SKIN CARE AND PREVENTION OF WOUNDS

PART I: SKIN CARE AND PREVENTION

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Abstract

Skin is the body's largest organ, and it serves as a vital shield of armor for the other organs. Protecting the skin is critical to maintaining good health and quality of life. Regardless of the cause, once chronic wounds affect the skin, patients suffer both physically and emotionally. Medical professionals are called upon to develop skin care and wound prevention strategies to ensure long-term patient skin health and to identify risk factors before they have a negative effect on their patients.
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Statement of Need
Health care professionals are increasingly relied upon to educate the public about the prevention of skin injury, inflammation and cancer. Helping individuals understand early recognition of skin disease can prevent spread, and support cure.
Course Purpose
To provide nursing professionals with knowledge of skin disease recognition, and the importance of prevention and early detection.

Learning Objectives
1. Define chronic wound.
2. Describe common prevention strategies for various wounds.
3. Identify risk and lifestyle factors that impede wound healing.
4. Identify types of pressure ulcers, and prevention strategies.
5. Describe medical-surgical and self-care measures to treat scars.

Target Audience
Advanced Practice Registered Nurses, Registered Nurses, Licensed Practical Nurses, and Associates

Course Author & Director Disclosures
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Please take time to complete the self-assessment Knowledge Questions before reading the article. Opportunity to complete a self-assessment of knowledge learned will be provided at the end of the course.
1. The _____ is the deepest layer of the epidermis, which contains the stem cells.
   a. Stratum basale.
   b. Stratum corneum.
   c. Stratum spinosum.
   d. Stratum granulosum.

2. All of the following are epidermal cells EXCEPT:
   a. Melanocytes.
   b. Islet of Langerhans.
   c. Langerhan cells.
   d. Keratinocytes.

3. The formation of early reparative coagulum and the activation of intrinsic and extrinsic clotting mechanisms occur during the _____ stage of the wound healing process.
   a. Remodeling.
   b. Inflammatory.
   c. Proliferative.
   d. Hemostasis.

4. The rearrangement and mixing of collagen fibers initially deposited in the wound bed during the proliferative phase occurs during the ______ stage of the wound healing process.
   a. Remodeling.
   b. Inflammatory.
   c. Proliferative.
   d. Hemostasis
5. All of the following are local factors that affect the wound healing process EXCEPT:
   a. Oxygenation.
   b. Infection.
   c. Sex hormones.
   d. Venous insufficiency.

6. True or False: Transient hypoxia in the wound stimulates the wound healing process.
   a. True.
   b. False.

7. ____ refers to the mere presence of infectious organisms on a wound that have not undergone replication.
   a. Contamination.
   b. Colonization.
   c. Local infection.
   d. Invasive infection.

8. What is the hallmark characteristic of diabetic foot ulcers?
   a. Elevated WBCs.
   b. Elevated levels of metalloproteases in the wound
   c. Elevated neutrophil count.
   d. Elevated renal albumin levels.
9. Proteins essential to wound healing include all of the following EXCEPT:
   a. Nerve growth factor.
   b. Substance P.
   c. Calcitonin gene-related peptide.
   d. Albumin.

10. True or False. Low dose aspirin is useful in managing pain and inflammation associated with diabetic foot ulcers.
    a. True.
    b. False.
Introduction

The skin is the most extensive organ in the body, made up of about 18 square feet. It consists of approximately 16% of the total body weight. It is a part of the integumentary system, which protects the body from different types of damage, such as dehydration, abrasions, and infections. The skin is essentially a barrier to the outside world protecting the body from infectious microorganisms, radiation, and extremes of temperature. It is also the organ responsible for the production of vitamin D.

The skin is not only a barrier; it is also a sensory organ, which interacts with the external environment. It has receptors that receive external stimuli and transmit signals to the brain about touch, pain, vibration, and position. For example, the pain receptors, also called nociceptors, are activated when the skin is pricked or injured, sending pain signals to the brain.

This course is a basic and useful resource for nurses to help increase knowledge about skin care and wound management. It is a useful beginner and refresher course for all members of the healthcare team involved in the assessment, treatment and ongoing management of wounds. The guidelines outlined in this course are by no means a substitute for professional judgment but should support clinical decision-making in professional practice.

Skin Anatomy And Physiology

The skin is made up of two layers: epidermis, and dermis. The epidermis is the outermost layer of skin. It is the part that is
visible to the eye and where new skin cells are formed and shed. It is also made up of different layers, which consist of four different types of epidermal cells. The different layers of the epidermis are:

- Stratum basale
- Stratum spinosum
- Stratum granulosum
- Stratum lucidum
- Stratum corneum

At the deepest region of the epidermis is the layer called stratum basale. It contains the stem cells, which give rise to the other epidermal cells. The cells found in this layer are cuboidal keratinocytes, melanocytes, and Merkel cells.

Above the stratum basale is the next layer called stratum spinosum. This is where Langerhans cells are found along with rows of cells called spiny keratinocytes. The spines are projections called desmosomes, which are located between keratinocytes to bind them together and oppose friction.

The layer above the stratum spinosum is called stratum granulosum. This is where keratinocytes start to generate waxy lamellar granules, which serve as an impermeable barrier to water. These cells are so far removed from the dermal layer that they die due to lack of nutrients.

There is another layer found in the thick skin of the hands and feet called stratum lucidum. It is made of many rows of clear, dead keratinocytes that serves to protect the underlying layers.
Finally, the outermost layer of skin is called the stratum corneum. It is made of many rows of flattened, dead keratinocytes, which also protects the underlying layers. Dead keratinocytes are continuously shed from the surface of this layer and replaced by newly formed cells from the deeper layers. The different types of epidermal cells are listed below:

*Keratinocytes*

Keratinocytes are the main skin cells. As its name suggests, it produces a protein called keratin that gives the skin its strength and flexibility. It also serves as a waterproof for the skin’s surface.

*Melanocytes*

Melanocytes produce the pigment called melanin, which gives the skin its unique color. Melanin protects the skin from the ultraviolet damage by absorbing solar radiation.

*Langerhans Cells*

Langerhans cells are produced in the bone marrow and migrate to the skin surface to help fight infection. It helps the immune system fight infections by processing antigens coming into contact with the skin.

*Merkel Cells*

Merkel cells are specialized skin cells that are involved in touch perception. They create a disk along the deep edge of the epidermis and connect with the nerve endings in the dermis. They are found on the tips of fingers and toes as well as other specialized areas.
The other major layer of the skin is called the dermis. It is the deeper layer of the skin and about 0.55 mm thick. It contains blood vessels and nerves. Like the epidermis, it is also made up of sublayers, which support the epidermis: the papillary dermis; and, the reticular dermis.

The papillary dermis is a thin layer of tissue found under the epidermis and contains capillary blood vessels and some elastic and collagen fibers.

The deeper layer, reticular dermis, contains big bundles of collagen and elastic fibers that are parallel to the surface of the skin. These fibers provide the skin its elastic characteristics, protecting it from
shearing injury and other kinds of trauma, as well as allowing it to return to its resting state after being stretched or compressed. This is also the layer where hair follicles, sweat glands, and sebaceous glands are found.

A subcutaneous layer, also called the hypodermis, provides extra support and cushioning to the dermis and epidermis. It is mostly made up of adipose, connective and vascular tissues. Beneath this layer lie the muscles and bones.

**Skin Injuries And Wounds**

Wounds are a result of broken, injured or damaged skin. As mentioned in the introduction, there are many causes of skin injury. Some of these include mechanical, chemical, electrical, thermal, or radiation sources. The type of wound depends on the cause and damage produced. Some of the most common types caused by traumatic injury are abrasions, lacerations, rupture injuries, punctures, and penetrating wounds. Some wounds are caused by disease and prolonged friction and pressure such as decubitus ulcers (bed sores) and diabetes ulcers.

Some wounds are superficial and only require local first aid attention such as cleansing and dressing. Other wounds are more severe and need medical attention to prevent infection and loss of function, due to injury to the deeper structures, such as bone, muscle, tendon, arteries, and nerves. The purpose of wound healing is to prevent complications and preserve function. The preservation of cosmetic appearance is also important, though not the primary consideration for wound repair.
**Wound Causes and Types**

As mentioned previously, skin injury and the type of wound produced depends on the mechanism of injury, such as:

- **Inflammation**
- **Superficial or surface wounds and abrasions:**
  These types of wounds leave the deeper skin layers intact and they are usually caused by friction rubbing against an abrasive surface.
- **Deep abrasions:**
  These injuries are cuts and laceration that penetrate all the layers of the skin and injure the underlying tissues such as muscle or bone.
- **Burns**
- **Cancer (i.e., melanoma)**
- **Puncture wounds:**
  These injuries are caused by sharp objects that enter the skin; *i.e.*, being stabbed with a knife or pricked by a needle.
- **Human and animal bites:**
  Bites are wounds that can either be puncture wounds, abrasions, or a combination of both.
- **Pressure and venous ulcers**
- **Decubitus and diabetic ulcers (bed sores):**
  Decubitus ulcers are wounds that result from lack of sufficient blood supply to the skin caused by chronic pressure or friction on a specific part of the skin. These wounds usually occur in patients who are bedridden. Diabetic ulcers are seen in the extremities of diabetic patients. They are caused by poor circulation due to peripheral vascular disease or malnutrition.
- Infections (i.e., impetigo, measles, necrotizing fasciitis, fungal skin infections)
- Allergic reactions (i.e., contact dermatitis)
- Inflammation (i.e., psoriasis, eczema)

As mentioned previously, proper wound care is essential to prevent infection and other associated injuries, and to promote healing of the skin. The sections below are intended to provide refresher information on wound care and prevention as well as general skin care measures to protect from disease.

**Wound Healing Process**

The wound healing process is a complex, dynamic process that is made up of several highly integrated and overlapping events. It involves replacement of devitalized and missing cellular structures and tissue layers through the four stages of hemostasis, inflammation, proliferation, and remodeling. It is during these stages that the immune system initiates a coordinated cascade of events, which include:

- Chemotaxis
- Phagocytosis
- Neocollagenesis
- Collagen degradation
- Collagen remodeling

Additionally, other events may also occur such as angiogenesis, epithelization, and the production of new glycosaminoglycans (GAGs) and proteoglycans, all of which are also essential to wound healing.
The conclusion of these events ultimately leads to the replacement of normal skin structures with fibroblastic mediated scar tissue. It is important to note that these events may not go as well as they should. For example, an overproduction of fibrous tissue may occur during the proliferation stage resulting in a hypertrophic scar. An exaggerated overproduction can further lead to keloid formation.

As mentioned briefly above, the wound healing process undergoes four stages, which are the:

- Hemostasis stage
- Inflammatory stage
- Proliferation or granulation stage
- Remodeling stage

**Hemostasis Stage**

At the onset of initial injury, there is a steady flow of blood and lymphatic fluid. The outflow of these fluids triggers the formation of early reparative coagulum and the activation of intrinsic and extrinsic clotting mechanisms. Thrombocytes dictate the intrinsic mechanism while the injured tissues lead the extrinsic mechanism.

After vasoconstriction, platelets attach to the damaged endothelium lining and release adenosine diphosphate (ADP). This chemical promotes the formation of thrombocyte clusters, which closes the wound and prevents further bleeding. Once this happens, vasodilation occurs and allows the entry of more thrombocytes and other blood cells into the injured area.
Inflammatory Stage

The inflammatory stage starts right after hemostasis has been established and typically lasts for 1-2 days. Some experts agree that both the hemostasis and inflammatory stages overlap. The arrival of more thrombocytes and other inflammatory mediators results in the release numerous healing factors to continue the healing process.
These include the activated complement, transforming growth factor-beta, circulating monocytes, tissue macrophages, neutrophils, platelets, clotting factors, and serum proteins, among others.

Alpha-granules release the platelet-derived growth factor (PDGF), platelet factor IV, and transforming growth factor (TGF–β). These factors along with cytokines trigger and mediate various processes, which are:

- Inflammation
- Collagen degradation and collagenogenesis
- Transformation of fibroblasts to myoblasts
- Angiogenesis
- Re-epithelialization

The interleukins is a very important mediator of the inflammatory process. Vascular endothelial growth factor (VEGF) and other factors promote angiogenesis, and some have several roles such as fibroblast growth factor (FGF)–2, which affects both angiogenesis but and re-epithelialization. Histamine and serotonin are also released from thrombocytes. PDGF exhibits chemotaxis, which is helpful in fibroblastic mitosis. This fibroblastic movement results in the productive formation of collagen fibril in later stages of wound healing. Fibrinogen is cut into fibrin, which forms the framework for coagulation. Fibrin is a structural protein that provides essential support for the other cells of inflammation.

Inflammation is an important stage of wound healing. But this only applies if it is short-lived. This is because an impaired regulation of the
inflammatory process (i.e., chronic inflammation) can lead to long-term complications, such as poor wound healing.

*Proliferation or Granulation Stage*

Like the hemostasis and inflammatory stages, the proliferative phase overlaps with the inflammatory phase. It also plays an important role in re-epithelialization, angiogenesis, release of fibroblasts, and formation of the collagen extracellular matrix (ECM).

The proliferation stage is named so because of its involvement in fibroblast proliferation inside the wound bed. Fibroblasts are important in the creation and alteration of the ECM. At first, rapid type III collagen deposition occurs followed by its replacement with type I collagen during the remodeling stage. The earlier release of cytokines from mitotic fibroblasts and activated macrophages further improve angiogenesis and the formation of granulation tissues in this stage.

After the deposition of collagen as an extracellular matrix (ECM) and new cells have grown on the wound bed, wound healing enters the last stage, the remodeling stage.

*Remodeling Stage*

The remodeling phase involves the rearrangement and mixing of collagen fibers initially deposited in the wound bed during the proliferative stage. This stage can last for 2-3 weeks while other wounds may go through remodeling for as long a year. In fact, it is the longest stage of the wound healing process. It is at this stage that the type III collagen is transformed into type I collagen and the existing fibers are rearranged into a more orderly manner, which contributes to
the wound’s tensile strength; however, it is important to note that the net amount of collagen at this stage remains the same and does not increase. When wounds are repaired well, the final wound strength can be restored to 70-80% of the pre-wound strength.

**Factors Affecting Wound Healing**

There are several factors that can lead to impaired wound healing. These factors are often interrelated and coexist with each other. Generally speaking, the factors that affect wound repair can be categorized into local and systemic. Local factors include those that directly affect the characteristics of the wound itself. These include:

- Oxygenation
- Infection
- Presence of foreign bodies, and
- Venous insufficiency.

The systemic factors, on the other hand, affect the overall health or disease state of patients, which in turn affect their ability to heal. These include:

- Age and gender
- Sex hormones
- Stress
- Ischemia
- Diseases, such as diabetes, keloids, fibrosis, hereditary healing disorders, jaundice, uremia
- Obesity
- Medications, such as glucocorticoid steroids, non-steroidal anti-inflammatory drugs, chemotherapy
- Alcoholism and smoking
• Immunocompromised conditions: cancer, radiation therapy, AIDS
• Nutrition

**Oxygenation**

Oxygen is essential for cellular function, especially in the energy production through formation of ATP, which is vital for almost all wound-healing processes. Specifically, oxygen performs the following functions:

• Protects wounds from infection
• Mediates angiogenesis
• Increases keratinocyte differentiation, migration, and re-epithelialization
• Promotes fibroblast proliferation
• Enhances collagen synthesis
• Promotes wound contraction
• Increases superoxide production, which is a vital component of oxidative management of pathogens by polymorphonuclear leukocytes

When vascular structures are injured, there is a state of hypoxia that ensues in the cellular tissues. This means that microenvironment of the early wound is starved of oxygen. Multiple disease states and condition can contribute to this hypoxic state, such as advanced age and diabetes. These conditions promote poor tissue perfusion and create a hypoxic state of the wound. Chronic wounds are particularly hypoxic.
Wounds that continue to remain hypoxic do not heal properly and rapidly. Although transient hypoxia following an injury stimulates wound healing, a chronic hypoxic state significantly retards wound healing. In acute wounds, hypoxia stimulates the body’s inflammatory response. Specifically, it stimulates cytokine production (i.e., PDGF, TGF-β, VEGF, tumor necrosis factor-α (TNF-α), and endothelin-1) and growth factor production from macrophages, keratinocytes, and fibroblasts.¹

Infections

A skin injury creates an opening for microorganisms to penetrate deeper tissues and possibly cause infections. The state of infection, status of the immune system, and the replication ability of the microorganisms will determine whether the wound is infected or not. Generally, infection proceeds through the following stages:

- Contamination
- Colonization
- Local infection, and/or
- Invasive infection

Contamination refers to the mere presence of infectious organisms on a wound that have not undergone replication. Colonization on the other hand, refers to the presence of replicating microorganisms on the wound without tissue damage. Local infection is the middle stage wherein the replicating microorganisms have begun to inflict tissue damage to the wound. Invasive infection refers to an infection, which has also spread to other surrounding tissues beyond the wound site.²
As discussed previously, transient inflammation is a normal part of the wound-healing process. It is vital to the elimination of infectious microorganisms and subsequent decontamination of wounds. Without it, infection can set in easily.

The presence of bacteria and endotoxins can keep cytokine levels (i.e., interleukin-1 and TNF-α) elevated and prolong the inflammatory stage of wound healing. If this remains unabated, the wound may become chronically inflamed and fail to heal that in turn can lead to an elevated level of matrix metalloproteases (MMPs). These enzymes can degrade the extracellular matrix (ECM) initially formed during the early stage of the healing process. The elevated protease levels can also lead to a reduced level of the naturally occurring protease inhibitors. This upsets the balance and may cause rapid degradation of growth factors that are present in chronic wounds. Like other bacterial infections, the bacteria in infected wounds aggregate in the with extracellular polysaccharide matrix in the form of biofilms.

Developed biofilms become protective microenvironments for bacteria, making their eradication with conventional antibiotics more challenging. These microenvironments are more resistant to the phagocytic activity of polymorphonuclear neutrophils (PMNs). The three most commonly found infectious bacteria in wounds are:

1. *Staphylococcus aureus* (S. aureus)
2. *Pseudomonas aeruginosa* (P. aeruginosa)
3. β-hemolytic *streptococci*
Many chronic skin ulcers are probably due to the presence of biofilms containing *P. aeruginosa*. This mechanism may help explain why certain antibiotics fail to treat chronic wounds.

*Advancing Age*

Advancing age is a major risk factor for impaired wound healing. Several clinical and animal studies at both cellular and molecular levels have found changes and delays in wound healing associated with advancing age. While older patients are more prone to temporary delayed wound healing, the quality of the healed wound was not affected. Delayed wound healing in older patients is linked with altered inflammatory responses, such as:

- Late T-cell infiltration into the wound area
- Changes in chemokine production
- Decreased macrophage phagocytic capacity

A study of these changes in wound healing showed that every stage of the wound healing process undergoes characteristic age-related changes, including:

- Improved platelet aggregation
- Improved secretion of inflammatory mediators
- Late macrophage and lymphocytic infiltration
- Dysfunctional macrophages
- Reduced secretion of growth factors
- Late re-epithelialization, angiogenesis and collagen deposition
- Decreased collagen turnover and remodeling
- Reduced wound strength
Sex hormones also contribute to the age-related wound healing impairment. Studies have shown that men, more than women, are more affected by the delayed wound healing effects of age. This may be partly explained by the presence of greater estrogen levels in women (estrone and 17β-estradiol). Recent studies have discovered significant differences in gene expression between elderly and younger male wounds which are almost completely estrogen-regulated. Estrogen exerts its influence over wound healing through its regulation of genes associated with regeneration, matrix production, protease inhibition, epidermal function, and inflammation. Moreover, other studies suggest that estrogen can indeed enhance wound healing processes in older patients while androgens impact cutaneous wound healing adversely.

**Stress**

Stress is implicated as a negative factor on the overall general health as well as social behavior of humans. It triggers and contributes to the severity of disease states as heart disease, diabetes, migraines, and impaired wound healing. Stress leads to dysregulation of the immune response through its effects on the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal medullary axes or sympathetic nervous system.

Studies have shown that psychological stress especially causes significant delay in wound healing in both humans and animals. This effect was seen by the investigators in patients with Alzheimer’s disease and students undergoing academic stress during examinations.
The hypothalamic-pituitary-adrenal (HPA) and the sympathetic-adrenal medullary axes are both responsible for the regulation of hormonal release from pituitary and adrenal glands. The hormones released are:

- Adrenocorticotrophic hormones
- Cortisol
- Prolactin
- Catecholamines (such as, adrenaline and noradrenaline)

When stress acts on the hypothalamic-pituitary-adrenal (HPA), there is an upregulation of glucocorticoids and reduction of inflammatory mediator levels at the wound site. Other inflammatory mediators are also affected, such as IL-1α and IL-8, both of which are vital to the initial stages of wound healing, hemostasis and inflammation. Additionally, glucocorticoids affect immune cells via the following:

- Suppression of the differentiation and proliferation processes
- Regulation of gene transcription
- Reduction of cellular expression of adhesion molecules

Cortisol is an essential part of the fight and flight response during inflammation. It also affects the Th1-mediated immune responses that are important during the early stage of wound healing. In summary, psychological stress causes impairment of cell-mediated immunity at the wound site, which results in substantial delay of the wound healing process.

Psychological stress also has an impact. It can cause anxiety and depression, which in turn may adversely affect physiological and behavioral mechanisms and affect the overall prognosis. Aside from its direct effects on endocrine, immune, and emotional integrity, stress
predisposes patients to adopt coping mechanisms that may not necessarily be healthy such as smoking, elevated alcohol consumption, substance abuse, binge eating, poor sleep and physical activity.

**Diabetes**

Diabetes is a metabolic disease, which affects millions of people all over the world. Patients with diabetes have impaired wound healing that is well documented by studies. In addition, diabetic patients are more predisposed to develop chronic non-healing diabetic foot ulcers, which occur in approximately 15% of this population.

Diabetic foot ulcers are one of the serious complications of diabetes. In fact, more than eighty percent of diabetic foot ulcers lead to lower leg amputations.\(^9\) The impaired wound healing of diabetic foot ulcers and other cutaneous wounds in diabetic patients is attributed to several complex pathophysiological processes.

Similar to venous stasis conditions, diabetic foot ulcers and pressure ulcers that are chronic are frequently accompanied by hypoxic state of the wound.\(^10\) As mentioned previously, prolonged hypoxic states due to impaired blood perfusion and inadequate vascular generation is detrimental for wound healing. The lack of oxygenation magnifies the inflammatory response of the body, thus extending the injury time by
elevating the free radical levels in the wound.\textsuperscript{11} Additionally, elevated blood sugar levels can also add to the oxidative stress especially when the formation of reactive oxygen species (free radicals) surpasses the antioxidant capacity.\textsuperscript{12}

A hallmark characteristic of diabetic foot ulcers is the elevated levels of metalloproteases in the wound, which is about 60 times higher than normal wounds. The up-regulation of this type of protein enzyme promotes tissue destruction and prevents normal repair processes.\textsuperscript{13}

There are multiple cellular function impairments in diabetic wounds, such as:

- Faulty T-cell immunity
- Leukocyte chemotactic impairment
- Impaired phagocytic activity
- Lowered bactericidal capacity
- Dysfunctional fibroblasts and epidermal cells

These defects result in poor bacterial decontamination and delayed wound healing in diabetic patients.

As mentioned many times previously, a hypoxic state impairs the healing of diabetic foot ulcers. There have been multiple studies conducted in recent years investigating the processes involved. Specifically, these studies have suggested impairment in the endothelial progenitor cell mobilization and homing as well as reduction in vascular endothelial growth factor and primary pro-angiogenic levels in the wounds of diabetic patients.\textsuperscript{9}
Another contributing factor to diabetic foot ulcers is neuropathy. Proteins essential to wound healing include:

- Nerve growth factor
- Substance P
- Calcitonin gene-related peptide

These proteins enhance cellular chemotaxis and proliferation as well as promote growth factor production. Decreased levels of these neuropeptides have been associated with the development of diabetic foot ulcers. Additionally, sensory neurons also contribute to the modulation of immune defense mechanisms, with denervated skin significantly reducing leukocyte infiltration.\(^1^4\)

**Medication And Wound Healing**

Medications are known to play a role in wound healing. They do so via a variety of mechanisms, such as, the impairment of platelet function, and induction of inflammatory responses and cell proliferation. Some of the common medications that act on these mechanisms include glucocorticoids, non-steroidal anti-inflammatory drugs (NSAIDs), and chemotherapeutic drugs. These are further explained below.

**Glucocorticoids**

Systemic glucocorticoids such as prednisone, prednisolone, hydrocortisone, triamcinolone, and methylprednisolone are a mainstay in a variety of disease states requiring anti-inflammatory therapy. Their ability to inhibit wound healing through widespread anti-inflammatory action and suppression of fibroblast proliferation and collagen synthesis is well recognized. They cause incomplete tissue granulation and decreased wound contraction. Additionally, they
prevent the production of hypoxia-inducible factor-1 (HIF-1), which is a vital transcriptional factor in healing wounds.\textsuperscript{15} Aside from their effects on the wound itself, these systemic medications also contribute to the risk of wound infection because they suppress the immune system response.

On the other hand, topical application of glucocorticoids produces more favorable effects on wound healing. A low dose topical corticosteroid applied on chronic wounds has actually been found to accelerate wound healing, decrease pain and exudate, and inhibit hypergranulation tissue formation in more than half of the cases. These beneficial effects are almost in stark contrast to the effects of systemic glucocorticoids. However, this doesn’t negate the necessity of careful monitoring to prevent potential complications such as higher risk of infections with chronic use.

**Non-Steroidal Anti-inflammatory Drugs (NSAIDs)**

Non-steroidal anti-inflammatory drugs (NSAIDs) are mostly over the counter painkillers that are widely used in a variety of pain and inflammatory conditions such as osteoarthritis, migraine headaches, muscular pain, and fever. Examples include:

- Ibuprofen
- Naproxen
- Diclofenac
- Piroxicam
- Meloxicam

There are some studies that indicate the negative effects of short-term use of NSAIDs on wound healing. On the other hand, the long-term
effects of NSAIDs on wound healing are still unknown. Animal studies have found that oral ibuprofen exhibits anti-proliferative effects on wound healing which led to reduced fibroblast levels, weaker breaking strength, decreased wound contraction, late epithelialization,\textsuperscript{16} and impaired angiogenesis.\textsuperscript{17}

The role of low-dose aspirin, such as those used for cardioprotection in wound healing, is not also well known. As of now, clinical recommendations do not support the use of low dose aspirin in patients about to undergo surgery due to the increased risk of bleeding. The exception to this recommendation is in patients who are at increased risk of cardiovascular events.\textsuperscript{18} In terms of topical application of ibuprofen foam on chronic wounds, studies have shown that it promotes moist wound healing, decreases constant and temporary wound pain, and promotes chronic venous leg ulcer healing.\textsuperscript{19}

**Chemotherapeutic Drugs**

The majority of chemotherapeutic drugs hinder several processes vital to wound healing, such as:

- Cellular metabolism
- Rapid cell division
- Angiogenesis

Because of their inhibitory effects on these processes, they inhibit many of the pathways that are essential to wound healing, such as:

- DNA synthesis
- RNA synthesis
- Protein synthesis
The inhibition of these pathways leads to reduced fibroplasia and neovascularization of wounds.\textsuperscript{20}

Chemotherapeutic drugs holdup cell migrations into the wound, reduce initial wound matrix formation and collagen production, weaken fibroblastic proliferation, and prevent wound contraction.\textsuperscript{20} Moreover, these drugs are immunosuppressants. They slow down the immune system, thereby hindering the inflammatory stage of wound healing and putting patients at greater risk of infection at the wound site. These drugs weaken the immune system by inducing neutropenia, anemia, and thrombocytopenia, resulting in wound vulnerability to infection, poor perfusion, and increased risk of prolonged bleeding. The administration of adriamycin within three weeks prior to surgery causes low albumin levels.\textsuperscript{21} In addition to low albumin levels, low post-operative hemoglobin, progressive diseases, and electrocautery use are all well-known risk factors for the development of wound complications.\textsuperscript{22}

Novel chemotherapeutic drugs, which inhibit angiogenesis such as bevacizumab neutralize the vascular endothelial growth factor. These drugs act together with older generation chemotherapeutic drugs to limit oxygenation to tumors and, hence, limit their growth. These drugs have been documented to increase wound dehiscence. However, since these novel drugs are often given in conjunction with older generation chemotherapeutics, it is hard to sort out whether they alone affect wound repair. Regardless of the lack of data, current recommendations call for the discontinuation of angiogenesis inhibitors ahead of surgical procedures.
Lifestyle And Wound Complications

This section discusses how lifestyle patterns can affect skin condition and, notably, influence wound healing. In particular, the impact of obesity, alcohol, smoking and nutrition relative to wound complications is discussed.

Obesity

Obesity is a complex disorder that has doubled in the United States since 1980. Moreover, a recent CDC survey documented that obesity continues to increase among adults, children, and adolescents, with greater than thirty percent of U.S., adults and fifteen percent of children and adolescents being classified as obese.23

Obese patients are more likely to experience wound complications, including:23

- Skin wound infection
- Wound dehiscence
- Hematoma
- Seroma formation
- Pressure and diabetic ulcers
- Venous ulcers

These risks have been seen in patients about to undergo both bariatric and non-bariatric procedures.24 Specifically, there is a higher rate of infection at the site of surgical incision in obese patients. The resulting complications may be attributed to poorer tissue perfusion and ischemia in the subcutaneous adipose tissues. Alternatively, this may also be attributed to reduced antibiotic levels that reach the site. In
surgical wounds, the elevated wound in obese patients also add to wound dehiscence. As explained previously, wound tension elevates tissue pressure, thereby decreasing microvascular perfusion and oxygen availability to the wound.\textsuperscript{23,24}

The higher rate of incidence of pressure ulcers or pressure-related injuries in obese individuals is also due to hypovascularity, which is also related to poor tissue perfusion. This is because poor perfusion puts tissues at greater risk of these injuries. Additionally, obese patients who are bedridden or use wheelchairs may find it harder to be able to reposition themselves as frequently as medically advised.

Obese patients have extra skin folds on their bodies, which can become breeding ground of infectious microorganisms if not washed and cleaned regularly and properly. Skin folds may be left moist by sweat and contribute to subsequent infection and tissue damage. Also, there is increased friction between skin surfaces due to their prolonged contact, which can result in ulcer formation.\textsuperscript{23,24}

Aside from the local conditions, there are also systemic factors that contribute to impaired wound healing and complications in obese patients. Obesity and overweight states are frequently associated with stress, anxiety, and depression, all of which can diminish immune system response to infections.\textsuperscript{23}
Adipose tissues secrete chemicals called adipokines, which are also thought to contribute to impaired wound healing. The exact mechanism by which they negatively impact wound healing remains unknown.\textsuperscript{25} Moreover, dysfunctional peripheral blood mononuclear cells, reduced lymphocyte proliferation, and elevated peripheral cytokine levels are also thought to occur more in obese patients.

**Alcohol Consumption**

Studies in the recent years have collected clinical evidence that demonstrate the negative effects of alcohol exposure to wound healing and infection.\textsuperscript{26}

Alcohol exposure reduces host resistance, and ethanol intoxication at the time of tissue injury is a risk factor for greater predisposition to contracting infections susceptibility in the wound.\textsuperscript{27} The precise mechanisms by which alcohol affect the immune response is lacking, although studies suggest that their effects are dependent on the type and characteristic of alcohol exposure (\textit{i.e.}, chronic or acute alcohol exposure, amount consumed, duration of consumption, time from alcohol exposure, and alcohol withdrawal). Generally speaking, short-term acute alcohol exposure can lead to suppression of pro-inflammatory cytokine release in the face of inflammatory events. This is in line with the fact that there is higher rate of infection in patients with neutropenia and decreased phagocytic function in acute alcohol exposure.
It appears that the most significant impairment caused by acute alcohol exposure is in wound angiogenesis, which is decreased by more than fifty percent after a single ethanol exposure. The result is reduction in vascular endothelial growth factor levels and nuclear expression of HIF-1alpha in endothelial cells. The alcohol-associated decline in wound vascularity leads to wound hypoxia and increased oxidative stress. Alcohol also affects collagen production and alters protease balance at the wound site. On the other hand, chronic alcohol exposure is also thought to negatively impact wound healing although the mechanisms involved are less clear and requires more investigation.

**Smoking**

Smoking is a well-known risk factor for cardiovascular disease and events as well as various cancers. Its negative effects on wound healing are also well known and well studied.

Smokers who have recently undergone surgery are more likely to experience delayed wound healing of their incision sites and at greater risk of postoperative complications including infections, wound rupture, anastomotic leakage, wound and flap necrosis, epidermolysis, and reduced wound tensile strength. These effects are most notable in smokers who have undergone routine oral surgery and dental implantation.

Smoking has also been linked with poorer cosmetic prognosis following plastic and reconstructive procedures. In fact, it is a well-known fact that many plastic surgeons are hesitant in performing cosmetic
surgeries on patients who refuse to quit smoking prior to their procedures.\textsuperscript{29}

Tobacco smoke contains a multitude of harmful substances, the most documented of which are nicotine, carbon monoxide, and hydrogen cyanide. Nicotine is thought to interfere with oxygenation by promoting tissue ischemia because of its vasoconstrictive effects. It also triggers sympathetic nervous stimulation, leading to the release of epinephrine, which also precipitates peripheral vasoconstriction and reduced tissue blood flow. It also makes the blood more viscous due to its inhibition of fibrinolytic activity and augmentation of platelet adhesiveness.

Additionally, nicotine and carbon monoxide in cigarette smoke cause tissue hypoxia. Carbon monoxide exhibits an aggressive reversible binding affinity for hemoglobin, which is primarily responsible for oxygen transport to cells. In fact, its affinity is 200 times greater than that of oxygen, which results in reduced fraction of oxygenated hemoglobin in the circulation.

Hydrogen cyanide is another toxic component of tobacco smoke with a well-studied effect on cellular oxygen metabolism. It causes impaired cellular function, which results in defective oxygen consumption in the tissues. Aside from these direct tissue effects, smoking significantly predisposes patients to atherosclerosis and chronic obstructive
pulmonary disease (COPD), both of which can also cause reduced tissue oxygen tension.\textsuperscript{29}

Tobacco smoke adversely affects the different stages of the wound healing process. During the inflammatory stage, it causes impaired white blood cell migration, which can lead to decreased monocytes and macrophage levels in the wound site. It is also at this stage that it decreases neutrophil bactericidal activity. Additionally, it also interferes with the normal lymphocyte activity, cytotoxic functions of natural killer cells, and production of IL-1. These effects lead to poor wound healing and greater risk of wound infection.

During the proliferative stage of wound healing, tobacco smoke causes depressed fibroblast migration and proliferation, decreased wound contraction, impaired epithelial regeneration, reduced extracellular matrix production (ECM), and protease imbalance. It is important to note that tobacco smoke has additional negative effects because it contains many toxic chemicals, which directly affect wound healing. It is therefore essential for patients to be counseled on smoking cessation to improve tissue repair and reduce the risk of wound infection.

**Nutrition**

Adequate nutrition is essential to wound healing. The vitamins involved in wound healing are primarily A and C. Some small studies have also found vitamin E to play a role in wound healing. Vitamin C is an essential cofactor in the healing process, directly affecting the synthesis of collagen and neutrophil function. Acting as a cofactor in the hydroxylation of proline and lysine, vitamin C allows the
cross-linking of collagen. Without adequate supply of vitamin C, skin breakdown and impaired wound healing occur. As an immunodefense cofactor, vitamin C acts as a potent antioxidant, which prevents toxic superoxide radical formation. Body stores of vitamin C last 4-5 months, and severe deficiency is unlikely to be observed in a person consuming the average Western diet.

Vitamin A deficiency impairs wound healing by decreasing collagen synthesis, its cross-linking, wound epithelialization, and tensile strength. Vitamin E is also a powerful antioxidant that is essential for the maintenance of healthy skin. Similar to vitamins A and C, it reacts with reactive oxygen species to prevent the formation of free radical, which are responsible for skin damage. It plays an important role in protecting the skin from the harmful effects of the sun. It affords photoprotection by absorbing ultraviolet (UV) light. In addition, some studies have also found that vitamin E contributes to anti-inflammatory activity.

Protein is also another essential nutrient in wound healing. In fact, protein deficiency can lead to dysfunctional:

- Capillary formation
- Fibroblast proliferation
- Proteoglycan synthesis
- Collagen synthesis
- Wound remodeling

Protein deficiency also adversely impacts the immune system response resulting in decreased leukocyte phagocytosis and greater susceptibility to infection.\textsuperscript{30} Collagen is an important structural protein
that makes up the connective tissues. It is mostly made up of the amino acids glycine, proline, and hydroxyproline. Collagen synthesis needs hydroxylation of lysine and proline as well as co-factors such as iron and vitamin C. Vitamin C deficiency and anemia can therefore lead to impaired wound healing.

Arginine is another amino acid that plays a role in wound healing. It is needed in times of maximal growth, severe stress, and injury. It exerts several effects in the body, such as:
- Modulation of immune function
- Wound healing
- Hormone secretion
- Vascular tone
- Endothelial function

Additionally, arginine is also a precursor to proline. Because of this, adequate arginine levels are required to support collagen deposition, angiogenesis, and wound contraction. Arginine also enhances the immune response. This is especially apparent during periods of psychological stress wherein the metabolic demand for arginine rises. In fact, exogenous supplementation of arginine has demonstrated efficacy as adjuvant therapy in wound healing.31

Another amino acid involved in wound healing is glutamine. It is the most abundant amino acid in plasma. It is also a primary source of energy for cellular metabolism of fibroblasts, lymphocytes, epithelial cells, and macrophages.31 There is a marked decrease in serum concentration of glutamine following major surgery, trauma, and sepsis. As such, its exogenous supplementation enhances nitrogen
balance and reduces immunosuppression. In addition, glutamine plays an important role in stimulating the initial inflammatory immune response during the early stages of wound healing. In fact, oral glutamine supplementation has demonstrated marked improvements in wound breaking strength and levels of mature collagen.

**Categories Of Wound Healing**

There are four different categories of wound healing, all of which have the same goal: to repair damaged tissues and reduce tissue defect. These categories are:

1. Primary healing
2. Delayed primary healing
3. Healing by secondary intention
4. Healing with partial thickness wounds

It is important to note that although these various categories exist, the cellular and extracellular processes and components involved are similar.

**General Wound Care Measures**

As mentioned previously, mild wounds can be treated at home with routine first aid measures such as thorough washing and dressing to prevent infection. However, some wounds are severe or pose greater health risk. As such, these wounds require immediate medical referral.

Some instances when referrals are needed are outlined below.

- Wounds caused by a significant trauma and other injuries are present.
- Hemorrhaging wounds that cannot be stopped with persistent pressure and elevation.
- Gaping wounds that require sutures. The size and location of the wound are important considerations. Most facial wounds may need to be repaired for cosmetic reasons, especially if they involve the lip or eye.
- Animal bites such as dog bites, cat bites, and human bites because of their high risk of infection and rabies.
- Dirty wounds that cannot be easily cleansed and dressed.
- Wounds with unmistakable evidence of infection such as redness, swelling, increased pain, and pus.
- Wounds in patients with outdated tetanus immunizations. These patients require a booster shot within 48 hours of injury. If the patient has never been immunized, the initial tetanus prevention with immunoglobulin should be administered as soon as possible.

Most minor and superficial wounds may be cared for at home. Superficial abrasions and lacerations can be cleansed; after cleansing, an antibacterial ointment may be applied, and then dressed appropriately.

In case of bleeding, direct pressure should be applied to the wound to control it, and if possible, the bleeding site elevated above the level of the heart. Doing so allows gravity to reduce blood flow to the injury. Most bleeding will stop within 10 minutes, at which point, a dressing can be placed over the wound.
In cases where bleeding is not a problem, a wound can be cleaned using tap water to clear it of debris to reduce the risk of infection. Only clean water should be used. Fresh water from rivers and lakes can contain many types of bacteria that may cause infection and should therefore be avoided. Deep wounds should not be scrubbed.

**Venous (Stasis) Ulcers**

Venous ulcers are also referred to as *stasis* ulcers. They commonly occur in the lower extremities. The most significant causes of venous ulcers include inflammatory events, which eventually lead to activation of leucocytes, endothelial damage, platelet aggregation and intracellular edema.\(^{32}\)

A venous ulcer is defined as a chronic sore on a leg or foot, which has failed to heal in the last four to six weeks. The most common site where a venous ulcer appears is the inside of the leg right above the ankle.\(^{35}\)

**Causes of a Venous Ulcer**

There are various factors that can lead to a venous ulcer of the leg including a minor injury exposed to constant high pressure which further damages the skin.\(^{35}\) In venous ulcers, veins fail to send the blood back to the heart resulting in an accumulation of blood and elevated pressure. If not treated on time, it eventually leads to the formation of an open sore.

Venous insufficiency is the condition which results from weakened veins in the leg being unable to push back the blood towards the heart and weakening the valves. The weakened valves cause blood to flow backwards and accumulate in the legs.
Venous insufficiency is one of the major causes of venous ulcers. Venous ulcers are a common problem especially in older patients. Other risk factors include osteoarthritis, leg fracture, obesity, paralysis, phlebitis, venous thrombosis, leg surgery, hip replacement, and knee replacement. In addition, people who have been diagnosed with varicose veins are also more susceptible to venous ulcers.

**Signs and Symptoms**

Venous ulcers can be identified through different signs and symptoms, such as:

- Pain
- Itching
- Swelling
- Discoloration or hardening of the skin

Sometimes, the discoloration and hardening are followed by a foul smelling discharge.

The earliest sign of venous insufficiency is stasis dermatitis wherein itchiness and thinning of the skin occurs. The early signs of venous ulcers include:

- Swelling of the legs
- Heaviness and cramping
- Dark red, purple or brown discoloration of skin along with skin hardening, itching and tingling

When venous ulcers have become established, other signs and symptoms emerge, such as:
- Shallow sores characterized by red colored base which may be covered with a yellow tissue
- Non-uniform shape of the borders of the sore
- Shiny, tight, hot or discoloration of the skin surrounding the sore
- Pain in the leg and bad odor or pus in case the sore has become infected

**Treatment**

When properly treated by a trained healthcare professional using compression therapy, venous ulcers can heal within 3 to 4 months. In spite of this, certain venous ulcers take longer time to heal while several others do not heal at all.\textsuperscript{35}

Treatment comprises of cleaning and dressing the wound, applying pressure or compression bandages to enhance or improve the blood circulation in the limbs, and using antibiotics in case of infection. However, it should be noted that antibiotics have no effect on healing the ulcer.\textsuperscript{35}

**Prevention**

Prevention of venous ulcers is extremely important to avoid further complications. The primary problem associated with venous ulcers is recurrence. A large number of preventive strategies have been used to stop the occurrence or recurrence of venous ulcers. Some of these are:\textsuperscript{36}

- Continued use of compression stockings
- Weight loss in case of obese individuals
- Regular exercise
• Leg elevation whenever possible

Studies have found that those who have already had an ulcer are at greater risk of developing another ulcer within a few months or years after the previous venous ulcer has been healed.\textsuperscript{38}

\textit{Compression Stockings}

A previous history of venous leg ulcer or noticeable skin hardening may be indicative of a new developing venous ulcer which requires compression therapy.\textsuperscript{38} Compression therapy has been cited to be the standard of care for both venous ulcers and chronic case of venous insufficiency. Compression therapy has been found to be useful in:

• Alleviating edema
• Improving venous reflux
• Accelerating ulcer healing
• Reducing pain

Studies have revealed that if compression therapy is maintained throughout life, then the risk of recurrence of venous ulcer is substantially reduced. There are different types of compression therapy including:\textsuperscript{38}

• Inelastic
• Elastic
• Intermittent pneumatic compression

\textit{Inelastic compression} therapy aims at providing high working pressure during ambulation and muscle contraction. It does not provide resting pressure. The most common inelastic compression method is comprised of the use of an Unna boot, which is a moist bandage
impregnated with zinc oxide that hardens once applied. A Cochrane review suggests that inelastic compression works best when given in conjunction with elastic compression.

A disadvantage with inelastic compression is that since it is inelastic in nature, the boot does not confirm to the change in leg size and may be uncomfortable to wear at times. It also requires frequent changes as it may emit a bad odor due to the accumulation of ulcer exudates.\textsuperscript{34}

\textit{Elastic compression} therapy, as the name suggests, is elastic in nature and conforms to the changes in leg size. It can endure pressure both during activity and rest. Stockings, bandages, or elastic wraps are used in elastic compression. Among these, the elastic wraps are not highly recommended because they are not capable of providing sufficient pressure. Compression stockings have graded pressure so that they apply highest pressure at the ankle and lowest pressure towards the knee and thigh. The recommended pressures on the knees and thighs are at least 20 to 30 mmHg and preferably 30 to 44 mmHg, respectively. Compression stockings are not to be worn at night. They need to be replaced every 6 years. This is because these stockings lose their compression with regular cleaning and washing.\textsuperscript{32}

Elastic bandages can also be used in place of compression stockings. A recent research study has found that elastic compression therapy is more effective than inelastic compression therapy. In addition to this, high compression is more beneficial than low compression. Multilayer bandages are also preferred over single layer bandages. On the other hand, multilayer bandages have an associated disadvantage that only
trained personnel in healthcare centers or physician’s office can apply them once or twice a week depending on the drainage of the wound.\textsuperscript{32}

\textit{Intermittent pneumatic compression therapy}

Intermittent pneumatic compression therapy utilizes a pump, which can deliver air to inflatable and deflatable sleeves which embrace the extremities, allowing for alternating pressure. The disadvantage associated with this type of compression therapy is that it is more expensive than elastic compression and also requires the patient to be restrained. Therefore, it is found to be more effective and useful in patients who are bedridden and cannot withstand continuous compression therapy.\textsuperscript{32}

Initially used to prevent deep vein thrombosis (DVT) and pulmonary embolism, the current use of intermittent pneumatic compression is in the reduction of swelling due to mobilization of excess tissue fluid. The use of compression pumps has been found to be useful in treating chronic venous insufficiency, which prevents the occurrence of venous ulcers. If intermittent pneumatic compression is not used appropriately, patients may experience substantial fluid accumulation and tissue edema.\textsuperscript{38}

\textit{Leg elevation}

Leg elevation, along with the use of compression stockings, is a standard measure of care and prevention of venous ulcers. It is suggested that the legs be kept elevated above the level of the heart. It can reduce the risk of edema, improve microcirculation and oxygen delivery within the tissues, and accelerate the rate of healing of existing ulcers. A research study has found that the simple procedure
of keeping a leg elevated increased the flow within veins by almost 45 percent. It has been medically recommended that leg elevation be performed for 30 minutes at least 3 to 4 times in a day.\textsuperscript{32}

\textit{Weight loss}

Obese patients should be encouraged to lose weight to prevent venous ulcers. This is because excessive weight puts a tremendous pressure on the veins in the legs, which can ultimately damage the skin. Research studies have reported that venous ulcers are more frequently found in obese people than those with normal weight. Daily exercise accompanied by a healthy and balanced diet are recommended for obese individuals to lose weight.\textsuperscript{36}

It has been suggested that at least 150 minutes of moderate physical activity is a must for those who want to lose weight. Walking is highly recommended as it is a great form of exercise. Sitting or standing in the same position for a prolonged period should also be avoided.\textsuperscript{36}

\textit{Exercise}

It is a well-known fact that alterations in the calf pump muscle function can raise the risk of chronic venous insufficiency, which can lead to an increase in the risk of formation of venous leg ulcers. Research studies have shown that the deficiency of the calf muscle pump can significantly cause severe cases of venous ulcerations in the lower limbs. An impaired calf-muscle pump is associated with an increase in the severity of venous ulcers. This is also an important contributing factor to reduced ejection volumes and fractions. Venous
insufficiency can also lead to a reduction in the range of motion at the ankle.\textsuperscript{38}

Exercise alleviates the harmful effects caused by impaired calf muscles. An exercise program incorporating a well-structured module of calf muscle exercise has been reported to improve hemodynamic performance as well as prevent recurrence of venous ulcers. Walking has been recommended for ambulatory patients. It has also been shown that an increase in the muscular activity and increased muscle mass may be helpful in enhancing the venous emptying of the calf. Patients should consult a physical therapist that can assist them with the correct exercises to perform to prevent venous ulcers.\textsuperscript{38}

_Nutrition_

Nutrition plays an essential role in wound healing. Studies have reported that patients who are either overweight or obese, but nutritionally deficient, fail to meet their dietary needs. Guidelines have recommended an appropriate daily intake of nutrients such as proteins, vitamins A and C, and zinc in order to prevent venous ulcers. Some scientists have even suggested a possible link between nutritional deficiencies and delayed wound healing. For example, many studies have claimed that zinc supplementation in patients with deficiency of the mineral can help speed up wound healing.

The guidelines for prevention of venous ulcers recommend nutritional assessment for patients undergoing treatment for venous ulcers. In addition, since overweight patients have reduced mobility, weight loss can prove to be highly advantageous. As mentioned previously, an increased body weight can lead to worsening of the calf pump muscle
function and venous hypertension. A nutritionally deficient patient should consult a dietitian for weight loss strategies and to maintain a well-balanced nutrition status.\textsuperscript{38}

**Manual Lymphatic Drainage**

Manual lymphatic drainage is comprised of manual tissue mobilization and stretching. It helps in returning the tissue fluid and proteins into the lymphatic vascular system. Guidelines from the Association for the Advancement of Wound Care have recommended the use of manual lymphatic drainage to reduce the swelling and inflammation of tissues in individuals with chronic venous insufficiency and, thus, prevent the recurrence of venous ulcers. This therapy has been proven to be useful in reducing the foot volume and also improve the quality of life in patients who have undergone surgical treatment. Manual lymphatic therapy is part of decongestive physiotherapy, which also includes skin care, compression bandaging, and exercise. Studies have demonstrated the effectiveness of decongestive therapy in the treatment of chronic venous insufficiency.

Manual lymphatic drainage enhances the venous blood circulation especially in the femoral and great saphenous veins. However, it requires special training and should only be performed by a physical or occupational therapist.

*Treatment of underlying problems*

Sometimes there are various underlying problems that may cause venous ulcers. The treatment of these conditions can help prevent venous ulcers. One good example is varicose veins which is a risk factor for venous ulcers.\textsuperscript{36}
Varicose veins can be treated with different procedures, such as:

- Catheterization
- High frequency radio waves or lasers

Sometimes even surgery is recommended to repair the damage on the leg veins.\(^3^6\)

**Self-Care**

Self-care is an integral part of preventing the recurrence of venous ulcers. Caring for the wound is of utmost importance. Patients can choose from a large variety of dressings available such as gauze, gel, foam or film. Some of the most important things to remember in the self-care of venous ulcers are outlined below.\(^3^3\)

*Keep the wound clean:*
The wound should always be clean and covered with a bandage to prevent exposure to any type of infectious organisms. The skin around the wound should also be kept clean and moisturized.

*Change the dressing frequently:*
The provider should inform the patient about the frequency of changing the dressing.

*Apply an appropriate cleanser:*
The wound should be thoroughly cleaned before applying a dressing.

*Apply compression stocking:*
Wear a compression stocking or bandage over the dressing.
During the course of treatment for a venous ulcer, patients themselves should take certain steps to assist the treatment and help improve blood circulation to the area. Some of these steps include:\(^{33}\)

- **Wearing of appropriate compression stockings or bandages:**
  These should be worn at all times as they are can help prevent the accumulation of the blood, reduce swelling, speed up the healing process as well as reduce the pain.
- **Elevating the foot:**
  The foot should always be above the heart.
- **Maintaining a regular exercise regimen to improve blood circulation in the extremities.**
- **Taking medications properly:**
  Any medications should be taken as prescribed.

**Wound Healing Society Guidelines**

The Wound Healing Society has compiled certain guidelines for the prevention of venous ulcers. These general guidelines were made based on a meta-analysis of multiple random clinical trials and are intended for preventing ulcers in patients at high risk of developing venous ulcers such as those with deep vein thrombosis, impaired function of the calf and ankle perforating veins, elevated ambulatory venous pressure or venous hypertension, dysfunctional calf pump pressure, fluid accumulation (edema), cellulitis, and lipodermatosclerosis. In addition, accumulation of fluid or edema in the lower extremities may be caused by a variety of causative factors such as congestive heart failure (CHF), lymphedema, pregnancy, obesity, trauma, and constriction close to the area of accumulation. If these conditions are alleviated or controlled, then the occurrence of venous ulcers can be prevented.
The guidelines suggest the following measures to prevent venous insufficiency and venous ulcers:

1. Continuous use of compression stockings:
   This is recommended in patients with venous hypertension or postphlebitic syndrome. Long-term compression is required to reduce the pre-existing conditions, which may lead to formation of venous ulcer. Health care professionals are required to help the patients in learning the techniques to use the compression stockings.

2. Regular exercise:
   The role of exercise in increasing the function of the calf muscle pump function is underlined in preventing venous ulcers.

3. Prophylactic treatment of venous thromboembolism:
   This is indicated following the diagnosis of deep vein thrombosis (DVT) to reduce its recurrence as well as postphlebitic syndrome. Recurrent venous thromboembolism can cause complications of postphlebitic or post-thrombotic syndrome, which is why their recurrence should be prevented.

4. Surgical intervention:
   This can help prevent backflow of fluid from the deep to the superficial venous system and, thus, prevent venous ulceration. The surgery of choice is called subfascial endoscopic perforator surgery (SEPS). In this procedure, the perforated blood vessels are interrupted resulting in a significant reduction of ambulatory venous pressure in the leg. Other surgical procedures that may be done are superficial venous ablation, endovenous laser
ablation, or valvuloplasty in conjunction with compression therapy.

5. Antibiotics:
The use of antibiotics to treat cellulitis is recommended to prevent venous ulcers. In cellulitis, the bacterial infection causes inflammation of the skin and subcutaneous tissues. If not treated, it may lead to edema in the lower extremities, which inactivates the fatty acids of sebum leading to the loss of normal bactericidal properties of the skin.

6. The use of anabolic steroids along with compression therapy helps in the enhancement of fibrinolysis, which can treat lipodermatosclerosis of the lower part. This reduces the induration and inflammation and hence prevents venous ulcer.

7. Micronized purified flavonoid fraction:
The oral use of micronized purified flavonoid fraction in conjunction with compression therapy has been found to be helpful in treating and managing chronic venous insufficiency. In addition, any agent that can prevent the formation of prostaglandins and free oxygen radicals can lead to a reduction in the microvascular leakage and, thus, prevent the activation of leucocytes, which in turn can reduce chronic venous insufficiency and prevent venous ulceration.

**Diabetic Ulcers**

One of the most common causes of amputation of the lower extremities is diabetic foot complications or diabetic ulcers. Statistics
show that the risk of amputation of the lower extremity is almost 15 to 46 times more in patients with diabetes mellitus when compared to those who do not have the disease.\textsuperscript{40}

Diabetic ulcers start with the formation of minor and superficial skin wounds. If detected and treated early, these formations can be prevented from developing into full-blown diabetic ulcers. In fact, studies have shown that amputations can be prevented in almost 85 per cent of patients if detected, diagnosed, and treated very early. Family physicians play a crucial role in detecting and providing early and optimal care for skin ulcers.\textsuperscript{40}

Diabetics with foot ulcers eventually require hospitalization and sometimes, amputation of the lower extremities. Generally, it takes many weeks to several months to heal a foot ulcer.\textsuperscript{39}

Foot ulcers are a breeding ground for many infectious organisms. They are almost always the primary source of infection, which can lead to complications. In many cases, the ulcer on the foot is covered by callus or fibrotic tissue, which is why eliminating these hyperkeratotic tissues is extremely important. This is also essential to allow for comprehensive evaluation of the wound.\textsuperscript{40}

The main reason why foot ulcers are treated too late and lead to amputations is the lack of sensation as well as poor blood flow and deformities of the foot. Nerve damage is a common complication of diabetes (diabetic neuropathy), which results in loss of sensory perception (lack of pain sensation, numbness, and tingling) in the extremities.\textsuperscript{39} A diabetic foot ulcer has various components including
biomechanical pressure, vascular supply compromise accompanied by neuropathy.\textsuperscript{42}

The presence of vascular conditions decreases the ability of the wounds to heal and thus, raise the risk of infection and foot ulcer complications. Hyperglycemia also decreases the general immune system function, reducing its ability to fight infections and the rate of wound healing.\textsuperscript{39} Peripheral neuropathy and the shearing forces on the foot through walking activities result in local inflammatory response, focal tissue ischemia, destruction of tissues involved, and eventually ulceration. The ulceration sites are usually observed in areas, which sustain the greatest plantar pressure points.\textsuperscript{46}

**Causes**

As mentioned previously, diabetes is one of the leading causes of foot ulcers. Studies have shown that Native Americans, African Americans, Hispanics, and older men are especially at greater risk of developing foot ulcers. Diabetics who use insulin are also at increased risk of developing a foot ulcer. Likewise, diabetic patients with nephropathic, neuropathic or ophthalmic complications are also at increased risk of developing foot ulcers. Obesity, excessive use of alcohol and tobacco are also contributing factors for the development of diabetic foot ulcers.\textsuperscript{39}

**Signs and symptoms**

Diabetic ulcers are primarily painless. In fact, patients with foot ulcers usually do not feel any pain or discomfort due to their neuropathy. This is true even if severe infection is present. However, they often express discomfort and ill at ease of the soiled footwear and
stockings. Sometimes, redness or swelling may be associated with the formation of ulcer and if the condition has progressed substantially, foul odor may also be present.

**Treatment**

Various novel approaches have been developed in the recent years to treat diabetic foot ulcers. Examples include:

- New dressings
- Growth factors
- Bioengineered skin and tissue substitutes
- Hyperbaric oxygen
- Negative pressure wound therapy

The primary treatment strategy involves sufficient vascular perfusion, control of infection if present, debridement, moist wound environment, and pressure dispersion. Among other offloading modalities are bed rest, use of wheelchairs and crutches, total contact casts, felted foam, half shoes, therapeutic shoes, custom splints, removal cast walkers, and total contact casting.

**Prevention**

Healthcare professionals agree that the best treatment for a diabetic ulcer is to prevent its development in the first place. Studies have found that the recognition of risk factors, preventive foot care and hygiene, patient education, and regular foot examination are essential preventive measures. However, these measures are not foolproof and foot ulcers may still develop.
The gold standard of care in preventing and managing diabetic foot ulcers includes: 

- Reduction of blood glucose levels
- Debridement of wound properly before inspecting the wound or foot
- Treatment of any infection present in the wound
- Reduction of friction or pressure on the foot
- Maintaining sufficient blood circulation

It is important to take care of and prevent diabetic complications since they are comorbidities of each other. Other preventive measures to manage diabetic ulcer formation include life style modification, blood pressure control, management of lipid levels, blood glucose control, and smoking cessation. A literature review concluded that prophylactic interventions such as patient education, prescription footwear, intensive podiatric care, and evaluation for surgical intervention are effective in preventing diabetic foot ulceration. In addition, it is imperative that patients understand the management process and be an active participant in making the aforementioned lifestyle changes.

*Early Identification*

The first and foremost step in preventing foot complications in diabetes patients is a careful inspection of the diabetic foot on a regular basis. In fact, it is the easiest, most inexpensive and effective means of preventing diabetic ulcer. Primary care physicians of diabetic patients should also perform foot examinations during routine visits. Likewise, hospitalized patients should also have their foot routinely
and adequately examined by nurses and caregivers.\textsuperscript{40}

An annual foot examination is recommended for all diabetic patients to prevent and treat high-risk conditions that can be identified as well as diabetic foot complications. During visits with a health provider, it is important to identify the risk factors associated with the development of foot ulcers for individuals by obtaining a thorough patient history and conducting physical examination of each foot. A complete evaluation must be performed of the sensation loss in the lower foot extremity, structure of the foot, restriction in the mobility of the joints, vascular status, previous case of diabetic foot ulcer, and amputation neuropathy.\textsuperscript{46}

Patients who are at greatest risk for diabetes should be examined frequently, preferably every 1 to 6 months. The absence of symptoms does not indicate healthy feet and circulation because of neuropathic loss of sensation, peripheral vascular disease or asymptomatic ulcers. The patient’s feet should be evaluated in both supine and standing positions. Aside from the feet, an examination of footwear such as shoes and socks must also be performed.\textsuperscript{41} Sometimes there is maceration present between the toes, which are generally caused by a fungal infection. To completely inspect this area and the plantar surface of the foot, a mirror should be used. In case of patients with
comorbid diabetic retinopathy, a caregiver or nurse should be asked to examine the foot of the patient instead.\(^{39}\)

**Patient History**

A patient’s history is essential in the overall treatment decision of diabetic foot ulcers. Current and past histories of ulcer, lower limb amputation, and neuropathic complications such as fractures are all risk factors for diabetic foot ulceration, infection, and eventual amputation. This information should be noted since patients with these risk factors, or who have undergone wound treatment, are more susceptible to develop foot ulceration within one year. This is mainly due to the fact that the plantar skin are less resilient and less tolerant of repetitive stress after a recent injury, and thus, more susceptible to breakdown and re-injury. This is why patients with these risk factors are said to be the highest risk group and require frequent foot assessment, intensive education, therapeutic shoes, padded stockings and blood sugar control to prevent recurring case of diabetic foot ulcer.\(^{46}\)

**Foot Care**

The cornerstone of preventing foot ulcer formation is regular foot care and proper management of minor foot injuries. This means daily self-inspection of the foot or with the help of a caregiver. Minor injuries should be kept clean by cleansing with gentle soap and water. The foot should be washed and dried at least once daily. After cleansing, it is very important to properly dry the inter-digital spaces between the toes to prevent ulceration and fungal infection.
The recommended water temperature for cleaning the foot is less than 37°C. A simple way to check for this temperature is to test the water using the elbows or forearms before using it to wash the foot. This is especially important for patients with sensory perception loss as in the case of many patients with neuropathy to prevent scalding or burning the foot.39

An appropriate moisturizer should also be topically applied in order to maintain healthy skin, which can resist regular pressure and trauma.40 A lubricating oil or cream should be used for dry skin but should not be applied between the toes. Lastly, chemical agents or plasters should not be used to remove corns or calluses.40

**Appropriate Footwear**

It has been stated earlier that the use of inappropriate footwear is one of the primary reasons for foot ulcers. This is why appropriate footwear should be worn at all times, both indoors and outdoors.

The footwear should easily adapt to the altered biomechanics and deformities of the foot to prevent formation of foot ulcers. Patients who do not have loss of sensory perception can choose their footwear off the shelf. On the other hand, patients with neuropathy or ischemia should choose their footwear with extra caution especially if foot deformity is also present.

Shoes should neither be too long or too short. Their interior is recommended to be 1-2 cm longer than the foot itself. The width should be equal to the size of the foot at the site of metatarsal phalangeal joints and the height should allow enough space for toes.
In certain conditions, patients are also recommended to wear special footwear such as insoles and orthoses. Also, special shoes of adequate size should be used when walking indoors on carpeted floors. Lastly, diabetic patients should always wear shoes with stockings.

Generally, diabetic patients should wear shoes made of canvas, leather, or suede to prevent foot ulcer formation. Shoes made from plastic and other materials should not be used because air does not pass through them. Shoes should also be adjustable such as those with laces, Velcro®, or buckles. Shoes that are pointed or with open toes, high heels, flip flops or sandals, should be avoided. Therapeutic shoes with pressure relieving insoles have proven to be essential in preventing ulcer formation and are also associated with a reduction in the development of ulcers.

**Pressure Mitigation**

Patients with diabetes should avoid putting too much pressure on one part of their foot. Sometimes, they may need to wear specially designed shoes, a brace, or a special cast if they already have foot ulcers. These help relieve pressure off the ulcerated area.

**Treatment of Underlying Conditions**

It is important for underlying conditions such as neuropathy, poor blood flow, deformity of foot, wearing inappropriate shoes, elevated
blood sugar, and a history of previous foot ulceration to be addressed and their recurrence prevented as much as possible. In addition, it is also recommended that diabetic patients address and eliminate lifestyle risk factors that are modifiable such as smoking, excessive alcohol consumption, high cholesterol levels, and elevated blood sugar levels.

Other skin related problems in diabetic patients such as dry or fissured skin, calluses, tinea or onychomycosis are also predisposing factors to foot ulcer formation and should be treated accordingly.46

**Patient Education**

Patient education plays a very important role in the treatment as well as management of the foot ulcers. Research studies have reported that patient education on the various aspects and implications of foot care and management to prevent foot ulcers have resulted in successful reduction of reported cases of diabetic foot ulcers.44

Generally, patients should be educated on proper foot hygiene and footwear, daily inspection, and the importance of prompt treatment for new lesions in order to prevent new or recurring cases of diabetic foot ulcer. It is important to note that patient education should extend to caregivers and family members as well who are living with diabetic patients to help them cope with their disease. Both patients and caregivers need to understand the implications of loss of protective sensation, proper nail trimming techniques, the importance of daily foot inspection, and proper foot care to prevent diabetic foot ulcers.46
Patient education should be provided in a structured and well-organized manner to make compliance easier for patients and caregivers. Ideally, it should be given in multiple short sessions over a period of time by using different methods of teaching. The educator should motivate patients to perform self-care measures. As mentioned previously, patient education should include self-detection of potential foot problems and how to respond appropriately to various skin conditions.

**Surgical Interventions**

Elective surgery may be used as a last measure to correct deformities of foot structures that cannot be rectified alone by specialized footwear. Various procedures to prevent the occurrence of foot ulcers include:

- Hammertoe repair
- Metatarsal osteotomies
- Plantar exostectomies
- Achilles tendon lengthening

Patients who have co-existing neuropathies can undergo these procedures with local anesthesia. These foot-correcting reconstructive surgical procedures can be performed following revascularization in ischemic patients.

**Cilostazol**

According to clinical trials, cilostazol has shown beneficial effects in the primary prevention of foot ulcer in diabetic patients. Cilostazol is an oral drug indicated in the treatment of intermittent claudication. It
reduces leg pain caused by impaired circulation to the lower extremities. It improves circulation by producing non-homogenous vasodilation in the femoral artery. Cilostazol does not affect the renal arteries. It inhibits the enzyme phosphodiesterase (PDE), especially PDE III, and produces temporary inhibition of platelet aggregation.

Treatment response is seen in patients as early as 2-4 weeks after initiation of therapy, although it is not unusual to see a response a few weeks after. Often, the optimal beneficial effects of Cilostazol are not seen until after a minimum of 12 weeks therapy. Metalloproteinase-9 is a biochemical marker that is implicated in chronic wounds, especially diabetic foot ulcers. Studies have shown cilostazol to be capable of reducing the metalloproteinase-9 levels in the body, thus preventing or reducing the rate of onset of diabetic foot ulcers.

**Multidisciplinary Approach**

It should be noted that healthcare centers and hospitals with such multidisciplinary teams in place have higher rates of recovery of foot ulcers and lower rates of lower limb amputations. Since diabetes is a multifactorial disease, it is essential that patients with diabetes should be under the simultaneous care of a primary care physician with input from specialists such as an endocrinologist, ophthalmologist, nephrologists, vascular surgeon, podiatrist, physical therapist, nutritionist, and diabetic educator.

Studies have found that the involvement of a multidisciplinary team can result in an almost 50% reduction of lower limb amputation in diabetic patients. The team should conduct proper screening and treatment protocols to decrease the risk of future hospitalizations and
lower limb amputations. Patients should also be stratified according to their risk level (i.e., high risk, medium risk, low risk) based on their comorbid conditions and complications such as neuropathy, peripheral vascular disease, deformities of the foot and pressures, and also a history of lower extremity pathology.46

During regular check-ups, medical providers should check the shoes of patients to identify areas of inadequate support or improper fit. Generally patients are recommended to wear athletic shoes and thick, adsorbent socks. Some patients with foot deformities may sometimes need to use custom made shoes for themselves. Stockings should be worn with seams inside out or without any seams.40

Diabetic patients should be cautioned about the use of home remedies for minor skin injuries and infections to the foot such as cuts, scrapes, blisters or athlete’s foot (tinea pedis). Home remedies that can severely aggravate foot ulcers or put it at risk of injury because they can inhibit the healing process. Any home remedy should only be used after consulting with a provider. Popular home remedies such as hot soaks and baths, heating pads, or topical products containing harsh chemicals such as hydrogen peroxide, iodine, and astringents should be avoided, unless otherwise advised by a provider.

Minor injuries or wounds should be properly cleaned and a topical antibiotic applied to keep the area around the wound clean, hydrated and prevent the formation of ulcer. A physician should immediately inspect wounds that do not show signs of healing. Members of the multidisciplinary team should stress the importance of good foot care habits to prevent diabetic foot complications.40
Pressure Ulcers

When tremendous or repeated pressure is exerted on a certain area of the skin, its blood circulation is impaired. As a result, the skin breaks down and pressure ulcer develops. The most common risk factors for developing pressure ulcers are:

- Use of wheelchair
- Being bed ridden for a long period of time
- Old age
- Immobility in certain part of the body due to a spinal or brain injury or a disease condition
- Condition affecting the blood circulation
- Presence of Alzheimer’s disease or any other condition affecting mental ability
- Fragile skin
- Urinary incontinence
- Bowel incontinence
- Malnourishment

The incidence rate of pressure ulcers have been reported to be increasing steadily in the last few years. This is primarily attributed to the growing, aging population, rising fragmented care, and shortage of nursing services. The prevention of pressure ulcers in hospitalized patients is one of the major concerns of nursing staff. In fact, the incidence of bed sores is often seen as a failure or gap in nursing care. However, the treatment and prevention of pressure ulcers is a responsibility of both medical providers and nurses. In fact, it is considered to be a multidisciplinary approach, with the nurses assuming the primary role. There are guidelines to prevent the
occurrence of pressure ulcers which include essential measures such as:

- Risk assessment
- Skin care
- Mechanical loading
- Patient and staff education

**Stages of Pressure Ulcers**

Pressure ulcers develop over a period of time and are classified accordingly as:

1. Stage I
2. Stage II
3. Stage III
4. Stage IV

The following is a description of the various stages of pressure ulcer formation and the National Pressure Ulcer Advisory Panel provides helpful education and downloadable illustrations for nursing education at: http://www.npuap.org/resources/educational-and-clinical-resources/pressure-ulcer-categorystaging-illustrations/.

**Stage I:**

At this stage, non-blanchable erythema is observed over an intact skin. There is redness that is localized over a bony prominence such as the heel of the foot. Darker skin tones may not reveal visible blanching, although its color will appear
significantly different from the surrounding area. There may also be pain, firmness or softness, warmness or coolness observed when touched compared to adjacent tissues. At this stage, the ulcer is difficult to detect in patients with dark skin tones.

Stage II:

At this stage, a partial thickness without bruising can be observed at the affected area, which is attributable to loss of dermis. There may be a shallow open ulcer with a red pink wound bed, without slough. There may also be an intact or open, ruptured, serum-filled or sero-sanginous-filled blister seen. Other presentation include shiny or dry shallow ulcer without slough or bruising. It is important to differentiate pressure ulcer at this stage from other similar looking condition, such as, minor skin tears, tape burns, and incontinence associated dermatitis, maceration or excoriation.

Stage III:

At this stage, a full thickness skin loss with visible subcutaneous fat may be seen. Although the injury has reached the deeper skin layers, it hasn’t penetrated into the bone, tendon or muscle at this stage. Slough may be visible but it usually does not obscure the depth of tissue
loss. In addition, undermining and tunneling may be observed. Other presentation is also possible, depending on the anatomical site involved. For example, the bridge of the nose, ear, occiput and malleolus do not have subcutaneous fat tissue, in which case, the ulcer may be shallow. On the other hand, other areas where fat tissue is present, a deep ulcer can develop.

Stage IV:
At this stage, there is an observable full thickness tissue loss with deeper tissue involvement. It is at this stage that bones, tendons or muscles may be exposed. Slough or eschar may also be present accompanied by undermining and tunneling. Similar to stage III, the presentation of ulcer differs with anatomical site. The bridge of the nose, ear, occiput and malleolus do not contain subcutaneous fat tissue resulting in pressure ulcers that are relatively shallow. However, the injury is deep and the exposed inner structures may be visible and at greater risk of complications, such as osteomyelitis or osteitis.

**Urinary Incontinence**

Urinary incontinence is an important cause of pressure ulcers. It is also known as bladder incontinence. It simply means loss of bladder control. Patients with urinary incontinence often have difficulty controlling their bladder.
There are four types of urinary incontinence defined in the Clinical Practice Guideline issued by the Agency for Health Care Policy and Research:

1. Stress incontinence
2. Urge incontinence
3. Mixed incontinence
4. Overflow incontinence

Stress incontinence occurs with increased abdominal pressure from physical stressors such as laughing, sneezing, coughing, and climbing stairs. Urge urinary incontinence occurs immediately before urinary urgency is experienced. Mixed urinary incontinence is a combination of stress and urge incontinence. It is characterized by involuntary leakage accompanied by both urinary urgency and physical stressors such as exertion, effort, sneezing or coughing. Overflow incontinence occurs when there is incomplete bladder emptying due to a dysfunctional detrusor contraction or outlet obstruction of the bladder.

There are many causes of urinary incontinence, including:
- Blockade in the urinary system
- Neurological disorders, such as dementia
- Dysfunctional urinary system

Some other long-term causes of urinary incontinence are prolonged bed rest which usually occurs following extensive surgery, medication use (i.e., diuretics, antidepressants, tranquilizers, cough and cold remedies, or antihistamines), mental confusion, pregnancy, prostate
infection or inflammation, severe constipation or bowel incontinence, urinary tract infection and weight gain.  

**Bowel Incontinence**

Another cause of pressure ulcer is bowel or fecal incontinence. It is a condition characterized by loss of bowel control resulting in psychological and social debilitation. This condition can vary in severity. The problem is more prominent in older patients, usually those over 65 years of age. It is also more commonly seen in women than men. Fortunately, bowel incontinence is treatable. With proper treatment and management, patients can recover coordinated functioning between the rectum, anus, pelvic muscles, and nervous system. There are many causes of bowel incontinence, including:

- Chronic constipation
- Fecal impaction
- Long term use of laxatives
- Bowel surgery
- Emotional stress
- Gynecological, prostate or rectal surgery
- Injury to the anal muscles during childbirth
- Nerve or muscular damage from a tumour, injury or radiation
- Severe case of diarrhea which may cause leakage
- Severe hemorrhoids
- Rectal prolapse
- Stress

The role of urinary and bowel incontinence in pressure ulcers

As mentioned previously, urinary and bowel incontinence are risk factors for pressure ulcers. People with urinary incontinence or bowel
incontinence are at an increased risk of skin problems such as pressure ulcers or bed sores around the buttocks, hips, genitals, and the area in between the pelvis and rectum. An increase in moisture around these parts can very well lead to redness, peeling, irritation, and even yeast infections.\textsuperscript{50}

Urinary and bowel incontinence have been well recognized to be a significant factor in causing development of pressure ulcers. Incontinence dermatitis leads to an inflamed, excoriated, infected, and damaged skin which can cause pain, discomfort as well as pressure ulcers. An increase in skin humidity can aggravate the localized shear and frictional forces on the skin, causing it damage. Bowel incontinence is one of the most significant indicators for the development of pressure ulcers in the elderly population. The excess moisture from urine, sweat, and frequent washing increases the coefficient of friction of the skin which in turn, increases its susceptibility to rubbing and scraping against fabrics and materials such as incontinence pads, clothing, and sheets. An increased coefficient of friction also increases the permeability of the skin to different chemical irritants.\textsuperscript{49}

\textbf{Risk Assessment}

There are many risk assessment tools which can act as adjunctive aids in the risk assessment for pressure ulcers. Some of these tools include:

\begin{itemize}
  \item The Braden Scale
  \item Norton Scale
  \item Waterlow Scale
\end{itemize}
The Braden Scale is the tool most commonly used in the U.S.\textsuperscript{52} It is used in adults and has 6 subscales which measure the sensory, perception, moisture, activity, mobility, nutrition, friction and shear. It utilizes the concept of finding the association between the above factors with the intensity and duration of pressure or tissue tolerance for pressure. The scores range from 6, which is the high risk, to 23, which is the low risk; and, 18 as the cut off score for the onset of pressure ulcers.\textsuperscript{52}

The Norton Scale was developed in the United Kingdom and uses 5 subscales made up of physical condition, mental condition, activity, mobility, and incontinence. The score ranges between 5 (high risk) to 20 (low risk). In addition to these scores, the nurses are required to use their clinical judgment before they initiate preventive care.\textsuperscript{52}

Risk assessment should be carried out on admission, at discharge, and during admission whenever the clinical condition of the patient changes. Basically, the risk assessment begins with identification of the risk factors and followed by a thorough inspection of the skin. The risk factors that may lead to pressure ulcers or bed sores are classified in two categories: intrinsic and extrinsic factors.\textsuperscript{48}

Intrinsic factors include the following:

- Limited mobility such as spinal cord injury, cerebrovascular accident, neurological diseases, pain, fractures, post-surgical procedures, sedation, coma or arthropathies
• Poor nutrition such as anorexia, dehydration, poor dentition, dietary restriction, reduced sense of smell or taste, or poverty or lack of access to bed
• Existing comorbidities such as diabetes mellitus, depression or psychosis, collagen vascular disorders, peripheral vascular disease, reduced sensation of pain, immunodeficiency, use of corticosteroid therapy, congestive heart failure, malignancies, end-stage renal disease, or dementia
• Aging skin problems such as loss of elasticity, decreased cutaneous blood flow, changes in pH of the skin and dermis, flattening of rete ridges, loss of subcutaneous fat and decreased dermal-epidermal blood flow

Extrinsic factors include the following:
• Pressure from any hard surfaces such as bed, wheelchair, stretcher
• Immobility of the patient which may cause friction
• Shear from movement of involuntary muscles
• Moisture due to bowel or urinary incontinence, excessive perspiration or drainage from wound

**Prevention**

After risk assessment, appropriate prevention measures should be taught to patients and implemented. Prevention measures include interventions related to:
• Pressure reduction
• Appropriate skin care
• Adequate nutrition
• First aid
• Self-care and assessment measures

**Pressure Reduction**

The mainstay in prevention of pressure ulcers is pressure reduction which helps preserve the microcirculation. There are no set schedules or optimal patient repositioning techniques which can be used to prevent pressure ulcers. Mechanical loading and use of support surfaces are two other ways of decreasing pressure. A reduction of mechanical load is one of the most important preventive care measures for pressure ulcers. This is done by turning or repositioning patients, either by themselves or with help from nurses and caregivers.\(^{52}\)

Ideally, patients should be turned at least every 2 hours and more frequently in case of critically ill patients to reduce the mechanical load. The positioning should also be considered. When repositioning the patient, head elevation should be maintained and the lateral turn should not be more than 30 degrees\(^{52}\) to reduce the shear forces exerted on the body. An appropriate head elevation should be maintained, preferably at the lowest degree which allows comfort to prevent other medical complications such as aspiration and worsening symptoms of congestive heart failure. Manual aids such as a trapeze bar can also be used by patients to reposition themselves.\(^{48}\)

**Support Surfaces**

The use of pressure reducing devices such as support surfaces can help reduce tissue pressure to less than capillary closing pressure of
32 mmHg. These devices help in distributing the pressure evenly throughout the body.\textsuperscript{52}

Specially designed support surfaces have been proven to be effective in preventing the development of pressure ulcers. Examples include dynamic mattresses, static foam mattresses, and seat cushions. There are two types of support surfaces: 1) Static support surfaces, and 2) Dynamic support surfaces.

Static support surfaces, as their name suggests, are static in nature. They make use of air, foam, gel and water overlays or mattresses. They decrease pressure by distributing the weight of the body over a larger surface area. They mold to the contours of the body and retain a constant level of inflation. They are most effective when used in work patients at moderate to high risk of developing pressure ulcers.

Dynamic support surfaces are dynamic and usually powered by electricity, pump, or battery pack. They are made up of alternating and low–air loss mattresses and designed to conform to the body contours. As their name suggests, they allow for the alteration of the level of support through inflation and deflation of air or movement of fluid. Because of this, dynamic support devices constantly alter the pressure of the support surface against the skin, specifically at the various pressure points. These mattresses are also suitable for patients at moderate to high risk for pressure ulcers and full-thickness pressure ulcers.

A subtype of dynamic support surfaces is made up of air-fluidized beds. These beds are electric and contain silicone-coated beads. They are highly effective for patients at high risk for pressure ulcers. They
are especially useful for non-healing full thickness pressure ulcers and those one full-thickness pressure ulcers on the trunk. However, they are not recommended for patients with pulmonary disease, delicate spinal conditions, or who are ambulatory. It uses a large amount of air to fluidize the total bed, putting patients at greater risk of dehydration due to the escaping heat from the body.

Some mattresses are a combination of an air-fluidized and low-air-loss bed. The lower half of the bed is made up of the air-fluidized component while the upper half of the bed is made up of the low-air-loss component. The sizes of these mattresses are like hospital beds with adjustable heads (allowing the bed to be raised) but lighter than a complete air-fluidized system.

While providing preventive care for pressure ulcers, nurses should choose a support surface based on several factors, including:

- Individual patient requirements
- Condition, number and location of pressure ulcers
- General health
- Mobility and ability to change positions
- Body build

Additionally, support surfaces should allow for the easy redistribution of pressure especially in patients who are not ambulatory and spend most of their time in bed or a chair.

Patients with urinary or bowel incontinence should consider a support surface that provides adequate airflow, such as air-fluidized and low-air-loss beds. These mattresses help intact skin "dry," which in turn,
maintain the integrity of the skin around the pressure ulcer and ultimately, prevent the development of additional pressure ulcers. When using these support surfaces, patients should be educated about the importance of not wearing incontinence briefs because they block the airflow to the skin, and using linen and underpads instead.

It is very important to note that the use of support devices does not negate the need for repositioning every two hours. Simply put, this means that even if patients are using a support surface device, they should still change their positions every 2 hours, if not more frequently.\(^5\)\(^2\)

**Skin care**

Skin care is the next important measure in preventing or arresting the development of pressure ulcers. In fact, stages 1 and 2 pressure ulcers can be managed and treated adequately with proper skin cleansing. As such, nurses and caregivers need to make sure that proper body hygiene products for washing and skin protection are used.

Other skin care recommendations include the following:\(^5\)\(^2\)

- Frequent and periodic skin assessments.
- Evaluation of skin temperature, color, turgor, moisture status and skin integrity.
- Proper skin hygiene and moisturization. However, excessive moisture should be
avoided to prevent greater friction against surfaces. Friction can lead to the breakdown of skin integrity and may be minimized with the use of lubricants, protective films such as transparent and skin sealants, protective dressings, such as hydrocolloids, and protective padding.

- Proper water temperature. Hot water should be avoided and only mild cleansing and non-drying agents should be used.
- Implementation of individualized bathing schedules.
- Use of appropriate topical products. Research studies have found that the use of topical hyperoxygenated fatty acid compounds reduced the risk of pressure ulcers by a significant percentage. The occurrence of pressure ulcers was also significantly lower in patients who use topical nicotinate.
- Avoidance of vigorous massage especially over reddened and bony prominences because it may cause or worsen skin trauma.

**Nutrition**

Nutrition plays a critical role in wound healing. In fact, the lack of proper nutrition or malnutrition, predisposes chronically ill patients to poor wound healing. The most serious form of malnutrition is protein-calorie malnutrition or protein-energy malnutrition. Proteins play an important role in maintaining the structural and functional integrity of several body tissues. Contractile proteins such as actin and myosin are essential in cardiac, skeletal, and smooth muscle functions fibrous proteins such as collagen, elastin, and keratin are important in maintaining structural support. In the proliferative stage of wound healing, collagen deposits on the injured area to increase its tensile strength.
About 40% of the body’s protein is found in skeletal muscles, which are a primary component of lean body mass, the metabolically active tissues. Age and critical illness decreases the lean body mass, considerably impairing the body’s ability to execute functions involving proteins. Various research studies concur that low levels of serum albumin, i.e., less than 3.5 gm/dl, increases the risk of pressure ulcers. This is why adequate protein intake should be implemented. Additionally, involuntary weight loss and the patient’s ability to chew and swallow should also be assessed in high risk patients.\textsuperscript{52}

Protein-calorie malnutrition is characterized by impaired or inadequate absorption of dietary sources of protein and energy. It causes the body to break down proteins for glucogenesis, decreasing the amino acid supply needed for maintenance of body proteins and healing. It is especially associated with the development of pressure ulcers. It is more commonly associated with chronic poor wound healing than any other nutrient deficiency conditions. This is why it is essential for institutionalized patients who are undernourished to receive proper nutritional management.

Nutritional management of institutionalized patients usually involves:

- Administration of adequate calories
- Prevention of protein-calorie malnutrition
- Promotion of wound healing
Nurses and caregivers must be able to recognize, monitor, and manage malnutrition in chronically ill patients. Early intervention measures may be needed to arrest the progress of malnutrition and correct current nutrient deficiencies. A good example is oral supplementation. Studies have shown that critically ill or older patients receiving oral supplements along with standard hospital diet are less predisposed to develop pressure ulcers.

In case of vitamin and mineral deficiency, supplementation has also been found to be beneficial in prevention of pressure ulcers. Care should be taken that if the patients do not have deficiency of proteins, vitamins or minerals then over supplementation should be avoided. In order to prevent pressure ulcers, high calorie foods and supplements should be taken to prevent malnutrition. Protein, vitamin C and zinc supplementation should be given if there is insufficient intake or deficiency detected.

The bottom line is adequate nutrition is needed to prevent pressure ulcers. A diet high in protein is important for patients with protein deficiency to promote wound healing.

**First aid**

First aid measures can initially help manage a developing pressure ulcer. Some of these measures include:

1. Relieving the pressure in the affected area with pillows that are specially designed for it. Other devices may be used such as foam cushions and sheepskin to alleviate the pressure from the area.
2. Treatment of emerging pressure sores as per instructions by the doctor, nurse or caregiver.
3. Protection of the area from further injury or friction. Sprinkle light powder on the sheets so the skin surface does not rub on the bed.
4. Treat malnutrition and other nutrient deficiencies with adequate dietary intake of nutrients and minerals and oral supplements.
5. Cleaning wounds and dressings to prevent any infection.
6. Rinsing with salt water to remove loose and dead tissues.
7. Dressing wounds properly with special gauze.
8. Using of prescribed medications as instructed by the doctor, nurse or caregiver.

Lastly, it is also important to check for developing ulcers or deteriorating changes to the injured area regularly.⁴⁷

**Self-care**

Patient participation is critical in preventing and healing pressure ulcers. Those with limited mobility due to existing medical conditions or on extended bedrest should have assistance. On the other hand, mobile patients can do most of the self-care measures by themselves independently.⁴⁷

**Self-assessment**

The most common areas where pressure ulcers can form should be checked during assessment such as the heels and ankles, knees, hips, spine, tailbone area, elbows, shoulders and shoulder blades, and back of the head and ears. Early signs of pressure ulcers include redness of
skin, warm areas, spongy or hard skin and breakdown of the top layers of skin or a sore. Other signs to look for are the presence of blisters, sores or craters on the body. Other self-care steps measures include:

- Changing position or repositioning at least every 2 hours to relieve pressure from specific regions on the body.
- Using support devices such as pillows, sheepskin, foam padding, powders.
- Keeping a balanced and healthy diet.
- Drinking plenty of water to stay hydrated.
- Keeping skin clean and dry.
- Using a soft sponge or cloth while washing.
- Avoiding scrubbing and massaging affected areas.
- Avoiding harsh products such as talcum powder or strong soaps.
- Cleaning urogenital areas after every urinary or bowel movement.
- Avoiding clothes with thick seams, buttons or zippers which can press on the skin.
- Avoiding very tight clothes which can hinder circulation.
- Avoiding wrinkling up or bunching up of clothes in pressure areas of the body.

Patients with limited mobility, such as those using wheelchairs, should see their medical provider or physical therapist at least twice a year. This is especially necessary if patients gain weight or use the wheelchair for the first time. Wheelchairs should fit patients properly. Foams or gel seat cushion covers should also fit wheelchairs well. Natural sheepskin pads can also be used to reduce the pressure on the skin. When on wheelchairs, patients should shift their weight every 15
to 20 minutes. To do this, they must first lean forward, then to one side and then the other side. Shifting the weight around redistributes the pressure evenly and also helps facilitate adequate blood circulation.\textsuperscript{53}

Patients who are bedridden should use a foam mattress or one filled with gel or air. Pads under the bottom help absorb wetness and keep the skin dry. Friction should be avoided between different body parts or between the body and the mattress with the use of soft pillows or piece of soft foams. While lying on the side, a pillow or foam between the knees and ankles may also help.\textsuperscript{53} Patients lying on their backs should place a pillow or foam under the:

- heels
- calves to lift heels and relieve pressure on from the heels
- tailbone area
- shoulders and shoulder blades
- elbows

Other self-care tips include the following:\textsuperscript{53}

- Avoid placing pillows under the knees to prevent pressure on the heels.
- Avoid dragging the body when attempting to change position or get out of bed.
- Lift patients or use a draw sheet to move patients.
- Ensure that sheets and clothing are dry, smooth, and not wrinkled.
- Sharp or injurious objects such as pins, pencils, pens, or coins should not be placed on the bed.
- Avoid sliding.
Management of Pressure Ulcers

The primary goal of the health care team is to close the ulcer and also to prevent its further deterioration. This can be achieved by:52

- Cleansing
- Debridement
- Bacterial decontamination
- Dressing
- Pain management
- Adjunctive management

Cleansing

As mentioned previously, pressure ulcers and the nearby area should be cleaned with a non-toxic, mild cleansing solution. Remove any debris and infectious sources from the ulcer bed to accelerate wound healing. Nurses should take care that the cleansers used for cleaning the sore do not further traumatize or disrupt the ulcer. The most recommended solution to clean the wound is saline solution.52

Debridement

Debridement is an important step in the overall management of the pressure ulcers because it can prevent the growth of infectious organisms and promote rapid healing of wounds. Choosing a method of debridement depends on the goals of the patient, absence or presence of infection, pain management, amount of the devitalized tissue involved, and financial considerations for the patient and the institution.52
**Bacterial Decontamination**

Bacterial contamination is a significant factor in preventive care. Bacterial contamination is present in all pressure ulcers. Oral antibiotics or topical sulfa silverdiazine has been found to be effective in reducing the rate of infection. Antiseptics are also useful in managing wound contamination.\(^{52}\)

**Dressing**

Dressings should be used to cover open and closed wounds. It also helps in maintaining an environment conducive for wound healing. A plethora of dressings are available to manage pressure ulcers. There are seven types of dressings used in pressure ulcers:\(^{52}\)

1. Transparent films
2. Foam islands
3. Hydrocolloids
4. Petroleum-based non-adherents
5. Alginates
6. Hydrogels
7. Gauze

Randomized controlled trials have shown that these dressings are efficacious in managing pressure ulcers. These studies also show that non-gauze protocols in pressure ulcer care can enhance the rate of healing.\(^{52}\)

**Pain Management**

Pressure ulcers may be painful especially those that are at stage IV. The goal of managing pain in pressure ulcers is to eliminate continuous
pain. Suitable dressing choices, management of wound by skilled health personnel, and using analgesic medications are all essential in the pain management of pressure ulcers.\textsuperscript{52}

\textit{Adjunctive Management}

Different adjunctive techniques may also be useful in managing pressure ulcers. These have been effective in healing and preventing further cases of pressure ulcers:

- Electrical stimulation
- Hyperbaric oxygen
- Growth factors
- Skin equivalents
- Negative pressure wound therapy

\textbf{Scar Tissue}

A scar is defined as a permanent patch of skin which grows over a wound. Simply put, it is a healed wound. It forms as a result of the body’s natural process of healing a cut, scrape, burn or a sore. Scars may also be a result of surgery that cuts through the skin, infections like chicken pox, or skin conditions like acne. Scars develop at different times. For example, some scars such as those after a burn develop within the first few months following the injury, peaks at around 6 months, and generally resolve within in 12-18 months.

As scars resolve, they gradually fade in color, become flatter, softer and generally less sensitive. However, they are never completely removed.
Scar formation is a natural process that follows a skin injury. Its appearance and treatment rely on a number of factors, such as:

- Depth of the wound
- Size of the wound
- Site of the wound
- Age
- Sex
- Ethnicity
- Genetics

**Types of scars**

There are four different types of scars:

1. Keloids
2. Contracture scars
3. Hypertrophic scars
4. Acne scars

*Keloids*

Keloids are formed as a result of a highly aggressive overgrowth of dense fibrous tissue which may extend further than the site of injury and do not revert on their own. They also project above the surrounding skin but do not extend to underlying subcutaneous tissues; and, usually recur even after surgical excision. They also generally affect dark skinned people and can impede physical movements. The name, keloid is derived from the old name cheloide. It was first used in 1806 by Alibert to refer to crab’s claw, which in Greek translates to *chele*. 
Keloids vary in consistency and are classified accordingly. Some are soft and doughy while others are rubbery and hard. In the early stages, the lesions appear erythematous. Later, they become brownish red and eventually pale. Lesions usually do not have hair follicles and other functioning adnexal glands.

The appearance of lesions differs depending on their clinical course (growth). Some lesions only grow for weeks and months while some grow for years. Keloids develop gradually, although they sometimes enlarge rapidly, doubling in size within months. As their growth stops, keloids remain stable and do not generally cause symptoms.

The shape, size, and overall appearance of keloids also depend on their anatomical location. For example, those found on the ears, neck, and abdomen are usually pedunculated. Keloids on the truncal region and extremities generally have elevated flat surfaces with wider base than the top. Keloids may be oval, oblong or round with regular or irregularly shaped boundaries. Others can take on the appearance of claws.

Keloids above a joint can impair movement. Generally, patients have one or two keloids, however, some patients, especially those with spontaneous keloids, may present with several lesions. Keloids differ from hypertrophic scars by their claw-like projections.

In Caucasian patients, keloids usually present in descending order of frequency. For example, a patient may present with keloids that are predominantly found on the face followed by the upper extremities,
chest, presternal area, neck, back, lower extremities, breasts, and least on the abdomen.

In Africans patients, the descending order of frequency tends start with the earlobes, face, neck, lower extremities, breasts, chest, back, and finally, the abdomen. In Asian persons, the descending order of frequency is earlobes, upper extremities, neck, breasts, and finally, chest.

*Contracture scars*

Contracture scars cause tightening of the skin that can restrict mobility. They usually happen after a burn. They may also the deeper underlying structures in the subcutaneous layer such as the muscles and nerves. Contractures due to severe burn injuries can result in long-term aesthetic and physical repercussions.

Skin contractures bridging or located adjacent to a joint may result in joint deformities that significantly reduce the range of motion (ROM) of the affected joint. Moreover, they are often accompanied by chronic pain that is a challenging to manage and requiring long-term dependency on pain medications. All these factors alone or in combination can seriously result in poor quality of life for the affected individual.

*Hypertrophic scars*

Hypertrophic scars are similar to keloids. They are usually red in color and appear above the area of injury. Unlike keloids, they do not
extend beyond the site of injury. They also don’t grow as big as keloids and may fade with time. They occur in all racial groups.

**Acne scars**

Acne scars are the result of healed acne lesions that range from deep pits to angular or wavelike in appearance. Acne scars are classified according to their appearance and presentation:

- Rolling
- Ice-pick
- Shallow box-car
- Deep box-car

*Rolling scars*, as their name suggest, appear like hills and valleys without sharp margins. They are especially common among individuals with chronic inflammatory acne. They are prone to becoming more pronounced with age when the skin gradually loses elasticity.

*Ice-pick scars* are also called pitted scars. They are the most common scars found among patients with inflammatory acne. They are round with deep depressions that culminate in a pinpoint base.

*Box-car scars*, as their name suggest, appear as relatively broad depressions with steep and well-defined margins. They also have a flat, "u-shaped’ base. *Shallow box-car* scars end in the shallow-to mid-dermis while *deep box-car* scars penetrate deeper into the reticular dermis. These scars usually cover smaller areas than rolling scars.

No matter the type of scar, patients experience varying degrees of its negative psychological effects. Some experience anxiety,
embarrassment while others may experience depression, social isolation, and emotional trauma.

Scars can be disfiguring and aesthetically unpleasant. Some are accompanied by undesirable physical effects such as severe itching, tenderness, pain, and sleep disturbances. And because most scars are difficult to treat and complete skin healing is often incomplete, prevention is the most important aspect of management following skin injury.

**Treatment and Management**

There are several sophisticated scar removal measures that are currently used:65

- Surgical revision
- Dermabrasion
- Laser treatments
- Injections
- Chemical peels, and creams

*Surgical revision*

Surgical revision is a procedure that improves or reduces the appearance of scars. Its primary goal is to restore function and correct disfigurement and deformities. It is also used to make existing scars look more aesthetically acceptable to patients. Although residual scars may not be aesthetically acceptable to patients, it is the restoration of form and function that must be considered first and foremost and not compromised at the cost of cosmesis.
Like other cosmetic surgeries, the general goal is to make patients more comfortable with their appearance. Teamwork between medical providers and patients can ensure the optimal results.

Appropriate preoperative screening of patients, proper nutrition, good technique, and appropriate wound care are all factors that yield rapid wound healing and more aesthetic results. Prior to surgery, a thorough patient history should be obtained. A history of diabetes mellitus and chronic cardiovascular diseases should be assessed carefully since these conditions impair microvascular circulation following surgical revision.

Chronic and occasional smokers are especially prone to flap necrosis and superficial epidermal slough, because of the microvascular constriction effects of nicotine. As such, these patients should be counseled about the higher failure rate of reconstructive procedures. They should also be advised that smoking cessation, even as early as 4 weeks before and after surgery, can significantly reduce the possibility of surgical failure. Other patient factors to consider are chronic steroid therapy, frequent UV light exposure (i.e., tanning and sunbathing) and history of premature return to activity since these predisposes patients to greater keloid formation.

Additionally, the patient's nutritional and immunologic status should also be assessed. As mentioned previously, vitamin and protein deficiencies impair wound healing which is why it is important to counsel preoperative patients about the importance of adequate nutrition. Since a lot of patients are often on self-directed programs of
nutritional and dietary modifications, surgeons must inquire about any non-traditional dietary or nutritional regimens.

Surgical revision is one of the many surgical options that patients may be presented with in treating their scars. Patients often visit plastic surgeons after already having exhausted other non-surgical options such as the use of other cosmetics, clothing, and hairstyle modifications. While it can significantly help with scar management, surgical revision is not perfect.

It is important that patients have a complete understanding of its limitations and be able to adjust their expectations accordingly. Patients should understand that the best results may only come about after several bouts of treatments and that initially, there may only be very little visible improvements over the pre-existing deformities.

Other factors to consider prior to surgical revision are:

- Skin type
- Availability of surrounding tissue
- Anatomical location of the scar
- Functional impairment from current scars, such as in burn patients
- Likelihood of functional impairment after scar revision (i.e., increased tension and reduced range of motion)

It is not unusual for patients to be advised to forego surgical treatment especially if the resultant scar may not be significantly improved. The goal of surgery is to make scars more aesthetically acceptable and less noticeable. Ideally, scars should match the characteristics of the
surrounding skin (*i.e.*, the same color, texture, distensibility, elevation). For example, patients with scars on areas where hair follicles should be present but are not, the ideal surgical result would be to restore these dermal appendages.

Technical details, such as wound characteristics and approximation, also come into play when considering surgical revision. For example, wounds that have undergone significant injury during closure are more to develop dehiscence or skin-edge necrosis, which often result in unpleasant scars. Similarly, wounds that did not heal well, *i.e.*, those that were repaired unevenly, developed infection, or sustained immunologic reactions to sutures, are more prone to yield poor cosmetic results following surgical revision.

Generally, wounds that sustained the greatest trauma yield the poorest eventual scars. Some of the most common causes of dehisced scars are:

- Wounds closed under tension
- Repairs not formed parallel to relaxed skin tension lines (RSTLs)
- Wounds on the trunk or extremities

**Surgical Techniques**

Scar revision techniques include:

- Fusiform excision
- Shave excision
- Z-plasty
- W-plasty
- Geometric broken line closure
- M-plasty
Fusiform excision:

Fusiform excision is a surgical technique that makes uses an elliptical incision around the scar. If needed, the scar should be rearranged in such a way so that it lies in the relaxed skin tension lines of the skin and subjected to axial forces. This is because forces opposite the long axis of the wound can result in longer healing time, widened the scar, and unsightly final result.

Large scars may require undermining and local tissue advancement. Successive excisions and undermining may be used create a final small ellipse. These yield a thinner surgical scar in place of a wider and larger scar.

Additionally, tissue expanders may be placed around the scar to expand healthy and non-scarred skin and permit a single-staged excision of scar tissue for primary closure.

Shave Excision:

The second surgical technique used in scar revision is called shave excision. It is especially indicated in raised scars relative to their surrounding skin. The raised part of the scar is shaved off parallel to the surrounding skin. The wound is then dressed with compressive-type or silicone dressings to prevent recurrence.

Z-plasty:

Z-plasty is a versatile scar revision technique and camouflage. It involves a transposition flap that uses qualities of advancement and rotation flaps. It may be used alone or together with other scar-
camouflage techniques. When done properly, it is a surgical technique that yields a high rate of positive aesthetic results.

The technique permits for two nearby undermined triangular flaps, created from the same central axis, to transpose over each other and to be placed in the other's original bed. Simply put, the two triangular flaps are transposed from rich areas to deficient areas and eventually placed at right angles adjacent to the original central axis.

Z-plasty has several advantages including its ability to:
- Rearrange a scar to be placed in a more desirable location which is in the direction of relaxed skin tension lines (RSTLs);
- Rearrange the scar into a more desirable site or position;
- Split the length of the scar to make it less noticeable;
- Lengthen the contracted scar to decrease the current contractile force and improve conformation to contoured surfaces; and
- Promote the revised scar to lie at an angle different from the older and established scar to reduce the chances of the final scar from becoming depressed.

W-plasty:

W-plasty is also referred to as a running W-plasty or zigzag plasty. It is most effective in correcting long linear and irregular scars. Additionally, it is also very useful in the closure of half-circle incisions wherein the running unbroken scar is more visible and subject to more tension, which over time is more prone to develop into a depressed scar. Unlike z-plasty, the final yield of W-pasty is usually readily
noticeable. Lastly, the final scar is not lengthened but only increased in the final total length.

The basic technique involves making a series of small triangular flaps on opposite sides of the wound, with sides measuring ≤6 mm and the corner angles ≤90°. There are important factors that must be considered in performing basic W-plasty, including:

- The use in curvilinear wounds, and
- The orientation relative to RSTLs.

W-plasty should be used carefully on curving wound borders. The external triangles must be larger in both side length and angle than their counterparts on the inner curve.

Geometric broken line closure (GBLC):

Geometric broken line closure (GBLC) is a type of W-plasty. It makes use of similar deceptive principles as a W-plasty to create greater irregularity in a linear scar, rendering it less noticeable to the eye. GBLC can yield greater aesthetic yield than the basic W-plasty because the resulting greater scar irregularity is a lot less noticeable. Similar to the basic W-plasty, GBLC does not elongate the original scar.

GBLC involves creating a pattern of irregular geometric shapes on either side of the wound. Various geometric shapes with corresponding dimension squares, rectangles, and triangles are placed together resulting in final closure, which interlock into opposite margin counterparts. These shapes must have their widths placed along the length of the scar, with the lesser placed first followed by the greater height close to the ends and the mid region of the scar.
M-plasty:

M-plasty is a surgical technique that is often used to preserve viable tissue and reduce the risk of secondary tissue disfiguration. The techniques in scar revision often make angles more than 30° at the lateral wound margins, which has a two-fold effect.

The first effect is that it preserves the integrity of adjacent tissues. Secondly, it creates a standing cone (i.e., dog-ear) deformity. Because the M-plasty makes two separate 30° angles instead of one, the loss of surrounding tissues are reduced to almost half.

Other Surgical Techniques:

Endoscopic surgical techniques may also be considered to achieve scarless healing since it allows mucosal wounds to heal with minimal scar formation. This is especially useful in larger wounds which are more prone to scar formation.61

Studies have also shown that prolonged intradermal suture support can bring about a significant reduction in the spread of the scar. It has been suggested that in clinical surgical practices, excisions in high-tension regions which are well known to spread over time can benefit from longer acting intradermal sutures.59

In case of burn scars, reconstruction surgery is the best option to prevent scars. The surgical aim is to promote wound closure, correction of tissue deficiencies, and allow postoperative splinting and compression therapy. Reconstruction is carried out in a stepwise approach to restore. First and foremost, it aims to restore active
function followed by passive function and then lastly, to correct aesthetics deficits.

Non-Surgical Options:

Scars formation may be minimized after primary and surgical revision. Additionally, scars may also be initially treated via non-surgical measures, including:

- Corticosteroid injections
- Cryotherapy
- Occlusive therapy
- Medications
- Laser therapy
- Radiotherapy
- Photodynamic therapy
- Combination therapy

*Corticosteroid Injections*

The first line of treatment for prevention and treatment of scars is the use of corticosteroid injections. It helps in the suppression of inflammation and mitosis. It also promotes vasoconstriction in the injured tissues. An injection of 10 to 40 mg per ml of triamcinolone acetonide suspension is usually administered to help flatten keloid. It usually causes mild discomfort and pain. To manage the pain and discomfort of the procedure, lidocaine, a local anesthetic, is usually combined with corticosteroid injections.

Generally, patients receive 2-3 injections, with each one administered one month apart. Sometimes, therapy can last for up to 6 months.
Corticosteroid injections provide the best outcomes when combined with surgical interventions.\textsuperscript{58}

\textit{Intralesional Cryotherapy}

In the past, cryotherapy had limited use in scar management. It was mostly limited to small scars because of its disadvantages:

1. It required several treatments,
2. It required prolonged healing time,
3. It carried the risk of permanent pigmentation, skin atrophy, and pain.

Several studies have shown that cryotherapy improves the following:

- Scar volume
- Scar hardness and redness
- Scar elevation
- Initial pain

In intralesional cryotherapy, a probe is inserted into a hypertrophic scar or keloid which is connected to a canister of liquid nitrogen. It freezes the scar tissue from the inside out and yields an almost 51\% reduction in the volume of the scar following a single cryogenic treatment. In fact, studies have demonstrated it to be more effective than other contact methods of managing scars.\textsuperscript{56}

In the recent years, adjunctive cryotherapy has also been used to augment other non-surgical measures. For example, it is used as an adjunct to corticosteroid injection. It is performed immediately prior to
the injection to help make the procedure easier on patients. Cryotherapy also helps in softening the scar.

Studies have shown that a combination of cryotherapy with corticosteroid injections improves the outcome of preventive and treatment of scars compared to their individual use alone. A disadvantage with the combined use of cryotherapy and corticosteroid injection is the increased risk of hyperpigmentation of the skin. Combination therapy is more effective for newer lesions as opposed to older and more established lesions.

**Occlusive Therapy**

Occlusive therapy includes the use of:

- Silicone gels and sheets
- Non-silicone occlusive sheets
- Cordran tape
- Scarguard

Occlusive therapy can minimize scar formation by reducing the pro-inflammatory or pro-fibrotic cytokine levels. Occlusion can also increase temperatures which can upregulate the expression of collagenase.61

Topical silicone gel has been used in preventive therapy to reduce the formation of scars. It enhances hydration of the wound. Scarguard is a newer over the counter topical product which is formulated from silicone, vitamin E, and hydrocortisone. A trial study has reported its use to substantially reduce erythema and cosmesis of the scar site. It
also stimulates the release of inactive collagenase precursors which may inhibit scar formation and reduce existing scars.\textsuperscript{61}

\textit{Silicone Sheeting}

Some research studies have found that the use of silicone sheets is associated with a reduced frequency of scar formation.\textsuperscript{57} Silicone elastomer sheeting is a non-invasive and a widely studied approach towards the prevention as well as treatment of keloids and hypertrophic scars. Silicone sheets increases the temperature, hydration and the tension of oxygen of the occluded scar thereby softening and flattening it.

Silicone sheets should only be used on healed wounds. It should not be used on open wounds. They should be worn over the scar for about 12 to 24 hours every day for a period of two to three months. They need to be washed regularly with mild soap and water, and reused until they show signs of disintegration. There is a wide range of silicone sheet products available in the market and can be obtained as over the counter products.

In addition to silicone sheeting, pressure dressings or garments can also be used. They are also effective in preventing the formation of burn scars. Pressure dressings (24 to 30 mm of Hg) are recommended to be used for a period of about 6 to 12 months.\textsuperscript{58}

\textit{Combination Therapy}

In case individual occlusive and corticosteroid therapies fail to yield desirable results within 12 months, both therapies can be used
together. Keloid surgery followed by corticosteroid injections can lead to a significant reduction of keloid recurrence. The excision on the scar may be complete or a minute remnant of a scar may be present on the margin of the wound to reduce the recurrence of the scar. A triple combination of silicone sheets, corticosteroid injection, and surgery have been used and found to be more effective in preventing recurrence of scars.\textsuperscript{58}

\textit{Barrier Film}

Barrier films are composed of certain type of absorbable components such as un-cross-linked, carboxymethylcellulose or sodium hyaluronate. They help in reducing scars by absorbing moisture from the site of application and converting into a dry film of gel consistency within 24 hours. This action promotes tissue repair by acting as an occlusive barrier between damaged tissue sites.\textsuperscript{61}

\textit{Laser Therapy}

Different types of laser therapy have been employed for the improvement of hypertrophic scars and keloids, with the best results obtained from pulsed dye laser therapy.\textsuperscript{56} Laser therapy or pulses of light are used to reduce erythema by improving blood flow within the excess scar tissues. In case of pitted scars, laser is used to flatten the scars and remove the epidermal layer of the skin to promote collagen production. Studies have proven that laser therapy is effective, beneficial, and safe in preventing scar formation.\textsuperscript{62}
**Pulsed Dye Laser**

The use of pulsed dye laser in treatment of keloids has only shown limited benefits. However, it has been found to be more effective if used early in the treatment and as an adjunct to other therapies. It yields significant improvement in erythema, pruritis, and scar height which lasts for at least 6 months. Its primary effect is on the scar microvasculature; it reduces erythema and pruritis and improves the skin texture.

Pulsed dye laser also reduces scar thickness to a significant extent.\(^{58}\) It works by causing capillary destruction which causes hypoxemia and hence changes the localised production of collagen.\(^{56}\)

**Dermal Fillers**

Dermal fillers are injections which are especially helpful in managing pitted scars. As their name suggests, they fill or plump up the scars. They are expensive and offer only short term aesthetic effects. The treatment needs to be repeated to maintain the effect.\(^{62}\)

**Skin Needling**

Skin needling makes use of a small device which is made up of small needles. The device is rolled across the skin. This procedure is most helpful in preventing scars. Like dermal fillers, it requires repeat treatments to achieve long-term effects.\(^{62}\)
Radiotherapy

Superficial radiotherapy in low doses is used to reduce the recurrence of hypertrophic and keloid scars following surgery. It is effective in almost 70% of the cases but there is a risk of side effects if used for long term. Radiotherapy is therefore only indicated in severe cases.\textsuperscript{62} Superficial X-rays, electron beams, and low or high dose rate brachytherapy have been effectively used as an adjunct to surgical removal of keloids. Radiation acts by inhibiting the neovascular beds and proliferating fibroblasts, leading to reduced collagen production. The radiation therapy is initiated early, right after the excision of the keloid.\textsuperscript{56}

Photodynamic Therapy

Topical photodynamic therapy has been widely utilized in the treatment of many skin conditions such as basal cell carcinoma and is considered to be a novel approach in the treatment and prevention of keloids. The photodynamic reaction is reported to release reactive oxygen species which leads to cell apoptosis, damage to cellular membranes and mitochondria, and reduces collagen synthesis which results in the improvement of the appearance of the scar.\textsuperscript{56}

Medications

Topical hyaluronic acid (HA) and saponins

The topical applications of hyaluronic acid and saponins enhance the function of hyaluronic acid which is said to leave an anti-scarring effect. Various studies have reported that the use of hyaluronic acid provides have increased the quality of tissue repair and reduced scar
formation. It is recommended to be used continuously on the wound site for a prolonged period of time.

The administration of glucosamine along with hyaluronic acid leads to an enhanced production of hyaluronic acid in the wound and thus, encourages a rapid healing of the wound. It is also said to eliminate complications associated with scarring such as adhesion bands.\textsuperscript{61}

\textit{Topical products}

Topical vitamin E is used by many patients due to their antioxidant properties. Antioxidant action is said to be instrumental in preventing scars. However, studies have suggested that the use of vitamin E may delay the healing of the wound and also reduce the tensile strength of the scar. Thus, its use is not recommended and should in fact, be discouraged.

Certain studies have suggested the use of over the counter topical gels containing onion extract. It is said to significantly improve the softness of the scar, redness, texture and overall appearance of the wound site. A plant extract with betasitosterol has been reported to improve hydration and enhance the rate of wound healing in exposed burn cases.

Another plant extract containing \textit{Centella asiatica} and \textit{Bulbine frutescens} have also been used in the preventive therapy of scars. These substances can increase the wound strength if used within the first six to eight weeks.\textsuperscript{58}
**Imiquimod**

Imiquimod is a 5% topical preparation which can also be used to enhance wound healing and prevent recurrence of scars. It acts as an immune response modifier to prevent keloid recurrence following surgical incision.

The cream is applied on the affected every alternate day for a period of 8 weeks after the surgery. This may cause some adverse effects such as skin irritation and hyperpigmentation.\(^{58}\)

**Bleomycin**

Bleomycin sulphate is a promising new agent that inhibits synthesis of collagen. It has been found to significantly improve hypertrophic scars and keloids.\(^{56}\)

**Interferons**

It has been reported that interferon leads to a significant reduction in the synthesis of collagen I and III and thus, improves and prevents excessive scarring.\(^{56}\)

**Self-care Measures**

According to dermatologists, there are many self-care measures which help reduce scarring. These include:\(^{63}\)

- Gently washing the site of injury with mild soap and water to eliminate debris and infectious microorganisms.
- Daily and regular cleaning of the wound
- Using antibacterial ointment.
• Using petroleum jelly on the wound to prevent drying and formation of a scab. Wounds which have a scab take much more time to heal. The use of petroleum jelly also helps to prevent the enlargement and itchiness of scars.
• Dressing the wound properly and covering it with an adhesive bandage.
• Changing the bandage daily to keep the wound clean while it heals.
• Using hydrogel or silicone gel sheets in larger scrapes, sores, burns or wounds.
• Removing stitches properly.
• Applying sunscreen on the affected area. Once the wound has completely healed, sunscreen should be used religiously prior to sun exposure. It protects the wound from direct sunlight to avoid further skin damage.

Summary

This course discussed skin as part of the integumentary system, and its role to protect the body from different types of damage, such as dehydration, abrasions, and infections. The skin is essentially a barrier to the outside world protecting the body from infectious microorganisms, radiation, and extremes of temperature. It is also the organ responsible for the production of vitamin D.

Underlying conditions leading to skin breakdown and wounds, such as disease (such as diabetes and poor circulation), lifestyle and other risk factors were discussed. Importantly, the role of health providers to educate patients about modifiable options to lifestyle patterns and
treatment for underlying causes of skin compromise and injury has been emphasized.

Predisposing factors leading to poor skin health are key to recognizing risk, intervention and treatment, and need to be included in patient education during routine physical evaluations and when a problem of skin health is identified. Helping patients to be aware of the important function of skin as a barrier to disease and what is needed to nourish and protect skin from injury is an essential part of a nursing clinician’s role.

Please take time to help the NURSECE4LESS.COM course planners evaluate nursing knowledge needs met following completion of this course by completing the self-assessment Knowledge Questions after reading the article. Correct Answers, page 113.
1. The _____ is the deepest layer of the epidermis which contains the stem cells.
   a. Stratum basale.
   b. Stratum corneum.
   c. Stratum spinosum.
   d. Stratum granulosum.

2. All of the following are epidermal cells EXCEPT:
   a. Melanocytes.
   b. Islet of Langerhans.
   c. Langerhan cells.
   d. Keratinocytes.

3. The formation of early reparative coagulum and the activation of intrinsic and extrinsic clotting mechanisms occur during the _____ stage of the wound healing process.
   a. Remodeling.
   b. Inflammatory.
   c. Proliferative.
   d. Hemostasis.

4) The rearrangement and mixing of collagen fibers initially deposited in the wound bed during the proliferative phase occurs during the ______ stage of the wound healing process.
   a. Remodeling.
   b. Inflammatory.
   c. Proliferative.
   d. Hemostasis
5. All of the following are local factors that affect the wound healing process EXCEPT:
   a. Oxygenation.
   b. Infection.
   c. Sex hormones.
   d. Venous insufficiency.

6. True or False: Transient hypoxia in the wound stimulates the wound healing process.
   a. True.
   b. False.

7. _____ refers to the mere presence of infectious organisms on a wound that have not undergone replication.
   a. Contamination.
   b. Colonization.
   c. Local infection.
   d. Invasive infection.

8. What is the hallmark characteristic of diabetic foot ulcers?
   a. Elevated WBCs.
   b. Elevated levels of metalloproteases in the wound
   c. Elevated neutrophil count.
   d. Elevated renal albumin levels.
9. Proteins essential to wound healing include all of the following EXCEPT:
   a. Nerve growth factor.
   b. Substance P.
   c. Calcitonin gene-related peptide.
   d. Albumin.

10. True or False. Low dose aspirin is useful in managing pain and inflammation associated with diabetic foot ulcers.
   a. True.
   b. False.

Correct Answers:

1. a
2. b
3. d.
4. a
5. c
6. a
7. a
8. b
9. c
10. b
REFERENCE SECTION

The reference section of in-text citations include published works intended as helpful material for further reading. Unpublished works and personal communications are not included in this section, although may appear within the study text.


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