skin is the largest and most visible organ of the body, comprising up to 15-20% of the total body weight. It receives approximately one third of the body's blood supply at a rate of 300 mls/minute.

Normal skin is composed of two layers: epidermis and dermis. Under the dermis lies the subcutaneous tissue (or hypodermis), a layer of loose connective tissue.



• Epidermis

- Thin, avascular, stratified, outermost layer of the skin. Ranges in thickness from 0.04 mm (eyelids) 1.6 mm (soles of the feet and hands).
- Composed of five layers of epithelial cells (squamous cells) each gradually differentiating into the next layer such that the final layer consists of dead, hardened, mature cells which are eventually shed as a result of external friction.
- The process of differentiation or regeneration of the epidermal layer is stimulated by growth factors. It takes approximately 3-4 weeks.
- The primary cells in this layer are Keratinocytes (protein producing cells) and Melanocytes (color producing cells).
- The primary functions of this layer are Protection and Regulation.

• Basement Membrane Zone

- Separates the epidermis from the dermis. Sometimes called the epidermal-dermal junction.
- Dermal papillae called rete pegs lock the epidermis and dermis together in a tongue and groove fashion.
- Consists of proteins, fibronectin (aids in water retention and adhesion of healing elements) and non-fiber forming collagen (adds thickness to the skin).
- o Anchors the epidermal appendages e.g. sweat glands & hair follicles.

• Dermis

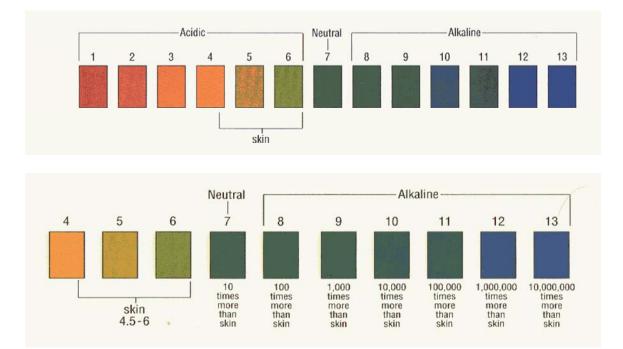
- o Is the layer between the Epidermis and the Subcutaneous Tissue.
- Ranges in thickness from 1 mm 4 mm. The most dense area is on the back.
- Contains blood vessels, nerve fibers, lymphocytes, sweat and sebaceous glands, and hair follicles.
- Main proteins present are collagen (which provides strength) and elastin (which is responsible for skin's ability to recoil back into shape).
- Contains the cells primarily responsible for wound healing: fibroblasts (secrete collagen), mast cells (initiate the inflammatory response), and lymphocytes (protect by controlling microbial invasion). The primary functions of this layer are strength, nutrition and structural support.

• Subcutaneous Tissue

- Supports and anchors the Epidermis and Dermis to the underlying body tissue.
- o Sometimes called the Hypodermis.
- Age, heredity, and nutritional intake influence the thickness of this layer.
- Consists of connective tissue and adipose tissue. Stores approximately 50% of the body's fat supply.
- The primary functions of this layer are protection, insulation, energy, and body shape.

• pH of Skin

The normal skin pH is 5.5. pH is a chemical symbol for the measurement of hydrogen ions in solution or the measurement of the acid-alkali units in a given substance. All of nature's and human chemicals are either acidic, alkaline or neutral. A skin pH of 5.5 means that the skin is acidic. If skin comes in contact something that has a pH outside of its normal range, the potential for harm to the skin is present. The closer something gets to a pH of 1 or a pH of 13, the more acidic or alkaline it becomes and it has the potential to cause serious burns to skin.



2.1.2. Functions of the Skin

The skin has six major functions. They are protection, thermoregulation, elimination of waste products, synthesis of Vitamin D, sensation and communication.

- Protection the epidermis acts as a barrier to protect underlying tissue from mechanical injury, dehydration and the effects of harmful substances. It also prevents many disease causing organisms from entering the body.
- 2. Thermoregulation capillaries in the dermis dilate and constrict in response to heat and cold. This process results in increased or decreased blood flow to the skin leading to a greater or lesser loss of body heat.
- 3. Elimination of Waste Products (Excretion) cellular waste products are excreted via the sweat glands.
- 4. Synthesis of Vitamin D Vitamin D is synthesized by the skin in the presence of ultraviolet radiation from the sun.

- 5. **Sensation** nerve endings that enter through the dermis provide skin sensations of pain, cold, heat, touch and pressure.
- 6. Communication skin serves as an organ of communication and identification. The skin over the face is important for identification of a person and plays a role in internal and external assessments of beauty. Scarring can affect self-image. Facial skin and underlying muscles are capable of expressions of smiling, frowning, and pouting.

2.1.3 Physiological Changes in Aging Skin

There are numerous changes to aging skin. They include:

- fewer active basal cells in the epidermis resulting in a thinning of the epidermis.
- slower cell turnover, resulting in slower healing.
- fewer Langherhan cells, which result in a delayed inflammatory response.
- tactile sensitivity lessens and patients experience reduced acuity of pain perception.

Major visible changes are also evident. They include:

- wrinkles, lines and sagging caused by loss of subcutaneous fat.
- pressure dissipation is reduced because of loss of subcutaneous fat.
- bruising circulation in skin is less efficient. Vascular walls become thinner and fragile and the skin bruises more easily.
- liver or age spots melanocytes are lost at a rate of 20% per decade after the age of 30. Those left behind clump to form irregular aggregates.
- skin tears dermo-epidermal junction is flattened, therefore the two layers become less secure.

2.2 TISSUE INJURY

2.2.1 Types of Tissue Injury

Type of Injury	Irritant
Environmental	Wind Temperature Irregularities Humidity Sunlight
Mechanical	Friction Shear Pressure Epidermal Stripping
Chemical	Fecal Incontinence Harsh solutions Drainage

Many factors are known to damage skin. Injuries can occur from environmental, mechanical, and chemical irritants. The impacts of these irritants are outlined below.

• Environmental Injuries

- *Wind* wind can have an excessive drying effect on the skin, leaving it more at risk of breakdown.
- *Temperature Irregularities* excessive cold can cause injury to skin in the form of frost nip or frostbite. A wind-chill temperature of -22 C can cause frostbite in 15 minutes.
- Humidity excessive moisture on the skin can cause alteration in the pH balance of the skin and can cause maceration, leaving the skin at risk of breakdown.
- Sunlight sunburn can result from even a short-term exposure to ultraviolet radiation (UVR). Exposure to UVR can lead to skin cancer, sunburn (first or second-degree burns), compromised immunity, and longterm skin damage.
- Mechanical Injuries
 - Friction Friction alone can also cause skin breakdown from two surfaces rubbing together. This can be two body parts rubbing together or from a body part, such as a heel rubbing on a mattress. The skin has the appearance of an abrasion or a blister. Common sites include the heels and elbows.

- Shear force This is created by the interaction of both gravity and friction (resistance) against the surface of the skin. Friction is always present when shear force is present. Shear force injuries cause significantly more devastating results because the damage usually starts at the level of the bone or deep tissue and then becomes evident later on the surface of the skin. A classic example of shearing occurs when a patient is sitting in a semi-fowlers position. As the patient slides downward towards the foot of the bed, the bed surface generates enough resistance that the skin over the sacrum remains in the same location. Ultimately, the skin is held in place while the skeletal structure pulls the body toward the foot of the bed. The blood vessels in the area are stretched and angulated and such changes may create small vessel thrombus and tissue death. This often occurs when the head of the bed is too high, above 30°, or when a patient is transferred using improper transferring techniques.
- *Pressure* the most common form of mechanical damage. When externally applied pressure exceeds capillary closing pressure, capillary occlusion occurs. Capillary closing pressure (also known as critical closing pressure) describes the minimal amount of pressure required to collapse a capillary. Capillary pressure, usually described as between 12 32 mm Hg, is exceeded with pressure and cellular damage occurs. With unrelieved pressure, tissue ischemia develops and metabolic wastes accumulate in the interstitial tissue. Anoxia and cellular death is the result. Prevention is the key.
- *Epidermal Stripping* is the inadvertent removal of the epidermis, with
 or without the dermis by mechanical means. It is often caused by frequent
 or careless tape application or removal, or it can also be caused by blunt
 trauma. It can be prevented by 1) recognition of fragile, vulnerable skin, 2)
 appropriate application and removal of tape, 3) use of solid-wafer skin
 barriers or skin sealants under adhesives, 4) use of porous tapes, and 5)
 avoidance of unnecessary tapes.

• Chemical Injuries

The presence of chemical irritants can cause a reaction known as irritant contact dermatitis. Skin damage may be evident in only a few hours in the presence of a strong irritant (such as diarrhea). Chemical dermatitis can be distinguished from an allergic reaction by its irregular borders and always requires the presence of drainage or chemicals. Subjectively chemical dermatitis is very uncomfortable for the patient because of the shallow (epidermal and dermal) nature of the lesions.

o *Fecal incontinence* - feces contains enzymes that are damaging to skin.

- Harsh solutions (for example, betadine, hydrogen peroxide, alcohol and salvodil) - cause chemical irritation by destroying or eroding the epidermis.
- *Drainage around percutaneous tubes, drains or catheters* the pH of drainage can erode the epidermis.

2.2.2 Nursing Strategies to Prevent Skin Problems

There are many nursing interventions that can be implemented to prevent skin damage and ulcer development.

- Avoid placing the head of the bed higher than 30°.
- Implement a regular turning schedule if the patient cannot change their own position.
- Change patient's position frequently. Only small, incremental changes to position are required to relieve pressure.
- Avoid dragging the skin across the bed or chair surface.
- Use lift sheets to assist with position changes.
- Use pressure relief or reduction surfaces.
- Use proper tape application and removal techniques. Anchor skin while removing tape. Use as little tape as possible.
- Assess skin daily.
- Cleanse skin, using gentle, pH balanced cleansers. Avoid overwashing skin. Ordinary washing can require 45 minutes to return the skin to the normal pH of 5.5. Prolonged exposure to soap can require 19 hours to return the skin to the normal pH of 5.5.
- Use moisture-barrier ointments to protect skin.
- Use caution with use of foot stools as pressure may occur to the ischial tuberosities.

2.2.3 Physiology of Wound Healing

There are four phases of normal wound healing. They are:

- 1. Hemostasis
- 2. Inflammatory Phase
- 3. Proliferative Phase (comprised of granulation and epithelialization)
- 4. Maturation Phase (also called reconstruction or remodeling phase)

• Hemostasis

Hemostasis begins immediately upon wounding. The body's natural defenses try to control bleeding first by constricting the local blood vessels, and then by creating a plug with circulating platelets. This temporary plug is later replaced by a more durable fibrin clot. This process is quick, occurring over several hours.

Inflammatory Phase



Inflammation is commonly referred to as the clean-up period. White blood cells (neutrophils and macrophages) invade the wound. Dead tissue, debris, and bacteria are first digested by these cells. Growth factors and other chemical messengers are then released. This starts the healing process.

Proliferative Phase

The process of "new" tissue growth or proliferation is subdivided into two phases depending upon the depth of injury: Granulation and Epithelialization.

o Granulation



All partial and full thickness wounds heal by the process of granulation. The epidermal layer has been destroyed so the natural healing process originates from dermal cells (Fibroblasts) in the wound bed and periwound margins. A new layer of protein

(Collagen) is deposited in the wound space. Because of the extent of the damage new blood vessel growth (angiogenesis - Endothelial cells) is required to bring the needed nutrients for healing to the area. Granulation will usually begin within 12 - 48 hours after the initial injury when hemostasis is complete and the inflammatory phase has subsided. This process can be very long, occurring over several months for full thickness wounds. However, only a minimal amount of granulation or the growth of scar tissue is required to fill a partial thickness wound, thus the granulation phase will be much shorter. Granulation, also called scar tissue, is relatively a vascular and is thus different in texture, appearance, and functions of normal skin. As the wound fills in with new tissue, the edges contract with the aid of specialized cells (<u>Mylofibroblasts</u>), after which epithelialization will commence to resurface the wound.

o Epithelialization

Superficial wounds heal by Epithelial Regeneration. The natural process of epidermal cell keratinocytes growth and differentiation will result in the resurfacing of the wound with natural skin. The growth originates from keratinocytes in the wound bed, periwound margins, and from islets of



epidermal cells (e.g. hair follicles, sweat glands) that remain scattered in the wound tissue. Regeneration will usually begin within 12 - 24 hours after the initial injury, when hemostasis is complete and the inflammatory phase has subsided. Because the damage is not too extensive the wound will regain near normal appearance and strength. The process is usually complete in 3 - 4 weeks.

Maturation Phase



The maturation phase, also known as reconstruction or remodeling, may take up to two years to complete. Newly formed scar tissue realigns its internal structure to increase its durability. The collagen deposits bundle up to increase the tensile strength of the wound. New tissue is quite fragile at this point in time and can be reinjured easily. The healed wound will only

regain up to 80% of its original strength.

2.2.4 Types of Wound Healing

Types of Healing	Description
Primary	In primary closure, such as with a surgical incision, wound edges are pulled together and approximated with sutures, staples, or adhesive tapes, and healing occurs mainly by connective tissue deposition. Epithelial migration is short- lived and may be completed within 72 hours. Within 24 - 48 hours, epithelial cells migrate from the wound edges in a linear movement along the cut margins of the dermis.
Secondary	In wounds that heal by secondary intention, wound edges are not approximated, and healing occurs by granulation tissue formation and contraction of the wound edges.
Tertiary	Wounds healing by tertiary intention (delayed primary intention). The wound is kept open for several days and the superficial wound edges are then approximated, and the center of the wound heals by granulation tissue formation.

2.2.5 Factors Affecting Wound Healing

Many factors affect wound healing. The clinician's role is to assess these factors and intervene or suggest to patients interventions or modifications that may assist in wound prevention and wound healing.

Factors Affecting the Health of Skin and Wound Healing

- Smoking 80 90% of people who have Peripheral Arterial Disease (PAD) report a history of tobacco use. Nicotine and its primary metabolite, cotinine, have serious effects on Endothelial injury, arthromatous lesion growth, smooth muscle tone and blood viscosity. The nicotine absorbed from cigarette smoking causes the peripheral blood flow to be depressed by at least 50% for more than an hour after smoking just one cigarette. Carbon monoxide binds to haemoglobin in place of oxygen, significantly reducing the amount of circulating oxygen, which can impede healing.
- *Stress* Stimulates the nervous systems to vasaconstrict peripheral blood vessels which ultimately can decrease tissue perfusion. Stress also increases the amount of circulating natural steroids that can decrease the inflammatory response and slow the growth of fibroblasts and keratinocytes.
- *Hypertension* in particular systolic hypertension is the second most predictive risk factor for PAD.

- Elevated Cholesterol levels, especially elevated LDLs, are thought to be an important risk factor for the development of atherolsclerosis and PAD.
- Metabolic Disorder A number of metabolic disorders can impair wound healing capacity. For example, diabetes directly affects the body's supply of glucose, status of peripheral circulatory vessels, and peripheral sensation that can influence awareness of injury or complications. High glucose levels reduce collagen synthesis and bundling processes. Renal disease can result in an accumulation or deficiency of metabolic by-products. Bowel disorders can interfere with nutritional absorption. Other diseases such as COPD, PVD, CHF, and Hypovolemia are all examples of disease states which can result in a decrease in the supply of oxygen to wounded tissue.
- Medications such as steroids can reduce the inflammatory response and suppress granulation. Chemotherapy and radiotherapy can effect the integrity of the adjacent cells which play an important role in proliferation. These treatments can also deplete essential immunologic agents, energy and oxygen sources including RBCs. Vasoconstrictors can limit the amount of circulatory volume available to healing tissue.
- Nutrition Normal healthy skin integrity is promoted by adequate dietary intake of protein, carbohydrate, fats, vitamins, and minerals. If skin becomes damaged, an increased dietary intake of some substances, such as Vitamin C, for collagen formation may be indicated and beneficial. Refer to Section 3 for more detailed information on nutrition.
- Surgery Certain anaesthetic agents cause vasodilatation that restricts the skin's natural ability to alter the diameter of peripheral blood vessels thus controlling thermoregulation. As a consequence, excess amounts of body heat can evaporate. Post operatively these clients can go into a phase of excess shivering. This reduction in body heat may influence healing. The use of warm blankets is critical to limit the amount of heat loss.
- Advanced Age A number of changes occur in the elderly that may limit healing potential. For example, there is a decrease in fibroblasts that are directly responsible for collagen deposition or new tissue growth. There also tends to be a decrease in the intake of nutrients and fluids. Concurrent diseases of the respiratory and circulatory systems that can limit tissue perfusion, are also common.
- Alcoholism Can impair liver functioning subsequently altering the production of protein and other essential elements needed for tissue repair.

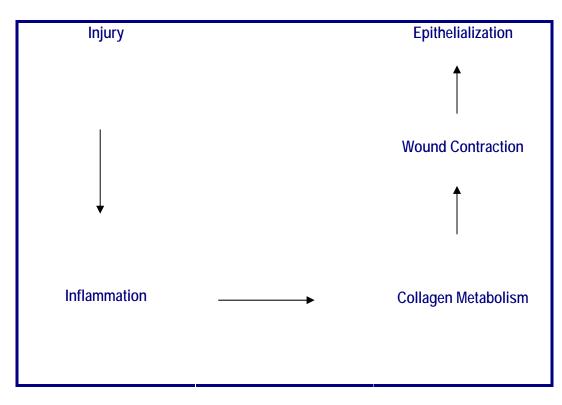
Section 3.0

NUTRITIONAL FACTORS IN WOUND MANAGEMENT

Prevention and treatment of nutritional deficiencies are critical in maintaining skin integrity, promoting tissue restoration and reducing wound complications. Malnourished patients are at greater risk for complications including longer length of stay and more infections leading to increased health care costs. Any strategy for wound management will not be effective unless nutritional deficiencies are corrected. A complete nutrition assessment by a registered dietitian should be an integral part of the evaluation of patients identified at risk for skin break down as well as those with existing wounds.

3.1 Biology of Wound Healing

Wound healing and tissue repair go through complex, multi-step processes, which include inflammation, collagen metabolism, wound contraction and epithelialization. An understanding of the nutrient utilization in each of these steps is helpful while assessing patients with wound complications and establishing care plans.



All tissue injury triggers an inflammatory response. Tissue destruction and vascular changes results in clotting and the release of platelet mediators. Small blood vessels dilate, capillary

permeability increases and leukocytes and macrophages ingest necrotic material and bacteria, fighting infection and preparing the area for new tissue growth.

Collagen metabolism provides tensile strength and integrity to healing wounds. Collagen formation is stimulated by fibroblasts the formation of which requires the specific enzyme lysyl and prolyl hydroxylase. The activity of these enzymes requires oxygen, Vitamin C and iron as co-factors. The glycosylation of collagen occurs through O-lysyl glactosyltranferase. This enzyme requires manganese as a co factor.

Once the basic collagen chains are in place they must be cross-linked, a process which is critical for the strength and elasticity of tissue. The final cross linking of the collagen requires the enzyme lysyl oxidase and requires copper as a co-factor.

Wound contraction, which helps to bring the dermal structures together is another important step in the spontaneous closure of wounds. Unless contraction occurs, the granulation surface is covered by a layer of epithelial cells that cannot provide adequate strength and integrity.

Epithelialization results from epithelial cells propagation and migration. The final result is the restoration of a barrier function similar to normal skin. A moist environment and oxygen are essential for epithelialization to take place.

3.1.1 Specific Nutritional Requirements

The complex process of wound healing involves numerous synthesis and energy consuming reactions, and requires optimal nutrient supply, adequate oxygenation and blood flow. The table, "Role of Specific Nutrients in Wound Healing" summarizes the functions of various nutrients in tissue repair and wound healing.

• Protein

Ongoing protein synthesis is essential to maintain tissue integrity. Protein and amino acids are essential for cell multiplication and protein synthesis including synthesis of enzymes involved in the healing process. Protein depletion impairs the immune system by altering antibody response time and decreasing resistance to infection. Lack of protein leads to hypoalbuminemia and to interstitial edema, which retards cellular exchange of nutrients and decreases skin integrity and resiliency, making it more susceptible to injury.

• Carbohydrates and Fats

Carbohydrates and fats are sources of cellular energy. Glucose, the simplest form of carbohydrate is the preferred fuel for wound repair. It serves as a source of energy for many tissues including leukocytes, fibroblasts cells and macrophages. When glucose is not available for cellular function, the body catabolizes protein and fat to meet energy requirements. Glucose meets the metabolic demand for wound healing and preserves the body's structural and functional protein.

Fatty acids are needed for cell membranes and deficiencies may impair wound healing. Unsaturated fatty acids such as linoleic and arachidonic are essential components of triglycerides and phospholipids, substrates that form cell membranes. Fatty acids are needed for the formation of prostaglandins and other regulators of the immune and inflammatory processes.

• Vitamins and Minerals

o Vitamin A

Vitamin A is required for maintaining the normal humoral defense mechanism and for limiting complications associated with wound infections. It promotes fibroplasia and collagen accumulation, hastens the healing process and enhances the tensile strength. Vitamin A has also been used to counteract the catabolic effect that steroids have on wound healing.

o Vitamin C

Vitamin C has an important function in wound healing. It functions as a cofactor in the hydroxylation of proline in the formation of collagen. It has also been shown to enhance the cellular and humoral response to stress. In Vitamin C deficiency old wounds may reopen due to loss of tensile strength and degeneration of the extra-cellular matrix.

o Vitamin K

Vitamin K is required for the hepatic synthesis of clotting factors II, VII, IX and X. Vitamin K deficiency can cause excessive bleeding and hematoma formation and predisposes wounds to infection and wound complications.

o B-Complex Vitamins

B complex vitamins are cofactors in enzyme systems and are essential for the protein, carbohydrate and fat metabolism and hence they are implied in the wound repair system. B-complex vitamin deficiency, particularly pyridoxine, pantothenic acid and folic acid results in suppressed antibody formation and leukocyte function predisposing individuals to infection and poor wound healing. Thiamin (B₁) deficiency could affect collagen synthesis. Alvarez et al. has postulated that thiamine has an important role at a cellular level related to energy metabolism in rapidly proliferating cells that secrete collagen.

o Zinc

Zinc has been identified in numerous enzyme systems that are responsible for cellular proliferation (e.g. the proliferation of inflammatory cells, epithelial cells, and fibroblasts). It is a component of biomembrane and is necessary for RNA, DNA and ribosome stabilization. Zinc deficiency impairs wound healing by reducing the rate of epithelialization, reducing wound strength and diminishing collagen synthesis.

o *Iron*

Iron is necessary for the hydroxylation of lysine and proline in the formation of collagen. Anemia could lead to hypovolemia and tissue hypoxia. Poor blood supply and low tissue oxygenation could lead to depressed inflammatory response, bacterial infection and delayed wound healing.

o Copper and Manganese

Copper is an integral part of the enzyme lysl oxidase that catalyzes the formation of stable collagen crosslinks. Manganese activates specific enzymes responsible for the glycosylation of procollagen molecules and synthesis of proteoglycans. Deficiencies of these minerals could cause altered collagen formation and delayed wound healing.

Nutrient	Function	Deficiency Effect
Protein/ Amino acids	 Required for cellular synthesis and cell proliferation Maintain tissue integrity Substrate for ptoteoglycan/glycoprotein Antibody production, resistance to infection Formation of granulation tissue/fibroblastic proliferation Collagen metabolism Several amino acids are enzymatic cofactors in healing 	 Delayed healing, decreased fibroblast proliferation, and collagen synthesis Impaired immune response Decreased skin elasticity and resilency making it susceptible to injury Leads to hypoalbuminemia, interstitial edema retarding cellular exchange of nutrients
Carbohydrate	 Energy source for tissues Essential for white blood cell function 	 Decreased energy for cellular metabolism causing protein breakdown for energy rather than wound repair Altered white blood cell function
Fat	 Membrane synthesis and proliferation 	Decreased tissue repair
Vitamin A	 Enhance fibroplasia and collagen synthesis Maintain normal humoral mechanism Counteract effects of steroids by reversing the effect on lysosomal membrane 	 Decreased collagen synthesis Decreased ability to prevent infection Decreased ability to counteract the negative effect of steroids
Vitamin C	 Cofactor in the hydroxylation of proline in collagen Enhance cellular and humoral response 	Altered collagen formation, delayed healing
Thiamine	Energy metabolism related to cell proliferation	Decreased cell proliferation and collagen metabolism
Vitamin K	Synthesis of clotting factors	Bleeding, hematoma and wound disruption
Iron	Enzyme cofactors in collagen metabolism	Anemia, hypoxia and hypovolemia
Copper, Mn	Enzyme cofactors in collagen metabolism	Altered collagen formation
Water	 Moist environment, electrolyte balance, faster epidermal cell migration 	Tissue breakdown, decreased tissue perfusion, volume depletion
Zinc	 Cofactor for the enzyme responsible for cellular proliferation Transcription of RNA 	 Decrease in enzyme production Altered cell replication

3.1.2 Hydration Status

Wound healing occurs more rapidly when dehydration is prevented. Adequate hydration is critical for electrolyte balance and for maintaining optimal intravascular volume and local tissue perfusion. It has been reported that epidermal cells migrate faster and cover the wound surface sooner in a moist environment. Appropriate fluid supplementation and preventing over-hydration and dehydration are critical in wound healing

3.1.3 Nutritional Screening/Assessment

Wound complications could be best prevented by use of risk assessment tools, which should not only focus on treatment of existing wounds, but also on the prevention of skin break down. When risk assessments are being performed, attention should be paid to those factors that could compromise skin integrity as well as nutritional deficits/imbalances that might negatively affect wound management. Several risk assessment tools such as Norton and Braden scales with nutrition screening components are recommended. However these scales are based only on present dietary intake and cannot determine the extent of malnutrition. Ideally, when risk assessments are completed, every patient must be individually assessed for co-existing risk factors for wound complications and malnutrition. A complete nutrition assessment should be an integral part of the evaluation of the patients identified at risk as well as those with existing wounds. Appropriate and timely referral/consultation would enable dietitians to initiate nutrition support.

3.2 Risk Factors for Malnutrition and Wound Complications

3.2.1 Admission Diagnoses

Patients admitted for neurological deficits, GI bleeding or obstruction, pulmonary, renal or hepatic failure and sepsis from pneumonia or urinary sources should be considered at nutritional risk. Poorly controlled diabetes could also lead to impaired wound healing and poor wound outcome. Certain treatments such as steroids may slow the rate of epithelialization, neovascularization and could decrease collagen deposition and tensile strength of the wound. In the presence of complex injuries involving different tissues and organs, infection, sepsis, and trauma there is increased energy and protein needs; in these circumstances if dietary intake is inadequate impaired healing could occur. Prolonged immobility secondary to fractures or decreased mental status places patients, particularly elderly patients, at greater risk of tissue breakdown and subsequent increase in morbidity and mortality.

Wagener has categorized the following common admission diagnoses as risk factors for wound complications and malnutrition.

Neurologic	Hepatobiliary / GI
CVA	GI Bleed
Dementia	Obstruction
Head Injury	Hepatic failure
Cord Injury/Paralysis	Pancreatitis
	Malabsorption/Diarrhea
Cardiopulmonary	Musculoskeletal
COPD/CHF	Fractures
Respiratory Failure	
Severe Cardiac Disease	
Other	
Sepsis	
Trauma	
Diabetes	
Systemic Steroids	
Connective Tissue Disease)

3.2.2 Weight Status

Body weight is one of the most commonly used and cost effective anthropometric indicators of nutritional status. Patients who had self reported unintentional weight loss of more than 10 pounds within 6 months prior to admission for elective surgery had significantly increased mortality rates compared to patients who did not lose weight. Unintentional weight loss may be associated with factors such as age and poor health and may increase mortality rate. It has also been reported that patients with decreased body weight are at increased risk of developing pressure sores. Blackburn et al. has defined significant weight loss as loss of more than 10% of usual weight over 6 months or less (See Table, Evaluation of Weight Change).

Evaluation of Weight Change		
Time	Significant Weight Loss	Severe Weight loss
1 week	1-2%	Greater than 2%
1 month	5%	Greater than 5%
3 months	7.5%	Greater than 7.5%
6 months	10%	Greater than 10%

Many factors could influence weight measurements. Rapid increase or decrease in weight status signal wide fluctuations in hydration status. The cause of these changes should be investigated. Gain or loss of more than 0.5 kg over 24 hours in the absence of significant fluid shifts may indicate measurement error and should be rechecked.

3.2.3 Biochemical Indicators

o Albumin

Serum albumin, an indicator of visceral protein status, is widely used in nutrition screening. This protein maintains plasma oncotic pressure, transports metabolites, enzymes, drugs, hormones and metals in the blood stream. Although serum albumin is subject to fluctuations in fluid status, it still remains a cost effective/sensitive indicator of changes in clinical status such as infection, hydration and starvation. Seltzer et al. has reported a fourfold increase in morbidity and six fold increase in mortality in patients with serum albumin concentrations less than 25 g/L.

The predictive value of decreased serum albumin concentration for wound related complications is well documented. Normal serum albumin levels range from 35-50 g/L. Blackburn et al. has classified serum albumin level 21-30 g/L as moderate malnutrition and less than 21 g/L as severe malnutrition. When screening for pressure ulcers, patients with serum albumin less than 25 g/L should be categorized at high risk.

o Transferrin

Transferrin, an iron binding protein, is frequently used in assessing nutritional status. It is more sensitive and specific than albumin due to its smaller serum pool and shorter half-life of 8-10 days. Kuvshinoff et al., in a retrospective study, found that serum transferrin measurements predicted spontaneous fistula closure. Albina had reported serum transferrin levels less than150 mg/dL (1.5 g/L) had significant predictive value for delayed healing or wound infections. Conditions such as iron deficiency anemia, transfusions, massive blood loss, chronic infections or inflammation could affect transferrin levels. Consideration should be given to these factors while using serum transferrin is present in the serum at a concentration of 2.12-3.66 g/L. Blakburn et al. has

categorized serum transferrin levels 100-150 mg/dL (1-1.5 g/L) as moderate malnutrition and less than 1.0 g/L as severe.

• Total Lymphocyte Count (TLC)

Total lymphocyte count (TLC) has been used as an indirect measure of nutritional and immune status. Pinchkofsky-Devin et al. and Allman et al. have found that lymphopenia (TLC less than 1200 and 1500 cells/ mm or 1.2 - 1.5 x 10 cells) was associated with pressure ulcers. Good prognostic value of lymphocytes has also been reported by Shaver et al. Total lymphocyte count of 800 -1200 mm or 0.8- 1.2 x 10 cells is classified as moderate malnutrition and less than 800 mm or less than 0.8 x 10 cells as severe malnutrition.

o Hemoglobin, Hematocrit and Serum Ferritin

Hemoglobin, hematocrit and serum ferritin have been correlated with the risk of developing pressure ulcers and are good predictors of eventual mortality. Since chronic anemia is common among patients with pressure ulcers, Wagner has recommended testing the triad of hemoglobin, hematocrit and ferritin to differentiate between anemia of chronic disease and true iron deficiency anemia. Determination of vitamin B₁₂ and folate levels could then be carried out to diagnose other common nutritional anemias.

o Serum cholesterol

Although serum cholesterol has received little attention as an assessment parameter for malnutrition, decreased serum cholesterol (less than 4.14 mmol/L) has been associated with measurable mortality and poor outcomes in older adults. Association between low cholesterol and decreased levels of membrane phospholipids has been reported. The decrease in membrane phospholipids will affect cellular function resilience and skin integrity.

3.3 Risk Assessment Tool

The Provincial Wound Management Clinical Nutrition Working Committee endorses the Braden Scale Nursing Policy to generate timely consults to dietitians to ensure therapeutic nutritional intervention is commenced. Since Braden Scale is intended for predicting pressure sore risk and not for identifying patients at high risk for delayed wound healing/wound complications, the committee recommends routine screening of pre/post surgical patients. The Committee recommends using one or more of the following criteria: (Indicative of somatic/visceral protein status immune status and hydration status) to generate dietitian consults for surgical patients.

Evaluation of Nutritional Indicators		
Serum Albumin	Less than 30	
Prealbumin	Less than .17	
Recent Weight Loss	Greater than 7.5% in 3 months	
Total lympocytes	Less than 1.8 x 10 ³ cells	
Urea	Greater than 7	

3.4 Therapeutic Plan

Provision of a diet that is complete in nutrient requirements provides the optimum environment for recovery and healing. Patients should be assessed by a dietitian to ensure that nutritional requirements are being met and to determine the need for nutrition supplements.

3.4.1 Nutrition Protocol of Pressure Ulcer Healing

The Provincial Wound Management Clinical Nutrition Working Committee recommends Jackob's Nutrition Protocol (Refer to Section 3 – Appendix 1) as a standard guide for nutrition intervention acknowledging that care plans need to be personalized based on individual assessments.

Energy, Protein and Fluid Requirements

Dietitians should use their clinical judgment to determine appropriate body mass when calculating energy, protein and fluid requirements.

o Energy Requirements

The first step in appropriate feeding plan is the calculation of energy and protein requirements. Wolfe et al. has determined that the maximum rate of glucose oxidation could be reached with 35 Kcal/Kg. Wagner recommends a range of 25 - 35 Kcal/Kg for estimating calorie requirements. The agency for Health Care Policy and Research (AHCPR) guideline Treatment of Pressure Ulcers recommend 30 – 35 kcal/Kg for patients with pressure ulcers. It would be prudent to agree with Wagner who suggests that elderly patients who are relatively unstressed as well as young unstressed malnourished patients generally fall at the lower end of the range and traumatized, septic, stressed patients fall at the higher end of the scale. Wagner also recommends Harris-Benedict formula to calculate energy requirements and to take into consideration adequate "injury factors" such as infections, fractures and open wounds that will increase the estimations of energy expenditure.

The Committee recommends using the energy requirements suggested by Jackob's protocol as a standard and guide (25-30 Calories/kg for stage I and Stage II wounds, 30 to 40 Calories/kg for Stage III and Stage 1V wounds).

o Protein Requirements

The Recommended Dietary Allowance (RDA) for protein based on 0.8 g/kg, is insufficient for patients with wounds. As the stress factor increases with injury, infection or open wounds, intake of protein should increase. AHCPR guidelines (36) recommend a protein intake of 1.25 to 1.5 g/kg, but acknowledge that some patients may need more protein depending on the type of wound and goals of care. Wagner recommends a protein intake of up to 2.5 g/Kg to avoid negative nitrogen balance. Chernoff et al. have documented that provision of a very high protein formula (up to 2.9 g/kg) was associated with improved nitrogen balance and improved healing of decubitus ulcers if adequate hydration is provided.

The Committee recommends using Jackob's protocol as a guide to determine protein requirements of patients with wounds (1 gm/kg for Stage I, 1.2 gm/kg for Stage II, and 1.5gm/kg for Stage III and 1V). However, protein requirements should be assessed individually for patients with compromised renal function e.g. patients with renal insufficiency versus patients on hemodialysis.

o Fluid Requirements

A general guideline to determine fluid requirement is to provide 1 mL fluid /kcal energy consumed with a minimum requirement of 1500 ml/day. When assessing fluid requirement, patients medical conditions such as cardiac status, renal function and exudative losses from open wounds should be taken into consideration. Patients on air fluidized beds need to increase their fluid intake to another 10-15 mL /kg of body weight above those recommended to prevent dehydration.

The Committee recommends using the fluid requirements suggested by Jackob's protocol, (25-30 cc/kg for Stage1 and Stage II wounds, 30–35 cc/kg for Stage III wounds and 30-40 cc/kg for Stage 1V wounds), except for patients on air fluidized beds.

o Vitamins and Minerals

When assessing vitamin/mineral supplementation, patients medical conditions such as renal status, gastrointestinal function and altered immune status should be considered.

Three nutrients frequently associated with wound healing are Vitamin C, Vitamin A and zinc. The AHCPR Guidelines recommend a vitamin mineral supplement if deficiencies are confirmed or suspected. When specific deficiencies are diagnosed, individual supplements of up to 10 times the

Recommended Daily Allowance for water-soluble vitamins may have to be added to the patient's daily dietary intake.

The recommended dietary allowance (RDA) for Vitamin C is 90 mg and 75 mg respectively for males and females above 19 years. This amount is easily achieved from dietary sources such as citrus fruits, green peppers and tomatoes. However, it has been reported that a significant proportion of elderly have low plasma and leukocyte ascorbate concentrations, as do smokers and patients with liver disease. It has also been shown that the Vitamin C status of hospitalized patients deteriorates during their hospital stay. A combination of injury or surgery with preexisting marginal Vitamin C status could detrimentally affect wound healing and supplementation of the diet of these patients may be beneficial. Although the exact amount supplementation differs, Dickerson reports dosages of 500 to 1000 mg per day. Levenson et al. reported that it was only after giving severely burned patients 2 g /day that various biochemical indices approached normal. They recommend that seriously ill and injured patients be given 1 to 2 g of ascorbic acid daily starting promptly and continuing "until convalescence is well advanced (until skin coverage is almost complete in burn patients)".

The Committee recommends caution when providing Vitamin C supplementation to patients with renal failure and kidney stones. For patients without these conditions, the Committee recommends supplementation according to Jackob's Nutrition Protocol (250 –750 mg/day for Stage II and Stage III wounds)

The RDA for zinc for adult males and female is 11 and 8 mg per day respectively. Dietary sources of zinc are seafood, oysters, liver, meat and milk. Zinc losses could occur through GI tract; so patients with diarrhea or high output ostomy or fistula drainage may be at risk of zinc deficiency. Patients with burns or other wound exudate also lose zinc. Typical oral supplementation is 220 mg zinc sulfate or 50 mg elemental zinc. Prolonged intake of more than 150 mg per day has been associated with copper deficiency. This would affect wound healing because copper is a constituent of lysyl oxidase which triggers the final cross linking of the collagen.

The Committee recommends zinc supplementation as per Jackob's Nutrition Protocol (25-50mg of elemental zinc for a period of up to 3 months for Stage II and Stage III wounds).

The RDA for Vitamin A is 900 RE (μ g) per day for adult males and 700 RE (μ g) per day for females. The major contributors of Vitamin A to our diets are liver, fish oils, fortified milk, eggs and dark green and orange vegetables such as carrots, spinach, broccoli, and squash. Levenson et al. have recommended that an administration of 25000 IU (4166 RE) is prudent for patients with severe injuries. Hunt recommends intravenous administration of 10,000 to 15,000 IU (1666 RE-2500 RE) per day for approximately a week to counteract

the tendency of anti-inflammatory steroids to suppress tissue repair. Animal sources of Vitamin A (retinol) are about six times more potent than vegetable sources and can be toxic if taken in excess. Because of this, Vitamin A supplements are restricted to carotenes (vegetable sources of Vitamin A).

The Committee does not recommend additional supplementation of Vitamin A above and beyond the amount that is contained in a multivitamin/mineral supplement.

The Committee reinforces the need for consulting pharmacists and reviewing clinical practice guidelines when implementing vitamin/mineral/oral nutrition supplements to rule out possible drug-nutrient-nutrient interactions (e.g. since iron, zinc and copper compete for common transport sites, patients taking therapeutic dose of 30 mg. iron should consider taking 15 mg zinc and 2 mg copper supplement). The Committee also underscores the importance of the dietitian/physician team exploring new, evidence-based options for wound management (e.g. use of erythropoietin (EPO) that stimulates neovascularizaiton and wound healing).

3.5 Nutritional Outcome Monitoring

The Provincial Skin and Wound Management Committee has developed a Nutritional Outcome Measurement Template (Refer to Section 3 – Appendix 2) to assist dietitians to evaluate nutritional progress of patients with wounds. This template uses a combination of objective and subjective indicators such as weight status, appetite, intake, affect, mobility status and serum albumin. Reviewing and comparing the initial assessment score with the score at a follow-up point will help to monitor/evaluate the nutritional progress of patients with wounds.

3.6 Patient Education Pamphlet

The Committee recommends using the newly developed pamphlet "Nutrition and Wound Healing" (Refer to Section 3 – Appendix 3) as a template for patient education.

3.7 Conclusion

A complete nutrition assessment by a registered dietitian should be an integral part of the evaluation of patients identified at risk for skin break down as well as those with existing wounds. Timely and appropriate nutrition intervention is an essential component of wound management. Optimal patient care must include measures to prevent or minimize metabolic and nutritional disturbances and to treat them promptly when they arise. Nutritional measures should be started early and consistently pursued. Even the best surgical care, wound care products and nursing care will not heal wounds if the nutritional status of a patient is compromised.

Section 3 – Appendix 1

Nutrition Protocol for Pressure Ulcer Healing, Jackobs, 1999

Stage I		Stage II	Stage III	Stage IV	
Diet or tube feeding	Diet or tube feeding as ordered.	Diet or tube feeding as ordered.	Diet or tube feeding as ordered.	Diet or tube feeding as ordered.	
	Assess adequacy of diet, eating environment, dentition, and food preferences.	Evaluate protein and nutrient needs.	Evaluate protein and nutrient needs.	Evaluate protein and nutrient needs.	
Vitamin/mineral supplement	Consider if intake is not adequate.	Recommend a multiple Vitamin And mineral supplement.	Multiple vitamin, mineral supplement, and Vitamin C at 250-750 mg/day and zinc at 25-50 mg/day for up to 3 months.	Multiple vitamin, mineral supplement, and Vitamin C at 250-750 mg/day and zinc at 25-50 mg/day for up to 3 months.	
Protein	1 gm/kg	1.2 gm/kg	1.5 gm/kg	1.5 gm/kg	
Calories	25-30 Calories/kg	25-30 Calories/kg	30-40 Calories/kg	30-40 Calories/kg	
Fluids	25-30 cc/kg	25-30 cc/kg	30-35 cc/kg	30-40 cc/kg	

Taken from: Jackobs, MK. The Cost of Medical Nutrition Therapy in Healing Pressure Ulcers. Topics in Clinical Nutrition. 1999; 14(2): 41-47.

These are guidelines only. The level of supplementation will vary depending upon individual patient assessments. Many facilities have their own supplementation guidelines based on best practice and knowledge to date.

Section 3 – Appendix 2

Nutritional Outcome Measurement Template For Wound Management

Assessment Indicators	Initial Assessment				SCORE	Follow up 1			SCORE	
Recent Weight	Severe Wt loss	Moderate Wt loss	Mild Wt loss	Stable Wt		Severe Wt loss	Moderate Wt loss	Mild Wt loss	Stable Wt	
	1	2	3	4		1	2	3	4	
Appetite	Poor	Fair	Good	Excellent		Poor	Fair	Good	Excellent	
	1	2	3	4		1	2	3	4	
Intake	Poor	Fair	Good	Excellent		Poor	Fair	Good	Excellent	
	1	2	3	4		1	2	3	4	
Mobility	Bedridden	Limited with chair/ walker/ assistance	Adequate with chair/ walker/ assistance	Independent		Bedridden	Limited with chair/ walker/ assistance	Adequate with chair/ walker/ assistance	Independent	
	1	2	3	4		1	2	3	4	
Affect	$\overline{\mathfrak{S}}$		©	\odot		$\overline{\mathbf{S}}$		\odot	Ü	
	1	2	3	4		1	2	3	4	
Weight Status	Underweight/ obese	Underweight/ob ese with no recent weight change	Underweight/ob ese with marginal weight gain or loss	Healthy weight		Underweight/ obese	Underweight/ obese with no recent weight change	Underweight/ obese with marginal weight gain or loss	Healthy weight	
	1	2	3	4		1	2	3	4	
Albumin	Less than 25	25-29	30-32	Greater than 33		Less than 25	25-29	30-32	Greater than 33	
	1	2	3	4		1	2	3	4	
	Total Score						Total Score			

Section 3 – Appendix 3

Nutrition and Wound Healing Pamphlet

See attachment.

How can you get all the nutrients you

need for wound healing?

- Make sure you choose foods from ALL four food groups as listed in Eating Well with *Canada's Food Guide*
- Make sure you drink enough liquids everyday. Have water, juice or milk with your meals and snacks.
- Talk to a health professional if you have:
 - An unplanned weight loss
 - Frequent diarrhea or vomiting
 - Loss of appetite
 - Trouble chewing or swallowing your food
 - Other health problems like diabetes or high cholesterol

IMPORTANT: You should talk to your doctor or a dietitian before taking a vitamin/mineral supplement

Notes

Our vision- to make a positive difference in the health and well being of people in our region

Contact information:

Eastern Health P.O.Box 13122 St, John's, NL A1B 4A4 (709) 752-4800



Nutritiion and Wound Healling



Who needs good nutrition? EVERYONE!

Who needs good nutrition for wound healing? YOU DO!

If you have a wound, the nutrients in food and liquids are very important.

These nutrients play a big role in wound healing:

- Calories
- Protein
- Water/Liquids
- Zinc
- Iron
- Vitamin A
- Vitamin C

Nutrient	Good Sources
Calories	All food and beverages contain calories, except water, coffee, tea and diet beverages
Protein	Beef, pork, chicken, turkey, fish, lamb, eggs, liver, dairy products (milk, cheese, yogurt, pudding), legumes (peas, lentils, beans), seeds and nuts
Water/ Liquids	Water, juice, milk, Jell-O™, sherbet, ice-cream, yogurt, pudding, soup, popsicles and other liquids except caffeinated beverages
Zinc	Seafood (especially oysters), beef, pork, chicken, milk, legumes (peas, lentils, beans), whole wheat pasta, wheat germ and nuts
Iron	Liver, beef, turkey (dark meat), legumes (peas, lentils, beans), baked potato with skin, fortified pasta and cereals
Vitamin A	Liver, milk, cheese, broccoli, frozen green peas, spinach, carrots, red peppers, carrots, red peppers, tomato juice, cantaloupe, mango, apricots and peaches
Vitamin C	Citrus fruits and juices (orange, grapefruit), cranberry juice, strawberries, broccoli, brussel sprouts, red peppers tomatoes, potatoes, cauliflower, melons (honeydew, cantaloupe)

Section 4.0

PRINCIPLES OF WOUND HEALING

In this section, the reader will learn the ten general principles of wound healing. An understanding of these principles will facilitate appropriate wound care.

Pr	Principles of Wound Healing		
1.	Risk Assessment and Prevention		
2.	Wound Assessment		
3.	Wound Cleansing		
4.	Debridement		
5.	Identification & Elimination of Infection		
6.	Elimination of Dead Space		
7.	Absorption of Exudate		
8.	Promotion of Moist Wound Healing		
9.	Provision of Thermal Insulation		
10.	Protection of the Healing Wound		

There are ten commonly accepted principles of wound prevention and healing. By applying each of these principles, the clinician implements appropriate interventions that will facilitate wound healing. By incorporating a holistic approach to the patient, an optimum wound healing environment will be achieved.

4.1 Risk Assessment and Prevention

- Complete Braden Scale Refer to Section 5 of this manual for more information on the Braden Scale for Predicting Pressure Sore Risk.
- Determine the patient's level of risk and implement interventions to prevent development of pressure ulcers.

4.2 Wound Assessment and Documentation Tool

- Complete the Wound Assessment Record when a wound is identified, and then in accordance with regional policies and procedures. The assessment will provide the clinician with the necessary information to implement interventions. This will help direct the appropriate intervention (for eg., wound bed dry add moisture; or, wound is too wet absorb exudate).
- Goals for Wound Assessment:
 - o focus on the clinical status of the wound;
 - o guide the appropriate intervention for the wound;
 - indicate that, if there is no change in wound status within a pre-determined timeframe, re-assess and alter the plan;
 - o monitor and evaluate overall client outcomes (progression or regression); and
 - o determine the effectiveness of treatment.

4.3 Wound Cleansing

- The purpose of wound cleansing is to remove foreign debris and surface contaminants from the wound.
- Cleanse wounds with sterile water, normal saline, or pH balanced wound cleansers.
- Commercial wound cleansers contain surface-active agents to improve removal of wound contaminants.
- Another form of wound irrigation is whirlpool. The whirlpool should only be used for wounds that contain slough and necrotic tissue. Once the necrotic tissue is removed, the whirlpool should be discontinued because it can damage granulation tissue.
- Antiseptics such as Povidone iodine, hydrogen perioxide, chlorhexidine, Dakins (javex), and acetic acid (vinegar) when used indiscriminantly, have been shown to be harmful to fibroblasts. Therefore routine use is not recommended. Having a "sterile" wound is not a pre-requisite for healing. Most chronic wounds are colonized yet only a few become infected.
- Pressures from 8-15 psi are considered safe and effective for wound cleansing and irrigation. Pressures greater than 15 psi can cause tissue trauma. Pressures of 8-15 psi can be obtained using an 18-20 gauge angiocath on a 30 to 60 cc syringe.

4.4 Debridement

Wound healing cannot take place until necrotic tissue is removed. Debride when there is deep eschar, purulence, infection or a large area of necrotic tissue. Do not debride if the wound has healthy granulation tissue and <u>no</u> necrotic tissue.

There are several ways to debride a wound. The more common methods are autolytic, mechanical, chemical and sharp debridement.

4.4.1 Autolytic

Autolytic debridement is a process whereby the body utilizes its own digestive enzymes to break down necrotic tissue. This is accomplished by keeping wounds moist with hydrogel or moisture retentive dressings. This allows the body's own enzymes to liquefy devitalized tissue.

This method is usually painless but is slower than sharp debridement. It may be used with full thickness wounds and Stage III and Stage IV pressure ulcers with small to moderate amounts of exudate and necrotic tissue. The wound must be watched closely for signs of infection.

4.4.2 Mechanical

This is the removal of devitalized tissue from a wound by physical forces rather than chemical (enzymatic), or natural (autolytic) forces. Two types of mechanical debridement include:

• Wet-to-Dry Dressings

- Wet-to-dry gauze dressings remove necrotic tissue and absorb small amounts of exudate, but as a nonselective method of debridement, it can harm healthy tissue in the wound.
- Wet-to-dry gauze dressings are not a preferred treatment for debridement as it may actually interrupt the healing process.
 Emerging granulation tissue dehydrate and new vessels are disrupted by the removal of the adherent dry gauze.
- These dressings can be extremely painful because they are put into the wound wet and removed dry. This removal method works by tearing out slough that becomes enmeshed in the weave of the gauze.

Irrigation

• Two common methods of wound irrigation are achieved through high pressure irrigation and pulsatile high-pressure lavage. A third method is through the use of whirlpool. Refer to Principle #3 Wound Cleansing for a discussion of irrigation.

4.4.3 Chemical

Chemical debridement is the removal of necrotic tissue through a chemical process that may include the use of enzymes, sodium hypochlorite (Dakin's solution), and maggots.

Enzymes

Enzymatic debriding agents work by either directly digesting the components of slough or by dissolving the collagen anchors that attach the avascular tissue to the underlying wound bed. The clinician must follow manufacturer's instructions regarding enzymatic debriding agents. Enzymes are not effective in a dry environment. If eschar is present, crosshatching of the eschar is recommended to facilitate. Enzymes must be discontinued once viable tissue is revealed and necrotic tissue is removed.

• Sodium Hypochlorite (Dakin's Solution)

Sodium hypochlorite was originally used as a topical disinfectant. It is nonselective, meaning viable tissue may be removed along with nonviable tissue, and it has cytotoxic properties. Sodium hypochlorite is most appropriately used when there is a large amount of slough on the wound bed and the wound is infected or malodorous. It should be used for a short-term, less than ten days. There is conflicting evidence regarding the effectiveness of sodium hypochlorite as a debriding agent. At concentrations of 0.025%, it can remain noncytotoxic and be an effective antimicrobial. At higher concentrations, it may be toxic and pose a risk of damage to fibroblasts, resulting in impaired wound healing.

4.4.4 Biological

Maggots

Sterile larvae are introduced into the wound bed. Larvae secrete proteolytic enzymes, including collagenase, that break down the necrotic tissue. Maggot therapy is considered for use in wounds that have not responded to conventional methods of debridement. While there are no reported side effects, care should be taken to avoid healthy skin contact. Some patients may feel a crawling sensation. The clinician can reduce this feeling by confining the larvae to the wound bed. This can be accomplished through the application of dressings designed to keep the larvae in the wound.

4.4.5 Sharp

This is the fastest type of debridement. Examples of sharp debridement are conservative sharp, surgical sharp, and laser debridement.

Conservative Sharp Wound Debridement

- o Removal of visible dead tissue above the level of viable tissue.
- Requires the use of surgical instruments (scissors, forceps, and scalpel).
- o Done by physician or other qualified health care professional.
- o If done correctly it usually causes the patient minimal pain.

• Surgical Sharp Wound Debridement

- Performed by a surgeon/physician.
- Surgical sharp debridement is usually reserved for the removal of massive amounts of tissue or when a patient's life is in jeopardy from an infectious disease.
- Penetration extends through viable tissue.
- Can help turn a chronic wound into an acute wound and thereby stimulate healing.
- Several disadvantages include the high cost of performing the procedure in the operating room, and the risk to the patient with anesthetic.

• Laser Debridement

- Uses focused beams of light to cauterize, vaporize, or slice through tissue.
- Two disadvantages may be damage to surrounding healthy tissue and delayed healing.

4.5 Identification and Elimination of Infection

There are four terms that the clinician should know when deciding whether a wound is infected. These terms are contamination, colonization, critical colonization and infection.

Difference	ce Between Terms
Contamination	Presence of non-multiplying bacteria within a wound which accounts for the majority of the microorganisms present on the wound surface.
Colonization	Presence of bacteria which are multiplying but are producing no host reaction. This includes skin commeusals such as Staphylococcus epidermis and corynebacterium species, whose presence has been shown to increase the rate of wound healing.
Critical Colonization	Refers to a wound in which the bacterial burden is rising due to multiplication of organisms which are now starting to cause a delay in healing. Critical colonization initiates the body's immune response <i>locally</i> but not <i>systemically</i> and will have an effect on healing.
Infection	Refers to the presence of multiplying bacteria that are causing an associated host response. Pathogenic bacteria multiply and invade surrounding tissue resulting in host injury. If untreated, this may lead to systemic infection.

4.5.1 Signs and Symptoms of Infection in Chronic Wounds

- abnormal odor malodorous after cleansing;
- changes in sensation or pain (type, intensity, duration);
- abnormal discharge purulent, sanguinous;
- warmth, redness, induration, edema, discoloration, erythema greater than 2 cm;
- prolonged inflammatory process;
- delayed wound healing;
- deterioration of wound site and surrounding tissue, tissue may be friable;
- poorly or abnormally granulating tissue; may be pale in color, uneven in growth pattern, have areas of pocketing;
- bridging of soft tissue and epithelium;
- increased temperature (may not be present in the elderly).

4.5.2 Diagnosis of Infection

To make an accurate diagnosis of infection, there must be an on-going holistic assessment including:

- client status
- wound status
- clinical signs and symptoms of infection
- microbiologic analysis to confirm diagnosis and identify causative agent

It is the interplay of all the above characteristics that determine whether or not infection is present. One factor alone does not confirm the diagnosis of infection. Swab analysis alone is not conclusive of an infection.

4.5.3 Identification of Infection

Types of Wound Culture		
Tissue Culture Obtained by taking a tissue biopsy for		
	culture. Considered the gold standard.	
Aspiration culture	Insertion of a needle into the tissue	
	adjacent to the wound to aspirate the fluid.	
Swab culture The collection of tissue fluid on a sterile		
	swab.	

Wound culture – there are several types of wound culture:

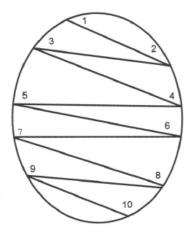
4.5.4 Guidelines for Swab Culture

Obtain a wound culture when clinical signs and symptoms of infection are present using "10-Point" technique.

Thoroughly cleanse the wound with sterile water or normal saline. Never take a swab of "old" surface drainage because this will only show what is growing on the surface drainage of the wound, not what is growing in the wound. If the wound is

dry, moisten the tip of the swab with sterile normal saline or with the medium found in the base of the culture tube.

Use a Zig Zag motion. Applying light pressure roll the swab on its side for one full rotation over as much of the wound surface as possible. In particular, sample the part of the wound with the most dramatic signs of infection or the area that is worsening. Be sure to rotate swab under wound margins and in any tunneled areas.



4.5.5 Local Versus Systemic Infection

A local infection can be identified by increased heat at the wound site, increased pain, increased swelling, increased redness, foul odor, increased drainage, change to the color of drainage.

A systemic infection, on the other hand, is manifested by fever (greater than 38.5°), increased tissue destruction and increased white blood cell count (WBC), and is much more serious than a local infection.

4.5.6. Treating Infections

There is significant controversy about the use of topical antibiotic therapy. If it is used, it should be used no more than 7-14 days. Their use may lead to local cell and tissue damage, systemic toxicity, or it could even lead to the development of contact sensitivity and allergic reactions, super infections and antibiotic resistance.

Systemic antibiotics should only be used when a definitive diagnosis of infection has been established. Collaborate with the patient's physician for appropriate antibiotics.

4.6 Elimination of Dead Space

Dead space refers to a hollow, cavity, or areas of tissue destruction underlying intact surface tissue as sinus tract formation. Dead space must be filled, though not overfilled, to promote healing and prevent premature closure of the wound. Wounds heal from the bottom upwards. Dead space provides a fluid medium for bacterial growth.

4.7 Absorption of Exudate

Excess exudate at the wound bed can cause maceration and tissue damage. It can pool and promote bacterial growth. Excess exudate is detrimental to wound healing and requires removal to achieve the optimal wound environment for healing. More frequent dressing changes may be initially required. Change dressing before break-through of drainage. Choose an absorbent dressing and change the dressing before it becomes entirely saturated.

4.8 Promotion of Moist Wound Healing

Maintaining a moist wound environment facilitates the wound healing process. Benefits associated with moist healing include:

 Increased rate of re-epithelialization – Wound healing is facilitated by a relatively hypoxic wound environment. Hydrocolloid dressings are capable of enhancing the process of angiogenesis. Moist wound healing helps to prevent crust formation, which leads to a faster epithelial migration across the moist wound bed.

- Bacterial barrier occlusive dressings act as a barrier to keep environmental microorganisms from coming into contact with the wound.
- Decreased pain local wound pain is significantly reduced in occluded wounds due to hydration of the wound by the dressing that insulates and protects nerve endings.
- Enhanced autolytic debridement moist wound healing can assist in the painless debridement of wounds.

4.9 Promotion of Thermal Insulation

Wound healing is accelerated when the wound bed is kept warm at body temperature, therefore, frequent dressing changes should be avoided when possible. Evidence-based practice indicates that the natural healing process should be disrupted as little as possible.

Local hypothermia can impair the healing process and the immune response. This impairment can increase the risk of infection because it causes vasoconstriction and increases hemoglobin's affinity for oxygen. Both these processes decrease availability of oxygen to the phagocytes. The consequence of hypothermia on phagocytes includes decreased phagocytic activity, decreased production of reactive oxygen products. Normal body temperature for optimal cellular function in humans is 36.4°C to 37.2°C. Above or below this range, the cellular reaction or process may be impaired or shut down.

The more occlusive a dressing is, the warmer the wound temperature remains. All moisture retentive dressings have different moisture vapor transmission rates (MVTR's)

4.10 Protection of the Healing Wound

Mechanical injury to the wound may occur because of shear, pressure or friction forces. Interventions to prevent reoccurrence:

- proper positioning and transferring techniques
- pressure redistribution support surfaces to reduce or eliminate pressure
- healed venous leg ulcers require compression hosiery for life
- frequent educational updates for the client with diabetes with attention to:
 - o proper foot wear
 - o proper foot care
 - o proper nail cutting
 - tight control of blood glucose, blood pressure, blood cholesterol and triglycerides
- education to all clients and their caregivers on prevention of reoccurrence

Section 5

BRADEN SCALE – FOR PREDICTING PRESSURE SORE RISK

5.1 Braden Scale – Policy

Please refer to Section 5 – Appendix 1 – for Regional Health Policy.

5.2 Braden Scale – For Predicting Pressure Sore Risk

Please refer to Section 5 – Appendix 2 – for Braden Scale.

5.3 Braden Scale – Copyright

Please refer to Section 5 – Appendix 3 – for Copyright.

Section 5 – Appendix 1

GENERAL NURSING MANUAL

REGIONAL HEALTH

NURSING POLICY

March 10, 2008

BRADEN SCALE – FOR PREDICTING PRESSURE ULCER RISK

POLICY

The Braden Risk Assessment Scale is used to identify individuals at risk for development of pressure ulcers. This validated and reliable measurement tool has been used for adult populations in hospitals, nursing homes and the community to link a score and level of individual risk to nursing interventions that promote, maintain and/or restore skin integrity.

Risk Factors for Consideration:

- Over 80 years of age
- Diastolic blood pressure less than 60 mmHg
- Cardiovascular disease
- Increased temperature
- Decreased dietary protein intake
- Chair/bed bound
- Impaired ability to reposition
- Extracorporeal oxygenation (the use of a heart-lung machine to take over the work of the lungs and sometimes the heart)

A Registered Nurse completes the Braden Scale as outlined below and whenever there is a significant change in an individual's health status.

Community Environment

Home/Personal Care Home

Complete on nursing admission for all clients who are chair/bed bound or who have limited ability to ambulate and for those clients who have **two** or more of the above risk factors for consideration. Repeat Braden Risk Assessment if score is less than or equal to 12, based on the stability/instability of the client and at a minimum of once per year.

Long Term Care Environment

Complete within 48 hours of admission, then weekly for four weeks, then quarterly.

Acute Care Environment

Complete on all adult inpatient admissions with the exception of mental health short stay and obstetrical patients.

If on initial assessment, the Braden Risk Score is 19 or above, the Braden Risk Assessment does not have to be repeated unless there is a change in the health status of the client.

If on initial assessment the Braden Risk Score is 18 or lower, the Braden Risk Assessment is repeated and interventions reviewed as per the following schedule:

Inpatient Unit	Frequency of assessment/review of interventions
Critical Care	Daily
Medical/Surgical	Monday/Wednesday/Friday
Extended Care (eg. Rehab, palliative care, comfort care, geriatric assessment, MH-long stay)	Weekly for one month and every three months thereafter.
Medically Discharged	On admission, weekly for one month, then every three months (as per LTC environment)

Criteria for Referral based on Braden Risk Assessment Score

The Braden Risk Assessment Score should not be the sole criteria for determining appropriate clinical interventions. Nursing interventions are to be initiated based on professional judgment and with consideration to available resources. The goal is to develop a plan of care that will promote, maintain and/or restore skin integrity.

Mild – Moderate Risk (total score 13-18)

Individuals who have Braden Risk Assessment score of Low to Moderate Risk (score 13-18) the RN **should consider** a referral to appropriate clinical discipline (for example, referral to dietitian if score on "nutrition" component is 2 or less; referral to physiotherapist and/or occupational therapy if score on "mobility" component is 2 or less).

High Risk (total score 12 or less)

Individuals who have Braden Risk Assessment score of High Risk (score 12 or less) the RN must send a referral to dietitian if score on "nutrition" component is 2 or less and a referral to physiotherapy and/or occupational therapy if score on "mobility" component is 2 or less.

References:

Ayello, E.A. (2001). Why is pressure ulcer risk assessment so important? *Nursing*, 31 (11), 75-79.

Bergstrom, N., Braden, B., Kemp, M., & Ruby, E. (1998). Predicting pressure ulcer risk. *Nursing Research*, 47(5), 269.

Braden, B. (2001). Risk assessment in pressure ulcers prevention. In D. L. Krasner, G.T.

Rodeheaver, & R. G. Sibbald, (eds.). Chronic wound care: a clinical source book for healthcare

professionals (3rd Ed.). Wayne: HMP Communications, pp 641-645.

Brown, S. (2004). The Braden Scale. Orthopedic Nursing, 23 (1), 30-38.

Quickfall, J. & Shields, D. (1998). Peak performance. Nursing Times, 94 (7), 74-77.

Smith, L.N., Booth, N., Douglas, D., Roberts, W.R., Walker, A., Durie, M., et. al. (1995). A critique of at risk pressure sore assessment tools. *Journal of Clinical Nursing*, 4, 153-159.

APPROVED BY:

DATE:_____

Chief Nursing Officer

BRADEN SCALE – FOR PREDICTING PRESSURE SORE RISK

Source: Barbara Braden and Nancy Bergstrom, copyright 1988.

Note: The lower the score, the greater risk of developing pressure ulcers. Refer to back for interventions.

RISK ASSESSMENT	SCORE/DESCRIPTION				
Sensory Perception Ability to respond meaningfully to pressure related discomfort.	1. Completely Limited Unresponsive (does not moan, flinch or grasp) to painful stimuli, due to diminished level of consciousness or sedation. OR Limited ability to feel pain over most of body surface.	2. Very Limited Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness. OR Has sensory impairment, which limits the ability to feel pain or discomfort over ½ of body.	3. Slightly Impaired Responds to verbal commands but cannot always communicate discomfort or need to be turned. OR Has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	4. No Impairment Responds to verbal commands. Has no sensory deficit, which would limit ability to feel or voice pain or discomfort.	
Moisture Degree to which skin is exposed to moisture Activity Degree of physical	 Constantly moist Skin kept moist almost constantly by perspiration, urine etc. Dampness is detected every time patient is moved or turned. Bedfast Confined to bed 	 2. Often moist Skin is often but not always moist. Linen must be changed at least once a shift. 2. Chairfast Ability to walk severely limited or nonexistent. 	 3. Occasionally moist Skin is occasionally moist, requiring an extra linen change approximately once a day. 3. Walks occasionally Walks occasionally during day but for very short 	 4. Rarely moist Skin is usually dry; linen only requires changing at regular intervals. 4. Walks frequently Walks outside the room at least twice a day and inside 	
Activity Mobility Ability to	1. Completely immobile Does not make even slight	Cannot bear own weight and/or must be assisted into chair or wheelchair. 2. Very limited Makes occasional slight	distances, with or without assistance. Spends majority of each shift in bed or chair. 3. Slightly limited Makes frequent though slight	room at least once every 2 hours during waking hours. 4. No limitations Makes major and frequent	
change and control body position	changes in body or extremity position without assistance.	changes in body or extremity position but unable to make frequent or significant changes independently 2. Probably inadequate	changes in body or extremity position independently. 3. Adequate	changes in position without assistance.	
Usual food intake pattern ¹ NPO: nothing by mouth ² IV: Intravenously ³ TPN: Total parenteral nutrition Friction and	Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement. OR Is NPO ¹ and/or maintained on clear liquids or IV ² for more than 5 days. 1. Problem	Rarely eats a complete meal and generally eats only about ¹ / ₂ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR Receives less than optimum amount of liquid diet or tube feeding. 2. Potential problem	Eats over ½ of most meals. Eats a total of 4 servings of protein (meat, diary products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered. OR Is on a tube feeding or TPN ³ regimen, which probably meets most of nutritional needs. 3. No apparent problem	Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and diary products. Occasionally eats between meals. Does not require supplementation.	
Shear	Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction. : Total score of 12 or less represen	Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.		

Date of Assessment:

Assessment Date	Signature/Status	Assessment Date	Signature/Status

Braden Risk Assessment – Intervention Tool

Score	Interventions
19 or above	 No special interventions
Mild to Moderate Risk (13-18)	 Observe skin for redness with attention to pressure points. Keep head of bed below 30° except for mealtime. Encourage adequate nutrition. Consider referral to dietitian if individual scores 2 or less on the "Nutrition" component. Encourage ambulation. Consider referrals to physiotherapy and/or occupational therapy if individual scores 2 or less on "Mobility" component. Reduce opportunities for excessive moisture from drainage, incontinence, perspiration, etc. Protect perineum with barrier product if incontinence present. Consider use of pressure reduction devices (eg. mattress, chair). Turn/reposition every 2 hours. Use pillows or covered wedges to help with repositioning small shifts in position frequently throughout the day. Protect heels by keeping heals off bed/chair (eg. place pillows lengthways under calf of leg). Moisturize dry/cracked skin.
High Risk (12 or less)	 As for Low to Moderate Risk and: Consider use of pressure relief surface(s). Consult dietitian if individual scores 2 or less on the "Nutrition" component. Consult physiotherapy and/or occupational therapy if individual scores 2 or less on "Mobility" component.

Risk Factors for Consideration:

Over 80 years of age
 Cardiovascular disease
 Decreased dietary protein intake
 Impaired ability to reposition
 Decreased dietary protein intake
 Chair/bed bound
 Extracorporeal oxygenation (the use of a heart-lung machine to take over the work of the lungs and sometimes the heart)

Community Environment

Home/Personal Care Home

Complete on nursing admission for all clients who are chair/bed bound or who have limited ability to ambulate and for those clients who have two or more of the above risk factors for consideration. Repeat Braden Risk Assessment if score is less than or equal to 12, based on the stability/instability of the client and at a minimum of once per year.

Long Term Care Environment

Within 48 hours of admission, then weekly for four weeks, then quarterly.

Acute Care Environment

Complete on all adult inpatient admissions with the exception of mental health short stay and obstetrical patients.

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Section 5-Appendix 3



Date: March 14, 2008

To: Nancy Wright, Wound Care Consultant Community Services Division, Central Regional Integrated Health Authority

From: Barbara Braden, PhD, RN, FAAN, Nancy Bergstrom, PhD, RN, FAAN

Permission to use the Braden Scale* RE:

As holders of the official copyright for the Braden Scale for Predicting Pressure Sore Risk and the interventions, we hereby grant permission for the use of the scale and the protocols in the provincial wound care manual to be used by all four health boards of Newfoundland and Labrador, Canada.

*It is understood that the name of the instrument and the indication that the copyright belongs to Braden and Bergstrom remain on any copies and that you do not make any changes to the wording or the scoring of this tool.

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Section 6.0

PREVENTION AND MANAGEMENT OF WOUNDS

This section presents an introductory discussion of various types of more commonly seen wounds, their prevention and treatment. All tissue heals the same way, regardless of the type of wound; the clinician must consider the etiology of the wound, in addition to co-morbidities that may be present in each patient they treat.

This section focuses on seven different types of wounds: pressure ulcers, venous leg ulcers, arterial leg ulcers, diabetic foot ulcers, surgical wounds, burns and oncology wounds. Each wound is presented with the following components: introduction, etiology, assessment, plan, intervention and evaluation. A discussion of prevention methods for each type of wound is included, if appropriate.

6.1 Pressure Ulcers

Pressure ulcers are "localized areas of tissue necrosis that tend to develop when soft tissue is compressed between a bony prominence and an external surface for a prolonged period". Capillary pressure, usually described as between 12 - 32 mm Hg, is exceeded with pressure and cellular damage occurs. There is a leakage of cells from this capillary damage and fluid accumulates. Local blood vessels dilate and the cascade of injury/repair begins (refer to Section 2 on Wound Healing).

6.1.1 Etiology

Factors that Increase Risk or Contribute to Pressure Ulcer Development

- immobility
- chair/bed bound
- impaired ability to reposition
- impaired perception or sensation
- over 80 years of age
- malnutrition
- decreased dietary protein intake
- dehydration
- cardiovascular disease
- moisture (such as incontinence)
- shear
- friction
- pressure
- increased temperature
- venous hypertension
- diastolic blood pressure less than 60 mmHg
- ischemia
- neuropathy
- extracorporeal oxygenation (the use of a heart/lung machine to take over the work of the lungs and sometimes the heart)

In addition to treating the wound, it is crucial that the clinician identify and eliminate any factors that may be contributing to the development of pressure ulcers. Many ulcers can be prevented.

6.1.2 Assessment

Pressure ulcers are identified according to a staging system developed by the National Pressure Ulcer Advisory Panel (NPUAP). It is based on the clinician identifying the level of tissue destruction or involvement and establishing a Stage. The ulcer, as it heals, will always be identified at the highest stage it developed.

For example, if a pressure ulcer involved the level of subcutaneous tissue but did not extend further, and it healed, it will always be known as a healed Stage III pressure ulcer. Ulcers are never reverse-staged (in other words, moved from a Stage III to a Stage II, etc). Conversely, if a pressure ulcer was identified as a Stage II ulcer, it could further deteriorate and become a Stage III or IV ulcer. It should then be identified at the deepest level of tissue involvement.

	STAGING OF PRESSURE ULCERS		
1	Non blanchable erythema of intact skin; the heralding lesion of skin ulceration. Area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue.		
II	Partial-thickness skin loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also presented as an intact or open/ruptured serum-filled blister. This stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation.		
III	Full thickness skin loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling. Note: the bridge of the nose, ear, occiput and mallelous do not have subcutaneous tissue and stage III ulcers can be shallow.		
IV	Full thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often undermining and tunnelling.		
Unstageable	Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and /or eschar (tan, brown, or black) in the wound bed. A pressure sore that cannot be accurately staged due to the presence of necrotic tissue covering the wound base.		

Deep Tissue Injury: Purple or maroon localized area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. Area may be painful, firm, boggy, warmer or cooler than adjacent tissue.

6.1.3 Plan

The clinician develops a plan that will consider treatment of an existing ulcer as well as prevent future skin breakdown.

6.1.4 Interventions

- Wound Care
 - Follow the principles of wound healing, select wound care products.
- Skin Care
 - Inspect skin daily and with each episode of incontinence.
 - Bathe skin using a mild pH balanced cleansing product and avoid hot water.
 - Use moisturizers liberally once to twice a day for patients with dry skin.
- Nutritional Support
 - Consult dietitian to develop a meal plan to increase protein and calories, if required.
 - o Provide nutritional supplements.
 - Maintain good hydration with 6 8 glasses of water per day, unless contraindicated.

• Pressure Reduction/Relief

- Consider implementing pressure reduction/relief mattress for all patients at risk or who have developed a pressure ulcer.
- Obtain pressure reduction/relief seating for patients who are chairbound.
- Use chairs with tilt/recline features rather than just recline features alone.
- Implement a repositioning schedule to reduce length of time spent in one position.

6.1.5 Evaluation

Evaluation indicators that reflect a positive outcome may include:

- Wound is healed.
- Wound is reduced in size (usually by 1 cm per week).
- Patient reports reduced pain.
- Patient reports improved quality of life.
- Wound is not deteriorating.

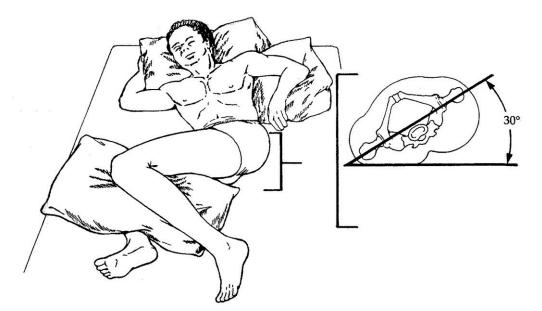
6.1.6 Prevention of Pressure Ulcers

- Skin Care Management
 - Use warm water, not hot water. Hot water can cause dryness, burns, and increase the skin's metabolic demands.
 - Use a pH balanced (5.5) cleanser. Astringent soaps can strip the skin of its natural protective oils and antimicrobial acid mantel.
 - Be gentle. Do not excessively rub the skin. Friction caused by rubbing can tear fragile skin. If skin is wet pat it dry.
 - Cleanse the skin ONLY as needed. A light daily wash is important to remove oily metabolic wastes secreted on the skin surface. Prompt cleansing of soiling from incontinence or drainage is critical. An increase in bathing frequency maybe required for clients with high levels of perspiration.
 - Moisturize regularly. This helps to keep the skin soft and reduce the chance of tearing.

• Injury Prevention

- Do not massage bony prominences or reddened areas. Bony prominences have less subcutaneous protection and are often exposed to increased pressure. Massaging can result in tearing underlying tissue (shearing effect), thus extending an injury.
- Maintain head of bed below 30° position. When the client is in bed, do not raise the head more than 30° unless clinically contraindicated i.e. CHF, patient eating. Slightly raise the knee gatch or use footboards to limit sliding. Lift clients to reposition, using draw sheets and/or overhead trapeze bars. Avoid pulling or dragging a client when repositioning.
- Position client to promote good blood flow. Be careful not to place the client in a jack-knife position which can compromise blood flow to lower extremities. This is a position where the head is raised above 30° and the legs may be elevated as well. Ensure that the client's feet are not exposed to unrelieved pressure from the footboard.

- Limit opportunities for friction injuries. When repositioning clients, lift don't pull. Use protective devices between the skin and the source of friction such as, socks or cotton bandages. Consider using cornstarch on sheets if the client is active in the bed.
- Do not use "Donut" type devices to remove pressure. These devices intensify the pressure produced under the ring part of the donut and can cause serious injury to tissue. Instead lift the entire area, for example, place a pillow lengthwise from the heel to the knee to reduce pressure on a heel. Do not use IV bags or other similar devices under the heels.
- Limit opportunities for pressure injuries. Reposition clients with limited mobility frequently. Chair bound clients should be moved every hour. Avoid positioning a client directly on their trochanters. This can be avoided by restricting the sidelying angle to less than 30°, see diagram below. Use appropriate support surfaces, such as cushions and specialty mattresses. Place pillows between bony prominences.



6.2 Leg Ulcers

There is a significant difference between venous and arterial disease as it pertains to the development and healing of ulcers. Different interventions are used and it is important for the clinician to understand the differences to be able to determine the appropriate course of both ulcer and patient treatment.

Causes of lower extremity ulcers may include one or more of the following:

- Venous hypertension
- Arterial disease
- Bacterial, fungal and syphilitic infections
- Diabetes mellitus
- Pressure
- Malignancy
- Squamous cell
- Kaposi's sarcoma
- Melanoma
- Lymphoma
- Sickle cell anemia
- Trauma
- Rheumatoid arthritis
- Lupus erythematosus
- Connective tissue disorders
- Insect bites
- Factitial (self-induced)

The two types of leg ulcers that are presented are venous and arterial leg ulcers. Diabetic foot ulcers are presented in a separate section.

6.2.1 Venous Leg Ulcers

a. Etiology

Venous Ulcers result from disorders of the deep venous system. Venous ulcers of the lower extremities affect 1% of the general population and 3.5% of persons over 65 years of age, with a recurrence rate approaching 70%.

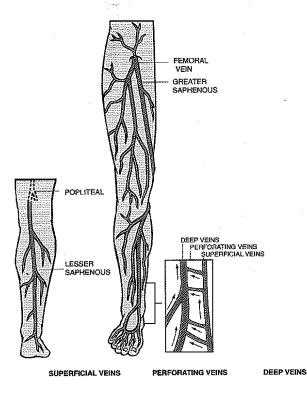
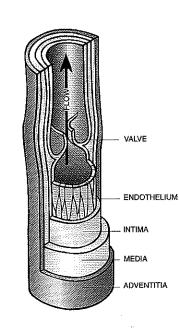


Figure 1: Significant venous anatomy of the leg.

Figure 2: Physiology and function of valves in veins of the lower limbs.





DURING SYSTOLE BLOOD IS PUSHED TOWARD THE HEART



DURING DIASTOLE GRAVITY CAUSES BLOOD TO FLOW TOWARD THE EXTREMITII CLOSING OF THE VALVES PREVENTS THIS RETROGRADE FLOW.



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The venous system of the legs comprises three parts:

- the deep venous system (includes the femoral, popliteal, and tibial veins);
- the superficial system, composed of the greater and lesser saphenous veins; and
- the perforator veins that join the deep and superficial systems.

Venous Ulcer



This is a fairly typical appearance of a healing venous ulcer on a lower limb. Note the area of healing that has separated one ulcer into two ulcers.

b. Assessment

The following table outlines assessment criteria when assessing a venous leg ulcer.

	DEFINING CHARACTERISTICS
Location	Anterior, pretibial and gaiter (sock) area. Medial malleolus
Skin Colour	Brown staining or discoloration due to deposits of hemosiderin
Skin	Warm, dry, pruritic
Pulses	Present; usually good popliteal and pedal pulses
Pain	Aching, heavy feeling in legs
Ulcer Base	Moist and shallow, deep red in color or yellow with fibrinous slough
Ulcer margin	Irregular configuration
Periwound	May be macerated if exudate copious; may also be dry and flaky
Edema	Increases with extremities dependent, decreases when limbs elevated to heart level or above. Mild ankle swelling
Discharge	Moderate to large amount of exudate

c. Plan

The gold standard for the management of venous ulcers lies in the application of compression therapy. Compression therapy is the application of external pressure to the lower extremity to facilitate venous blood return.

Compression Therapy is shown to:

- 1. Reduce distension in the superficial veins, counteracting the high pressure.
- 2. Encourage and enhance blood flow in the deep veins.
- 3. Restore damaged valve function in some patients
- 4. Facilitate the action of the calf muscle pump; restricting the muscle which directs pressure inwards on to blood vessels, thereby increasing venous return.
- 5. Force fluid into both the venous and the lymphatic system, thus reducing edema.
- 6. Reduce the symptoms of venous disease such as aching limbs and pain from the ulcer.
- 7. Increase the healing rate of venous ulceration.
- 8. Improve skin condition; some studies report increased removal of fibrin during compression therapy.
- 9. Enhance fibrinolytic activity needed to increase the healing rate of venous ulceration.
- 10. Promote growth of healthy tissue and prevents concomitant infection.

d. Compression

Compression is created by the use of elastic or rigid external layers of bandages. The amount/type applied is dependent upon the extent of peripheral edema - Measured by ankle circumference, and the expected amount of normal calf muscle flexion. LePlace's Law* for compression bandages explains the effect.

LePlace Law*

SubBandage = <u>Number of Layers x Tension</u> Pressure Ankle Circumference x Bandage Width

Therefore an increase in the number of layers applied and/or the stretch pulled on the elastic wrap will increase the amount of compression obtained. Likewise a decrease in ankle size or bandage width will also increase the compression.

Approximately 40mm Hg pressure is required to effectively and safely facilitate venous return and decrease peripheral edema. The pressure will be the greatest at the ankle (usually the smallest portion of the leg) and the least just below the knee.

e. Application Pointers

High compression is contraindicated for patients with arterial disease (ABI less than 0.8). However, lower compression can be used in patients with an ABI of 0.6 to 0.8.

- o Always read and follow manufacturers' instructions carefully.
- Confirm with physician or client records that the client has adequate blood flow to safely use compression. This is usually done through vascular studies that include an ABI reading.
- o Always wrap from the toes to the knee. Never stop mid-calf.
- Measure the ankle circumference before and periodically thereafter to evaluate the effectiveness of the treatment to decrease edema.
- Use tape, not safety pins or clips to secure bandage.
- Never completely encircle a limb with a strip of tape to secure the bandage.
- Loose stocking style bandages can be applied over a compression bandage to reduce friction/adhesion problems between the bandage and clothing.

f. ABI (also known as Ankle-Brachial Index)

The Ankle-Brachial Index is a noninvasive test used to detect evidence of significant arterial insufficiency. It is also used to assess client's need for further testing. Results of testing allows the clinician to feel confident that compression can be used safely. Usually compression is used in clients with an ABI reading greater than 0.8. Lower compression can be used in patients with an ABI between 0.6 - 0.8

ABI Reading	Results indicate
Greater than1.0	Normal Arterial Circulation
Less than 0.9	Mild Degree Arterial Disease
0.5 - 0.8	Mixed arterial and venous disease
Less than 0.5	Arterial disease

Refer to Section 6 – Appendix 1 for Procedure for ABI.

g. Interventions

Interventions focus on the principles of wound care management and the application of appropriate wound care products including compression.

If compression therapy is applied inappropriately or to patients with arterial occlusion, it could result in the need for amputation of the limb. An appropriate assessment must be done to verify that a patient can tolerate compression therapy. Refer to physician regarding Doppler studies and other vascular assessment to evaluate arterial perfusion.

h. Evaluation

Evaluation indicators that reflect a positive outcome may include:

- Wound is healed.
- Wound is reduced in wound size (usually by 1 cm per week).
- Patient reports reduced pain.
- Patient reports improved quality of life.
- Wound is not deteriorated.
- Wound does not reoccur.

i. Prevention

Prevention of a venous ulcer should focus on:

- Maintenance of compression therapy for life.
- Maintenance of healthy skin integrity.
- Prevention of injury to the lower limbs.

6.2.2 Arterial Leg Ulcers

Arterial Ulcers are caused by insufficient arterial perfusion to an extremity or location and are also termed "ischemic ulcers." In other words, not enough oxygen is able to be transported via the arteries to distal tissue, for example, a foot. The term "ischemic ulcer" denotes a skin lesion with tissue loss related to arterial disease and is not used to describe the actual perfusion state of the ulcer. Arterial ulcers are not as common as venous ulcers and they are often more complex to manage because of coexisting diseases and complications.

Peripheral Vascular Disease (PVD) is a term commonly associated with arterial insufficiency. However, PVD includes the arteries, veins and lymph vessels and results in chronic, systemic health problems. There is no cure for PVD. The following discussion relates to the arterial component of PVD, Peripheral Arterial Disease (PAD).

The pathogenesis of PAD is arteriosclerosis, a thickening and decreased elasticity of the arterial walls. Atherosclerosis, a form of arteriosclerosis, develops as a result of the accumulation of plaque, lipids, fibrin, platelets, and other cellular debris into and along the wall of the artery. The dynamics of blood flow are affected by atherosclerotic plaque. When resting, a person can tolerate up to 70% occlusion of the artery. However, with exercise, the increased demands for blood flow cannot be met and muscle ischemia occurs, causing crampy leg pain; 90% or greater occlusion will reduce flow resulting in pain even at rest. Although the exact initiating mechanism of atherosclerosis is unknown, the aging process, life-style habits, and disease can combine to affect both large and small arteries.

a. Risk Factors for PAD

Smoking - Pathogenesis is unknown, atherosclerosis may be related to:

- 1. carboxyhemoglobin, which can injure vessel walls;
- 2. altered platelet function with resultant thrombus formation; and
- 3. decrease in prostacyclin, a prostaglandin that prevents platelet aggregation and promotes vasodilation. In a smoker, 5 15% of the oxygen in the blood is replaced with carbon monoxide.
- b. Diabetes Mellitus the longer a person has diabetes mellitus, the more likely the person is to develop PAD. Patients with diabetes who smoke are severely jeopardizing arterial perfusion because of diabetes-associated PAD. Peripheral vasoconstriction effects of nicotine reduce absorption of insulin from s/c tissue. Good control of blood glucose levels may prevent, stabilize, or improve the microangiopathies (retinopathy, nephropathy, and neuropathy) but, according to several studies so far, preventing hyperglycemia does not seem to affect macrovascular atherosclerosis.
- c. Hyperlipidemia (hypercholesterolemia and hypertriglyceridemia) significantly affect atherogenesis. A serum cholesterol level greater than 220 mg per 100 ml is considered a sign of hyperlipidemia. Elevated triglyceride levels should be evaluated and may also be indicative of hyperlipidemia.

d. Signs and Symptoms of Peripheral Arterial Disease

- *Pain* pain may be with exercise (intermittent claudication) or nocturnal or it may simply be pain at rest.
- *Impaired Circulation*, manifested by:
 - o Decreased pulses
 - o Skin-temperature changes
 - o Delayed capillary and venous filling

- o Pallor on elevation
- Dependent rubor

• Ischemic Skin Changes

- o Color
- Atrophy of subcutaneous tissue
- o Shiny, taut epidermis
- o Loss of hair
- Gangrene
- e. Assessment



Photograph of a typical arterial ulcer caused by pressure on the lateral malleolus in a patient with an ABI of 0.5. Note the round punched-out appearance and pale wound base.

f. Diagnostic Tests

An accurate diagnosis is essential to determine appropriate interventions to treat the ulcer. The main determination that must be done is whether the arterial blood supply is adequate to attempt to heal the wound. If the arterial blood supply is inadequate, the clinician will employ interventions aimed at reducing risk of infection and spread of the ulcer (palliation/maintenance) as opposed to healing. Ankle Brachial Indices (ABI readings) and Transcutaneous Oxygen (TcpO₂) measurements are simple bedside methods to determine the status of blood perfusion. Other invasive/noninvasive vascular studies are available. A consult to a special vascular assessment laboratory is recommended. Bypass surgery, antithrombolytics, and angioplasty are viable treatment options. Noninvasive tests may include segmental pressures, ultrasonic doppler waveforms, pulse volume recording (PVR), and transcutaneous oxygen measurements.

Invasive tests include angiography and digital subtraction angiography.

DEFINING CHARACTERISTICS	
Location	Anywhere on the legs or feet, especially pressure points, distal to impaired arterial supply
Skin Colour	Blanching with limb elevation to 30 degrees. Rubor with limb dependence
Skin	Thin, shiny, and hairless
Nails	Hypertrophied, yellow, and fragile, clubbing of nails, loss of hair on feet
Pulses	Absent or faint
Pain	Severe pain, intensified with activity or limb elevation
Ulcer Base	Pale, grey, or yellow, with no evidence of new tissue growth
Ulcer margin	Well defined, "punched-out" appearance. These can be deep wounds
Periwound	Dry, no surrounding inflammatory response. Shiny red
Edema	Minimal
Discharge	Minimal serous or purulent
Capillary Refill	Greater than 4-5 seconds

g. Wound Assessment

h. Plan

Following a thorough assessment, a goal for healing or palliation/ maintenance is then decided. Depending on this goal, the clinician then selects interventions that will work towards that goal.

i. Interventions

Invasive procedures - are used in an attempt to restore blood flow in the extremity. This may include vascular surgery.

Goals of a comprehensive management plan for arterial ulcers are to:

- 1. reduce or eliminate the cause
- 2. optimize the microenvironment
- 3. support the host and
- 4. provide education

Debridement of eschar may be contraindicated. If the arterial disease is severe, wound debridement will only increase the risk of infection. The wound will not receive the needed WBC's and nutrients to heal. The exposed tissue would then be vulnerable to microorganisms. Occlusive moisture retentive dressings are not recommended in these circumstances. Dry gauze is an appropriate option to keep the wound dry and prevent further deterioration of the wound.

j. Evaluation

Evaluation indicators should evaluate the goal of treatment. If the goal is to heal the wound, a positive outcome may include:

- wound is healed
- wound is reduced in wound size (usually by 1 cm per week)
- patient reports reduced pain
- patient reports improved quality of life
- wound is not deteriorating

If the goal is to palliate/maintain the wound status, a positive outcome may include:

- patient reports reduced pain
- patient reports improved quality of life
- wound does not deteriorate
- wound does not become infected

k. Prevention

Prevention of Arterial Ulcers might include:

- 1. cessation of smoking
- 2. implementation of a walking program to improve collateral blood flow
- 3. prevention of injury

4. Maintenance of skin integrity.

6.3 Diabetic Foot Ulcers

Foot complications are one of the most common reasons for hospital admissions in patients with diabetes. It is estimated that, of the two million persons in Canada with diabetes, approximately 4 - 10% (80,000-200,000) will develop a foot ulcer. Of those people who develop a foot ulcer, approximately 14 - 24% (11,200 – 48,000) will require an amputation because the ulcer does not heal.

The foot complication experienced by people with diabetes is most often related to diabetic neuropathy. The neuropathic foot is often termed the "insensate foot" because the patient has a diminished or absent ability to feel pain and temperature. The cause is unknown but probably involves multi system changes resulting from diabetes. There are three types of diabetic peripheral neuropathy.

Sensory - Sensory neuropathic changes, considered to be the most disastrous type of neuropathic change, puts the client at risk for mechanical, chemical, and thermal trauma. At a foot temp of 21°C, a patient requires one millilitre of blood flow per 100 grams of tissue per minute. A client with even moderate PAD can manage this requirement for oxygenation. Soaking the foot in hot water can quickly raise the skin temperature to 40°C. This requires an increase of 10 times the flow of blood. A client with PAD is not able to achieve this level of oxygen requirement. The results: blistering, ulceration, infection and/or gangrene, and, not infrequently, amputation.

Motor - Motor neuropathy results in muscular atrophy in the foot, creating two basic problems. Foot deformities such as cocked-up toes, or hammertoes, develop and the patient's gait changes. These gait changes cause repetitive stresses on areas of the foot, usually a metatarsal head, rather than distributing the stresses of walking more uniformly. Callus build-up is the first sign of repetitive stress and will progress to ulceration if the weight is not properly redistributed with special shoes (orthotics). These ulcers are sometimes referred to as neuropathic, neurotrophic, trophic, perforating, or malperforans ulcers.

Autonomic - autonomic neuropathy is the third category of peripheral neuropathy, with distal anhydrosis as its principal symptom. Anhydrosis refers to the absence of sweating. Anhydrosis results in xerosis (dry skin) and predisposes the client to develop cracks and fissures. A chronically dry or moist interdigital environment on the foot is a perfect breeding ground for selective bacterial or fungal flora. These bacteria or fungi that gain entry to soft tissues through the cracks and fissures penetrate further into the soft plantar tissues with repetitive stresses of ambulation and may cause infection, gangrene, and even ultimately amputation.

6.3.1 Assessment

A comprehensive assessment of the diabetic foot and the diabetic ulcer must be performed.

	Diabet	ic Foot Assessment			
Ischemia	Ischemia results from atherosclerosis of the arteries of the leg. Assess pedal pulses, skin colour (dusky red or cyanotic blue) and capillary refill.				
Deformity	Deformity often leads to the development of vulnerable bony prominences, which are associated with high mechanical pressures on overlying skin. This usually results in ulceration in the absence of protective pain sensation and particularly on those who wear unsuitable shoes.				
		Foot Deformities			
	Clawed Toes	Fixed flexion deformities at the interphalangeal joints			
	Abnormally high medial longitudinal arch, leading to abnormal distribution of pressure and excessive callus formation under the metatarsal heads				
	Hallux Rigidus Limited joint mobility of the first metatarso-phalangeal joint with loss of dorsiflexion leading to excessive pressure causing callus formation				
	Hammer ToeFlexion deformity of the promiximal interphalangeal joint of a lesser toe with hyperextension of the associated metasophalangeal and distal interphalangeal joints leading to ulceration.				
	Charcot Foot Bone and joint damage in the metatarsal-tars which results in rocker bottom foot and media convexity.				
Callus	Thickened area of epidermis which develops at sites of high pressure and friction.				
Swelling	Predisposes to ulceration; impedes healing of ulcer.				
Skin Breakdown	Any break in the skin over the entire surface of the foot, ankle, between the toes and back of heel.				
Infection	Signs could include ulceration, cellulitis, purulent discharge, pain in an insensate foot.				
Necrosis	Black/brown devitalized tissue.				

	Diabetic Foot Ulcer Assessment			
Location	Most often on the feet, especially weight bearing surface or pressure points. May be located between the toes.			
Surrounding Skin	Dry, thin, crack and/or fissured. Thick callous pressure point(s).			
Ulcer Base	May be dry or covered with eschar. Often has deep necrotic areas that go undetected until opened surgically.			
Border	Undefined; ulcer may be small at surface and have large subcutaneous abcess.			
Pain	Absent, burning or numbness. (Mild to severe).			
Drainage	Varies; an infected ulcer may have purulent exudate; others may have little serosanguinous discharge.			
Pulses	Usually present (dependent on involvement of arterial component).			
Skin Color	Normal; pallor if arterial disease involved.			

6.3.2 Semmes-Weinstein Monofilament

The Semmes-Weinstein monofilament is an easy and inexpensive way to test a client for neuropathy. The device has filaments of varying diameters that are touched to various areas of the plantar surface of the foot, avoiding areas of heavy callus build-up. The 5.07 g monofilament it the preferred size monofilament to assess for loss of protective sensation in a person with diabetes. The client with neuropathy is unable to detect the presence of the 5.07 g monofilament.

Refer to Section 6 – Appendix 2 *"Diabetic Foot Screen for Loss of Protective Sensation"* for procedure on monofilament testing .

Wagner Grading System for Vascular Wounds on Extremities

Grade	Characteristics
0	Preulcerative lesion
	Healed ulcers
	Presence of bony deformity
1	Superficial ulcer without subcutaneous tissue involvement.
2	Penetration through the subcutaneous tissue; may expose bone, tendon, ligament, or joint capsule
3	Osteitis, abscess, or osteomyelitis
4	Gangrene of digit
5	Gangrene of foot requiring disarticulation



Diabetic Foot Ulcer on the 1st Metatarsal Phalangeal Joint (MTP) in a patient with sensory neuropathy who wore an inappropriate insert in his shoe.

6.3.3 Plan

Following a thorough assessment, a goal for healing or palliation/maintenance is then established. The interdisciplinary team is involved, to establish treatment parameters to address all the components of wound care and offloading that will be required to work toward that goal. Nursing interventions address wound management, nutritional support, management of diabetes, appropriate use of offloading equipment.

6.3.4 Interventions

Consult with the physician regarding vascular studies to evaluate arterial perfusion and potential for healing.

Consult with the physician to determine surgical and medical intervention and management.

Refer client to professional who will determine appropriate offloading strategies. Offloading strategies may include orthotics and contact casting. Orthotics can be a simple insert to place inside footwear or it can be a type of shoe specially designed to redistribute pressure across the surface area of the foot and leg. Orthotics can also be a splint type device with a rocker bottom. A contact cast is a toe to knee cast that is applied to a client with an existing ulcer. It is a temporary intervention that redistributes the weight of the foot to an insignificant amount.

Elevate heels of bedbound patients on pillows, if patient has adequate lower limb circulation. Support the leg with a pillow placed lengthwise from behind the knee to the foot and allow the heels to be suspended off the end of the pillow.

Wash and inspect feel daily, dry carefully, especially between the toes. Lubricate dry feet. Do not put oil or cream between the toes.

Do not use chemical agents to remove corns or calluses. Consult a physician or foot care specialist.

Debride callus frequently to promote healing of neuropathic wounds promptly. The presence of callus impairs healing.

Intervention focus on management of wound and appropriate wound care products.

6.3.5 Evaluation

Evaluation indicators may include:

- Wound is healed.
- Wound is reduced in wound size (usually by 1 cm per week).
- Patient reports reduced pain.
- Patient reports improved quality of life.
- Wound is not deteriorating.
- Function is restored.
- Maintenance of wound condition.
- Maintenance of skin integrity.

6.3.6 Prevention

Goals of a comprehensive management plan for prevention of diabetic foot ulcers include:

- 1. Avoid smoking.
- 2. Wash and inspect feet daily.
- 3. Avoid exposing feet to temperature extremes.
- 4. Do not walk barefoot.
- 5. Do not soak feet.
- 6. Wear proper-fitting socks and change them daily.
- 7. Wear proper-fitting shoes and inspect shoes daily for any areas of wear or roughness inside the shoes.
- 8. Cut nails straight across or, if unable to cut their own nails, have a health professional, who is foot care specialist trained, cut their nails.
- 9. Manage their diabetes closely and work with health professionals to try to achieve and maintain good glycemic control.
- 10. See a health professional immediately if a cut, blister, or sore develops.
- 11. Do not cut corns and calluses. See a health professional.

6.4 Surgical Wounds

An acute surgical wound is a healthy, uncomplicated breach in the normal skin barrier as a result of surgery.

6.4.1 Methods of Surgical Wound Closure

Surgical wounds are closed using one of four methods of wound closure:

- 1. Suture materials
- 2. Skin staples or clips
- 3. Adhesive skin closures
- 4. Skin closure adhesive

6.4.2 Dressings for Surgical Wounds

The purpose of applying a dressing to a surgical wound is:

- to protect it against pathogens
- to protect the skin from exudate and for aesthetic reasons

Within 48 - 72 hours, the wound is sealed with fibrin and so becomes impervious to bacteria. However, it may be appropriate to continue to cover the surgical wound, depending on patient preference, amount of exudate, risk of injury to the incision line, approximation of suture line, medical diagnosis, such as diabetes. Within 5 - 9 days after surgery, the clinician should be able to palpate the healing ridge beneath the skin extending to about 1 cm on each side of the wound.

6.4.3 Surgical Wound Healing

Most surgical wounds heal, without incident, by primary intention. Refer to section 2 for a discussion of primary, secondary and tertiary healing.

There are certain complications that may prevent this from happening. Examples of complications that may interfere with healing include:

- Wound infection
- Wound dehiscence
- Hemorrhage
- Evisceration
- Hematoma
- Poorly approximated incision line

6.4.4 Surgical Site Infection (SSI)

Surgical Site Infections are defined as occurring within 30 days of the operative procedure. If an implant is used, such as a mesh, the time for the SSI may extend to one year.

The following are types of surgical site infections:

- 1. *Superficial infection* includes the skin or subcutaneous tissue of the incision.
- 2. *Deep infection* involves the deep soft tissue of the incision, ie. fascial and muscle layers.
- 3. *Organ/space infection* involves any part of the organs or spaces that was manipulated during surgery other than the skin.

6.4.5 Interventions

Following the principles of wound healing, the clinician chooses the most appropriate dressings to manage the wound.

6.4.6 Prevention

There is a paucity of information on the prevention of surgical wound complications. As with all wounds, there should be appropriate attention paid to sterile technique. Client conditions, such as good nutrition and hydration, are also important, as is control of any factor that may place the client at increased risk of complications.

6.5 Burns

Burns result from many sources: thermal, chemical, electrical, and radiation. A burn injury creates a significant alteration in the functioning and structural integrity of the skin. Care of the patient with the burn is a complex process that requires a multidisciplinary team approach. All burn team members must be knowledgeable about the concept of wound care, wound healing and pathophysiology.

6.5.1 Severity of the Burn Wound

Treatment of the burn is based on the amount, depth, and severity of the injury. The depth of the injury is based on the number of cells injured or destroyed and on the functional capacity of the level of the skin. Classification of burns is now identified as epidermal, partial-thickness (superficial), partial-thickness deep, and full-thickness injury. • *Epidermal burn*: Sometimes referred to as a first degree burn involves only the skin surface and skin function is largely preserved. Usually cause is from a sunburn or flash flame burn from a gas stove.

Clinical features include:

- o Pain; nerve endings intact
- o Erythema
- o Slight edema
- o Absence of blisters
- Mild to moderate pain
- o Heals within a few days
- o Usually no scarring



Second degree burns are now divided into superficial and deep-partial thickness injuries.

• *Partial-thickness (superficial) burn*: Involves both the epidermis and superficial dermis and skin functions are lost. Generally caused by flash accidents, scalds or brief contact with hot objects i.e. stove element or curling iron.

Clinical features include:

- o Pain
- o Erythema
- o Marked edema
- o Blister formation
- o Moist appearance
- o Moderate pain
- o Heals within 14 -21 days



• *Partial- thickness (deep) burn*: Involves the epidermis, superficial and deep dermis and may be difficult to differentiate from a full-thickness injury.

Clinical features include:

- Epidermal and more dermal involvement with skin appendages (hair follicles, sebaceous and sweat glands) intact
- o Edema
- o Blistering thick walled and will increase in size
- o Dry, mottled appearance
- o Waxy-white color
- o Capillary refill (may or may not be present)
- Moderate to severe pain
- o Heal within 4-6 weeks
- Scar formation and possible contracture formation
- *Full-thickness burn*: Destroys the epidermis, dermis and epidermal derivatives i.e. hair follicles. It can also include deeper structures such as fat, muscle, nerves and bone. Skin functions are lost. Usually caused by flame, high intensity flash, chemical, electrical or prolonged contact with heat source. They cannot heal spontaneously and require surgical intervention.

Clinical features include:

- May be little sensation or pain due to destroyed nerve endings
- o Edema
- o White leathery or charred skin
- o No blisters
- o No capillary refill



6.5.2 Long Term Management of Burns

Once the initial acute management of the burned area is under control and the patient is stable, local management of the wound follows the same course of wound healing as other chronic wounds and the goals of wound care are "to control the growth of microorganisms, reduce the potential for invasive wound infection, prevent the wound from being a source of sepsis, and prepare the area for closure".

Deeper burn wounds may require specialized dressings, skin grafts, surgical interventions, etc. and these are usually performed through a specialized burn treatment center or unit.

6.5.3 Objectives of Wound Care

• Prevention of conversion

 wounds that dry out or develop an infection can become deeper. A partial-thickness wound could then convert to full-thickness and require skin grafting.

• Removal of devitalized tissue

• debridement, either through dressing changes or surgery, is necessary to clean the wounds and prepare for spontaneous healing or grafting.

• Preparation of healthy granulation tissue

 healthy tissue, free of eschar and nourished by a good blood supply, is essential for new skin formation.

• Minimization of systemic infection

• eschar contains many organisms. Removal is essential in order to decrease the bacterial load and reduce the risk of burn wound infection.

• Completion of the autografting process

 full-thickness wounds require the application of autologous skin grafts from available donor sites.

Limitation of scars and contractures

wounds that heal well the first time tend to have fewer scars and contractures.
 Some degree of scar and contracture formation are, however, part of the healing process and cannot be entirely prevented.

(Refer to Section 8, Products and Product Categories under antimicrobials for available dressings.)

6.5.4 Guidelines for Minor Burn Wound Care

Note: Clients should be considered for outpatient burn care only if the following considerations have been addressed.

- Intravenous fluid resuscitation is completed or not necessary.
- The client is able to maintain fluid balance with oral intake.
- Facilities for physical and occupational therapy on an outpatient basis are adequate.
- Pain control is adequate using oral medications.
- Family support and follow-up are arranged, and any abuse or neglect issues have been addressed.

• Follow-up is arranged at a facility with appropriate burn expertise for continued evaluation and treatment of infection, function, wound care and scarring.

• Tetanus Prophylaxis

Even clients with small burns are at risk of developing tetanus. All clients should receive appropriate tetanus prophylaxis unless they have been fully immunized or received a booster within the previous five years.

• Minor Burn Wounds

All minor burn wounds should be cleansed thoroughly with normal saline and all foreign material removed. Loose, devitalized tissue should be trimmed away. Blisters should be punctured and trimmed in areas where range of motion is inhibited, if it is a chemical burn injury and/or the blisters are large in size.

• Facial Burns

Action	Rational
Normal saline-soaked gauze dressings	Removes exudates and debris from face to
applied directly on burned areas and left on for	promote effective wound healing.
about 15 minutes twice daily.	
Polymyxin B Sulphate (Polyspoirn®) ointment	Prevents desiccation and infection, and
applied thinly to burned areas twice daily	possible conversion of the wound.
following saline soaks and as necessary	

• Tar Burns

- If the tar is not cool to touch, it should be actively cooled to stop further thermal damage by washing the skin with room temperature saline, (NEVER USE ICE).
- Do not rub as this can cause further damage.
- Tar can be removed using a number of emulsifying agents i.e. mineral oil or polymyxin B sulphate(Polyspoirn[®]).
- Several applications and gentle attempts at removal, without causing further damage to the underlying epidermis, may be required on an inpatient basis over the course of a few days.

6.5.5 Burn Unit Referral Criteria

Burn injuries that should be referred to a burn unit include the following:

- Partial thickness burns greater than 10% total body surface areas (TBSA).
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
- Third degree burns in any age group.
- Electrical burns, including lightning injury.

- Chemical burns.
- Inhalation injury.
- Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality.
- Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgement will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
- Burned children in hospitals without qualified personnel or equipment for the care of the children.
- Burn injury in patients who will require special social, emotional, or long term rehabilitation intervention.

6.6 Oncology Wounds

Wounds that result from cancerous tumors and metastasis present unique challenges to clinicians.

Types of Oncology Related Wounds

6.6.1. Etiology

- *Fungating wounds* are ulcerated "malignant skin lesions" which are open and drain. They can result from a primary cancer, a metastasis to the skin from a different tumor site, or a tumor at a distant location on the body. The lesions may look like a rapidly growing fungus or it can present as a cauliflower-like appearance that may ulcerate and form craters. Fungating wounds may result from almost any type of cancer but are most commonly associated with breast cancer. The wounds often become infected with anaerobic and aerobic organisms. The pungent odor that results is distressful to patients, families, and their caregivers.
- Radiation induced wounds are skin reactions and complications occurring from radiation therapy. A reaction may develop that progresses through erythema to dry desquamation and then moist desquamation when the skin receives a significant does of radiation therapy.
- Extravasation wounds results from the leakage of vesicant intravenous fluids or medications into the interstitial tissue surrounding an intravenous site. The injury to the tissue is dependent on the specific drug administered, its concentration, the amount of drug extravasated, the length of time the extravasation was occurring and the site of the extravasation.

 Malignant cutaneous wounds – are characterized by visible changes in the skin where an extension of cancer cells is observed through the epidermal/dermal layer. These lesions may result from a primary cancer or it may develop as a secondary infiltration in the late phases of disease.

6.6.2 Assessment

 Fungating wounds and malignant cutaneous lesions - The assessment and management of a fungating wound or cutaneous lesions is the same. It is based on identifying the objectives of symptom control and patient comfort as priorities rather than wound healing. It is often best to begin the assessment by asking the patient what aspect of the wound is most disturbing for them.

The assessment parameters for fungating wounds include:

- Appearance
- o Odor
- o Drainage/exudate
- Presence of infection
- o Periwound Skin
- o Size and shape of site
- o Pain
- o Bleeding
- Irradiated skin looks dry because sweat glands and sebaceous glands are destroyed. There may also be loss of elasticity. Other skin complications may include ulceration, necrosis, shedding or nail deformity and malignant tumors. Fibrosis of the lymph glands may cause lymphedema.
- Extravasation wounds frequently manifests as a burning pain and occasionally erythema at the injection site. Often, there may be swelling or bleb formation. The clinician should observe for swelling, stinging, burning, bleb formation, pain or redness.

6.6.3 Interventions

 Interventions may or may not support wound healing when caring for a fungating wound or cutaneous lesion. The wound is usually not healable.
 Dressing selection is based on a number of factors, in addition to efficacy and possibly even excluding efficacy.

The considerations for dressing selection should also include the following:

- o Cost
- o Reimbursement
- o Local availability

- Number of applications required
- o Complexity of the procedure
- Additional patient care needs
- Anticipated care provider
- Education requirement of care providers
- o Client comfort

There are three key objectives when managing a fungating wound. They are:

- o Wound pain management
- Odor management
- Control of exudate
- *Radiation wounds* experts disagree on the appropriate approach to treat radiation wounds. Some require leaving the area open to air while others suggest covering the area with a cream or nonadherent dressing. Nursing care is aimed at optimizing client comfort, promoting healing, and reducing the effects of radiation.
- *Extravasation wounds* look for the extent of tissue damage as it may not become evident for several weeks and require excision and skin grafting.

6.6.4 Pain Management

- Fungating wounds and cutaneous lesions are typically painful and trauma associated with dressing changes are a primary source of pain for patients. When selecting dressings for a fungating wound, it is important to consider using products that do not stick, may act as a hemostatic dressing to control bleeding, and provide protection to periwound skin. Appropriate medication for pain is crucial.
- Radiation wounds may be extremely painful, depending on the depth of tissue involvement and the outward signs of damage. Cool compresses may help and systemic analgesics should be administered as appropriate.
- Extravasation wounds can be painful. Depending on which agent has been infused, cold compresses may be contraindicated so the clinician should know all potential reactions and antidotes before beginning infusion.

6.6.5 Odour Management

 It is important to try to determine if the odor is caused by necrotic tissue, infection, or by saturated dressings. Sometimes, odor can come from all three of these. Odor can be controlled by wound cleansing, use of wound deordorizers, debridement, and treatment of infection. The topical application of the antimicrobial metronidazone has been reported to be effective in the reduction of odor by managing anerobe growth and infection. • Odor is not usually a problem in radiation or extravasation wounds unless the tissue becomes necrotic.

6.6.6 Debridement

- The use of products to promote autolytic debridement in a fungating wound is indicated if necrotic tissue is present. It is not recommended to mechanically debride because these wounds often bleed easily and are quite painful.
- Radiation wounds unless areas of necrosis occur, there may be no need for debridement in many radiation wounds.
- Extravasation debridement and surgical repair with skin graft is indicated in some types of extravasation wounds. As few as one third of all vesicant extravasations will develop ulcerations, therefore surgical debridement is not indicated in all extravasation wounds.

6.6.7 Exudate Control

• The use of exudate management products that can absorb high volumes of exudate will provide appropriate exudate management in the wound and facilitate a dressing change schedule that will not be too traumatic to the patient. The clinician should observe the dressing to look for strike through and base the decision to change the dressing on the level of exudate present in the dressing and the patient's report of comfort with the dressing. Radiation and extravasation wounds do not usually have exudate management problems.

6.6.8 Evaluation

The key indicator when evaluating the success or effectiveness of care to a patient with any oncology wound is to ask the patient if the care being provided has met their needs and determine if it has improved their quality of life.

6.6.9 Quality of Life

Quality of life is multifactoral and different for each client with an oncology-related wound. Key areas that comprise quality of life for clients include:

- Lifestyle factors: social, cultural and economic
- Aesthetics
- Management of bleeding
- Odor and exudate control
- Comfort: psychological and spiritual
- Pain management
- Control of treatment induced side effects

• Infection control

Section 6 – Appendix 1 Determination of Ankle Brachial Index (ABI)

Indications

Doppler ABI ratios are useful for determining the quality of underlying arterial blood flow for patients with peripheral limbs ulcers. This simple bedside check is not meant to replace formal vascular assessment. However, it is an additional way to validate the use of compression therapy if a thorough vascular assessment is unavailable.

For some patients such as diabetics, the values obtained may be influenced by concurrent disease processes. Calcification of blood vessels makes the underlying arteries resistant to compression causing a falsely high ABI value

Equipment BP cuff and manometer Portable Doppler Conducting gel

Procedure

- Have the patient resting in a supine position for at least 10 minutes before you begin.
- Place a standard BP cuff around the calf of the affected leg.
- Palpate the dorsalis pedis or posterior tibial pedal pulse.
- Place the doppler probe at a 45 degree angle (via a mound of conducting gel) in region of the pulse.
- When the pulse is heard (whooshing sound), inflate the BP cuff until the signal disappears.
- Release the cuff slowly listening for the signal to return.
- Record the value on the BP meter where the signal resumed. This is the ankle systolic pressure.
- Repeat this procedure over the brachial artery to find the brachial systolic pressure.



<u>Note:</u> To ensure reliability, readings should be repeated 2-3 times with the highest value used for the calculation of the ratio.

Results

Calculate the ABI value by dividing the ankle pressure by the highest brachial pressure. The results are presented as a ratio **ABI = Ankle pressure/Brachial pressure**

```
For example: Ankle pressure = 80
Brachial pressure = 120
A/B = I
```

```
ABI = 80/120 = 0.66
```

ABI Reading	Results indicate	
Greater than 1.0	Normal Arterial Circulation	
Less than 0.9	Mild Degree Arterial Disease	
0.5 – 0.8	Mixed arterial and venous disease	
Less than 0.5	Arterial disease	

Warning:

- Repeated or prolonged inflation of the cuff can cause the ankle pressure to falsely drop due to a hyperaemic response.
- If the arterial pulse is irregular the systolic pressure may vary from beat to beat. Be sure to repeat assessments on each limb using the highest value.
- Expert opinion varies regarding the absolute values for each category. It is better to err on the side of caution using higher values for each category until a formal vascular assessment can be done.

Note: Values less than 0.5 indicate limb threatening disease. A surgical consult is recommended. Use of high compression is contraindicated with anyone who has an ABI less than 0.8.

Section 6 – Appendix 2

Diabetic Foot Screen for Loss of Protective Sensation

Attached.

Diabetic Foot Screen for Loss of Protective Sensation

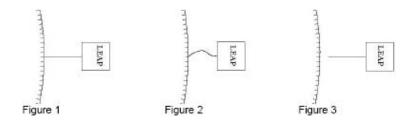
National Hansen's Disease Programs, LEAP Program, 1770 Physicians Park Dr., Baton Rouge, LA 70816

Filament Application Instructions:

- 1) Show the filament to the patient and touch it to his/her hand or arm so that he/she knows it does not hurt.
- 2) Use the 10 gram filament to test sensation at the indicated sites on each foot as shown. Apply the filament along the perimeter of and NOT on an ulcer, callous, scar, or necrotic tissue.



3) Hold the filament perpendicular to the skin and use a smooth motion when testing. Use a 3 step sequence that includes (1) touch the skin, (2) bend the filament, and (3) lift from the skin (See Figures 1-3). Do not use rapid movement. The approach, skin contact, and departure of the filament should be approximately 1½ seconds duration.



- 4) Ask the patient to respond "yes" when the filament is felt. If the patient does not respond when you touch a given point on the foot, continue on to another site. When you have completed the sequence, REPEAT the area(s) where the patient did not indicate feeling the filament.
- 5) Use the filament in a random sequence.
- 6) On the form, indicate with a minus sign, "—", the areas where the patient did not respond to the filament. LOSS OF PROTECTIVE SENSATION AT ANY ONE OF THE EIGHT SITES INDICATES A FOOT AT HIGH RISK.
- If you wish to clean the filament, use sodium hypochlorite (household bleach) 1:10 solution or follow the infection control disinfecting guidelines in your facility.

JF/06/2005

Section 7.0

WOUND ASSESSMENT AND DOCUMENTATION

7.1 Wound Assessment - Policy

The Registered/Licensed Practical Nurse must assess, manage and document wounds upon initial visit or occurrence of wound. The nurse must apply the Principles of Wound Care Management based on evidence researched practices as outlined in the Newfoundland and Labrador Provincial Skin and Wound Care Manual.

Wound assessment and documentation will be completed initially, weekly and with significant change in the patients condition or the wound following all the parameters as found on the wound assessment record.

Wounds requiring frequent dressing changes i.e. daily, assessment and documentation will include all the parameters as found on the wound assessment record with the exception of wound measurement and undermining tunnel/sinus.

The wound treatment plan will be evaluated at least every 2 weeks and when there is significant change in the wound.

Physician/Nurse Practitioner orders for wound management are not routinely required however, an order is necessary for certain management plans such as; conservative sharp debridement, compression, removal of sutures/clips, treating infection, the use of adjunctive therapy such as negative wound therapy and the application of silver nitrate (this list is not all inclusive).

When the order does not support evidence based practice the nurse will initially consult with the Physician/Nurse Practitioner to discuss the management plan. If a mutually agreed treatment plan cannot be developed with the ordering Physician/Nurse Practitioner the nurse will contact the wound resource person/manager and expected outcomes and resource implications associated with the initial order will be reviewed.

7.2 Wound Assessment Record

Please refer to Section 7 – Appendix 1 for "Wound Assessment Record".

Type of wound____

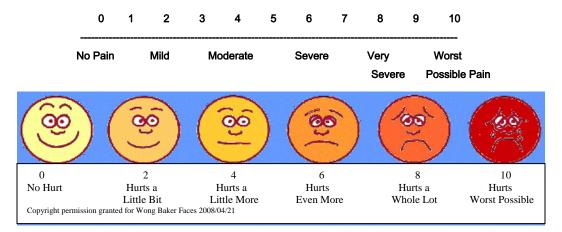
_

Location of Wound

Present on Admission
Yes No, Diabetic Yes No, Braden Score: Date: Date of Initial Assessment Identify client specific factors that could influence wound Healing e.g. allergies, smoking, etc.

DATE				
EXUDATE	Amt: Heavy Mod Light Nil Type: Serous Serosanguinous Purulent Sanguinous Tophi Other Odour: None Mild	Amt: Heavy Mod Light Nil Type: Serous Serosanguinous Purulent Sanguinous Tophi Other Odour: None Mild Foul	Amt: Heavy Mod Light Nil Type: Serous Serosanguinous Purulent Sanguinous Tophi Other Odour: None Mild Foul	
WOUND BED	Tissue type:EpithelializationGranulationHypergranulationSloughNecroticColor:PinkRedBlackGreenOtherFriableFriable	Tissue type:EpithelializationGranulationHypergranulationSloughNecroticColor:PinkRedBlackGreenOtherFriableFriable	Issue type: Epithelialization Granulation Hypergranulation Slough Necrotic Color: Pink Red Yellow Black Green Other Friable Friable	
WOUND EDGES	Attached Unattached Other	Attached Unattached Other	Attached Unattached Other	
PERIWOUND SKIN	Intact Induration Erythema Maceration Callus Other	Intact Induration Erythema Maceration Callus Other	Intact Induration Erythema Maceration Callus Other	
WOUND MEASURE	Lengthcm Widthcm Depthcm	Lengthcm Widthcm Depthcm	Lengthcm Widthcm Depthcm	
UNDERMINING TUNNEL/SINUS CLOCK METHOD (cm)	UnderminingTunnel/ SinusLocationLocationLengthLocationLocationLocationLengthLength	UnderminingTunnel/ SinusLocationLocationLengthLengthLocationLocationLengthLength	UnderminingTunnel/ SinusLocationLocationLengthLengthLocationLocationLengthLength	
WOUND PAIN	Intermittent Constant Nocturnal At intervention Other Pain Scale	Intermittent Constant Nocturnal At intervention Other Pain Scale	Intermittent Constant Nocturnal At intervention Other Pain Scale	
Management Plan	Infect. suspected: Yes No Swab C&S: Yes No	Infect. suspected: Yes No Swab C&S: Yes No	Infect. suspected: Yes No Swab C&S: Yes No	
Dressing				
Signature			:	

Analog Pain Scale



Definitions

Exudate Type	Color	Consistency	Description
Serous	Clear	Thin watery	Clear fluid absence of blood, pus debris
Serosanguineous	Light red/pink	Thin watery	Blood mixed with clear fluid
Sanguineous	Red	Thin watery	Bloody(not frank blood)
Purulent	Yellow/green	Thick, opaque	Pus, cloudy, viscous, often malodorous
Tophi	White	Thick white curds	Deposit of urates in tissue

Wound Bed	Description		
Epithelialization	Light or dark pink skin, regeneration of epidermis		
Granulation	Beefy red/pinkish red, bumpy, shiny tissue.		
Hypergranulation	Excessive production of granulation tissue		
Slough	Moist yellow/whitish or green-gray tissue, can be stringy, thick or thin		
Necrotic	Thick, black leathery crust		
Friable	Fragile wound tissue that bleeds or tears easily		
Wound Edges	Description		
Attached	No sides or wall present, even or flush with wound base, flat		
Unattached	Sides or wall are present, base of wound is deeper than edge		
Intact	Physically and functionally complete		
Other	- Fibrotic - Rolled under		
Periwound skin	Description		
Induration	Hardening of the tissue, sometimes elevated and extends out from wound margins		
Maceration	Softening of the skin caused by extended contact with excess fluids. Skin looks white, wrinkled and		
	waterlogged.		
Callous	Build up of hardened dead skin, usually on feet. Can occur around an ulcer. May indicate repeated		
	prolonged pressure or gait changes.		
Erythema	Redness of the skin usually caused by vasodilation, infection or injury.		
Other	- Dermatitis: Presents as scaly red papules or plaques (elevated spots)		
	- Dry - Scaling - Edematous -Tender		
Wound Measure	Description		
Length	Longest measurement		
Width	Widest measurement, as perpendicular to the length		
Depth	Deepest area of wound base		
Undermining	Extension of wound under peri-wound margins, can go in any direction. Indicate location using clock		
	method. (i.e., 4cm undermining from 6:00 to 8:00 o'clock)		
Tunnel	Narrow extension of wound in one direction from the wound bed. Indicate location using clock method.		
Sinus A narrow tunneling extension from the wound base extending into deeper body tissue. Indica			
	using clock method.		

Section 8.0

PRODUCTS AND PRODUCT CATEGORIES

The variety of wound care products available provides unique challenges to clinicians faced with selecting an appropriate dressing to apply to a wound. Often, there are several appropriate options. The decision is based on understanding the principle of wound healing that the clinician is trying to achieve and understanding what the dressing can contribute to the wound healing environment.

8.1 Wound Care Products

There are five rules to understand dressing choices. They are:

a. Categorization

Learn about the dressing by its generic category and compare new products with those that already make up that category.

b. Selection

Select the safest and most effective, user-friendly and cost effective dressing possible.

c. Change

Change the dressing based on the patient, wound and dressing assessment, taking into consideration manufacturer's guidelines, not on a standardized routine.

d. Evolution

As the wound moves through the phases of the wound healing process, evolve the dressing protocol to optimize wound healing. The same dressing approach will not necessarily be appropriate through all phases of wound healing.

e. Practice

Practice using dressing materials to learn how they perform and what "tricks of the trade" will optimize their performance.

8.2 Wound Care Product Categories

Dressings are divided into a category, based on how they behave and contribute to the healing process. A category is a generic way to identify what the product contributes to the wound healing environment. By understanding which category a dressing belongs to and by knowing what is desired for the wound care environment, the clinician will know immediately what products to select for the dressing.

Wound care product categories include:

- Wound Cleansing
- Wound Hydration
- Moisture Retention
- Exudate Management
- Specialty Products
 - Odor Management
 - Cover Dressing
 - Compression Bandages
 - o Antimicrobials

Section 8 - Appendix 1 identifies some of the commonly available products for each wound care category. The manufacturer of each product can be determined by comparing the number located over the product name to the manufacturers list provided in Section 8 - Appendix 2.

Section 8 – Appendix 1 Wound Care Products

Dressing Category	Examples	Principle Function	Advantages	Disadvantages
Non-adherent Dressings (Impregnated and non-impregnated with moisturizer) Passive Contact	Mepitel ¹⁰ Adaptic ⁸ Jelonet ¹¹ Sofratulle ¹¹ Petrolatum ⁹ Tegapore ⁴	Protect from injury	 Separates wound base from outer dressing, decreasing risk of damaging wound bed upon removal. Multiple layers can create partial occlusion thereby reducing moisture loss. 	 Can damage wound bed if particles dry into wound or granulation tissue grows over dressing. Requires secondary dressing to secure. Can develop sensitivity to some product components, e.g. antibiotics. Impregnated products are hydrophobic. They will not absorb or transmit drainage from wound to secondary dressing.
Transparent – Film Dressings Passive Primary or Secondary	Opsite ¹¹ Tegaderm ⁴ Bioclusive ⁸ Blisterfilm ⁹ Polyskin II ⁹ Comfeel Film ⁸ Cutifilm ² Mefilm ¹⁰	 Protect from injury. Maintain moist wound environment. Maintain a therapeutic environment. Film dressings with high moisture vapor permability are designated for intravenous sites e.g., IV 3000. 	 Provides a barrier to invasion and spread of microorganisms. Supports autolytic debridement. Allows visualization of wound. Self-adhesive product that allows gas and water vapor exchange. 	 Not recommended for draining wounds. Requires a border of intact, dry skin for securing dressing. Adherence may be difficult in highly mobile joint areas or over wet wounds. Adherent material is in direct contact with wound bed. Removal could strip off new tissue growth.

Legend: Active Versus Passive indicates presence or absence of interactivity between the wound and the product. Primary Dressings are placed in direct contact with the wound bed. Secondary Dressings are used over a primary dressing. Contact Dressings separate the wound bed from a secondary dressing.

Dressing Category	Examples	Principle Function	Advantages	Disadvantages
Hydrocolloid Dressings (Wafers, Pastes or Powders) • Active • Primary or Secondary	Tegaderm Hydrocolloid thin ⁸ Tegaderm Hydrocolloid ⁸ Duoderm [®] Extra Thin ¹ Duoderm [®] Signal ^{TM1} Duoderm [®] CGF ^{®1} Restore ⁷ Comfeel ³ Ultec Pro ⁹ Cutinova ² Hydrocol ⁵ Replicare ¹¹	 Protect from injury. Maintain moist wound environment. Maintain therapeutic environment. 	 Provides a barrier to invasion and spread of microorganisms. Supports autolytic debridement. Provides scant to moderate absorption. Conforms to fit. Water-resistant. 	 Not recommended for heavily draining wounds. Some controversy over use with infected wounds. Requires a border of intact, dry skin for securing dressing. Can appear "mucky" as product does not allow water vapor loss so drainage accumulates under dressing. Requires heat to ensure good adherence. Opaque texture limits wound visualization.
Hydrogel Dressings (gel or sheet form) Active Primary	Hypergel ¹⁰ Intrasite gel ¹¹ Restore gel rope ⁷ Duoderm [®] Hydroactive [®] Gel ¹ Norm gel ¹⁰ Woun'dres ³ Purilon ³ Tegagel ⁴ Restore ⁷ Hydrogel ⁷ Cutonova gel ² NuGel ⁸	 Maintain moist wound environment. Fill dead space. Support debridement. 	 Some products provide minimal absorption. Can be soothing, reduce pain. Some products contain ingredients claimed to speed healing e.g. collagen. Semi-transparent permeable to water vapored gas. 	 Not recommended for heavily draining wounds. Requires occlusive secondary cover to prevent leakage. Some gels become watery with body heat and will leak out on periwound skin. Protect periwound skin from excess moisture.
Absorbent Dressings (Hydrofiber®1 or impregnated gauze) • Active	Aquacel ^{®1} Mesalt ¹⁰	 Absorb excess drainage. Fill dead space. 	 Can absorb large amounts of drainage. Keeps wound bed moist without being soaked. Easy to remove – 	 Not recommended for wounds with minimal to no drainage. Can dry out wound bed.

Dressing Category	Examples	Principle Function	Advantages	Disadvantages
			flush with N/S.	 Requires secondary cover dressing to secure. Risk of "packing" dressing into wound too tightly.
 Foam Dressings (sheets or cavity) Passive Primary or Secondary 	Tielle Heel ⁸ Adhesive ⁸ Allevyn Heel ¹¹ Nonadhesive ¹¹ Optifoam ¹² Mepilex Border ¹⁰ Mepilex Nonadhesive ¹⁰ LyoFoam ^{®1} Allevyn ¹¹ Biatain ³ Cutinova ³ Reston ⁴ Flexzan ⁵	 Absorb excess drainage. Protect from injury. Maintain therapeutic environment (warmth). 	 Can absorb moderate to heavy amounts of drainage. Cavity foams can help fill dead space. Easy to remove. Physical structure provides "padding" which increases patient's comfort. Non-adherent, thus friendly to fragile peri-wound skin. 	 Not necessary for wounds with minimal to no drainage. Shape not conformable to irregular shaped wounds or wounds with undermining. May require secondary cover dressing to secure. Cutting cavity forms can lead to "chips" entering wound. Will not draw moisture from a non-exudatory wound.
 Alginate Dressings (fibre/rope or sheets) Active Primary or Secondary 	Curasorb ⁹ Kaltostat ^{®1} Medline dressing Sorbsan ⁵ AlgiSite ¹¹ SeaSorb ³ Dermacea ⁹ Restore ¹¹ CalciCare ⁷ Tegagen ⁴ Melgisorb ¹⁰ Cutinova ² FibraCol ⁸ Agicell	 Absorb excess drainage. Fill dead space. 	 Can absorb moderate to heavy amounts of drainage. Can be cut to size and layered to improve absorbency. Support removal of infected drainage. Product gels when wet which keeps wound bed moist. Some have hemostatic ability (calcium based). Easy to remove – rinse with N/S. 	 Not recommended for dry or lightly draining wounds. Require secondary cover dressing to secure. Some products fall apart in wound and may be difficult to remove.
Odor-Control Dressings Passive Primary or	Hollister Odor ¹¹ CarboFlex ^{™1} Carbonet ¹¹ Actisorb Silver 220 ⁸	Control odour	 Minimal absorptive ability. 	 Can lose effectiveness when wet. Need cover dressing or tape to secure wound.

Dressing Category	Examples	Principle Function	Advantages	Disadvantages
Se condar y Composite Dressings. Consists of a combination of non-adherent inner layer absorption layer, occlusive and/or adherent outer layer. Passive Primary or Secondary	Combiderm [™] ACD ^{™1} Combiderm [™] Non- Adhesive ¹ Versiva ^{®1} Allevyn ¹¹ Adhesive ¹¹ AllDress ¹⁰ Tielle ⁸ Biatain Adhesive ⁹ Ventex ⁹ Tegaderm ⁴ Coverlet ² Viasob ⁹	 Absorb excess drainage. Maintain moist environment. Protect from injury. 	 All in one dressing. Self-adhesive. Conforms well to fit. Water resistant (wash and wear). 	 Not recommended for dry or lightly draining wounds. Requires a border of dry and intact skin for securing. Deep wounds will also require a primary filler dressing.
Skin Solvents and Sealants (tube, spray, wipes)	3M No Sting ⁴ Skin Prep ¹¹ Sween Prep ³ AllKare Barrier and Remover ¹ No Sing Skin Gel ¹¹	 Protect form injury. 	 Decrease irritation and maceration to periwound skin. Increase adhesiveness of dressings/tapes. 	 May sting or irritate skin. Skin needs to be dry before secondary tape or dressing can be applied.
Compression Dressings (Elastic multi-layered bandages) Passive Primary or Secondary	3M Coban ⁴ Profore Light ¹¹ Dufore ¹³ SurePress ^{®1} Profore ¹¹ CircAid ³ Thera-Boot ³ CircAid Circplus ³	Supportive Therapy.	 Increases venous return decreasing venous congestion and edema. Decreases hypertrophic scarring. Comfort fit. Amount of support is variable depending upon number of layers. 	 Most require training to learn how to apply properly. Be sure to, follow manufacturers instructions. NEVER use on arterially compromised limbs. Can be warm to wear and noticeable. Lifetime commitment to use a compression bandage.
Collagen and Oxidised Regenerated Cellulose Dressings Freeze dried matrix • Active Primary	Promogran ⁸	 Binds and inactivates matrix metalloproteas- es which can be harmful to a wound in excessive quantities. Binds to naturally 	 Management of chronic wounds that are free of necrotic tissue and visible signs of infection 	 Known hypersensitivity to either components of the product, i.e collagen or ORC Oxidised regenerated cellulose).

Dressing Category	Examples	Principle Function	Advantages	Disadvantages
		occurring growth factors.		
nzymes (Creams, ointments) Active Primary	Collagenase ¹² Varidase Elase Travase Accuzyme Llypeigel ¹⁰ (gel)	Debride necrotic tissue.	 Can be applied at the bedside. Selective debridement (only works where placed). Painless when used correctly. 	 Requires moisture to work so needs moisture retentive cover dressing. Works better if eschar is scored or slit with scalpel. May require prescription. Can be inactivated if used with silver, iodine or mercury releasing products.
Other Products Active Primary 	Seasorb Ag ³ Silvercel ⁸ Aquacel [®] Ag ¹ Iodosorb ^{*11} Acticoat ¹¹ Prisma ⁸	Treat infection.	 Reduces the need for topical or systemic antibiotics. Iodosorb* changes color from brown to yellow as activated particles are depleted. 	 Wound must be moist for product to work Acticoat – can be inactivated by NaCI. May be slight burning sensation upon application. If these dressings dry out they can be difficult to remove. Acticoat has no method to "signal" end life of product.
Negative Pressure Wound Therapy	VAC ATS ¹⁴ Blue Sky	Promotes wound healing by increasing granulation tissue growth, removing infectious materials or other fluids and providing a moist healing environment.	Refer to Section 10	Refer to Section 10.

Section 8 – Appendix 2 Company Index

- 1. Convatec Canada 1-800-465-6302 www.convatec.com
- 2. Beiersdorf-jobst, Inc. 1-800-795-6278 www.jobst.net
- 3. Coloplast Sween Corporation 1-800-533-0464 www.coloplast.com
- 4. 3M Health Care 1-800-644-0197 www.mmm.com/healthcare
- 5. Dow Hickman, Pharmaceuticals Inc. 1-304-285-6420 www.bertek.com
- 6. Cook Canada Inc. 1-800-668-0300 www.cookgroup.com
- 7. Hollister Ltd. 1-800-263-7400 www.hollister.com
- Johnson & Johnson Medical, Inc. 1-800-423-5850 www.jnj.com
- 9. Kendall Sherwood 1-800-325-7472 www.kendallhg.com
- 10. Molnlycke 1-800-494-5134 www.molnlycke.net
- 11. Smith & Nephew 1-800-463-7439 www.smith-nephew.com

- 12. Medline 1-800-396-6996 <u>canada@medline.com</u>
- 13. Dermascience 1-800-387-5302 www.dermasciences.com
- 14. KCI Medical 1-800-668-5403 www.kci-medical.com

SECTION 9.0

DATA COLLECTION AND REPORTING

The Provincial Skin and Wound Management Committee approved the following three data collection and reporting processes, to be implemented in a three year, staged implementation plan.

These indicators are:

- 1. Chart audit of Braden Skin & Risk Assessment
- 2. Wound care best practice chart audit
- 3. Incidence tracking of various types of wounds

9.1 Chart Audit of Braden Skin & Risk Assessment

See Section 9 – Appendix 1 for Braden and Risk Assessment Audit

9.2 Wound Care Best Practice Chart Audit

Agency should conduct regular audits of health records to ensure that provincial standards and guidelines for wound care are met.

Audits should evaluate:

- Documentation of wound assessment;
- Wound assessment includes all parameters outlined in wound assessment record.

9.3 Incidence Tracking of Various Types of Wounds

Incidence tracking of various types of wounds will be the third phase of the data collection and reporting structure.

Section 9 – Appendix 1

Audit of Skin Risk Assessment – Braden Skin Risk Assessment

Pressure ulcers are a common problem across all health care settings. A preventative approach should include three steps:

- 1. Identifying individuals at risk for pressure ulcers
- 2. Implementing a preventative plan
- 3. Auditing the prevention plan

Using the steps listed above is an important means of reducing pressure ulcer prevalence and incidence.

Organizations need to monitor its success in preventing the development of pressure ulcers and make improvements to its prevention strategies and processes. Audits should be scheduled on a regular basis, and spontaneously audit to monitor changes. Organizations and/or practice settings will need to determine the required frequency of audits to meet individual needs.

An audit tool has been provided in the manual for organizations to use to monitor practices and processes regarding skin risk assessment and prevention strategies. The tool provided includes eight sections and will allow for the documentation of 10 chart audits. Appropriate answers to the questions will depend on the policy in place for each particular practice setting, e.g. "Was the Braden score reassessed appropriately for the practice area?". Depending on the practice setting, the information required for the audit will be obtained from paper format resident charts or from electronic resident charts.

Organizations should utilize the information obtained from this audit to identify goals the organization needs to meet and to make practice changes to ensure prediction of skin risk and to implement strategies to prevent pressure ulcers.

Section 10.0

SPECIAL PROCEDURES

10.1 Negative Pressure Wound Therapy Systems

Negative Pressure Wound Therapy is used in the treatment of complex wounds and/or wounds unresponsive to conventional therapy. Negative Pressure Wound Therapy (e.g. VAC), is indicated for clients who would benefit from a subatmospheric pressure device which may promote wound healing. This includes clients who would benefit from drainage and removal of infectious material or other fluids from wounds under the influence of continuous and/or intermittent negative pressure.

• Contraindications

Types of wounds for which Negative Pressure Wound Therapy currently is contraindicated include:

- o Malignant wounds
- o Untreated osteomyelitis
- o Necrotic tissue with eschar
- o Exposed blood vessels or organs
- o Non-enteric and unexplored fistula

• Precautions

Precautions should be taken for clients with:

- Active bleeding
- o Difficult wound haemostasis
- Bone fragments or sharp edges as they could puncture protective barriers, vessels or organs
- o Close proximity to weakened, irradiated or sutured blood vessels or organs
- Anticoagulant therapy Note: Acceptable INR level is less than 2.0.
- Policy
 - 1. A physician or a wound care specialist in consultation with a physician will determine the need for Negative Pressure Wound Therapy and to determine pressure settings, intensity and discontinuation of therapy.
 - 2. The Registered Nurse will review the Guidelines for Negative Pressure Wound Therapy and obtain the necessary competency.

- 3. Please refer to the "VAC. Therapy Clinical Guidelines" for procedures on application and removal of VAC. dressings, operating instructions and special considerations.
- 4. The Registered Nurse is responsible for performing the wound assessment and dressing application/change. The wound is measured weekly.
- 5. A Nutritional Consult should be considered on all clients requiring VAC Therapy.
- 6. Negative Pressure Wound Therapy should not be used or should be discontinued if:
 - A client has an allergic reaction, bleeding, bruising or unmanaged pain in response to Negative Pressure Wound Therapy
 - Negative Pressure Wound Therapy does not reduce wound size or granulation growth is not witnessed after 2 to 3 weeks
 - The wound deteriorates
 - An occlusive seal cannot be attained
 - A client cannot adhere to therapy, or it is not feasible to use Negative Pressure Wound Therapy in a given setting.

• Evaluation Process

 If the wound shows progress in healing during first 2 weeks of Negative Pressure Wound Therapy continue with treatment plan and reassess every 2 weeks to a maximum of 4 – 6 weeks or until wound is no longer appropriate for Negative Pressure Wound Therapy. If the wound shows no progress in healing during first 2 weeks of Negative Pressure Wound Therapy, consult the physician about the discontinuation of Negative Pressure Wound Therapy and other treatment options that will achieve moist wound healing.

• Dressing Change

Routine dressing changes should occur three times a week.

• Canister Change

The canister should be changed on a weekly basis or PRN if full.

• Infection Control

For further information on weekly cleaning, quality control checks and disposal, please refer to Guidelines for Cleaning and Disinfection of VAC System, As per regional policy.

10. 2 Silver Nitrate

• Policy

Application of silver nitrate is a specialty procedure. Application of silver nitrate requires a written physician's and/or nurse practitioner's order and will be performed by a registered nurse who is competent in the procedure. Prior to a registered nurse applying the silver nitrate independently, an education program and competency must be assessed.

Application of silver nitrate by a nurse will be limited to hypergranulation tissue in wounds, peristomal and peritube area after other interventions have been determined ineffective.

• Procedure

This is a specialty nursing skill. Nurses must be competent in use of silver nitrate to perform this procedure. Silver nitrate will be used by a registered nurse for removal of hypergranulation tissue from a wound, peristoma or peritube after other strategies have been tried.

• Supplies

Silver nitrate sticks Sterile water Appropriate dressing Gauze sponges Protective barrier wipe Normal saline Plastic apron/gown Non sterile gloves

Note: Silver nitrate stains clothes and inanimate surfaces.

- i. Check client's file for physician/nurse practitioner orders.
- ii. Activate silver nitrate stick by applying a drop of sterile water to silver tip of applicator. <u>Note:</u> Silver Nitrate is activated with sterile or distilled water, not tap water.
 - The strength of the action is controlled by the amount of water used to moisten the tip.
 - The more water on the tip, the less burn but the greater spread.
 - The less water on the tip, the more burn and less spread.
- iii. Apply activated silver nitrate tip to area of hypergranulation rotating the applicator until all silver nitrate is used or targeted area is covered.
- iv. Deactivate silver nitrate with normal saline.

- Cleanse wound with normal saline and apply dressing. ۷.
- Document amount of hypergranulation present at beginning of a. procedure, number of silver sticks used and client's vi.
 - tolerance of procedure.

Section 11.0

GLOSSARY OF TERMS

Adjunctive therapy – non-traditional methods of healing. Used in conjunction with wound dressings. Usually associated with advanced technology.

Aerobic bacteria – bacteria that thrive in an oxygen-rich environment.

Alginate – dressings derived from seaweed to produce a hydrophilic gel on contact with exudate. Some alginate dressings have hemostatic properties.

Alternating pressure mattress – dynamic air mattress in which the cells alternately inflate and deflate to reduce interface pressure.

Anaerobic bacteria – bacteria that thrive in an oxygen-free environment.

Angiogenesis – the production of new blood vessels in a wound – resulting in granulation tissue.

Ankle Brachial Index (ABI) – the ratio of systolic pressure in the ankle to that of the arm. It is measured with a Doppler and standard BP cuff. Used to determine arterial compromise in lower limbs.

Ankle Flare – the characteristic clinical sign evident in the region of the ankle associated with venous hypertension/varicose veins visible as a result of a number of engorged veins in the area.

Antibacterial - a substance that kills or inhibits bacteria.

Antibiotic – a chemical substance that kills or inhibits bacteria.

Antimicrobial – an agent that inhibits the growth of microbes.

Antiseptic (Topical) – product with antimicrobial activity designed for use on skin or other superficial tissues; may damage cells.

Assessment – information obtained via observation, questioning, physical examination and clinical investigations to establish a baseline for planning care.

Atherosclerotic – a thickening, hardening, and loss of elasticity of the blood vessel walls.

Athromatous – deposit or degenerative accumulation of lipid containing plaques on the innermost layer of the wall of an artery.

Atrophie Blanche – white avascular areas of scar tissue that are susceptible to skin breakdown. These areas can be quite painful.

Autolysis – the body's natural capacity for breaking down necrotic tissue.

Bacteremia – the presence of viable bacteria in the circulating blood.

Bacteriocidal – agent that destroys bacteria.

Bacteriostatic – agent that is capable of inhibiting the growth or multiplication of bacteria.

Bioburden – the presence of multiple microorganisms in the wound, originating from both endogenous and exogenous sources.

Biofilm – formed from communities of organisms. Usually encased in an extracellular polysaccharide called glycocalyx that they themselves produce. This glycocalyx protects the bacteria from antibiotics and accounts for the persistence of the infection.

Blanching – skin becomes paler when compression is applied as a result of local occlusion of capillaries. Limbs can also blanch with limb elevation.

Bottoming Out – expression used to describe inadequate support from a mattress overlay or seat cushion as determined by a "hand check." To perform a hand check, the caregiver places an outstretched hand (palm up) under the overlay or cushion below the pressure ulcer or that part of the body at risk for a pressure ulcer. If the caregiver feels less than an inch of support material, the patient has bottomed out and the support surface is therefore inadequate.

Callus – the build-up of hardened dead skin, usually on the feet. Can occur around an ulcer and is usually a sign of changed gait and repeated prolonged pressure.

Cell Migration – movement of cells in the repair process.

Cellulitis – inflammation and infection of the cells, associated with heat, redness, swelling and pain.

Champagne Leg – wide calf and narrow, woody ankle.

Charcoal Dressing – dressing composed of activated charcoal. Used to control odor.

Charcot Joint – a neuropathic deformity that occurs in the presence of diabetic neuropathy. Often as a result of trauma; may cause collapse of the arch of the foot and a rocker bottom deformity.

Chronic wound – a wound that has remained unhealed for more than 6 weeks.

Claudication – inadequate blood supply that produces severe pain in calf muscles during walking; subsides with rest.

Clean – containing no foreign material or debris.

Collagen – the most abundant protein in the body; found in bone, teeth, skin, hair, etc.

Colonized – the presence of replicating bacteria on the surface or in the tissue of a wound without indications of infection such as purulent exudate, foul odour, or surrounding inflammation. All Stage II, III, and IV pressure ulcers are colonized.

Compression – the deliberate application of pressure using elastic bandages. Application of a bandage or stocking which provides sufficient external pressure to relieve venous congestion or to reduce scarring.

Contaminated – containing non-replicating bacteria, other microorganisms, or foreign material. The term usually refers to bacterial contamination and in this context is synonymous with colonized. Wounds with bacterial counts of 10⁵ organisms per gram of tissue or less are generally considered contaminated; those with higher counts are generally considered infected.

Contact Layer – the first layer of dressing in contact with the wound bed.

Contraction – the pulling together of wound edges in the healing process.

Critical Colonization – the bacterial burden is rising due to multiplication of organisms where are now starting to cause a delay in healing. Critical colonization initiates the body's immune response locally but not systemically and will have an effect on healing.

Culture (Bacterial) – removal of tissue, fluid and/or bacteria from wound for the purpose of placing them in a growth medium in the laboratory to propagate to the point where they can be identified and tested for sensitivity to various antibiotics. Swab cultures are generally inadequate for this purpose.

Culture (Swab) – techniques involving the use of a swab to remove bacteria from a wound and place them in a growth medium for propagation and identification. Swab cultures obtained from the surface of a pressure ulcer are usually positive because of surface colonization and should not be used alone to diagnose ulcer infection.

Cytokine – substances other than growth factors that contribute to the regulation of cellular function and wound repair.

Dead Space – the space created by tissue loss. When tissue is debrided or lost from injury, an empty cavity, sinus tract, or tunnel is created between the wound base and the skin surface.

Debridement – the removal of devitalized or contaminated tissue through surgery, sharp debridement, larval therapy, autolysis or occlusive dressings.

- Autolytic Debridement the use of synthetic dressings to cover a wound and allow eschar to self-digest by the action of enzymes present in wound fluids.
- Enzymatic (Chemical) Debridement the topical application of proteolytic substances (enzymes) to breakdown devitalized tissue.
- Mechanical Debridement Removal of foreign material and devitalized or contaminated tissue from a wound by physical forces rather than by chemical (enzymatic) or natural (autolytic) forces. Examples are wet-to-dry dressings, wound irrigations, whirlpool, and dextranomers.
- Sharp Debridement removal of foreign material or devitalized tissue by a sharp instrument such as a scalpel. Laser debridement is also considered a type of sharp debridement.

Debris - remains of broken down or damaged cells or tissue.

Dehiscence – separation of the opposed edges of a surgical wound.

Demarcation – a line of separation between viable and non viable tissue.

Dependent – a dependent position is the fallen, limp or relaxed position of a limb or extremity.

Dessication – drying up of; dehydration.

Deterioration – negative course. Failure of the pressure ulcer to heal, as shown by wound enlargement that is not brought about by debridement.

Diabetic foot ulcer – ulceration of the foot as a result of underlying diabetic pathophysiology.

Doppler Ultrasound (in leg ulcer assessment) – the use of very high frequency sound in the detection and measurement of blood flow.

Edema – the presence of excessive amounts of fluid in the intercellular tissue spaces of the body.

Electrical Stimulation – the use of an electrical current to transfer energy to a wound. The type of electricity that is transferred is controlled by the electrical source.

Emollients – a mixture of water with a suspension of oil usually with emulsifiers and preservatives.

Epithelialization – when a wound bed is level with the surface, epithelial cells will migrate over the wound to complete healing.

Erythema – redness of the skin caused by inflammation or prolonged pressure.

- Blanchable Erythema reddened area that temporarily turns white or pale when pressure is applied with a fingertip. Blanchable erythema over a pressure site is usually due to a normal reactive hyperaemic response.
- Nonblanchable Erythema redness that persists when fingertip pressure is applied. Nonblanchable erythema over a pressure site is a symptom of a Stage I pressure ulcer.

Eschar – thick, hard, black, leathery, necrotic, devitalized tissue.

Evaluation – a critical appraisal or assessment; a judgement of the value, work, character or effectiveness of interventions.

Excoriation – where the skin has been traumatized – worn away or eroded as a result of incontinence, inappropriate dressings, or as a result of bodily fluids such as gastric fluids.

Exudate – serous fluid that has passed through the walls of a damaged or overextended vein. Accumulation of fluid in the wound. The fluid may be serous, serosanguineous or sanguinous. Consistency of the exudate may be thin, thick, milky or purulent.

Factitious wound - a self-inflicted wound

Fascia – a sheet or band of fibrous tissue that lies deep below the skin or encloses muscles and various organs of the body.

Fibrin – an insoluble protein that is essential to clotting of blood, formed from fibrinogen by action of thrombin.

Fibroblast – in wound healing, fibroblasts stimulate cell migration, angiogenesis, embryonic development and healing.

Fibrotic – formation of fibous connective tissue, usually as a repairative process.

Fistula – a passage that has formed between two organs, i.e. the bowl and the skin.

Friable – fragile, tears easily or bleeds easily. A term used to describe wound tissue that tears or bleeds easily.

Friction – trauma from heat caused by the movement or rubbing of the skin against an external surface.

Full Thickness Skin Loss – the absence of epidermis and dermis.

Fungating – a cancerous lesion involving the skin which is open and may be draining.

Gaiter Area – 2.5 cm below the malleolus to the lower one third of the calf.

Gangrene – devitalized, dead tissue caused by failure of the blood supply.

Granulation Tissue – the pink/red, moist tissue that contains new blood vessels, collagen, fibroblasts, and inflammatory cells, which fills an open, full-thickness wound when it starts to heal.

Growth Factors – proteins that affect the proliferation, movement, maturation, and biosynthetic activity of cells. For the purposes of this guideline, these are proteins that can be produced by living cells. Cytokines and peptides vital for proliferation in wound healing.

Haemotoma – a bruise or collection of blood in the tissues.

Haemostaisis – the control of bleeding.

Healing – a dynamic process in which anatomical and functional integrity is restored. This process can be monitored and measured. For wounds of the skin, it involves repair of the dermis (granulation tissue formation) and epidermis (epithelialization). Healed wounds represent a spectrum of repair: they can be ideally healed (tissue regeneration), minimally healed (temporary return of anatomical continuity), or acceptably healed (sustained functional and anatomical result). The acceptably healed wound is the ultimate outcome of wound healing but not necessarily the appropriate outcome for all patients.

- **Primary Intention Healing** closure and healing of wound edges using sutures, staples, steristrips or skin grafts.
- Secondary Intention Healing closure and healing of a wound by the formation of granulation tissue and epithelization.

Hemosiderin – brown staining or discoloration of the tissues due to deposits of iron byproduct.

Homoeostasis – the body's natural mechanism for maintaining health constancy and ensuring survival.

Hydrocolloid – hydrocolloids are formulations of elastomeric, adhesive, and gelling agents.

Hydrofiber – hydrofibers are nonwoven, white, cotton-like products comprised of fibers of carboxymethylcellulose.

Hydrogel – water-based products for re-hydrating necrotic tissue.

Hydrophilic – water loving – absorbent dressings.

Hydrophobic – water hating – non-absorbent dressings.

Hyperbaric Oxygen – oxygen at greater than atmospheric pressure that can be applied either to the whole client inside a pressurized chamber or to a localized area (such as an arm or leg) inside a smaller chamber.

Hypergranulation tissue (overgranulation) – excessive production of granulation tissue.

Incidence – the occurrence of an event over time. Data reflects the total number of new cases in relation to the total population of interest during a specified period of time.

Induration – term used to describe a hardened, elevated area of inflammation. Extends out from wound margins.

Infection – the presence of bacteria or other microorganisms in sufficient quantity to damage tissue or impair healing. Clinical experience has indicated that wounds can be classified as infected when the wound tissue contains 10⁵ or greater microorganisms per gram of tissue. Clinical signs of infection may not be present, especially in the immuno-compromised client or the client with a chronic wound.

Inflammatory Response – a localized protective response elicited by injury or destruction of tissues that serves to destroy, dilute, or wall off both the injurious agent and the injured tissue. Clinical signs include pain, heat, redness, swelling, and loss of function. Inflammation may be diminished or absent in immunosuppressed clients.

Insulation – maintenance of wound temperature close to body temperature.

Interactive dressing – a dressing that mediates changes within the wound bed or fluid.

Intermittent Claudication – cramplike pains in the legs caused by reduced arterial circulation, often exacerbated with exercise.

Irrigation – cleansing by a stream of fluid, preferably saline.

Ischemia – deficiency of arterial blood supply to a tissue, often leading to tissue necrosis.

Keratin – one of the components of the stratum corneum, the outermost layer of the epidermis.

Laceration – a tearing or splitting of the skin caused by blunt trauma.

La Place's Law – the theoretical pressure produced beneath a bandage can be calculated as follows:

 $P = 4630 \times N \times T$ C x W Where P = sub-bandage pressure (mmHg) N = number of layers T = tension within bandage (Kgforce) C = limb circumference (cm) W = width of bandage (cm)

A bandage applied with constant tension to a limb of normal proportions will automatically produce graduated compression with the highest pressure at the ankle. This pressure will gradually reduce up the leg as the circumference increases.

Leg ulcer – wound of the lower limb that is frequently chronic in nature.

Lipodermatosclerosis (LDS) – firm fibrotic skin and subcutaneous tissue. Gives the lower leg a "champagne glass" look.

Low Air Loss – a series of interconnected woven fabric air pillows that allow some air to escape through the support surface. The pillows can be variable inflated to adjust the level of pressure relief.

Lymphadema – a condition that develops when there is a tremendous increase in the volume of lymph produced and/or a major reduction in the capacity for lymph transport. Classified as high output or low output.

Maceration – the softening of tissue that becomes white and soggy after being moist or wet for a long time. In this context, it refers to degenerative change and disintegration of skin that has been kept too moist.

Macrophage – a phagocytic cell derived from the blood monocyte which plays a vital role in inflammation and initiates angiogenesis.

Malodour – unpleasant odour from a wound.

Maggot therapy – (also called larval therapy) – the use of live maggots in a wound to digest necrotic tissue.

Malnutrition – state of nutritional insufficiency due to either inadequate dietary intake or defective assimilation or utilization of food ingested.

Moisture – in the context of this document, moisture refers to skin moisture that may increase the risk of pressure ulcer development and impair healing of existing ulcers. Primary sources of skin moisture include perspiration, urine, feces, drainage from wounds, or fistulas.

Moisture retentive wound dressing – general term that refers to any dressing that is capable of consistently retaining moisture at the wound site by interfering with the natural evaporative loss of moisture vapor.

Moisture vapor transmission rate (MVTR) – moisture vapor transmission rate; measured in units of weight of moisture vapor per area of material per time period (eg., g/m²/day).

Monofilament – a device with a hairlike filament of different diameters that are touched to various areas of the body. Inability to sense the 5.07 monofilament correlates with neuropathy.

Multidisciplinary – a process where health care professionals representing expertise from various health care disciplines participate in a prevention or restorative based program standardizing and practicing pressure ulcer management. Multidisciplinary teams function across all sectors of health care.

Necrosis – death of tissue or an organ in response to injury, disease or occlusion of blood flow.

Necrosis/Necrotic Tissue – describes devitalized (dead) tissue, e.g., eschar and slough.

Needle Aspiration – removal of fluid from a cavity by suction, often to obtain a sample (aspirate) for culturing.

Negative Pressure Wound Therapy – a closed wound management system which facilitates a negative pressure across the complete wound interface through suction, thereby stimulating improved circulation and a reduction in exudates production.

Neuropathy – the impairment of nerve function and is one of the most frequently reported complications of diabetes.

Occlusive wound dressing – no liquids or gases can be transmitted through the dressing material.

Oedema – an unnatural accumulation of fluid in the interstitial spaces.

Offloading – the avoidance of mechanical stress to a wounded area. Commonly used for diabetic and neuropathic foot ulcers.

Osteomyelitis – a bone infection which can be both localized and generalized.

Partial Thickness – loss of epidermis and possible partial loss of dermis.

Pathogen – any disease producing agent or microorganism.

Peri-Wound – the skin region immediately surrounding the wound.

Phlebitis – inflammation of a vein.

Physiological wound environment – in a wound, the presence of the physical, chemical, and biotic (living) factors that are characteristic of healthy intact skin; desirable to facilitate the natural process of wound healing.

Pliable – supple; flexible.

Pressure ulcer – an area of localized damage to skin and underlying tissue caused by pressure, shear, friction and/or a combination of these.

Prevalence – assessment of the frequency of occurrence of an event, at a single point in time.

Proliferation – to produce new growth or offspring rapidly; to multiply.

Pyogenic – producing pus.

Purulent Discharge/Drainage – a product of inflammation that contains pus - e.g., cells (leukocytes, bacteria) and liquefied necrotic debris.

Pus – a product of inflammation usually caused by infection, containing used cells, debris and tissue elements.

Reactive hyperaemia – observed as red flushing of skin following a period of occlusion and ischaemia.

Recalcitrant – a recalcitrant wound is a chronic wound which has failed to respond to optimal standard wound care.

Rubor –a purple-red discoloration of the dependant lower limb that is thought to be caused by pooling of the blood within the chronically dilated arterioles.

Scab – collection of dried exudate attached to a wound, after injury to the skin that has caused bleeding.

Scaling – Abnormal shedding or accumulation of an upper layer of skin.

Segmental Pressures – similar to the Ankle Brachial Index (ABI), but instead of only doing the ankle blood pressure measurement, the clinician obtains additional blood pressure measurements on different sites along the leg. This helps determine the quality of arterial blood flowing down the extremeties with the specific purpose of determining the level of potential occlusions.

Semiocclusive dressing – no liquids are transmitted through dressing naturally; variable levels of gases can be transmitted through dressing material; most dressings are semiocclusive.

Sepsis – the presence of various pus-forming and other pathogenic organisms or their toxins, in the blood or tissues. Clinical signs of blood-borne sepsis include fever, tachycardia, hypotension, leukocytosis, and a deterioration in mental status. The same organism is often isolated in both the blood and the pressure ulcer.

Septicaemia – presence of pathogenic organisms or toxins in the bloodstream.

Sharp debridement – a method of debridement using scalpel or scissors to remove necrotic tissue.

Shear – mechanical force that acts on a unit area of skin in a direction parallel to the body's surface. Shear is affected by the amount of pressure exerted, the coefficient of friction between the

materials contacting each other, and the extent to which the body makes contact with the support surface.

Sinus Tract – an epithelial cell-lined tube from the outside of the body to inside. A cavity or channel underlying a wound that can involve an area larger than the visible surface of the wound. It is a pathway that can extend in any direction from the wound surface, which results in dead space with potential for abscess formation.

Skin Equivalent – a material used to cover open tissue that acts as a substitute for nascent (beginning) dermis and epidermis and that has at least some of the characteristics of human skin (e.g., amniotic tissue, xenografts, human allografts). For the purpose of this guideline, only tissue with viable, biologically active cells is given this designation.

Slough – necrotic (dead) tissue is the process of separating from viable portions of the body. It is seen as the accumulation of dead cellular debris on the wound surface, and tends to be yellow in colour due to the large amounts of leukocytes present. However, yellow tissue is not always indicative of slough but may be subcutaneous tissue, tendon or bone instead. Yellow or grey stringy necrotic tissue. A mixture of dead white cells, dead bacteria, rehydrated necrotic tissue and fibrous tissue.

Stasis – stagnation of blood caused by venous congestion.

Static Air Mattress – a vinyl mattress overlay composed of interconnected air cells that are inflated with a blower before use. The shifting of air among the cells distributes pressure uniformly over the support area to create a flotation effect.

Static Device – pressure-reducing devices designed to provide support characteristics that remain (or Static Support Surfaces) constant - i.e., do not cycle in time. Examples include foam overlays, cushions, and water mattresses.

Strike-through – evidence of wound exudate appearing on the outside of the wound dressing indicating a need for dressing change.

Synthetic wound dressing – dressings that are composed of man-made materials, such as polymers, as opposed to naturally occurring materials, such as cotton.

Telangiectasia – permanent dilation of superficial capillaries and venules. This is often referred to as areas of "starburst" vessels.

Tissue Biopsy – use of a sharp instrument to obtain a sample of skin, muscle, or bone.

Tissue viability – the ability of tissue to perform its normal function optimally.

Tophi – Deposits of uric acid crystals in the skin or around joints associated with gout.

Topical Antibiotic – a drug known to inhibit or kill microorganisms that can be applied locally to a tissue surface.

Topical Antiseptic – product with antimicrobial activity designed for use on skin or other superficial tissues; may damage some cells.

Transcutaneous Oxygen Measurements (Not tensions) – provides information about the ability of oxygen, being transported through the blood, to be delivered to the skin and underlying tissue. The process is completed with electrodes and the results help clinicians determine the healing potential in the tested areas.

Trochanter – bony prominence on the upper part of the femur.

Trophic – changes that occur as a result of inadequate circulation, such as loss of hair, thinning of skin, and ridging of nails.

Tunneling – a passageway under the surface of the skin that is generally open at the skin level; however, most of the tunneling is not visible.

Ulcer – a lesion of the skin which can be accompanied by necrotic tissue and caused by a number of factors.

Underlying Tissue – tissue that lies beneath the surface of the skin such as fatty tissue, supporting structures, muscle, and bone.

Undermining – a closed passageway under the surface of the skin that is open only at the skin surface. Generally it appears as an area of skin ulceration at the margins of the ulcer with skin overlaying the area. Undermining often develops from shearing forces.

Ultrasonic Doppler Waveforms – are obtained with a special doppler probe that produces a traced image of the amount of blood flowing through a vessel during the cardiac cycle. The shape of the waveform helps determine the amount of blood flow in the vessel.

Vacuum Assisted Wound Closure – a closed wound management system which facilitates a negative pressure across the complete wound interface through suction, thereby stimulating improved circulation.

Varicose Veins – a distended, engorged vein, usually as a result of incompetent valves or local trauma. The long saphenous vein is most commonly affected.

Varicosities – dilated tortuous superficial veins.

Vasoconstriction – the arteries and arterioles constrict under the influence of drugs, hormones or cold.

Vasodilatation – the lumen of blood vessels opens and widens, blood flow slows, and more oxygen can reach the tissues.

Venous Eczema – Eczema associated with the development of venous ulcers. Also known as venous or stasis dermatitis.

Venous Hypertension – back pressure on the venous system exerted either from central or pulmonary sources, or from extrinsic compression syndrome. For example, a mass, tumour, or tight girdle.

Venous Insuficiency – an obstruction which blocks outflow, valvular incompetence, which permits retrograde flow, or muscle pump failure, resulting in incomplete emptying of the venous system in the lower leg.

Venous Leg Ulcers - wounds that usually occur on the lower leg in people with venous insufficiency disease. Venous leg ulcers are also known by such terms as venous stasis ulcer and venous insuficiency. Ulcers result from chronic venous hypertension caused by the failure of the calf muscle pump.

Wound – a break in the epidermis that can be related to trauma or pathological changes within the skin or body.

Wound bed – (also called wound base) – uppermost viable tissue layer of the wound; may be covered with slough or eschar.

Wound margins – rim or border of wound.

Xenograft – another species (such as a pig) serves as donor for the tissue; also known as heterograft.

Section 12.0

BIBLIOGRAPHY

- Advanced Wound Care Manual. Health Care Corporation of St. John's and Lynn Power, Nursing Consultant, ARNNL, 2003.
- Agency for Health Care Policy and Research. Treatment of Pressure Ulcers. Clinical practice guidelines. Number 15 (1994). AHCPR Publication Number 95-0654. Rockville (MD): Agency for Health Care Policy and Research, Public Health Services, U.S. Department of Health and Human Resources, 5-11.
- Albina, J.E. Nutrition and wound healing. JPEN 1994:18:367-376.
- Allman, R.M.; Goode, P.S.; Patrick, M.M.; et al. Pressure ulcer risk factors among hospitalized patients with activity limitation JAMA 1995;273:865-870.
- Allman, R.M.; Laprade, C.A.; Noel, L.B.; et al. Pressure sores among hospitalized patients. Ann Int Med 1986: 105:337-342
- Alvarez, O.M.; Gilbreath, R.L. Thiamine influence on collagen during the granulation of skin wounds. J Surg Res 1982;32: 24-31.
- AMG Medical Inc. Material Safety Data Sheet. Montreal, Quebec.
- American Dietetic Association. Nutrition Care of the Older Adult: A handbook for dietetic professionals working throughout the continuum of care. 2nd ed. Chicago: Diana Faulhaver; 2004: 134-152 and 185-195.
- Arredondo, M.; Martinez, R.; Nunez, M.T.; et al. Inhibition of iron and copper uptake by iron, copper and zinc. Biol Res 2006; 39(1): 95-102.
- Baracos, A.; Nutrition assessment practical approaches. Clin Geriatr Med 1995; 11:674-708.
- Baronoski, S.; Ayello, E. (2004). "Wound Assessment," in Baronoski, and Ayello, Wound Care Essentials Practice Principles. New York: Lippincott Williams & Wilkins.
- Barton, P.; & Parslow, N.; (2001). Malignant wounds: Holistic assessment and management. In: Krasner, D. L.; Rodeheaver, G. T.; Sibbald, R. G. (Eds). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Third Edition. Wayne, PA: HMP Communications, 699-710.

- Barton & Parslow, 1996; Central West Corporation, 1998; Convatec, 1997; Iocono, Ehrlich, Gottrup, & Leaper, 1998; Ovington, 1998; University of Dundee, 1993; Van Rijswijk, 1995.
- Benbow, M. (2005). Evidence-based Wound Management. Whurr Publishers: London.
- Benbow, M. (2005). Evidence-Based Wound Management. Whurr Publishers: London, West, J.M. & Gimbel, M. L. (2000). Acute surgical and traumatic wound healing. In: R. A. Bryant, Acute & Chronic Wounds. Second Edition. Mosby: Elsevier, 189-196.
- Bergstrom, N. ; Bennett, M.A.; Carlson, C.E. et al. Treatment of pressure ulcers. Clinical Practice Guidelines. No 15. AHCPR Publication No 95-0652. Rockville, Md: Agency for Health Care Policy and Research, December 1994.
- Berslow, R.A.; Nutrition and air-fluidized beds: A literature review. Advances in wound care 1994; 7(3): 57-62.
- Blackburn, G.L.; Bistrian, B.R.; Maini, B.S.; et al. Nutritional and metabolic assessment of the hospitalized patient. J Parental Enter Nutr 1977; 1:11-22.
- Book by Bryant and Dix Acute & Chronic Wound, 3rd edition, 2007 Mosby.
- Book of Chronic Wound Care by Morrison, Ovington & Wilkie, Mosby 2004.
- Braden, B. J. (2001). Risk assessment in pressure ulcer prevention. In: Krasner, D. L.; Rodeheaver G. T.; Sibbald, R. G. (Eds). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Third Edition. Wayne, PA: HMP Communications, 641-651.
- Bray Group Ltd., Product Safety Data Sheet. Farington, Oxfordshire, UK.
- Bryant, R.A. & Nix, D.P. (2007). Acute & Chronic Wounds: Current Management Concepts (3rd ed.). Philadelphia, PA: Mosby.
- Bryant, R. A. (2000). Acute & Chronic Wounds: Nursing Management (2nd ed.). Mosby: St. Louis.
- Burrows, C.; Miller, R.; Townsend, D.; Bellefontaine, R.; MacKean, G.; Orstead, H.; & Keast, D. Best practice re commendations for the prevention and treatment of venous leg ulcers: update: 2006. Wound Care Canada 2006, 4 (1): 30-37.
- Canadian Diabetes Association. (2006). Diabetes and Footcare. http://www.diabetes.ca/Section_About/FactsIndex.asp

Center Inc. www.BurnResource.com

Charney, P.J.; Nutritional screening and assessment. In: A Skipper (Ed). Dietitian's hand book of enteral and parenteral nutrition. Maryland: Aspen Publication Inc. 1998. 3-24

- Chernoff, R.S. Milton KY, Lipschitz. The effect of a very high protein liquid formula on decubitus ulcer healing in long term tube fed institutionalized patients JADA 1990; 90 (9): 130-133.
- Coloplast Sween Skin Pamphlet , Moisturizing Products: Product Line Skin Care Without Compromise, M0402N06.06. Courtesy of Coloplast.
- Cutting, K. F.; and White, R. J. (2005). Criteria for Identifying Wound Infection Revisited, Ostomy/Wound Management, 51(1), 28-34.
- Demetriou, A.S.; Levenson, S.M.; Rettura, G.; et al. Vitamin and retinoic acid induced fibroblast differentiation in vitro. Surgery 1985; 98:931-934.
- Department of Health, Government of Nova Scotia. (2000). Evidence Based Wound Management Protocol.
- Dickerson, J.W.T. Ascorbic acid, zinc and wound healing. J Wound Care. 1993;2:350-353.

Dickhaut, S.C.; DeLee, J.C.; Page, C.P. Nutritional Status: Importance in predicting wound healing after amputation. J Bone Joint Surg 1984;66:71-75.

- Doughty, D.B.; Waldrop, J.; and Ramundo, J. Lower-Extreminity Ulcers of Vascular Etiology. In: Bryant, R.A. (2000). Acute and Chronic Wounds: Nursing Management. 2nd ed. Mosby: Toronto.
- Ehrlich, H.P.; Hunt, T.K. Effect of cortisone and vitamin A on wound healing. Ann Sur 968;167:324-328.
- Fish, J.; Knighton, J.; and Pape, S. A Systematic Approach to Acute Burn Management. Courtesy of Smith & Nephew.
- Goldberg, M. T. and McGinn-Byer, P. (2000). Oncology-related skin damage. In: R. A. Bryant, Acute & Chronic Wounds. Second Edition. Mosby: Elsevier, 367-386.
- Government of Nova Scotia. (2000). Evidence Based Wound Management Protocol.
- Hadley, A.; Fitzsimmons, L. Nutrition and wound healing. Top Clinical Nutrition 1990; 5(4): 72-81.
- Ham, R.J. Indicators of poor nutritional status in older Americans. Am Fam Phy 1992; 45(1); 219-226.
- Health Canada. Nutrition for a Healthy Pregnancy: National guidelines for the childbearing years. Ottawa: Minister of Public Works and Government Services Canada; 1999
- Heughan C.; Grish G.; Hunt, T.K. The effect of anemia on wound healing. Ann Surg 1974; 179: 163-165.

- Holloway, A.; Ooi, S.; & Weingarten, M. (2000). Anatomy and Physiology of the venous system. In: Management of Venous Insufficiency and Ulceration. Curative Health Service Inc.
- Holloway, G. A. (2001). Arterial ulcers: Assessment, classification, and management. In: Krasner, D. L.; Rodeheaver G. T.; Sibbald, R. G. (Eds). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Third Edition. Wayne, PA: HMP Communications, 495-503.
- Hunt, T.K. Critical care of wounds and wounded patients. In: Shoemaker, W.C.; Ayers, S.; Grenvik, A.; Holbrook, P.R.; and Thompson, W.L..(Eds). Text book of critical care. Philadelphia, Pa: WB Saunders. 1989.
- Jackobs, M.K. The cost of medical nutrition therapy in healing pressures. Topics in Clin Nutr 1999; 14(2): 41-47.
- Keast, D.H.; and Fraser, C. Treatment of chronic skin ulcers in individuals with anemia of chronic disease using recombinant human erythropoietin (EPO): A review of four cases. Ostomy and Wound Management. 2004;50:65-70.
- Keast, D.H.; Parslow, N.; Houghton, P.E.; Norton, L.; & Fraser, C. Best practice recommendations for the prevention and treatment of pressure ulcers: 2006. Wound Care Canada 2006, 4 (1): 19-29.
- King, J.C.; and Keen, C.L. Zinc. In: Shills, M.E.; Olson, J.A.; and Shake, M. (Eds). Modern nutrition in health and diseases. Philadelphia: Lea and Berger. 1994. 214-230.
- Knighton, J. (2007). Innovations in Burn Wound Care. Burn Resource Center Inc. www.BurnResource.com
- Knighton, J. (2007). Sunnybrook Health Sciences Center. Ross Tilley Burn Centre.
- Krasner, D.; Rodeheaver, G.; & Sibbald, R. (Eds.). (2007). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals (4th ed., pp. 29-43). Wayne, PA: HMP Communications.
- Krasner, D.; Rodeheaver, G.T.; & Sibbald, G. R. (Eds.). (2001). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals (3rd ed.,). Wayne, PA: HMP Communications.
- Krasner, D. (1997). Dressing decisions for the twenty-first century: on the cusp of a paradigm shift. In Krasner, D.; and Kane, D. (Eds.) Chronic Wound Care: A Clinical Source Book for Healthcare Professionals (pp. 139-150). Wayne, PA: Health Management Publications, Inc.
- Kuvshinoff, B.W.; Brodish, R.J.; and McFadden, D.W.; et al. Serum tranferrin as a prognostic indicator of spontaneous closure and mortality in gastrointestinal cutaneous fistula. Ann Surg 1993; 217:615-623.

- Levenson; S.M.; and Demetriou, A.A. Metabolic factors. In: wound healing; biochemical and clinical aspects. Cohen, I.K.; Dieglemann, R.F.; and Lindflad, W.J. (Eds). Philadelphia: WB Saunder Co, 1992. pp 248-273.
- Levenson, S.M.; and Seifter, E. Dysnutrition, wound healing and resistance to infection. Clin Plast Surg 1977; 4:375-388.
- Long, C.L.; Nelson, K.M.; Akin, J.M., Jr. A physiologic basis for the provision of fuel mixture in normal and stressed patients. J Trauma 1990; 30:1077-1086.
- Meltzer, A.; Everhart, J.E.; Unintentional weight loss in the United States. Am J Epidemiol 1995; 142:1039-1046.
- Moffatt, C. (2007). Compression Therapy in Practice. Trowbridge, Wiltshire: Wounds UK Publishing.
- Moffatt & O'Hare, 1995; O'Brien, Mureebe, Lossing, & Kerstein, 1998; Thomas, 1997; Seaman, 2000; Vowden, Goulding, & Vowden, 1996; WOCN, 1996.
- Morrison, M. & Moffatt, C. (1997). Leg ulcers. In: Morrison, M.; Moffatt, C.; Bridel-Nixon, J.; Bale, S. Nursing Management of Chronic Wounds, Second Edition. Mosby: Barcelona, Spain.
- National Academy of Sciences. Dietary Reference Intake for Vitamin C, Vitamin E, Selenium and Carotenoids. Food and nutrition board, Institute of Medicine, 2000.
- National Academy of Sciences. Dietary Reference Intakes for Vitamin A, Vitamin K, Aresenic, Boron, Copper, Iodine, Iron, Manganese, Molybedenum, Nickel, Silicon, Vanadium and Zinc. Food and nutrition board, Institute of Medicine. 2001.
- National Hansen's Disease Programs, LEAP Program, 1770 Physicians Park Drive., Baton Rouge, LA 70816.
- National Pressure Advisory Panel. New Staging Guidelines NPUAP. Washington, D.C. February 2007.
- Osterweil, D.; Wendt, P.F.; and Ferrell, B.A. Pressure ulcer and nutrition. In: Morely, J.E.; and Glick, Z. (Eds). Geriatric nutrition. A comprehensive review. New York: Raven Press.1995; 335-354.
- Pape, S.A.; Judkins, K.; and Settle, J.A.D. (2001). *Burns The first five days.* (2nd edition). Smith & Nephew, (2001)
- Payne, R. L.; & Martin, M. L. (1990). Defining and classifying skin tears: need for a common language, Ostomy and Wound Manual.

- Pinchcofsky-Devin, G; . Nutritional assessment and Intervention. In: Krasner, D.; and Kane, D. (Eds). Chronic Wound Care. Wayne, PA: Health Management Publications Inc. 1977; 73-83.
- Pinchofsky-Devin, D.; and Kaminski, M.V. Correlation of pressure sores and nutritional status. J Am Geriatr Soc 1986; 34:435-440.
- Prasad, A.S. Discovery of human zinc deficiency and studies in an experimental human model. Am J Clin Nutr 1991:53:403-412.
- Rayfield, E.J.; Ault, M.J.; and Keusch, G.T. et al. Infection and diabetes: The case for glucose control. Am J Med 1982; 72:439-450.
- Rodeheaver, G.T.; & Ratcliff, C.R. (2007) Wound Cleansing, Wound Irrigation, Wound Disinfection. In: Krasner, D. L.; Rodeheaver, G. T.; and Sibbald, R. G. (Eds). *Chronic wound care: A clinical source book for health care professionals* (4th ed.). Malvern, PA: HMP Communications. 331-342.
- Rolstad, B.S.; Ovington, L.G.; & Harris, A. (2000). Principles of wound management. In R.A. Bryant (Ed.), *Acute and Chronic Wounds: Nursing Management* (2nd ed.); 85–34. St. Louis: Mosby.
- Rudman, D.; Mattson, D.E.; Feller, A.G.; and Nagraj, H.S. A mortality risk for men in a Veterans Administration extended care facility. JPEN 1989;13(2):189-195.
- Rudman, D.; Mattson, D.E.; Nagraj, H.S. et al. Prognostic significance of serum cholesterol in nursing home men. 1988; JPEN 12(2):155-158.
- Sabioncell, A.; Rabatic, S.; Kadrnda-Loverence, M.; et al. Decreased phagocytosis and antibody dependent cellular cytotoxcity (ADCC) in type-1 diabetes. Biomedicine 1981; 35:227-229.
- Sandstrom, B. Micronutrient interactions; effects on absorption and bioavailability. Br. J Nutr 2001; 85 Suppl (2): S181-185.
- Seltzer, M.H.; Slocum, B.A.; Cataldi-Betcher, E.L..; et al. Instant nutritional assessment: absolute weight loss and surgical mortality. J Parental Enter Nutr 1982;6:218-221.
- Seltzer, M.H.; Bastidas, J.A.; Cooper, D.M. Instant nutritional assessment. JPEN 1979; 3:157-159
- Shaver, H.J.; Loper, J.A.; Lutes, R.A.; Nutritional status of nursing home patients. JPEN 1980 :4(4):367-70
- Sibbald, R. G.; Orsted, H.; Schultz, G. S.; Coutts, P.; and Keast, D. (2003). Preparing the Wound Bed 2003 Focus on Infection and Inflammation. Ostomy/Wound Management, 49(11), 24-51.

- Sibbald, R. G.; Williamson, D.; Falanga, V.; Cherry, G. W. (2001). Venous leg ulcers. In: Krasner, D. L.; Rodeheaver, G. T.; and Sibbald, R. G. (Eds). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Third Edition. Wayne, PA: HMP Communications, 483-494.
- Sussman, C.; and Jensen-Bates, B. (2001). Wound Care. 2nd edition. Aspen Publishers Inc.
- United States Department of Health and Human Services. Health Resources and Services Administration. Lower Extremity Amputation Program, LEAP. June 2003.
- Wagner D. Nutritional management of the pressure ulcer patient. Nestle Nutrition 1999.
- Weinsier, R.L.; Hunker, E.K.; and Krumdieck, C.L. Hospital malnutrition: A prospective evaluation of general medical patients during the course of hospitalization. Am J Clin Nutr 1979; 32:418-426.
- West, J.M., & Gimbel, M.L. (2000). Acute surgical and traumatic wound healing. In R.A. Bryant (Ed.), (Acute and chronic wounds: Nursing Management (2nd ed.). St. Louis: Mosby. 186-189,
- Wiersema-Bryant, L. A., & Kraemer, B. A. (2000). Vascular and neuropathic wounds: The diabetic wound. In R. A. Bryant, Acute & Chronic Wounds. Second Edition. Mosby: Elsevier, 301-315.
- Williamson, D.; Paterson, D. M.; and Sibbald, R. G. (2001). Vascular assessment. In: Krasner, D.L.; Rodeheaver, G. T.; Sibbald, R. G. (Eds). Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Third Edition. Wayne, PA: HMP Communications, 505-516.
- Wilson, R. (2000). Massive tissue loss: Burns. In R. A. Bryant, Acute & Chronic Wounds. Second Edition. Mosby: Elsevier, 189-196.
- Winkler, M. Surgery and wound healing. In: A Skipper, Ed. Dietitian's hand book of enteral and parenteral nutrition. Maryland: Aspen Publishers Inc. 1998;383-417.
- Winter, G.D.; Scales, J.T.; Effect of air drying and dressing on the surface of a wound. Nature 1963;197;91-92.
- Wolf, R.R.; O'Donnell T.F.; Stone, M.D.; et al. Investigations of factors determining the optimal glucose infusion rate in total parenteral nutrition. Metabolism 1980;29(9):892-900. www.BurnResource.com.

Web Site Resources

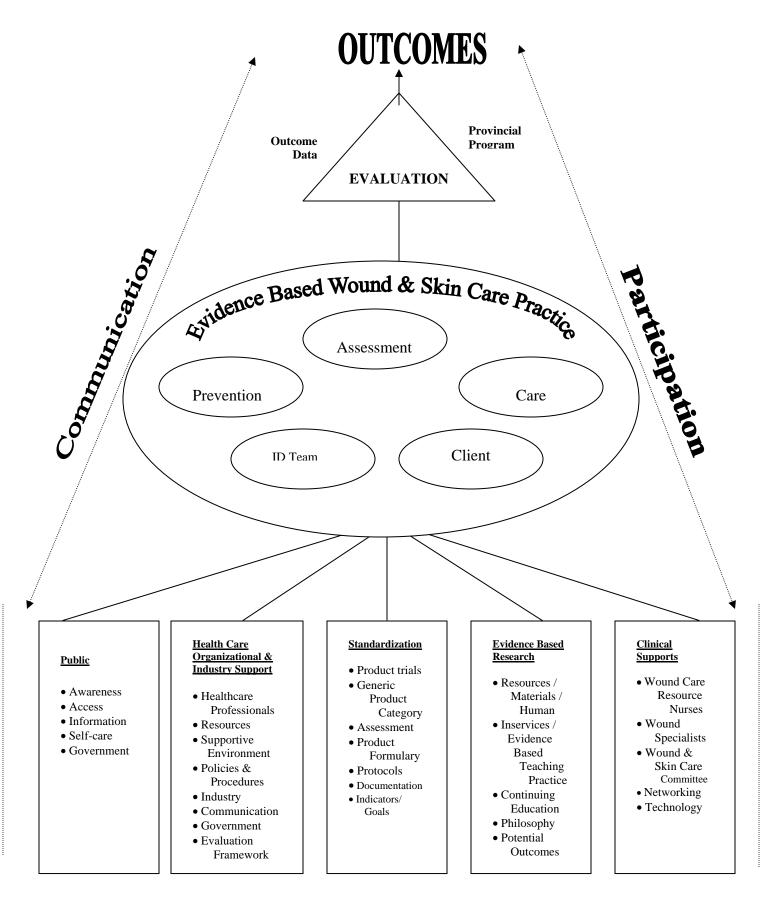
www.wocn.org	Website for the Wound, ostomy and continence nurses society (WOCN). A forum for discussion online journals, and access to professional resources.
www.woundsource.com	Access to Wound Product Source Book online, monthly newsletter, and professional resources.
www.smtl.co.uk/World-Wide.Wounds	Electronic journal of wound management practice, newsletter, and discussion.
www.woundcarenet.com	Online resource of the wound care communication network (WCCN) Springhouse corporation.
www.medicaledu.com/wndguide.com	The wound care information network education, discussion forums, and updates.
www.npuap.org	Site for the national pressure ulcer advisory panel (NPUAP). Provides information on PUSH tool.
www.ahrq.gov	Site for the Agency for Healthcare Research and Quality (AHRQ - Formally known as AHCPR) Provides guidelines, technology assessment, and outcomes.
www.woundheal.org	Site for the Wound Healing Society (WHS). Non- profit organization of clinical and basic scientific investigators interested in wound healing.
www.evidence.org	Online journal of Clinical Evidence-BMJ Publishing group and American College of Physicians.
www.cochrane.org	Collection of systematic reviews of the effects of health care interventions

Section 13.0

CONCEPTUAL MODEL

PROVINCIAL SKIN AND WOUND CARE PROGRAM

See attached.



EDUCATION

Structure

- Public
 - Facilitates health promotion/prevention strategies
 - o Promotes client teaching
 - Provides information to all sectors
 - o Facilitates marketing and awareness
 - Ensures access to prevention and care strategies
 - primary health care
 - availability
 - affordability (Blue Cross, financial resources)
 - o Fosters a self-care model
 - Encourages community partnerships

• Health Care and Industry Support

- o Is essential to support changes to clinical wound care practice and standardization
- o Supports and approves consistent policies and procedures
- Works closely with industry to meet efficient and effective wound healing goals
- Requires support and commitment of healthcare professionals nurses and physicians, etc.
- Provides effective communication
 - marketing to all key stakeholders and comprehensive data base
- Supports and provides adequate financial and human resources for program development and implementation
- Fosters supportive clinical environment

• <u>Standardization</u>

- Supports evidence-based practice
- Provides standardized product formulary based on generic product categories
 - Wound Cleansing
 - Wound Hydration
 - Moisture Retention
 - Exudate Management
 - Odor Control
 - Antimicrobial Management
- o Contains consistent policies and procedures
- o Provides standardized product formulary
- Ensures consistent assessment parameters
- Provides standardized documentation tools

• Evidence Based/Research

- o Is based on needs assessment
- Facilitates on-going research-based education for health care professionals
- Provides training based on Adult Learning Principles (engages learner, active learning, evolutionary, self-evaluation)

- Ensures adequate resources and materials (posters, reference information, videos, in-service sessions)
- Ensures a consistent evidence-based teaching package
- Provides a forum for evaluation
- Promotes client teaching and self-care
- Incorporates the RN, LPN enhanced Scope of Practice and skill development
- Targets all members of the Interdisciplinary Team to include nursing students, nursing staff, physicians and other healthcare providers
- o Participates in ongoing research
- Supports continuing education
- Fosters a change in philosophy around wound healing practices to include:
 - changes in attitudes and beliefs of the healthcare provider, client and administration
 - changes in clinical wound care practices (i.e. moist wound healing)

<u>Clinical Supports</u>

- Provides protocols, policies and procedures
- Supports mentoring to assist in changing clinical practice
- Ensures availability of wound care resource nurses/wound care specialists
- Supports networking and linkages (provincial initiatives ARNNL SIG, national organizations - Canadian Association of Wound Care (CAWC)
- Uses technology to advance knowledge and support evidence-based decisionmaking through:
 - information technology
 - workload measurement
 - data base
 - web sites

Process

Evidence Based Wound & Skin Care Practice

- <u>Client</u>
 - Collaborates and participates in interdisciplinary team
 - o Partners in care and service
 - o Consents to services freely
 - Involved in self-care and learning

• Interdisciplinary Team Approach

- o Leadership from provincial committee
- o Fostered through the wound and skin care committees and clinical practice areas
- o Provides a method for marketing/PR
- o Ensures various needs are met through referrals, assessment and interventions
- o Supports holistic and collaborative decision-making
- Supports interdisciplinary care planning, better utilization of human (regulated and unregulated) resources and supplies and ensuring improved client outcomes
- Ensures the client is an active participant within the interdisciplinary team

- Develops community partnerships
- <u>Prevention</u>
 - o Public information teaching
 - o Assesses risk for skin impairment
 - Based on a detailed history comorbidity
 - Promotes skin care measures social factors
 - Provides prevention strategies/interventions (support surfaces, pressure relief/reduction, off-loading, turning and positioning, chair tilt vs recline)
 - Promotes client teaching (causes/preventative measures, nutrition and hydration, activity, mobility and hygiene)

• <u>Assessment</u>

- o Provides assessment criteria/baseline data
- o Facilitates effective decision-making and care planning
- Provides reassessment of the wound to guide treatment decisions based on classification of wounds, measurement, color, exudates, etc.
- o Supports principles of wound healing/management
- o Requires documentation
- <u>Care</u>
 - o Promotes moist wound healing
 - Defines wound healing phases
 - o Provides reassessment criteria
 - Assesses client outcomes
 - Teaching
 - o Referrals
 - o Holistic care psychosocial/physical
 - o Rehabilitation
 - o Client
 - self-care
 - learning environment
 - access

OUTCOMES

- <u>Standardized Provincial Program</u>
 - Supports product category evaluation process:
 - Wound Cleansing
 - Wound Hydration
 - Moisture Retention
 - Exudate Management
- Specialty Products
 - Odor Management

- Cover Dressings
- Compression Bandages
- Antimicrobial Management
- Provides evaluation of education
- Enables review of program goals and objectives
- Standardized documentation tools:
 - Audits
 - Data analysis
- Outcome Data

•

- o Establishes Indicators
 - Client
 - Healing
 - Satisfaction
 - Professional/Provider
 - Knowledge
 - Attitude/motivation
 - Ownership
 - EBP
 - Collaboration
 - Population Health Status
 - Self-care
- o Facilitates an evaluation process
- Ensures standard definitions across the province
- o Captures and assesses provincial data to include:
 - cost analysis/benefits
 - prevalence rates
 - incidence/occurrence rates
 - infection rates
 - healing/success stories
 - self-care
 - time of referral
 - appropriateness of referrals
 - healing rates

Outcomes

- Monitors wound/skin management through data collection
- Assesses improvements on quality of life and client satisfaction
- Prevention
- Self-care