APPENDIX 6: GENERAL WOUND HEALING INFORMATION

WOUND HEALING

The wound healing process is defined by the Wound Healing Society (WHS) as “a complex and dynamic process that results in restoration of anatomic continuity and function”.

Wound healing, though often taken for granted, is a very dynamic and delicate process. The wound healing process is a cascade of events beginning with injury to tissue. Appropriate wound management is dependent on an understanding of the normal repair process, the factors affecting this process and the interventions that can impact either positively or negatively on the outcome. Healing progresses in a series of overlapping phases.

The following are the phases of wound healing:

Hemostasis
Hemostasis is characterized by clot formation resulting in cessation of bleeding. This process is enhanced by a 5-10 minute phase of vasoconstriction. The ultimate effect of coagulation is the conversion of fibrinogen to fibrin stabilizing the initial clot. Clot formation facilitates the migration of cells and begins the process of fibroblast proliferation into the wounded area.

Platelet activity, integral to the establishment of a hemostatic plug, is mediated by the release of platelet-derived growth factor, platelet factor 4, transforming growth factor α and transforming growth factor β. These substances stimulate the synthesis of extracellular matrix and initiate tissue repair.

Medical conditions or medications that alter the body’s ability to clot will slow down the initiation of healing.

Inflammation
This is the body’s defense mechanism against bacterial invasion.

Neutrophils and monocytes are attracted at the same time to the wound site. The neutrophils are the most abundant cells present in this phase of healing. Neutrophils migrate through the blood vessels to accumulate in the periphery of the wounded tissue. The main function of these cells is to phagocytose local bacteria and debris. The inflammatory environment stimulates the release of toxic reactive oxygen intermediates that destroy the contaminating bacteria.

The later phases of inflammation are characterized by conversion of monocytes to macrophages. Wound macrophages continue the process of phagocytosis. They generate chemotactic factors and these factors attract additional inflammatory cells. Macrophages are also thought to release growth factors critical to the coordination of granulation tissue formation.
Any medical conditions (autoimmune diseases) or anti-inflammatory medications will alter this phase and delay healing.

**Proliferation**
The phase of proliferation overlaps inflammation. The key cells of this phase of healing are the fibroblasts and myofibroblasts. Fibroblasts secrete a loose extracellular matrix that is rich in collagen. Collagen synthesis peaks 5-7 days post injury. Once this extracellular matrix is deposited, the fibroblasts cease collagen production.

Myofibroblasts have been implicated in a process of wound healing called wound contraction. Wound contraction is the reduction of a tissue defect by the centripetal movement of the surrounding skin. This process of contraction will peak at approximately two weeks after the injury and varies in its ability to reduce wound size by the depth of the wound and the tension of the surrounding tissue.

Angiogenesis or the formation of new blood cells occurs simultaneously with the above two cellular processes. This is the process where capillaries bud from pre-existing small vessels found in close proximity to the wound to form granulation tissue. Granulation tissue is very metabolically active and requires a rich blood supply. If there is insufficient arterial inflow to support the formation of this tissue, fibroblasts and macrophages will stop proliferating and healing will be altered.

Re-epithelialization occurs as keratinocytes migrate across the wound defect. This process is aided by moisture at tissue level. This epithelial tissue is very fragile and is unattached to the wound surface until complete coverage has occurred.

**Maturation**
Remodeling of the immature tissue matrix occurs at the same time as granulation tissue formation. The very vascular granulation tissue is gradually replaced and remodeled forming scar tissue. The composition of the extracellular matrix is in a state of constant change from the time it is first deposited. Tensile strength of the scar tissue is created by collagen deposition and also realignment and remodeling of the tissue. This process occurs for a period of months or years after initial wound closure. The functional strength of scar tissue can only regain up to 70-80% of its pre-injury strength and so is less resistant to tissue breakdown than it was previous to the injury.
### Factors Affecting Wound Healing

| Metabolic Disorders | Systemic diseases, like hepatic, renal, cardiac or lymphedema can lead to increased wound edema and subsequent increased risk of infection and a reduction in epithelialization.  
| | Individuals with diabetes have an altered immune response.  
| | Optimal wound healing is achieved by a holistic approach including blood sugar and blood pressure management. |
| Nutrition | Nutritional deficiencies are associated with a prolonged inflammatory response and delayed wound healing. Lack of key vitamins and minerals can be a significant deterrent to healing. |
| Medications | The most frequently encountered medications that are detrimental to wound healing are: systemic steroids, alcohol, NSAIDS and antineoplastics. |
| Ischemia | Wound hypoxia is harmful to all wound healing. Adequate evaluation of the individual's circulatory status is essential. |
| Infection | All chronic wounds harbor bacteria, but as long as there are no overt clinical signs and symptoms of infection, treatment is not indicated.  
| | The presence of foreign fibres (gauze) or necrotic debris in a wound may predispose it to infection.  
| | Topical antibiotics should not be chosen at random and should be used for a maximum of 14 days. |
| Treatment Regimes | Wound cleansing should be performed using normal saline or sterile water at a pressure per square inch (psi) of 4-15. This can be achieved through the use of a 35 ml syringe and 19 gauge angiocath or 100 ml single use saline squeeze bottle.  
| | Wipe down the wound edges only. Gauze wiped across newly epithelializing tissue can remove all new cell growth.  
| | Consider non-adherent dressings to reduce the risk of tissue trauma in minimally exudating wounds. Maintain a moist, not wet, wound environment. |
| Aging | Aging reduces the rate of healing and alters all phases of healing. Elderly individuals typically have increased skin fragility and special care must be taken to reduce tissue damage with dressing changes. |
| Smoking | Nicotine is a vasoconstrictive drug and will limit oxygen supply to tissues and reduce the potential for healing. Smoking has been shown to cause an increase in scar formation and an increased risk of infection. |
| Malignancy | Wounds are at risk for malignancy. If healing does not occur, a biopsy should be obtained. |
| Psychological Well-being | Stress and insomnia may impact upon wound healing. |