WOUND HEALING DRESSINGS AND DRUG DELIVERY SYSTEMS: A REVIEW

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Abstract

Wound and its healing process are thought to be complicated process in the world. In fact, a wound heals in an orderly effective and in a systematic manner. The process of wound healing can be divided into four distinct phases but seen to be overlapping with each other. To distinguish the various phases of wound healing biological markers are used. This review describes the details regarding the wound and its types, phases of healing, biological process involved in normal and pathological wound healing. When the skin is wounded, it is healed through a series of processes, such as via the haemostasis/inflammation phase, migration phase, proliferation phase, and maturation phase, during which time exudate containing various growth factors is released at the wound. Wound dressings were employed by dry dressing in the past, and this process heals the wound by drying it and creating crust, but, recently, wet dressings have been used in locations where appropriate temperature and humidity are maintained. Also, besides wet dressings, drug delivery systems and tissue engineering have been used recently for wound treatment, both of which utilize wound dressing products, such as hydrocolloid, alginate, hydrogel, foam, and biological dressings. And now, reaching beyond the existing role of just protecting the wound, wound dressings for drug delivery systems containing drugs and growth factors are being researched so that they can be used for difficult wound treatments, such as chronic wounds or burns. This article reviews the developments of various wound dressings that have been made to date.

Keywords: Wound healing, Phases of wound healing, Control factors, Dressings and bandages.

Introduction: Wound healing is “the provision of the appropriate environment to cure wound by both direct and indirect methods together with the prevention of skin breakdown”[1]. In other words wound care means more than just
putting a dressing onto a wound. It means looking into patient’s general health, lifestyle and factors that might slow healing down. A wound is a physical injury to the body consisting of a laceration or breaking of the skin or mucous membrane, or an opening made in the skin or a membrane of the body incidental to a surgical operation or procedure.\textsuperscript{2, 3} Wounds may be acute or chronic trauma resulting from an injury where, because of a number of factors, the injury does not heal. Acute wounds may be a planned or unplanned event, and healing typically proceeds in an orderly and timely fashion. Examples of acute wounds include a cut, graze or burn. Examples of chronic wounds include leg ulcers, pressure wounds and diabetic wounds.\textsuperscript{4}

**Basic Principles of Wound Care\textsuperscript{5}**

There are three basic principles which underlie wound healing.

1. Identify and control as best as possible the underlying causes.
2. Support patient centered concerns
3. Optimize local wound care.

**Common types of wound\textsuperscript{6}**

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<tr>
<th>Black necrotic wound</th>
<th>Yellow necrotic wound with high exudate</th>
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<th>Cavity wound with high exudate</th>
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<th>Exudating wound with slough and clinical signs of infections</th>
<th>Superficial wound with clinical signs of infection</th>
<th>Malodorous Wounds</th>
<th>Cavity wound with low exudate</th>
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<th>Superficial granulating wound with high exudate</th>
<th>Superficial granulating wound with low exudate</th>
<th>Epithelizing wounds</th>
<th>Skin tears</th>
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There are four phases of wound healing:

- Inflammatory
- Migratory
- Proliferative
- Maturation

During all of these phases there are a number of cells that are essential to the process of the healing including platelets, neutrophils, macrophages and fibroblasts. Some of the cells which you may think are only present for one particular phase of the healing processes are there from the very beginning of the wound, through to the ultimate healing of the wound. The critical thing is that the phases of healing are a continuum. Each phase continues in a steady process merging with the next phase. In fact, one wound may be in more than one phase at one time.

Figure 1. Schematic representation of the phases of wound healing (a) infiltration of neutrophils into the wound area (b) invasion of wound area by epithelial cells (c) epithelium completely covers the wound (d) many of the capillaries and fibroblasts, formed at early stages have all disappeared.

**Phases of Healing**

1. **Haemostasis and Inflammation**

  Bleeding usually occurs when the skin is injured and serves to flush out bacteria and/or antigens from the wound. In addition, bleeding activates haemostasis which is initiated by exudate components such as clotting factors.
Fibrinogen in the exudate elicits the clotting mechanism resulting in coagulation of the exudates (blood without cells and platelets) and, together with the formation of a fibrin network, produces a clot in the wound causing bleeding to stop. The clot dries to form a scab and provides strength and support to the injured tissue. Haemostasis therefore, plays a protective role as well as contributing to successfully wound healing. The inflammatory phase occurs almost simultaneously with haemostasis, sometimes from within a few minutes of injury to 24 h and lasts for about 3 days. It involves both cellular and vascular responses. The release of protein-rich exudate into the wound causes vasodilation through release of histamine and serotonin, allows phagocytes to enter the wound and engulf dead cells (necrotic tissue). Necrotic tissue which is hard is liquefied by enzymatic action to produce a yellowish coloured mass described as sloughy (Tab. 1). Platelets liberated from damaged blood vessels become activated as they come into contact with mature collagen and form aggregates as part of the clotting mechanism.

2. Migration

The migration phase involves the movement of epithelial cells and fibroblasts to the injured area to replace damaged and lost tissue. These cells regenerate from the margins, rapidly growing over the wound under the dried scab (clot) accompanied by epithelial thickening.

3. The proliferative phase

The proliferative phase occurs almost simultaneously or just after the migration phase (Day 3 onwards) and basal cell proliferation, which lasts for between 2 and 3 days. Granulation tissue is formed by the in-growth of capillaries and lymphatic vessels into the wound and collagen is synthesized by fibroblasts giving the skin strength and form. By the fifth day, maximum formation of blood vessels and granulation tissue has occurred. Further epithelial thickening takes place until collagen bridges the wound. The fibroblast proliferation and collagen synthesis continues for up to 2 weeks by which time blood vessels decrease and oedema recedes.

4. The maturation phase

The maturation phase is the final stage of healing. During this stage the fibroblasts decrease in number, vascularization decreases, and the tensile strength of the wound increases. Maturation is the most misunderstood phase of healing. It is assumed that a wound is healing once the epithelium has closed the surface of the wound. Tensile strength of the wound may in fact take quite a considerable time to develop, and in some patients it can take up to twelve months. It is often incorrect to view a re-epithelialized wound and think that the wound is totally healed.
beneath the surface. The lack of tensile strength in a wound will increase the risk of breakdown that may be related to tension of the tissue below the surface.

**Moist wound management**

Knowledge and understanding of wounds, tissue and healing have grown rapidly over the past 40 years, resulting in a major change in the method of wound management. There has been a growing awareness that traditional wound-care products do little to aid healing, and in many cases actually delay it. Traditional theory has always been that wounds should be kept clean and dry so that a scab may form over the wound, the wounds should be exposed to the air and sunlight as much as possible, and where tissue loss is present, the wound should be packed to prevent surface closure before the cavity is filled, and then the wound should be covered with a dry dressing. The clear disadvantages of these principles are that the scab, which is made up of the dehydrated exudate and dying dermis, is a physical barrier to healing, which is then delayed because the epidermal cells cannot move through the scab formed. This may ultimately lead to a poor cosmetic result, even scarring. Exposure to the air reduces the surface temperature of the wound causing peripheral vasoconstriction affecting the flow of blood to the wound which further delays healing. This reduced blood flow will also affect the supply of oxygen, nutrition and other factors to the wound. Air exposure will also cause the wound to desiccate and form a scab. Where a wound is packed with dry gauze, the quality of healing is impaired due to adhesion of the material to the surface of the wound, causing it to dry out. Equally, covering the wound with a dry dressing that adheres to the wound may traumatize the wound surface on removal. Wounds covered by an occlusive dressing do not form a scab, so epidermal cells are able to move rapidly over the surface of the dermis through the exudate which collects at the wound/dressing interface. The application of a totally occlusive or semi-permeable dressing to the wound can also prevent secondary damage as a result of dehydration.

**Control Factors Affecting Healing**

Most wounds heal readily whereas others are slow or remain unhealed for a considerable length of time. There are a number of factors which affect the healing of a wound, and these factors are both intrinsic and extrinsic.

**Intrinsic factors**

- health status
- immune function
- diabetes
age factors
body build
nutritional status.

**Health status:** Good circulation, both arterial and venous, is essential for good wound healing. Anaemia, regardless of type, reduces the capacity of the blood to provide oxygen to the tissues, since haemoglobin transports oxygen to the cells.

**Immune function:** Normal immune system function is required for the inflammatory phase of healing. A reduction in immune function slows the cleansing of the wound bed and reduces the ability of the body to fight invading pathogens (This is likely to be due to a reduction of the number and activity of the white blood cells).

**Diabetes:** Diabetes is one of the major problems for chronic wounds. Diabetics have a delayed capillary response to injury, reduced cellular function at the injury site, and defects in collagen synthesis and wound strength. Hyperglycaemia (caused by reduced insulin availability and increased insulin resistance) appears to be a major predisposing factor in delaying healing in diabetic patients and increasing the rate of infection.

**Age factors:** As we age our skin and tissues change. We lose the sensory cells, as well as the secretory cells which are so essential for the maintenance of skin moisture and flexibility. We lose the vasculature within the skin, and hair follicles. The skin becomes thinner, dryer, and far more prone to destruction whether by physical or by chemical means.

**Body build:** Because of the adipose tissue being poorly vascularized, an obese patient will have a great deal of trouble healing due to the inability to deliver oxygen and nutrients to the wound site. Underweight individuals may also experience difficulties in the healing process.

**Nutritional status:** Nutrition is one of the most important factors in the healing of wounds. Proteins, carbohydrates, fats, vitamins, trace elements and fluids all play a vital role in wound repair. Research has shown that amino acids (eg. Arginine), when given as a supplement, will improve the rate of wound healing.

**Extrinsic factors**
- mechanical stress
- debris
- temperature
- desiccation / maceration
- infection
- chemical stress
other factors.

**Mechanical stress:** When a patient is immobile and pressure is exerted locally, especially over a bony prominence, for more than two hours, at a pressure exceeding 30 mm of mercury, localized micro vascular ischemia will occur. This will ultimately lead to tissue destruction both at the surface and deeper into the wound-leading eventually to a pressure ulcer. Equally, shearing forces and friction occur when the tissue below the skin is forced to move while the skin itself is restrained by contact to a surface, such as the bed sheet. This is particularly evident in the patient’s heels.

**Debris:** Debris-whether slough, eschar, scab, wound dressing residue, gauze fibers or sutures- will impede wound healing. Their presence will prolong the inflammatory phase, as well as predisposing the wound to infection. Debris should be removed, either surgically or by the use of hydrogels, proteolytic enzymes or hydrocolloids.

**Temperature:** The optimum temperature for the growth of human cells is 37 degrees centigrade. It is therefore essential to maintain the wound environment at body temperature. A drop in body temperature will lead to peripheral vasoconstriction- affecting the flow of blood through the wound- and it will markedly reduce the activity of growth factors and proteases. Increased body temperature can lead to changes at the wound site increasing the risk of cellular breakdown and limiting the healing process.

**Desiccation:** If a wound dries, healing is either delayed, or will cease. Exposed, dry wounds are more inflamed, painful, itchy, and have more scab material during the early stages of wound healing.

**Maceration:** Maceration may be due to incontinence, perspiration or excessive exudation. Maceration will cause the destruction of tissue and slow the healing process. It is essential to maintain the moist environment without excessive exudation.

**Infection:** All wounds will have some level of bacterial colonization; however, this does not mean that the wound is infected. The presence of erythema, discharge, fever, pain with elevated white blood cell count, and sometimes odour, is evidence that the wound is infected. If clinical signs of infection are present, the use of systemic antibiotics is mandatory. If there are no clinical signs of infection there is little reason to use either systemic or topical antibiotics. An exception to this may be the use of very specific topical antibiotics in very specific cases to reduce the level of bacteria in wounds of compromised patients (eg. the use of topical metronidazole in anaerobic colonized wounds).
Chemical stress: Iodine, peroxide, Chlorhexidine, alcohols, hypochlorite and acetic acid are commonly used antiseptics and cleansing agents. Use of these agents is often responsible for delayed healing, since they are non-selective in their activity and will kill healthy cells as well as bacteria. It is preferable to avoid the prolonged use of these products on a granulating wound. Even their use in infected wounds is somewhat dubious, as research has shown that although they may reduce the surface load of bacteria in an infected wound, they do not penetrate below the surface and therefore have no real effect on the infection in the tissue itself. They may be of use in dilute forms when applied to some chronic wounds and left in place for no more than five minutes and then washed off.

Other factors

A) Systemic medications

The effects of systemic medications on the healing wound vary greatly. We commonly see medicine prescribed for a condition which is unrelated to the wound, but may have side effects which could either inhibit or stimulate healing. Medications can therefore be divided into two groups; the stimulatory drugs and the inhibitory drugs. The stimulatory drugs affect the inflammatory response, epithelialisation, fibroblast activity, fibrinolysis, and cell stimulation, whereas the inhibitory medications affect tensile strength, cell activity, capillary proliferation, and fibroplasia.

B) Alcohol

Excessive and/or chronic alcohol intake can lead to health problems affecting wound healing. Alcohol-induced digestive problems may lead to malnutrition and anemia. Liver damage can result in chronic disturbances due to a reduction in platelet levels, and subsequent circulatory damage that may reduce the blood flow that is required for wound healing.

C) Smoking

The adverse effects of smoking and the potentiation of cancer in various parts of the body have been understood for many years. However, it is clear that the toxic constituents of smoking such as nicotine, carbon monoxide and cyanide have a dramatic and inhibiting effect on healing. Nicotine will diminish red blood cells, fibroblasts and macrophages, and increase platelet adhesiveness. This will produce cutaneous vasoconstriction. Carbon monoxide has an affinity for haemoglobin 200 times greater than that of oxygen. This will have a major effect on the oxygen-carrying capacity of the blood and may potentially lead to ischemia. Hydrogen cyanide inhibits the enzyme-systems
necessary for oxygen transport at the cellular level, as well as oxidative metabolism. Smoking can therefore be a major cause of the non-healing of wounds.

General Overview of Wounds

Complex and chronic wounds

A large proportion of wounds seen in clinical practice are chronic in nature. The epidemiological studies indicate that one percent of the population has a chronic wound, and of that group some twenty percent have had the wound for more than two years.

More studies indicate that the level of chronic wounds in older patients is considerably higher than that percentage.

Chronic wounds may be classified into the following groups:

- Leg ulcers
- Pressure wounds
- Post-operative wounds
- Neoplasia (Cancer)
- Chronic infected wounds
- Diabetic wounds

The difficulty in the management of any chronic wound is that there is always an underlying physiological cause of the wound which must be treated, but many patients have multi-factorial issues and co-morbidities. For best results the basic cause of the problem must be addressed, and any negative factors altered. It must be understood that some patients may never heal due to the basic pathophysiology of the disease process and our inability to alter some or all of the major factors influencing the non-healing of the wound. However, even in the most extreme cases, good wound care can be a great help in minimizing the worst effects of such chronic wounds.

Acute wounds

Acute wounds may show some of the following characteristics:

- higher risk of infection due to debris contaminating the wound
- inflammation occurs
- may heal by primary intention
- may require antiseptic use
Chronic wounds may show the following characteristics:

- lower risk of infection
- symptom of underlying condition
- heal by secondary intention
- May be sloughy and moist, or dry and scabby.

**Chronic Wounds includes:**

**a) Leg ulcers**

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

**b) Venous ulcers**

Venous ulcers result from the breakdown of the venous circulation of the leg, and are an association of 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 masses. 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:** They are most often found in the lower 1/3 of leg (in the gaiter area) are usually irregular in shape, may not be painful, and oedema is often present. The skin is often stained around the ulcer area due to hemosiderin deposition. Part of the underlying cause is past fractures or trauma with a possible silent deep vein thrombosis (DVT). Skin changes such as eczema and atrophy blanche (white stippled scars on the skin) are often present, and ankle flare, distended small veins on the medial aspect of the foot are seen and there is 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 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.

**Ischemia, 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 ischemic 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 fibrino genesis, (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 Index (ABI), weak/absent pulses, and sluggish/poor capillary refill. The ulcer site is usually below the ankles to 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 the 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 callipers or braces
- post-burn ulcers
- Those caused by intra-lessional injections (in an area with an already impoverished blood supply).

Treatment of arterial ulcer 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 that between 10 percent 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 is 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 (eg. rheumatoid arthritis, lupus and polyarthritis).

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

Hematological problems such as thalassemia or leukaemia.

Polycythaemia and skin conditions like pyodermagangrenosum and epidermolysis bullosa.

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.

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 hyperkerototic nature of the skin.

The Diabetic

The prevalence of diabetes in Australia is on the rise with estimates of 3 to 4 percent of the population currently having diabetes. The trend is of concern as the number of people diagnosed with diabetes, as of 2006, is 1925 people every week.

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 a small and minor skin breakdown which they may not consider important however, due to their disease, these minor wounds have the potential of becoming serious.
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.

Pressure Ulcers

Pressure ulcers are the most preventable of all of the chronic wounds. Pressure wounds may be as simple as the blister most of us may have experienced over the years from footwear, to the extensive pressure sores experienced by patients suffering from mobility decreasing conditions. It has been estimated that between six to twelve percent of all patients treated in hospital develop pressure wound, but sadly, this number increases to about 30 percent in the elderly.

Pressure wound develops when the capillary blood flow to the skin and tissue over a bonyprominence 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 wounds are**

- **Pressure**: Direct pressure on tissue over a bony prominence in excess of 30mm of mercury will cause ischemia 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 wound. 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.

**Wound Dressings and Bandages**

**Wound pharmaceuticals / Wound healing product classification**: The history of the development and use of dressings has seen an evolution through many centuries from inert and passive products such as gauze, lint and fibre products to a dazzling range of modern moist wound dressings. The range of dressings increases every year and is often a source of confusion when attempting to differentiate between both similar and different dressings. The simplest way of classifying dressings is by their functionality.
Wound products can be divided into two broad groups:

- Passive products
- Interactive products

Within these two groups, the passive dressings can be sub-classified into absorbing and non-absorbing dressings whereas the interactive dressings can be sub-classified as absorbing, non-absorbing and moisture donating. The interactive group has six different dressing types.

**Ideal properties of wound dressing**

1. Remove excessive exudate from the wound without allowing the wound to dry out thereby maintaining a moist environment
2. Allow gaseous exchange so that oxygen, water vapour and CO2 may pass in and out of the dressing
3. Be thermally insulating so as to maintain the wound core temperature at approximately 37 degrees C
4. Be impermeable to micro-organisms in order to minimize contamination of the wound from outside the wound itself
5. Be free from either particulate or toxic contamination
6. Be non-traumatic and not adhere to the wound, so that at dressing change it will not damage granulating tissue.

**Passive dressings**: For many years the products used were of the 'passive' or the 'plug and conceal' concept and included gauze, lint, non-stick dressings, and tulle dressings. Passive dressings fulfil very few of the properties of an ideal dressing and have very limited (if any) use as a primary dressing, however, some are useful as secondary dressings.

In addition to gauze, lint and cotton dressings, other simple modified absorbent pads covered with a perforated plastic film to prevent adhering to a wound (such products include Melolin™, Cutilin™ and Telfa®) are used both as primary and secondary dressings. They are used in minor and low exuding wounds.

**A modern inert dressing**: Exudry™ and Mesorb® are examples of products with a highly absorbent pad and a non-stick, nonshear surface. They can be used as a secondary dressing over moderate to highly exuding wounds and over hydrocolloid paste, cadexomer iodine, alginate and other primary dressings.
The non-absorbent passive dressings

The non-absorbent passive dressings such as paraffin gauze (tulle) dressings are among the earliest modern dressings. Many variations have been developed over the years by changing the loading of paraffin in the base. In general, these dressings produce a waterproof paraffin cover over the wound which may lead to maceration in that water vapour and exudation may not pass through and so become trapped within the wound. These products:

- are permeable to bacteria
- are known to adhere to the wound causing trauma on removal
- require a secondary dressing.

Their use is limited to simple, clean, superficial wounds and minor burns. They are also used as a primary dressing over skin grafts. There are modern alternative dressings composed of synthetic fibres tightly meshed and impregnated with materials that allow moisture to pass through and thus minimize any maceration of the wound and tissues. Examples include Adaptic™, Cuticerin™ and Atrauman®.

Non-Absorbing dressings

1. Film Dressings (for wounds with no to low exudate)

These dressings consist of a thin, poly-urethane membrane coated with a layer of acrylic adhesive and:

- are waterproof
- are gas/vapour permeable
- are flexible
- protect from shear, friction, chemicals and microbes
- are transparent
- spread tension forces.

They are particularly useful in superficial, clean wounds and in the prevention of breakdown and preulcers in pressure wounds. They are also used as a post operative dressing over sutures and to reduce sub-tissue tension over a closed sutured wound after removal of the sutures or clips. Film dressings should not be used for infected wounds.

2. Hydrocolloid dressings (for wounds with low to moderate exudate): Hydrocolloids are a combination of polymers held in a fine suspension, and often contain polysaccharides, proteins and adhesives. When placed on a
wound the polymers combine with the exudate and form a soft, moist gel-like mass. They also encourage autolysis to aid in the removal of slough from a wound.

They:

- are flexible and waterproof
- provide a physical barrier
- form a gel with exudate
- aid in debriding
- need no secondary dressing
- are available in a thin form (transparent).

The early forms of hydrocolloids are occlusive, so as to make them impermeable to gases and water vapour. They do, however, act as a barrier to external bacteria and are waterproof enabling the patient to shower. The thin, transparent hydrocolloids have a polyurethane film backing, are nonocclusive, thereby allowing the passage of water vapour and gases.

Hydrocolloids should be applied over the wound with at least 3-4 cm extra product greater than the size of the wound. The skin should be dry and free from creams, ointments or oil to ensure good adhesion. The dressing should be placed 1/3 above the wound and 2/3 below the wound, as this will prolong the wear time of the dressing. The dressing can remain in place for up to 7 days with removal dependent on the level of exudate and when ‘strikethrough’ has occurred (i.e. the exudate has migrated to the edge of the dressing)\textsuperscript{29}.

Hydrocolloid products are used in low to moderately exudating wounds- including ulcers, donor sites (after haemostasis) and granulating wounds\textsuperscript{30}. They may also be used in conjunction with a paste or powder form. The paste or powder is used in a deeper ulcer or cavity. These convert to the same hydrophilic gel and are covered with the normal hydrocolloid wafer.

3. Foam dressings (for wounds with medium to high exudate)\textsuperscript{31}

These products are soft, open-celled hydrophobic/ hydrophilic non-adherent dressings that may be single or multiple layered and meet many of the properties of an ideal dressing.

They:

- allow the passage of exudate through the non-adherent surface to be absorbed in the main body of the product
are absorbent
- maintain a moist environment
- are thermally insulating
- are cushioning
- are non-adherent
- are non-residual.

Foams are mainly used in moderately to heavily exudating wounds- including ulcers, donor sites and minor burns, and they act as a secondary dressing, particularly as a covering with the use of amorphous hydrogels\textsuperscript{32}. In addition to standard and waterproof foams there is also a charcoal impregnated version, Lyofoam C\textsuperscript{TM}, as well as shaped cavity devices which may be inserted into cavity wounds or gaping surgical wounds.

4. Alginate dressings (for wounds with medium to high exudate)\textsuperscript{33}

Alginates are the calcium or sodium/calcium salts of alginic acid, composed of manuronic and guluronic acids obtained from seaweed. When applied to a wound, the sodium salts present in the wound exchange with the calcium in the alginate to form sodium alginate which is a hydrophilic gel. This gel has the ability to absorb exudate into itself while maintaining a moist environment at the interface between the dressing and the tissue.

Alginate dressings\textsuperscript{34}:

- are highly absorbent
- form a gel with exudate
- create a moist interface
- are easily removed
- are haemostatic.

Alginates are used on donor sites, bleeding sites, exuding leg ulcers and cavities. They are not considered to be of value in low exudating wounds or dry wounds with eschar. They come in a number of different forms, including sheets, packing rope and in combination with charcoal for exuding malodorous wounds\textsuperscript{35}. Sheet alginates should be cut to the shape of the wound, placed on the wound and covered with a suitable secondary dressing (eg. foam and non-stick dressings). They should then be held in place with a cohesive or tubular bandage. If the wound is highly exudating then an outer absorbent pad may be added. In the case of a moist cavity wound, the rope or packing ribbon is gently placed into the cavity taking care not to pack the material tightly into the space. In general, alginates should
be changed when they have fully converted to a gel, this will vary from 1 to 3 days depending on the level of exudate in the wound. When used on a donor site, the material is placed over the area after skin harvesting and covered with a film or a foam dressing, which can remain in place for up to 7 days.

5. Hydrofibre dressings

Hydrofibre dressings have some of the properties of alginates in that they are a fibre rope or dressing that forms a firm gel in contact with fluid.

They:

- are composed of a synthetic fibrous mat
- form a firm gel in contact with exudate
- are highly absorbent
- have no lateral wicking → protects peri-skin

An example of this product is Aquacel®.

6. Hydroactive dressings (for wounds with medium to high exudate)

These multi-layered highly absorbent polymer dressings with a surface adhesive and a waterproof outer layer are similar to hydrocolloids; however, instead of forming a gel in contact with exudate, the fluid is trapped within the product itself, to maintain a moist environment.

Hydroactive dressings are:

- highly absorbent polymer dressings
- waterproof
- expandable
- non-residual
- Semi-permeable.

Hydroactive dressings are indicated for use in highly exudating surface and cavity wounds including leg ulcers, pressure wounds and minor burns. They are particularly useful over joints such as elbows, knees, fingers and toes due to their ability to expand and contract without causing constriction. Hydroactive dressings are not indicated for dry or lightly exudating wounds.
Hydrogels (For dry or sloughy wounds)\(^{39}\)

Hydrogels are a group of complex organic polymers having a high water content ranging between 30 to 90 percent. This broad class of polymers swells extensively in water, but does not dissolve in water. They have the properties of both rehydrating dry tissue, and absorbing certain amounts of fluid into themselves. They are provided as either amorphous gels or sheet gels. These products are used to help re-hydrate sloughy wounds and necrotic tissue to aid in the autolytic debridement of wounds. They are also used in the management of burns, including sunburn, scalds and other partial thickness burns. Amorphous hydrogels have also been used in the management of chicken pox and shingles, applied to the eruptions three to four times a day. They provide a moist environment and relieve the discomfort of the lesion, and also reduce the probability of scarring. Hydrogels are also available in sheet form consisting of a cross-linked polymer and water held in abacking. These products are particularly useful in the management of burns, and also to aid the removal of necrotic tissue in pressure wounds\(^{40}\).

Conclusion

Wound healing is a complex biological process that consists inflammation, proliferation, and remodeling. New wound dressings and bandages can help facilitate eventual healing by providing prophylaxis against barriers to healing, augmentation of wound healing factors, assistance in temporizing and bridging time to definitive repair, and optimization of the ultimate results of wound reconstruction. Current wound healing products and modalities increase the armamentarium of the wound practitioner to address all aspects of wound care.

References


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