WREN

(Wound Resource Education Nurse)

Regional Wounds Victoria
Loddon Mallee

Pre-Education Learning Package

Due Date: Sunday 20th February at 10pm

Please contact the RWV CNC who covers your health service with any queries, or to discuss anything you don't understand. See contact details on the RWV Loddon Mallee Webpage:
https://www.regionalwoundsvictoria.com/loddon-mallee

Booklet

Please allow at least 4 hrs to complete this booklet and do the quiz.

You'll also notice 'questioning WREN' icon (above) throughout the booklet.
Each time you see it, please stop reading and consider the reflective question posed.
Within the booklet there are also links to additional resources.
Please take the time to follow these links and read the extra material prior to completing the quiz.
You don't need to submit this booklet.

Online Quiz

Your link to the quiz will be emailed to you.

Please check the email you registered with. This link is unique to you, so please don't share it.

You don't need to complete the quiz in one sitting.

You may exist and re-enter with your unique link, as often as you like until the due date, however your answers may not be saved for each attempt, so jot down your
Your answers are not 'final' until the due date, so feel free to go back in and review them until then.

We recommend 60mins to complete the quiz.

The aim is for you to learn as much as you can, so next to each question you'll find a 'hint' telling you which section of this booklet to review to be sure your answer is correct.

Contents

1. Objectives

a) Identify basic skin anatomy, its functions and the major changes associated with ageing

b) Recall ways to maintain healthy skin, thereby reducing the risk of developing a wound

c) Identify initial causes of wounds and reasons why some wounds become hard-to-heal

d) State the major events that occur for each of the three phases of wound healing

e) Recognise necrotic, sloughy, granulating and epithelialising wound tissue

f) Recognise peri-wound maceration and excoriation

g) Recall several initial causes of wounds

h) State several reasons why wounds become hard-to-heal

2. Main Functions of the Skin

a. Protection

The skin helps protect internal tissues by acting as a barrier to disease producing micro-organisms (pathogens), mechanical trauma and dehydration. Some cells in the skin also play an active part in our immune system.

How might large wounds affect fluid balance?

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b. Thermoregulation

Thermoregulation is the body's ability to keep its temperature within certain boundaries even when the surrounding temperature fluctuates.

What would this mean for people with large burns?

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c. Sensation

Sensation of pain, heat, cold, touch, pressure and vibration result from stimulation of nerve receptors in the skin. Some stimuli are unpleasant and initiate a reaction that helps protect us from injury, while other stimuli are pleasant, and contribute to our quality of life

People with diabetes sometimes lose sensation in their feet due to peripheral neuropathy. How might this affect their risk of injury?

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It is sometimes assumed that someone who is ambulant and self-caring will not be at risk of pressure injury. Can you think of a few examples where an ambulant, self-caring person might be at risk of pressure injury as a result of reduced sensation?

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d. Absorption

The skin is able to absorb some substances such as some medications, plant toxins and other chemicals through direct contact.

Can you name some medications that are delivered through skin patches?

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What is the scientific definition of a 'chemical'? (Try 'googling' it)

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e. Excretion

The skin also excretes some substances, such as salt and other minerals.

f. Storage and Production / Synthesis

Water, fats, cholesterol and vitamins A, D, E & K are all stored by the skin, while vitamin D is an example of something produced by the skin. Vitamin D production occurs as a result of interaction with UV light from the sun.

We all need exposure to small amounts of sunlight to produce vitamin D. A balance is required between avoiding an increase in the risk of skin cancer by excessive sun exposure and achieving enough exposure to maintain adequate vitamin D levels.

g. Cosmesis

Cosmesis is defined as "the preservation, restoration or bestowing of bodily beauty". The condition of the skin plays an important part in how we feel about ourselves and respond to others.

Have you ever suffered from significant acne, a scar, rash or wound? How did you feel about it at the time?

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3. Skin Anatomy

Our skin is one of the largest organs of the body. It has three main layers, the epidermis, the dermis and they hypodermis. Figure 1 shows the main layers and some of the major structures of the skin.

On the following pages, these are discussed in more detail.

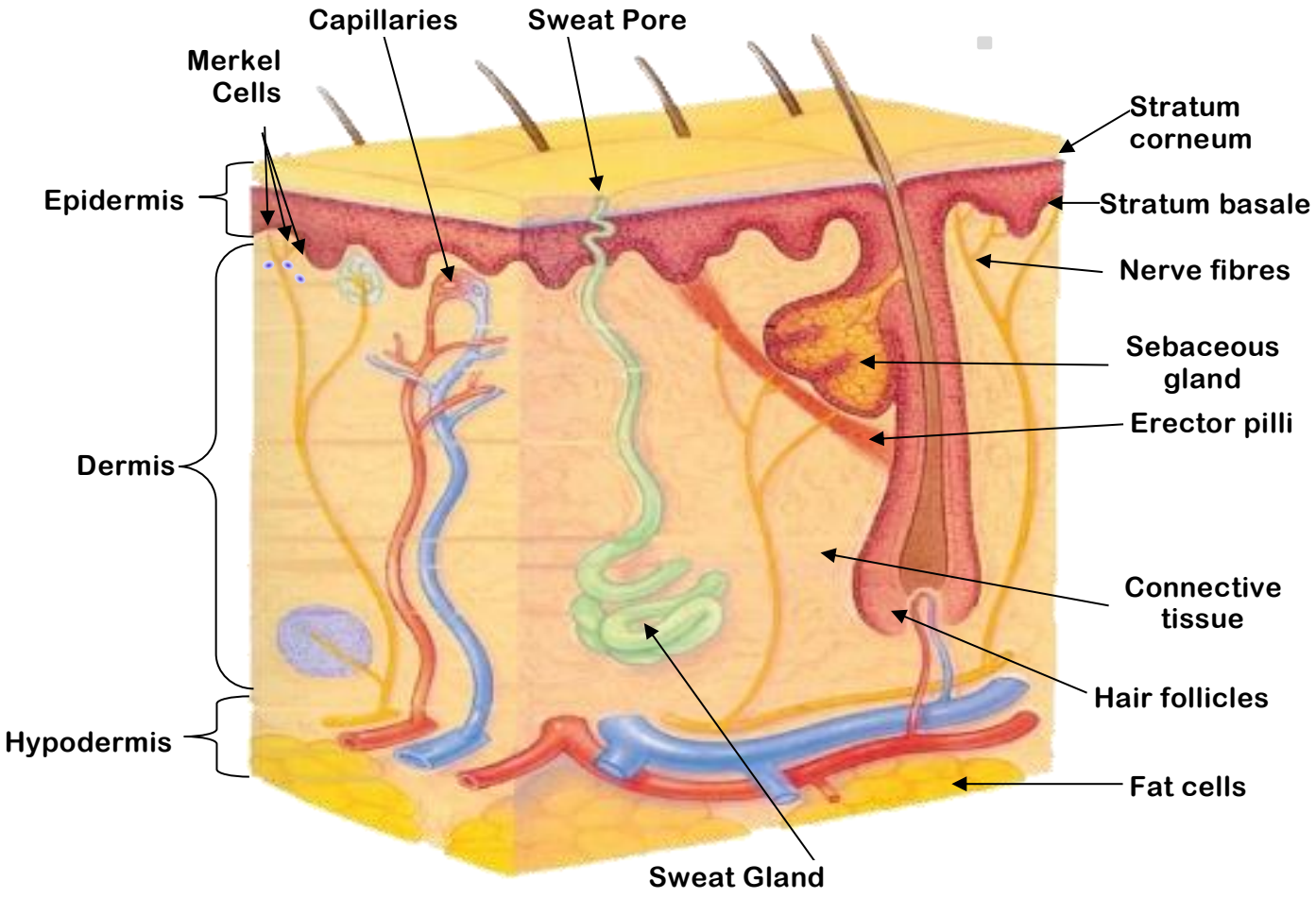

Figure 1 - Major structures of the skin

a. The Epidermis

The epidermis is the outermost layer of the skin. It doesn't contain any blood vessels and is nourished by diffusion from underlying dermis.

Consider a partial thickness skin tear. The skin flap is comprised of the epidermis. When you realign the skin flap, the cells in the skin flap require nutrition to remain alive. How is the skin flap nourished?

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Sometimes, following a partial thickness skin tear, a blood clot forms on top of the dermis. We are taught to clean under the skin flap. How might healing be affected if this wasn't done, and the skin flap was laid on top of the blood clot, instead of directly on the dermis?

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i. Layers of the Epidermis

The epidermis contains five distinct layers, as illustrated in figure 2. It is not necessary to commit the five layers to memory, but a brief understanding will help you understand the pathophysiology of various skin diseases and wounds later on.

The main cells of the epidermis are known as keratinocytes, because they produce keratin. Keratin is a very tough, fibrous, insoluble protein. 'Hard keratin' is found in the hair and nails, while 'soft keratin' in found in the skin.

Only the bottom layer, known as the *stratum basale*, or basal layer of cells divide. As the basal cells divide, they gradually push older cells up towards the surface of the skin. During this journey, they gradually dry out, flatten and die. This process normally takes about two weeks. Eventually the dead keratinocytes are shed from the skin surface (desquamate)

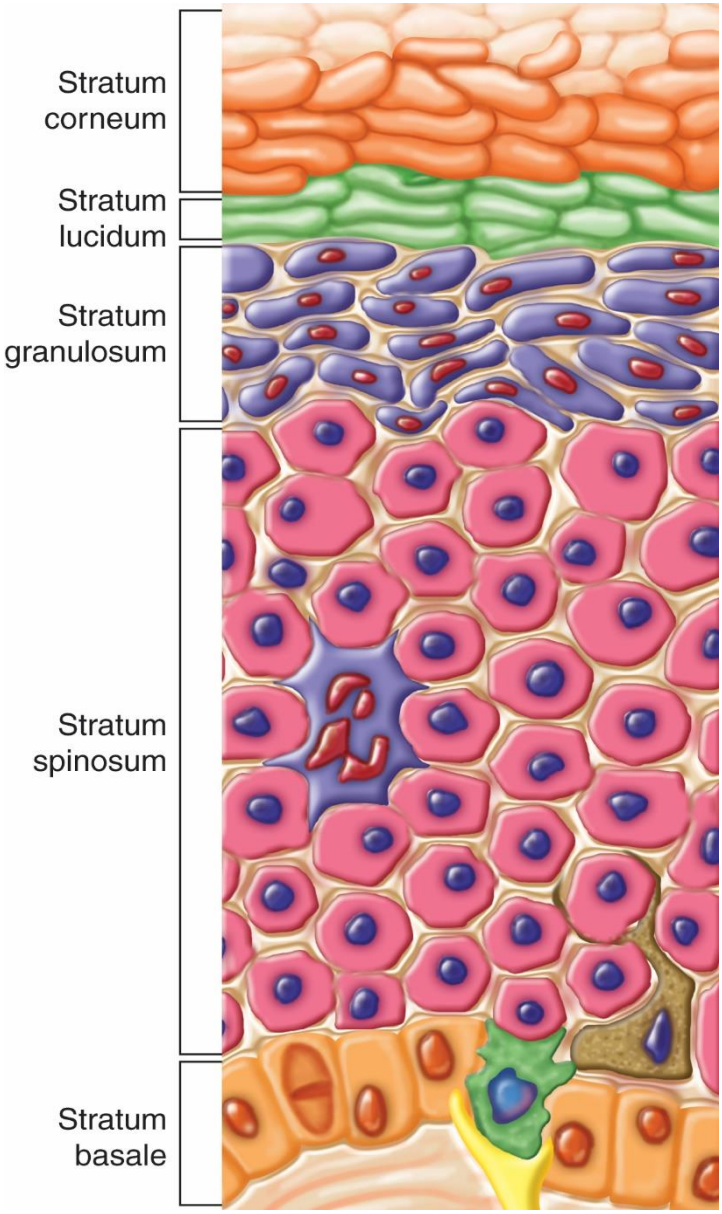

Figure 2 – Layers of the Epidermis

Stratum basale

The bottom layer of the epidermis also known as the *basal cell layer*. Most of the cells in this layer are live ***keratinocytes***, but there are also some ***Melanocytes*** (cells which produce the pigment, *melanin* which is largely responsible for skin colour) and ***Merkel cells,*** which are involved in the sensation of light touch.

This is the only layer in which keratinocytes undergo *mitosis* (divide to make new cells). Cells in the stratum basale lie on the ***epidermal basement membrane***, which separates the dermis from the epidermis.

The stratum basale is only one cell thick in most skin, although it may be two or three cells thick in the ***glabrous*** (hairless) skin. Glabrous skin is found on the palms of hands, soles of feet, lips, labia minora and glans penis.

Stratum spinosum

This layer is also known as the 'spinous' or 'prickle-cell' layer, because the particular way the keratinocytes adhere to each other give 'spiny' or 'prickly' appearance.

The keratinocytes in this layer are still alive, but they do *not* undergo mitosis (divided). Various blistering conditions (e.g. bullous pemphigoid) are characterised by cell adhesion problems within this layer.

Langerhans cells (antigen presenting cells, which function as part of the immune system) are also found in this layer.

Stratum granulosum

The stratum granulosum is composed of one to five layers of diamond-shaped keratinocytes that still have an active nuclei (ie, still 'alive').This layer has a grainy appearance due to the presence of granules, which contain substances that promote strengthening of the keratin fibres through aggregation and cross linking.

An oily substance is produced in the stratum granulosum, which helps to waterproof and moisturise the skin.

Stratum lucidum

The stratum lucidum is not found in all areas of skin. It is only found in thick skin such as the palms of the hands and the soles of the feet. Where present, it is composed of one to five layers of dead, flattened keratinocytes with a translucent appearance under the microscope.

Stratum corneum

The stratum corneum is the outermost layer of skin and is composed of 12 – 16 layers of dead, flat, dehydrated, keratinised cells, now called ***corneocytes***.

Once the corneocytes reach the top of the skin, they detach and flake off in a process called ***desquamation***, or exfoliation.

Desquamation is a complex process, regulated by processes that involve enzymes.

Although corneocytes are dead, the stratum corneum is biochemically active. It not only functions as a water barrier, but also protects against UV radiation, pathogens and mechanical stress.

Figure 3 - Corneocytes

ii. Psoriasis

In areas of psoriasis, inflammatory processes cause the basal cells divide much more rapidly than in normal skin. Basal cells take only 2 – 6 days to move to the surface. The stratum granulosum is thinned or absent, and the keratinocytes in the stratum corneum haven't fully matured into corneocytes. The cells on the top of the epidermis do not shed normally, and so build up into plaques.

Without the barrier function provided by mature corneocytes, areas with psoriasis become dehydrated, and are more susceptible to irritation from external friction and chemicals.

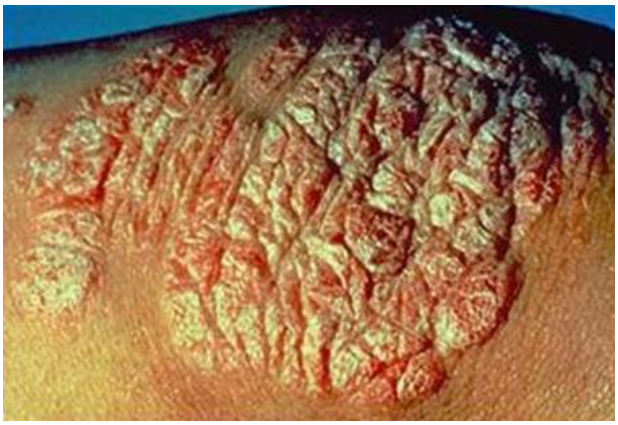

Figure 4 – Psoriasis
from www.healthy-skin-guide.com

b. The Basement Membrane Zone (BMZ)

The junction between the epidermis and the dermis is known as the basement membrane zone (BMZ), dermal-epidermal junction.

Notice the BMZ is not flat, but 'bumpy'.

The epidermis projects 'finger-like' structures, called **rete ridges** down into the dermis, while the dermis projects similarly shaped structures, called **dermal papillae**, up into the epidermis.

These projections function to:

- help anchor the epidermis to the dermis
- increase the surface area through which oxygen and nutrients move from the capillaries at the top of the dermis, into the non-vascular epidermis to keep it viable (alive).

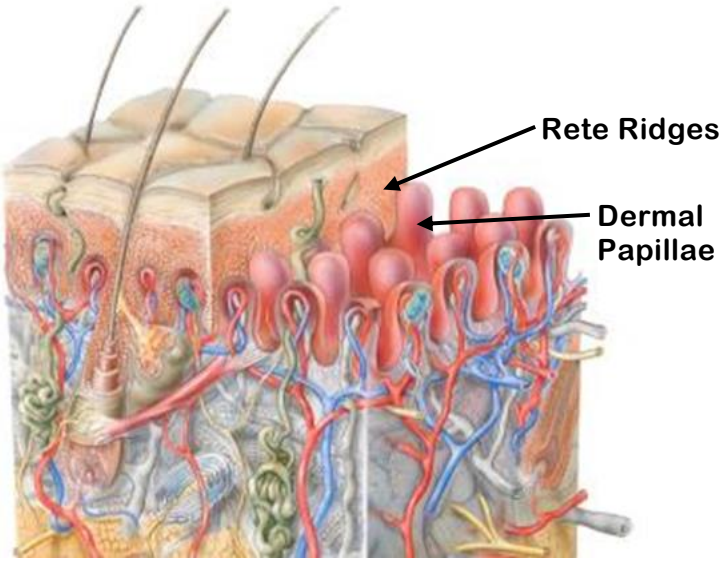

Figure 5 – The Basement Membrane Zone
From www.scf-online.com/english/27_e/frontpage27_e.htm

c. The Dermis

The dermis is a tough layer of skin composed mainly of connective tissue. The following structures are found within the dermis:

- hair follicles
- blood and lymphatic vessels
- nerve fibres (many types)
- sweat glands (produce sweat) and ducts (deliver sweat to the surface of the skin)
- erector pilli muscles (give you 'goose bumps' by pulling hair shafts into a more perpendicular position)
- sebaceous glands (secrete sebum)

i. Fibroblasts

Fibroblasts are the principle cell in all connective tissue, including the dermis. They function to maintain the extracellular matrix by producing collagen and other proteins. Fibroblasts also play a key role in wound healing.

ii. Extracellular matrix (ECM)

The extracellular matrix is the defining feature of connective tissue. Although it is made entirely of non-cellular components, fibroblasts have a large role in maintaining the ECM by producing collagen and various other proteins.

Collagen & Elastin Fibres

Collagen fibres provide dermal tensile strength. Elastin fibres allow flexibility for the skin to stretch and return to its original state as the body moves. Elastin and collagen work together to provide structure to the skin.

Ground Substance

Ground substance is a non-cellular, amorphous, gelatinous material that is transparent, colourless and fills the spaces between fibres and cells. It contains a variety of large molecules (such as hyaluronic acid) that are excellent at absorbing water, thereby providing dermal volume and cushioning.

iii. Layers of the Dermis

There are two layers in the dermis, however, there is no sharp junction between the layers.

Papillary Dermis

The papillary dermis lies immediately below the BMZ, and contains capillary loops and lymph channels. The collagen fibres in the papillary dermis are much smaller compared with those in the reticular dermis.

Reticular Dermis

The reticular dermis forms the base of the dermis. In this layer of the dermis, the collagen fibres are much larger, and there is less ground substance.

d. The Hypodermis (Subcutaneous Tissue)

Underneath the dermis is the hypodermis, or subcutaneous layer. This region is characterised by fat cells (also known as adipocytes or adipose cells).

Have you ever seen a skin tear where fat cells are visible under the skin flap? , What layer of skin were you observing? Was it a partial-thickness or full-thickness skin tear?

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4. Skin Changes with Ageing

Many changes occur to skin as it ages. The onset of most of these can happen earlier as a result of smoking or excessive sun exposure.

a. Total Skin Thickness

- Total skin thickness reduced to approximately 65 – 70% of normal adult

b. Epidermis

- ~ 10 – 50% thinning
- Decreased active melanocytes
- Increased time for epidermal turnover

c. Dermal-Epidermal Junction

- Flat with loss of rete pegs / dermal papillae

d. Dermis

- Decrease in number of fibroblasts
- Decrease synthesis of elastin and collagen fibres leads to loss of dermal thickness (about 1% per year)
- Slight decrease in cushioning proteins in the ground substance

e. Hypodermis

- Decrease in amount of subcutaneous fat

f. Sweating

- 15% less sweat glands.
- 70% reduction in sweating

g. Sebum

- Reduction in size of sebaceous glands, and amount of sebum produced

How might these age-associated skin changes affect a person's risk of sustaining and skin tear?

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How might these age-associated skin changes affect a person's risk of sustaining a pressure injury?

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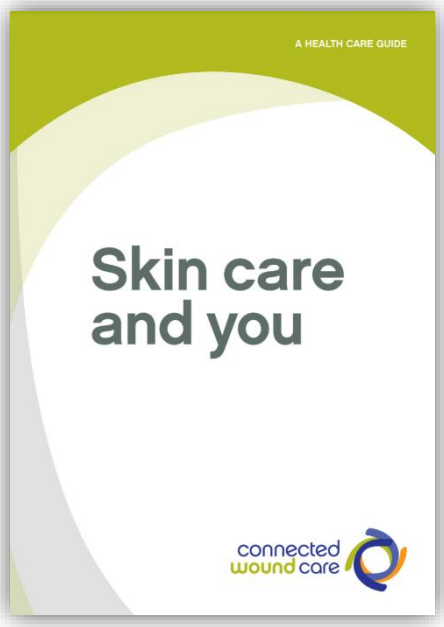
5. Keeping Skin Healthy

Many wounds can be prevented by keeping skin as healthy as possible because healthy skin is better able to resist damage from physical forces and microbes that cause injuries and infections.

Please read the Connected Woundcare pamphlet titled "Skin care and you". It is a good teaching aid to use educating clients. This pamphlet, as well as several other wound-related health care guides can be download free of charge from the RWV Website → Resources →Connected Woundcare Healthcare Guides:

https://www.regionalwoundsvictoria.com/connected-woundcare-health-care-guides



The Acid Mantle

The acid mantle forms on top of the epidermis. It is an oily substance with a slightly acidic pH of 4.5 – 5.5. It forms from sweat and sebum and contains various substances such as amino acids, lactic acid, fatty acids and hormones. Certain microbes reside in the acid mantle, but these are protective and make up our normal *skin flora*. The acid mantle has a role in protecting us from infection with pathogenic (disease-producing) microbes.

Maintaining the Health of the Acid Mantle

Protect the acid mantle. Use a pH balanced soap substitute when showering or bathing. Then, immediately after patting the skin dry, apply a moisturiser. This is especially important for people with dry, frail or aged skin. These people should never use 'normal' soap, so facilities that care for these people should stock a pH balanced soap substitute instead. These simple measures have been shown to significantly reduce the number of pressure injuries and skin tears in aged care facilities.

Avoid Smoking.

Smoking has many effects on skin health. One of the main effects is a reduction of blood supply to the skin. As a result skin cells receive less nourishment which affects their ability to function.

Manage Chronic Illnesses

Management of chronic illnesses such as diabetes, heart disease, lung disease and arthritis is *extremely important*. This may involve referring to other members of the health care team, arranging treatment reviews by medical staff, providing educational resources or simply offering ongoing support and encouragement. In particular, consider the important part that allied health services play in improving chronic disease management.

Remember that some medications affect the condition of the skin. One commonly seen example is Prednisolone. Prolonged use can cause skin to become thin, shiny and fragile. It is only prescribed to treat medical conditions where the benefits outweigh the risk of unwanted side effects such as this.

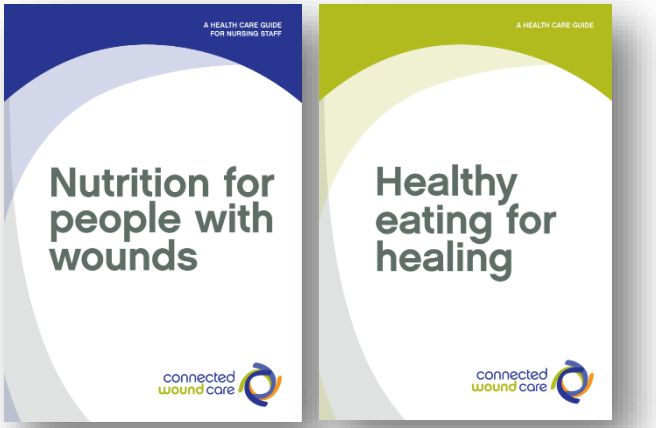
A Nourishing Diet and Adequate Hydration

A screening tool should be used on admission to any health service to identify individuals who are malnourished or *at risk* of malnutrition. If found to be at risk, a referral to a dietician is recommended for a comprehensive assessment.

Several screening tools are available at https://www2.health.vic.gov.au/hospitals-and-health-services/patient-care/older-people/nutrition-swallowing/nutrition-and-hydration/nutrition-identifying

Whenever there is a change in the individual's condition, nutritional screening must be repeated.

The presence of wounds always increases nutritional needs. Please read the Connected Woundcare Healthcare Guide for nursing staff called Nutrition for people with wounds, and the guide you can use to help educate clients called Healthy eating for healing.



Both can be download free of charge from the RWV Website → Resources →Connected Woundcare Healthcare Guides: https://www.regionalwoundsvictoria.com/connected-woundcare-health-care-guides

Protect skin from sun damage

Australia has the highest rate of skin cancer in the world. Exposure to UV radiation damages the DNA in skin cells, producing mutations that can lead to premature ageing and skin cancer. Higher doses lead to cell death (i.e. sunburn).

Please read the position statement titled Sun exposure and vitamin D – risks and benefits (approved by the Australian and New Zealand Bone and Mineral Society, the Australasian College of Dermatologists, Cancer Council Australia, Endocrine Society of Australia and Osteoporosis Australia).

https://wiki.cancer.org.au/policy/Position_statement_-_Risks_and_benefits_of_sun_exposure

Have you seen the cancer council's SunSmart App?

At what UV index should sun protection measures be used?

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How does sun exposure increase skin tear risk? Which skin care practices should you teach clients to reduce their risk of skin tears?

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6. Phases of Wound Healing

No matter what the cause of a wound, the body attempts to heal it using the same three phases of healing. These phases over-lap and vary in how long they take depending on the situation:

The three phases of wound healing are:

Phase	Main Functions	Tissue types seen
Inflammation	**'Cleaning the wound bed'** of • Damaged tissue • Excessive microbes • Dirt • Foreign bodies	Inflamed tissues Non-viable tissue • Slough • Necrosis Foreign substances • Retained suture • Dirt or environmental contamination
Proliferation	**Repairing** by: • Filling the deficit by growing new blood vessels and forming granulation tissue • Covering with new epithelium	Granulation Tissue Epithelialising Tissue
Maturation	**Strengthening**	Epithelial Tissue Scar Tissue

Figure 6 – Phases of wound healing: Main functions and tissue types seen

Wound healing involves a complex matrix of biochemical events. The cells involved at each stage produce a different cocktail of chemicals. As one stage is nearing the end, the cocktail of chemicals being produced changes to trigger the next stage.

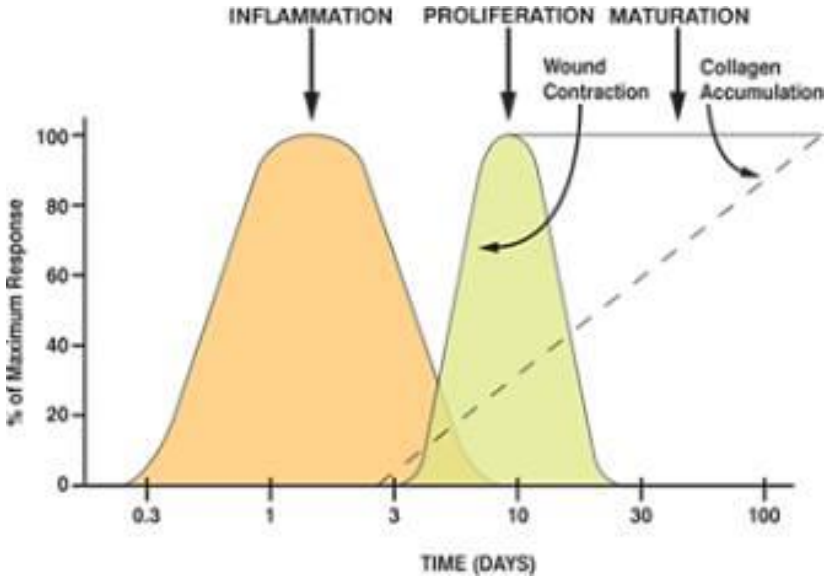

Figure 7- Phases of wound healing

a. Inflammation: The Cleaning Phase

The first part of this phase involves haemostasis. When wounding occurs, collagen in the blood vessels (which is normally covered by endothelium) is exposed. This exposed collagen triggers platelets to become 'activated'. Substances released by activated platelets as well as those released by injured tissue cells result in brief vasoconstriction and formation of a blood clot. Substances produced by these processes attract cells involved in inflammatory processes (neutrophils and macrophages) and the entire wound healing cascade is initiated.

During inflammation, leukocytes (white blood cells) including neutrophils and macrophages are busy cleaning the wound by engulfing and destroying microbes and non-viable (dead) tissue. Inflammatory substances, are released during this phase that result in:

- vasodilation → this causes the area to look red
- capillaries becoming 'leaky' → this causes exudate and swelling
- stimulation of pain nerve receptors → this causes pain
- attraction of leukocytes that engulf microbes and non-viable tissue → this causes heat from increased metabolism

After clean surgery, the inflammation phase can be expected to last only last 2 – 5 days. The inflammatory phase will dominate while:

- new tissue damage continues to occur
- there is dead tissue (e.g. slough, necrotic tissue), bacterial imbalance or foreign substances (e.g. a retained suture, or dirt) in the wound bed

In ***hard-to-heal wounds*** (formerly known as 'chronic' wounds), the inflammation phase may be prolonged for weeks, months or even years!

Why might the inflammatory phase continue to dominate for weeks, months or even years in hard-to-heal wounds?

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Necrosis and slough are often seen in hard-to-heal wounds. While these tissues continue to be present, the inflammation phase will continue to dominate as the body continues to attempt to 'clean' the wound.

Necrosis

Necrotic tissue is dead tissue. It can be jet black if dry, or some shade of grey or brown if moist. It used to be coded as 'black' for documentation purposes, but we now prefer to document tissue types rather than colour codes.

When tissue cells become necrotic, certain enzymes are released from the dead cells. These 'proteolytic' enzymes break down proteins and cause necrotic tissue to liquefy in a process known as **autolysis**. Autolysis is a naturally occurring form of debridement that breaks down necrotic tissue. It will only occur in a moist wound environment.

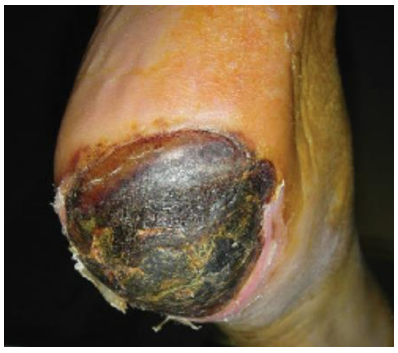

Figure 8 – Dry, stable necrotic eschar. Safer to keep dry and prevent autolytic debridement

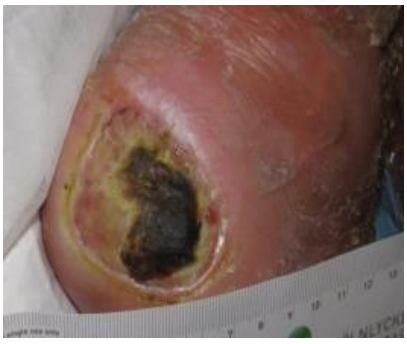

Figure 10: This necrosis is a little dry, but moist underneath. Keeping it moist will encourage autolytic debridement.

Figure 9: Moist necrotic tissue undergoing autolysis

Sometimes necrotic tissue presents as dry, stable eschar. Without moisture, autolysis does not occur. The body will attempt to grow epithelium beneath the dry, stable eschar in the same way it does under a scab. Given enough time, the dead tissue/ body part will 'auto-amputate'. This is a much safer, and less painful way to manage dry, stable necrosis on the foot than to moisten it to encourage autolysis. Do not moisten dry, stable eschar anywhere on the body until blood supply and healing potential is assessed.

If the aim is to promote autolytic debridement, this can be achieved by:

- Keeping the wound bed quite moist, and
- Applying a dressing that can 'take up' the thick exudate and wound debris.

In the 1990s we used to use an ointment called Elase, which contained proteolytic enzymes to help break down necrotic tissue. It is no longer in common use, because studies showed that simply keeping necrotic tissue moist (promoting autolysis) was just as effective.

Necrotic wound exudate contains proteolytic enzymes. Where do these enzymes come from?

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Hydrogels (e.g. Intrasite, Solosite) do *not* contain proteolytic enzymes, so how do they encourage autolytic debridement?

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Why is applying a hydrogel to a wound with high levels of exudate not effective at improving autolytic debridement? What problems might this practice cause? What alternative might you suggest?

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Enzyme Alginogels (Flaminal Hydro, Flaminal Forte) are promoted as debriding agents. The enzymes they contain are antibacterial, not proteolytic. What type of debridement do they promote?

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Slough

Slough is non-viable (dead tissue) that is yellowish in colour.

Slough is usually some shade of creamy yellow. It may be shaded by red or orange if blood is present, or green if certain strains of bacteria (e.g. pseudomonas) are present. We used to code slough as 'yellow' in wound documentation, but now prefer to describe tissue types rather than use a colour code.

Slough is a complex substance, containing dead tissue, fibrin (from exudate), white blood cells and a microbial community.

It's important to understand that we are not looking at biofilm in the photos below. Biofilm is not visible to the naked eye and is likely to be present on all hard-to-heal wounds, whether is slough present or not.

You'll learn a lot about preventing and treating biofilm during the WREN education days.

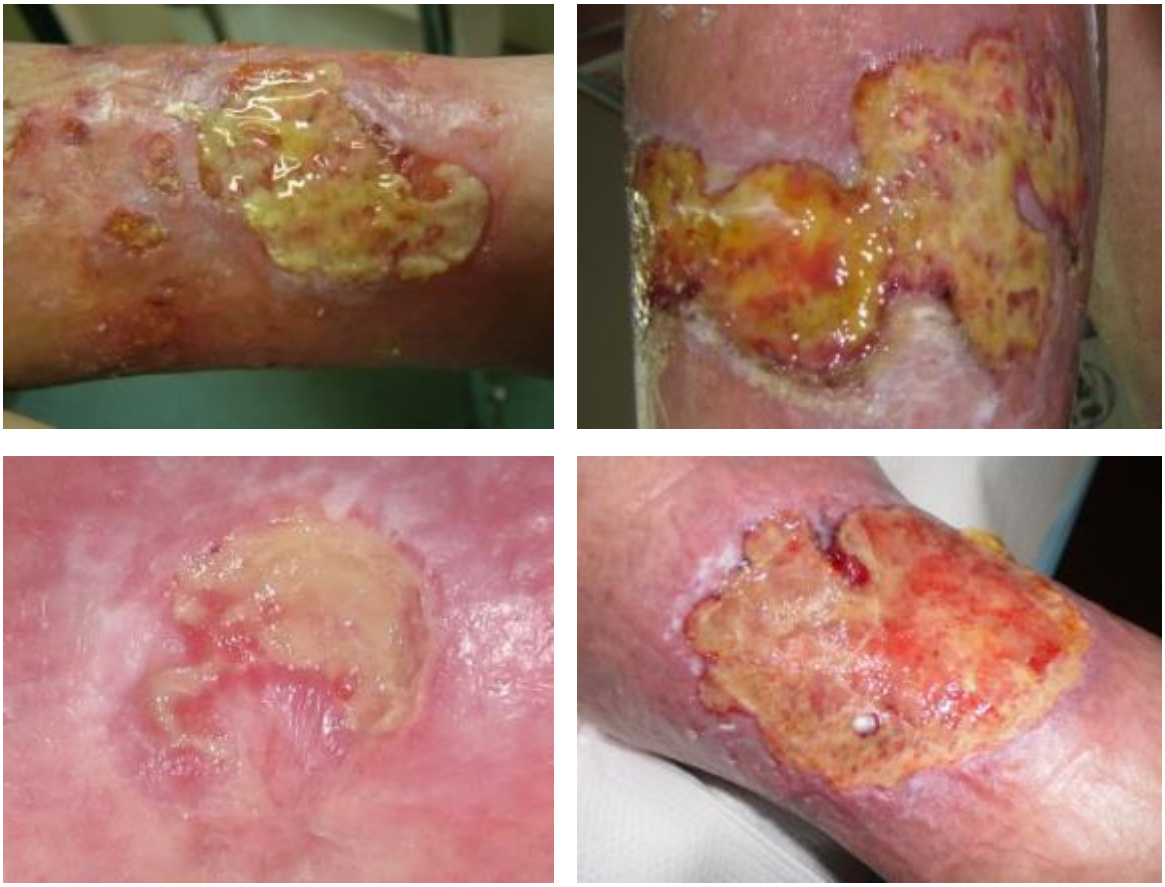

Figure 11: Sloughy Wounds – These wounds are stuck in the inflammatory phase of wound healing

b. Proliferation: The Re-building Phase

Once the inflammatory phase has settled down, the proliferative phase can begin in earnest. During the proliferative phase, tissue defects are filled with granulation tissue, and covered with epithelial tissue.

Granulation

Granulation tissue consists of a collagen 'scaffold' (produced by fibroblasts) upon which capillaries grow. The production of new capillaries is called *angiogenesis*. These new capillary loops give the granulation tissue a 'granular' appearance. Note that granulation tissue does not appear 'striated' like muscle tissue.

We used to code granulating wounds as 'red' for documentation purposes, but now prefer to use the name of the tissue type, rather than a colour code.

Granulation tissue in acute wounds is very delicate and must be treated more gently than tissues in hard-to-heal wounds.

Over time, the granulation tissue begins to contract, making the wound smaller.

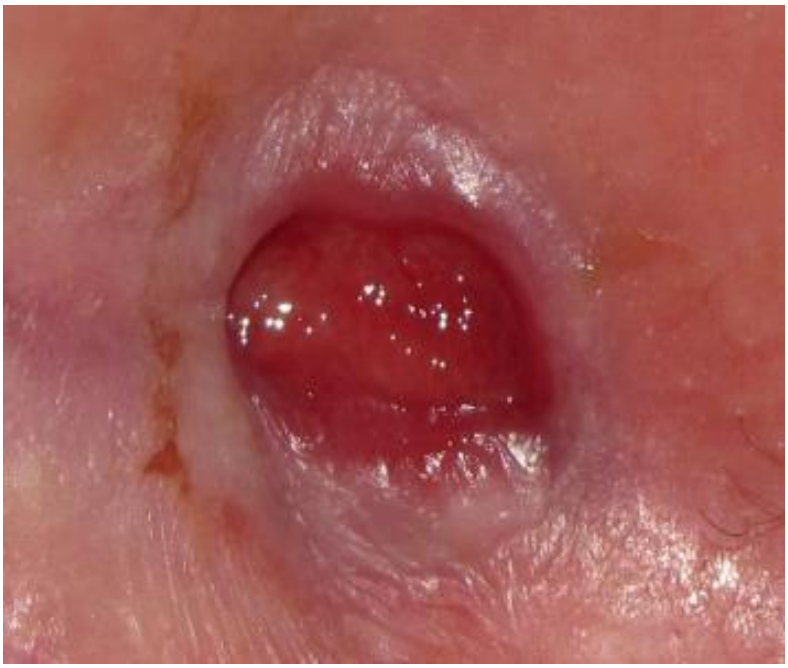

Figure 12: Granulating wound:
Granulation tissue is growing to fill a grade 3 PI

Epithelialisation

During re-epithelialisation, remaining epithelial basal cells detach and migrate across the wound. The new epithelial skin layer then thickens as the basal cells divide and move upwards. It is very delicate, and must be treated gently. We used to code epithelial tissue as 'PINK', but now prefer to describe tissue types rather than using a colour code..

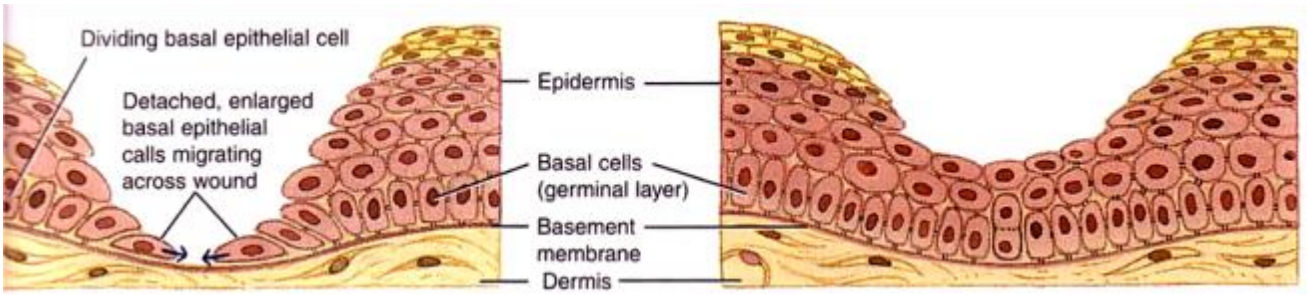

Figure 14: Re-epithelialisation

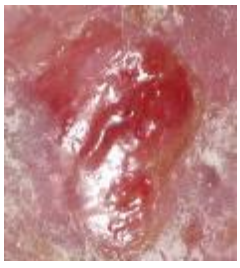

Figure 13: Epithelialising Wound
Pink, shiny new epithelium can be seen.
In this case, the wound is having trouble re-epithelialising because the granulation tissue has grown above the level of the surrounding skin (hypergranulation). Hypergranulation will be discussed during the WREN education days.

c. Maturation: The Strengthening Phase

The maturation phase is known as the strengthening phase.

Although it is accepted practice to record a fully epithelialised wound as 'healed', in reality wound healing is not complete.

Collagen Changes

The collagen initially laid down in granulation tissue is a *type III collagen*, but during maturation, it is replaced with the stronger *type I collagen*.

During the maturation phase, collagen, and the extra-cellular matrix is continually being broken down and reformed in a balanced way. This balance is controlled by several different processes. If any of the controlling processes becomes disturbed, the result can be:

- too little collagen formation → a weak scar → risk of dehiscence
- too much collagen formation → a hypertrophic scar

Contraction

Scar contraction continues to occur during the maturation phase.

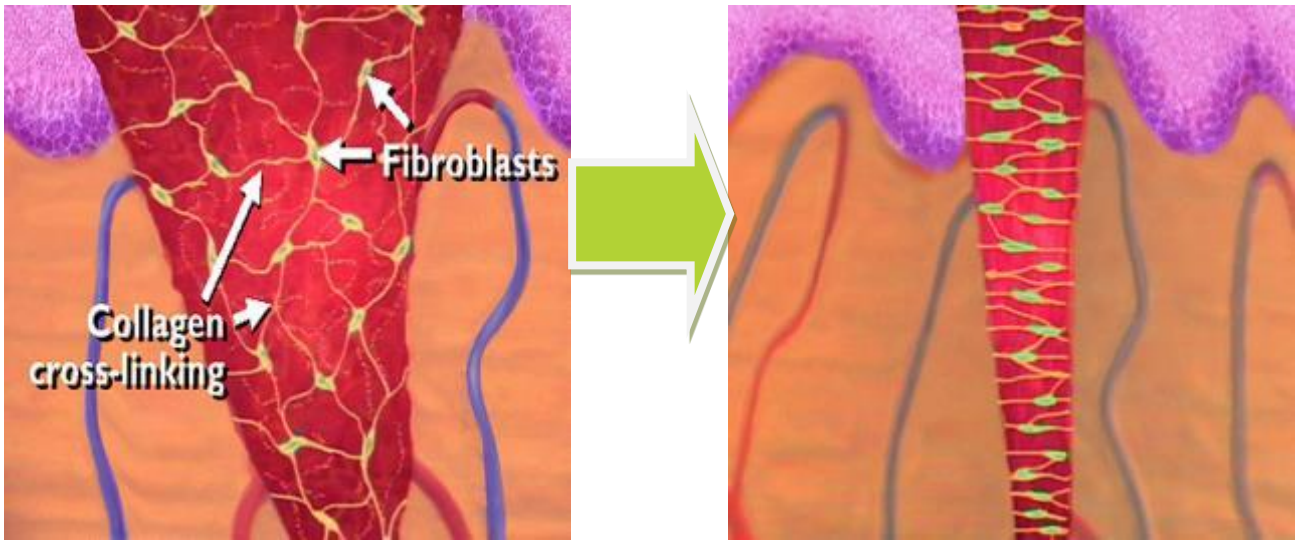

Figure 15: Maturation Phase

Scar tissue

Scar tissue is what is left after most of the live cells in the granulation tissue die away. As this happens, the original scar strengthens, and fades from a purple/red colour to a pale silver/pink colour. This process continues over a year or two.

The scar tissue will never regain the strength or function of the original tissue.

Scenario: You are providing wound management for a person with paraplegia who suffered a stage 3 PI over their ischial tuberosities ('sit bones') following a prolonged car trip.

The wound has now almost healed.

Has the stage 3 PI resulted in any permanent damage?

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Knowing what you do about the tensile strength of scar tissue compared with original tissue, how might their previous PI prevention plan need to be adjusted?

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7. Initial Causes of Wounds

Wounds can initially be caused from:

- Physical forces (e.g. Surgery, trauma, pressure, friction, shear)
- Radiation (e.g. sunburn, radiotherapy)
- Infection (usually not the initial cause of a wound, but it is possible for some highly virulent organisms to *cause* wounds in otherwise healthy skin or skin that has very minor trauma only)
- Medical conditions that result in:
    - Oedema (e.g. Lymphoedema, chronic venous insufficiency, heart failure)
    - Reduced blood flow to wounded area (e.g. peripheral arterial disease)
    - Fibrotic changes to the tissues (e.g. chronic venous insufficiency)
    - Abnormal cell growth (e.g. malignancy)
    - Immune system dysfunction (e.g. Auto-immune diseases such as vasculitis, pyoderma granulosum, bullous pemphigoid)

Some of these medical conditions may go unnoticed until a wound caused by something else (e.g. a skin tear) fails to heal. A common example is when a lower leg wound initially begins as a skin tear, but after weeks, months or years, presents as a venous ulcer due to the pathophysiology of chronic venous insufficiency.

How often have you seen wounds heal, or get close to healing, only to have them break down again? Can you think why this might be happening?

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If new tissue damage occurs within an existing wound, which phase of wound healing will be re-stimulated? How will this affect the healing time?

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It is essential that the
initial cause of the wound
be **identified** and **addressed** as per best practice guidelines.

If all the causes of the wound aren't removed or adequately addressed, there will be **continuous re-wounding,** and continuation of the **inflammation phase** of wound healing.

8. Why Some Wounds Become Hard-to-Heal

Why is it that some wounds heal, and some wounds become hard-to-heal?
Common reasons include:

- The cause (aetiology) of the wound hasn't been identified and adequately addressed, so the wounding process is still continuing.
- Oedema in the wounded area not managed.
- Wound bed problems haven't been identified and addressed. The mnemonic TIME helps us to identify problems with the wound bed.

T	Tissue	Non-viable (dead) tissue in the wound Foreign body in the wound (eg. a retained suture, dirt)
I	Inflammation / Infection	Local, spreading or systemic infection Other causes of inflammation
M	Moisture	Too little → Wound bed dehydrated Too much →Periwound moisture-associated skin damage (MASD): Maceration (white, pale or grey skin that is softened and/or wrinkled) or periwound moisture-associated dermatitis (where erythema is present)
E	Edge	Edge not advancing, presenting as hypergranulation, rolled edges (epibole) and/or undermining.

- Poor communication between health professionals (including poor documentation).
- Failure to prevent or manage wounds according to best available evidence.
- Medical conditions such as those listed in section 7. Not only can these conditions *cause* wounds, they can also *delay* healing in other types of wounds.
- General (systemic) factors that delay wound healing haven't been identified and addressed properly. These include things like inadequate diet, smoking or comorbidities like diabetes.

If a wound isn't healing in a timely fashion, the cause(s) of the wound must be identified and addressed.

Dressings only help manage wound-bed problems, so simply changing the dressing plan rarely results in wound healing.

Where you work, is the aetiology accurately recorded for all wounds?

Are you certain the main aetiology has been identified and addressed correctly for all hard-to-heal wounds that are more than 3 weeks duration?

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How do you find out what current best practice is?

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Regional Wounds Victoria provide the following links to help you keep up-to-date:
Education

- Recorded educational webinars and e-learning
- Upcoming live events and webinars
- Post graduate wound education

Best Practice Documents

Company websites (for information on wound products and dressings)

References

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