

## 4. Wounds

### 4.1 Acute wounds

#### 4.1.1 Skin tears

The ageing process will impact on most of the structures of skin. Skin loses hair follicles, sebaceous glands that supply natural moisture to the skin, receptors, blood supply and sweat glands. The result of these tissue changes is that the skin becomes thinner, brittle, avascular and more prone to injury. Skin integrity reduces with age, dermal thickness is reduced, and there is a weakened dermal-epidermal junction. The level of vitamin D, collagen and moisture is reduced. Migration of capillary epithelial cells, epidermal turnover is reduced and fragility of capillaries is increased.

The major acute wound in the ageing population are skin tears. The main causative factor is **TRAUMA** from; manual handling e.g. transferring from bed to chair, removing adhesive tapes, falls, cot sides and wheel chair foot plates. Identification of risk factors and the introduction of prevention strategies is essential. In particular, there is good evidence that the use of a moisturising lotion twice a day will reduce the incidence of skin tears by 50 percent. Skin tears may be classified by the Star System or Payne Martin classification.

#### Prevention of Skin tears

Skin tears are a very common acute wound in older patients or people with fragile skin. It is essential to identify risk factors for development of skin tears such as:

- History of previous skin tears
- Dry skin
- Dependent in activities of daily living and ageing
- Paper-thin skin.

It is essential to institute prevention programs to include:

- Choosing appropriate dressing products; silicone coated dressings are the best choice.
- Educating staff, family care-givers, and home healthcare assistants on the importance of maintaining adequate hydration and nutrition.
- Adequately hydrating dry skin with moisturising agents. Note: try to use lotions twice a day on dry skin areas and extremities.
- Dressings should be removed gently and with the use of a no sting adhesive remover if required.

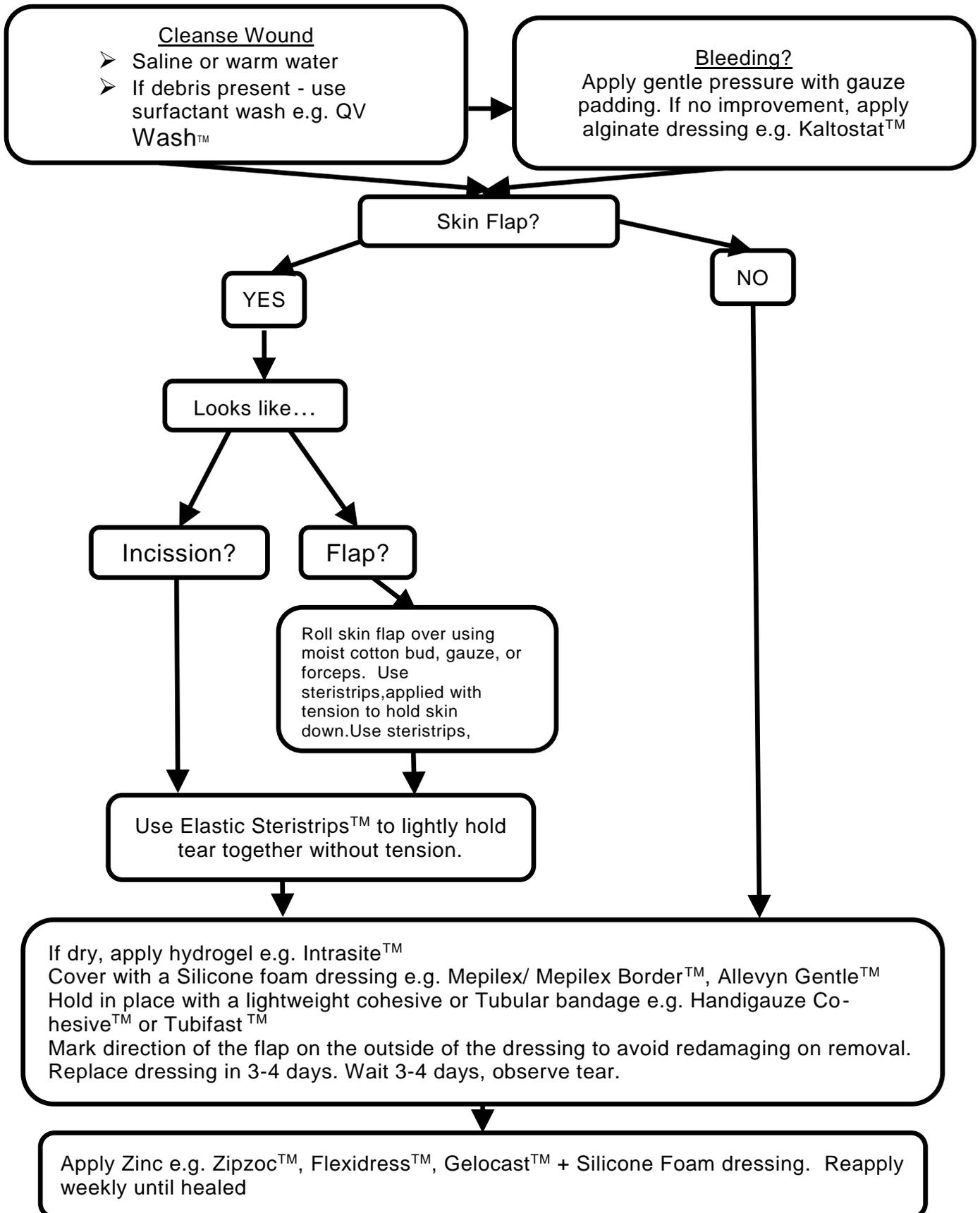
Use of emollient liquid soaps (soft, soothing, moisturising) not bar soap as they are alkaline and will further damage the skin. As a general rule do not use any adhesive products on fragile skin.

## Protocol for wound management of skin tears

- Clean the wound with water or saline. If there is contamination, use a surfactant wash product (e.g. QV Wash™) to help remove any debris.
- If bleeding, apply gentle pressure with a piece of gauze padding, if unsuccessful then apply a haemostatic alginate dressing (e.g. Kaltostat®) to aid in haemostasis.
- If there is major separation of the skin edges, apply an elastic sterile strip, preferably 3-4mm in width to several parts of the skin tear to hold the skin flap in place.
- If injury occurred with contamination apply a low strength povidone iodine solution to the wound, leave in place for 3 minutes and then wash off.
- Apply a small amount of an amorphous hydrogel (e.g. IntraSite Gel™) to the peri wound.
- Cover with a foam dressing, silicone coated are best. Draw the direction of the flap on the outside of the dressing to prevent reinjury on removal (e.g. Lyofoam Max™, Allevyn™, Mepilex®).
- Hold in place with a light weight cohesive bandage, or light weight tubular bandage

This system is left in place for 4 to 5 days at which time the dressing is removed, the wound cleaned and the system replaced. After the next 4-5 days the dressing is removed and replaced with a patch of non-preserved zinc paste bandage (e.g. Gelocast®, ZipZoc™). This is then covered with foam and held in place with a lightweight cohesive bandage (e.g. Handygauze cohesive™) and left for seven days. Upon removal the wound should be healed. If not, then repeat the zinc paste patch dressing system for a further seven days.

## Flow Chart Skin Tear Management



## **4.1.2 Difficult wounds (i.e. animal or insect bites)**

- Clean
- Decontaminate
- Dress

NB. May need systemic antibiotics as infection may be due to atypical micro-organisms.

## **4.1.3 Post-operative wounds**

- Dress over sutures with a film or thin hydrocolloid. Leave in place for 2-3 days or remove if exudate is high or bleeding, or pain or odour is present.
- If exuding apply an island film or an island foam dressing.
- For an open cavity wound, pack lightly with alginates or alginate alternate and/or foam.
- Use strips covered with a film dressing over the incision once sutures or clips are removed. If a hypertrophic scar is observed, then apply a silicone dressing or gel.

## **4.1.4 Burns**

- For sunburn, apply cold running water for 20 to 30 minutes then amorphous or sheet hydrogels.
- For simple burns and scalds of partial thickness, apply cold running water for 20-30 minutes then hydrogels (either sheet or amorphous). Once burn is close to healed, apply a retention tape (e.g. Fixomull® or Hypafix®).
- In general, simple burns do not require silver dressings. If a silver dressing is considered, then use a silver impregnated dressing not a cream as creams will cause a mucilaginous film to form on the surface of the wound.

## 4.2 Chronic wounds

### 4.2.1 Ulcers

Ulcers have a number of different causes, including venous insufficiency, arterial disease, diabetes mellitus, vascular complication of auto-immune disease (such as rheumatoid arthritis), malignant disease, trauma and deliberate self-injury.

#### **Venous ulcers**

Venous ulcers result from the breakdown of the venous circulation of the leg. They are associated with the inability of the leg to force the passage of blood through the various connecting veins via the bicuspid valves by muscular contraction. Deep veins are supported by thick connective tissue and their surrounding muscle mass. Superficial veins dilate easily under sustained back pressure. Communicating veins connect the two systems. Valves, usually bicuspid, are found in all three systems and they may become damaged, thickened or may degenerate with age. Thrombosis can cause their destruction.

#### **General features of venous ulcers**

Venous ulcers are mostly found in the lower third of leg, in the gaiter area. They are usually irregular in shape, can be painful, and oedema of the lower leg is often present. The skin is often stained around the ulcer area due to hemosiderin deposition. Part of the underlying cause can be due to past fractures, trauma or a possible silent deep vein thrombosis (DVT). Skin changes such as eczema and atrophy blanche (white stippled scars on the skin) are often present. Ankle flare (distended small veins on the medial aspect of the foot) may also be seen. There may also be a history of varicose veins. The main feature is a lack of venous return caused by a malfunction of the valve system either in the deep or the peripheral system. There is often a history of obesity, past DVT, and/or poor mobility resulting in venous stasis. Venous leg ulcers are usually painless, irregular in shape, and may have copious exudate.

The treatment for venous incompetence includes surgery in some cases; however, the main stay of treatment is the application of compression therapy- toe to knee 30-40mmHg at the ankle. It is, however, essential to exclude arterial involvement. Exercise should be encouraged and occupational factors such as long periods of standing which leads to venous stasis, should be avoided.

#### **Ischaemia, or arterial ulcers**

The death of skin automatically follows occlusion of its arterial blood supply unless this occlusion is gradual enough to allow a collateral blood supply to be established. Atheroma (thickening) is the most common cause of arterial ulcers of an ischaemic nature. The loss of arterial circulation may be due to extramural strangulation. Scar tissue or other factors may cause strangulation of the arterioles, or fibrosis resulting from longstanding, chronic oedema. Chronic infection may also obstruct arterial flow. Arterial ulcers can result from mural and intramural changes to the vessel walls:

- Mural changes: Atherosclerosis, or plaque formation reduces the blood flow until thrombosis; embolism or infection cause complete closure.
- Intramural changes: Occlusion of small vessels by changes in blood viscosity, platelet adhesiveness or fibrinogenesis, (especially in small painful ulcers of the feet and ankles).

## **General features of arterial ulcers**

Arterial ulcers are very painful, especially at night. This is as marked in small ulcers as in larger ulcers. Their edges are sharply defined, and the ulcer is 'punched out'. The base is often covered with slough, which may deepen to bare the tendons. There is often a history of intermittent claudication (pain on exercise), dependent foot (dusky foot) white on elevation, a history of peripheral vascular disease, lower Ankle Brachial Pressure Index (ABPI), weak/absent pulses, and sluggish/poor capillary refill. The ulcer site is usually below the ankles to the toes. The skin is often shiny and friable. Uncontrolled diabetes and smoking are significant factors causing arterial insufficiency. Healing is often slow and may depend on control of the underlying cause.

Some examples of arterial ulcers are:

- traumatic ulcers on the shin and ankles
- ulcers following fractures
- ulcers caused by ill-fitting calipers or braces
- post-burn ulcers
- ulcers caused by intra-lesional injections (in an area with an already impoverished blood supply).

Treatment of arterial ulcers may involve a surgical intervention - angioplasty, stenting, bypass, grafting and ultimately amputation. Pain control is an important aspect of the management of arterial ulcers.

## **Venous/Arterial (mixed ulcers)**

It is important to note between 10 and 15 percent of leg ulcers are of mixed aetiology. These ulcers are often hard to heal due to associated oedema, cellulitis, thrombophlebitis, diabetes or underlying vascular disease, rheumatoid diseases especially in bed-ridden patients, and general conditions of the skin in elderly patients which are often associated with malnourishment.

## **Other causes of ulcers**

In addition to the more common forms of ulceration, there are a number of less familiar causes. Vasculitic ulcers may develop as a result of other medical conditions, such as those that affect the immune system (e.g. rheumatoid arthritis, lupus and polyarthritis).

Infections of the skin can produce ulcers especially if necrotising bacteria are involved. Other potential causes of ulcers are:

- Haematological problems such as thalassaemia or leukaemia.
- Polycythaemia and skin conditions like pyoderma gangrenosum and epidermolysis bullosa.
- Neoplasia: Some ulcers may be as a result of neoplasia (cancer) which may develop into non-healing ulcers. The most common of these are squamous cell and basal cell carcinomas.
- Lymphoedema ulcers may also form in patients with lymphoedema, caused by a reduction in the function of the lymph vessels to drain extracellular fluid. The resultant oedema will place the patient at risk of ulcer development as a result of minor trauma and by the hyperkeratotic nature of the skin.

## **4.2.2 Wounds in diabetics**

The prevalence of diabetes in Australia is on the rise with estimates of 4 to 6 percent of the population currently having diabetes<sup>5</sup>. The trend is of concern as the number of people diagnosed with diabetes, as of 2015, is 2,000 people every week. Data from 2010 shows that only half of Australians with diabetes were achieving adequate control of their blood glucose levels. Poor control of blood glucose means a higher risk of developing diabetes complications.

This constitutes a very large number of people in this country and many health professionals will be confronted by patients with the problem of diabetic foot ulcers. Many diabetics may have small and minor skin breakdowns which they may not consider important; however, due to their disease, these minor wounds have the potential of becoming serious.

The diabetic patient's foot is subject to neuropathy, ischemia, and infection. There are two major wound types, neuropathic where there is a loss of sensory perception and ischaemic where there is a loss of arterial blood supply. Preventing the diabetic foot should be the first priority. This can be achieved by identifying the high-risk individuals, such as those with peripheral neuropathy, peripheral vascular disease, foot deformities, and presence of callus. The management of the diabetic limb wound is multifaceted with the application of wound products being only one aspect of patient treatment.

Wound management principles are:

- cleansing with minimal trauma
- removal of slough/necrosis where it is safe to do so
- adequately absorption of exudate, prevent/reduce contamination/infection
- protection of damaged/healing tissue and
- off-loading to remove any pressure.

### **How does diabetes affect wound healing?**

Patients with diabetes are prone to have:

- Impaired inflammatory response
- Association of atherosclerosis (small vessel disease)
- Damaged nerves which diminish pain sensation and nerve response
- Up to a fivefold risk of infection.

## **4.2.3 Risk factors in diabetes**

### **Peripheral vascular disease**

A major consequence of diabetes is the damage to both macro-vascular and micro-vascular systems. The resultant reduction in perfusion will contribute to the development of ulcers and also to a delay in wound healing.

### **Peripheral neuropathy**

The lack of feeling or diabetic peripheral sensory neuropathy is the major risk factor for foot ulceration. The fact that the diabetic patient is unable to detect even minor injuries or discomfort in the feet will often place the patient at risk of developing a small wound. Due to the lack of sensation, the patient is unaware of the tissue damage and the wound will progress and only be noticed when it is larger in size. The other indicator may be the presence of odour which may indicate an infected wound. In addition to sensory neuropathies there may be autonomic and motor neuropathies present.

### **Callus formation**

The development of excess callus will elevate plantar dynamic pressure and when combined with peripheral neuropathy, may lead to ulcer development.

### **Limited joint mobility**

This will increase foot pressures and therefore increase the risk of ulcer development.

### **Bony deformity**

Deformities of the ankle, feet, bunions and toes will all increase the risk of ulcers forming.

## 4.2.4 Pressure injury

Pressure injuries are the most preventable of all of the chronic wounds. Pressure injuries may be as simple as the blister most of us may have experienced over the years from footwear, to the extensive pressure injury experienced by bedridden patients suffering from:

- stroke
- spinal injury
- multiple sclerosis
- dementia.

It has been estimated that between six to twelve percent of all patients treated in hospital develop a pressure injury, but sadly, this number increases to about 30 percent in the elderly.

A pressure injury develops when the capillary blood flow to the skin and tissue over a bony prominence is decreased for a sufficient period of time.

The capillary pressure in the arterial blood system is some 32mm of mercury. It therefore requires a pressure of only about 30mm of mercury to restrict the arterial blood flow. The consequence of this restricted blood supply is a reduction in oxygen supply and nutrition to the tissue, accompanied by the problem of waste products not being removed from the site.

The result of this is hypoxia, tissue acidosis, increased capillary permeability (which allows intravascular fluid to escape causing oedema), and cell death. The main causes of pressure injury are:

- pressure
- friction
- shear

### **Pressure**

Direct pressure on tissue over a bony prominence in excess of 30mm of mercury will cause ischaemia in the surrounding tissue. This will occur not only from a patient being in bed, but also on a trolley or sitting in a chair. The extent of tissue damage will depend on the intensity of the pressure, and the length of time the pressure remains unrelieved. The tissue can tolerate pressure for short periods of time; however, even low pressure over a long period of time will have some detrimental effect.

### **Friction**

Friction occurs when the top layers of skin are worn away by continued rubbing against an external surface. This can manifest itself in a simple blister or tissue oedema, or an open pressure injury. This can be caused by ill-fitting footwear, or even bed linen.

### **Shearing forces**

Shear is when the skin remains in place, usually unable to move against the surface it is in contact with, while the underlying bone and tissue are forced to move. This force will contribute to the destruction of microvasculature in a manner similar to direct pressure. This type of pressure injury is seen in patients left sitting up in bed or on a chair, while gravity causes the patient to slide down with the skin adhering to the bed linen or the surface of the chair.

## **4.2.5 Incontinence associated dermatitis and skin injury (IAD)**

IAD is now the accepted term for skin damage caused by exposure to stool or urine. Elderly adults, and especially those in long-term care facilities, are at risk for urinary or faecal incontinence and IAD. It is sometimes confused with stage 1 or 2 pressure injury.

Current clinical consensus supports the following as key components of an effective program for IAD prevention: gentle cleansing with a no-rinse cleanser with pH range similar to normal skin. Moisturisation to maintain normal levels of intercellular lipids and the skin's normal barrier function. Application of moisture barrier product (petrolatum-based, dimethicone-based, zinc oxide-based, or liquid film-forming acrylates).<sup>18</sup>

## **4.2.6 Neoplasia**

Neoplasia may be the cause of skin damage resulting in a wound or skin lesion requiring surgical removal. Wounds may also result from post cancer surgery with active cancer. Neoplasia may also develop in non-healing leg ulcers e.g. marjolin ulcer. Most commonly out of all the neoplasms, squamous cell carcinomas may develop in chronic non-healing venous ulcers.

Sunlight can cause premature ageing of the skin. Moles can be induced by sunlight. Solar keratosis is a form of dysplasia of keratinocytes. Skin cancers include basal cell carcinoma, squamous cell carcinoma and melanoma. It is important if skin cancer is suspected to perform a biopsy to either confirm or exclude cancer.