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Firstly, why do we care about skin?

Function of the Skin
The skin is the body’s largest organ. In a 70kg person it weighs over 5kg and covers a surface area of almost 2 m². It performs the functions of[6, 7]:

- Barrier to mechanical, chemical and physical insults
- Barrier to biological invasion
- Moisture retention
- Heat retention
- Sensory perception (pain, heat, pressure, vibration, itch)
- Communication
- Synthesis of vitamin D
- Immune surveillance

The barrier function of the skin relies on a well hydrated epidermis. It is also this level of moisture in the stratum corneum that helps to regulate the pliability and elasticity of the skin[8].

When we have changes in, or injury to, the skin we alter the functions listed above. From a medical perspective it means we are at increased risk of infection, possible reduction in Vitamin D, reduced ability to regulate temperature and potentially a reduced ability to heal. Functionally, how we interact with our environment can change as sensory perception changes, possibly losing some protective mechanisms (ie – “ow, I feel pain, I had better move…”). But there are also a number of psychosocial impacts:

- ADLs – what was once a regular task (cooking) can now result in bruises and skin tears from seemingly inconsequential knocks to cupboards and countertops, as well as burns from only the briefest of contact with hot oven racks.
- Comfort – These injuries, which are so easy to get, can be quite painful. Also, aging skin is dry skin, this results in itchiness which can impact on all aspects of daily living, including sleep[9],
- Self-image – Skin changes, wounds, bruising and scarring which are visible to others also change how we see ourselves; changing self-esteem and self-confidence[10].
- Mental Health – and when you live with pain, pruritus and changes to your self-image, it becomes understandable that there may also be some associated mental health concerns[11].

So, why does skin change over time?

Physiological skin changes over time
As we get older we undergo changes that make our skin more susceptible to damage. A number of specific skin changes have been related back to changes in hormone levels as we age, such as the effects of estrogen on inflammation and dryness[4, 12]. The accumulation of radical oxygen species (ROS) in the mitochondria of cells has also been shown to induce senescence and increase terminal differentiation; this has been implicated in the thinning of aging skin[13, 14]. But as well as the intrinsic factors of aging the skin also has to face extrinsic
influences. The two most significant of these are sun exposure and smoking, which have a significant effect on the skin’s elastin and collagen\textsuperscript{(15, 16)}. A complex diagram of some of the biochemical changes can be seen in Appendix A. Nutrition and hydration also play a role in the resilience of the skin. Some of the physiological changes include:

- the dermal papillae flatten out, reducing the strength of the interface between the dermis and the epidermis leading to easier separation of these two layers\textsuperscript{(17)}
- a reduction in sebum production and reduced moisture to the skin from both reduced movement of water from dermis to epidermis as well as a reduction in fluid intake means dried skin and less resilience\textsuperscript{(17-19)}
- loss of hypodermis (~50% by age 80) reduces cushioning and temperature regulation\textsuperscript{(20)}
- decrease in vascularity and structures supporting the vasculature lead to bruising and bleeding\textsuperscript{(17)}
- loss of collagen and elastin fibres in the dermis means lower tensile strength and less structural support
- immune changes\textsuperscript{(17)}

Appendix B has a larger table of the clinical implications of aging skin.

Not only do these changes have an obvious cosmetic and self-esteem impact but they can also impact on quality of life with skin conditions such as xerosis cutis (dry skin), pruritus (itchy skin), and eczema being widespread in the older population\textsuperscript{(17)}. And, not only that, but the simultaneous changes in immune function as well as skin structure and function produces higher levels of autoimmune skin disorders. The exposure to new pharmaceuticals increases the risk of autoimmune drug reactions that manifest in the skin. These may include pemphigoid or pemphigus disorders and small vessel vasculitis. Then there’s the possible re-activation of dormant viruses such as Shingles\textsuperscript{(17)}.

But it’s not just the changes in the skin that make us more likely to get a skin injury. Other changes that may be experienced include (certainly not comprehensive):

![Table 1. Changes in the older population specifically related to healing](https://www.remeka.hu/index.php/magazinxmszakmai-frissxm/single-column-blog-190/kozmetikus-szemmel/805-az-antiaging)

<table>
<thead>
<tr>
<th>Haemostasis</th>
<th>Inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased platelet aggregation</td>
<td>Decreased vascular permeability</td>
</tr>
<tr>
<td>Increased release of alpha-ganules</td>
<td>Increased secretion of inflammatory mediators Delayed infiltration of macrophages and lymphocytes Impaired macrophage function</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Proliferation</th>
<th>Remodelling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delayed re-epithelialisation</td>
<td>Reduced collagen turnover and remodeling</td>
</tr>
<tr>
<td>Delayed angiogenesis</td>
<td>Delayed tensile strength</td>
</tr>
<tr>
<td>Delayed collagen deposition</td>
<td>Decreased tensile strength</td>
</tr>
</tbody>
</table>

Figure 1 - image from http://www.remeka.hu/index.php/magazinxmszakmai-frissxm/single-column-blog-190/kozmetikus-szemmel/805-az-antiaging
• Centre of balance and gate changes
• Immobility
• Peripheral neuropathy
• Altered vision
• Incontinence

So let’s say we do have a skin injury, and now we have to heal it. It is argued that the effects of aging do not directly, negatively impact on wound healing but that it is all the confounding factors that go along with aging\(^{(21)}\) such as polypharmacy, comorbidities, and alterations in nutrition and hydration. The reasoning behind this thought is that the body has so many redundant systems for healing, that no matter what becomes less efficient, during the healthy aging process there are enough systems remaining to make up the difference ... if just a little bit slower\(^{(22)}\). So when we do our assessment of systemic features that may impair healing, while we may list aging as ‘slowing healing’ it will be other assessment items like venous insufficiency or low serum albumin that will need to be targeted in the management plan.

**Factors that affect the rate of deterioration**

**Hormones**

Estrogen is probably the most researched female hormone with regards to skin and wound management. Estrogen receptors are in many organs and the skin is the largest non-reproductive target on which estrogen acts. For several years before the menopause, estradiol production declines and after menopause estrone becomes the predominant estrogen. Estrone is a less potent form of estrogen than estradiol. Changes in estrogen levels can also be surgically induced\(^{(12)}\).

The effects of reduced estrogen can include wrinkling, dryness, atrophy, laxity, poor wound healing, hot flashes, and vulvar atrophy. The use of estrogen replacement, either orally or topically has been widely studied. While results vary there is some evidence to support that it encourages thickening of the epidermis, improved skin hydration, increased skin surface lipids, and (in a very small study) improved wound healing. Interestingly, scar appearance is improved in the older patient. The scar formation is paler, flatter and with less (but more structured) collagen deposition compared to younger patients\(^{(12)}\).

**Smoking**

Studies have shown that smoking results in more premature facial wrinkling than sun exposure. Lines around the eyes called “crow’s feet” and multiple vertical lines around the mouth called “smoker’s lines” develop at an earlier age. The damage is irreversible and the effects continue into old age. By the age of 70 years, smoking 30 cigarettes a day could lead to the equivalent of an extra 14 years of skin ageing\(^{(1)}\).
Cigarette smoking negatively impacts on wound healing in several ways. Mostly associated with the lack of oxygen delivery to the wound site resulting in reduced collagen and reduced proliferation and migration of key cells involved in wound healing. It is thought to also change how the skin looks (ie wrinkles and grey colour), related again to collagen changes, reduced oxygenation and also to the changes in muscle use: puckering of the mouth and hollow cheeks from repeated drawing on cigarettes, squinting from smoke.

Research has shown that smokers are more likely to experience skin flap failure. Microcirculation has been assessed using laser Doppler flowmetry: bloodflow decreased by 23.8% and 29% immediately after smoking one and two cigarettes, respectively, and subcutaneous wound tissue oxygen tension was noted to be significantly reduced and remained low up to 80 minutes after smoking. This means that in a typical pack-per-day smoker, tissue hypoxia can be expected for most of the day.

Sun exposure
Premature skin aging and development of malignant cutaneous tumors are interrelated issues that are increasingly important problems. Skin aging is important aesthetically, whereas skin cancer is a direct threat to health. Photoaging and photocarcinogenic mechanisms are predominantly the effect of solar ultraviolet (UV) radiation that induces reactive oxygen species (ROS) and alters DNA/cellular homeostasis, which together alter signal transduction pathways and inflammatory cascade and induce immunosuppression and extracellular matrix (ECM) remodeling.

Data from the Cancer Council of the ACT which shows that in 2013 there were over 500 non-melanoma skin cancer (NMSC) related deaths reported. The estimated total treatment cost for NMSC during 2010 was $500 million making skin cancer, in financial terms, the most costly cancer burden to the health system.

Photoprotection is the main preventative measure with frequent and appropriate use of sunscreen important. Most sunscreen contain both physical filters (inorganic micropigments) and organic/chemical filters (these can absorb short
wavelength UV photons and to transform them into heat\(^{(15)}\). For prevention of skin cancer (and by association skin aging) the Australian Cancer Council recommends:

- **Slip** on some sun-protective clothing that covers as much skin as possible.
- **Slop** on broad spectrum, water resistant SPF30+ (or higher) sunscreen. Put it on 20 minutes before you go outdoors and every two hours afterwards. Sunscreen should never be used to extend the time you spend in the sun.
- **Slap** on a hat – broad brim or legionnaire style to protect your face, head, neck and ears.
- **Seek** shade.
- **Slide** on some sunglasses – make sure they meet Australian Standards.

**Co-morbidities and their Medications**

With increase in age may also come an increase in co-morbidities and the medications to manage them. While there are a few medications that directly, negatively, impact the skin, such as corticosteroids, most are beneficial as they manage the underlying co-morbidity which is affecting the skin.

Two significant examples of medications that have a big impact on the skin are corticosteroids and vasoconstrictors. Corticosteroids are known to decreased tissue formation due to: (i) reduced cytokine stimulation from decreased presence of macrophages and platelets; (ii) reduced mitotic activity leading to decreased re-epithelialization, fibroplasia, and angiogenesis; (iii) reduced protein synthesis causing decreased synthesis of collagen, proteoglycan, and glycosamine. They also decrease tissue remodeling due to reduced number and synthetic activity of fibroblasts\(^{(23)}\). Skin is constantly remodeling and corticosteroids interfere with this process. However, they are also used in managing skin conditions such as eczema and a number of auto-immune diseases. Therefore their use needs to be carefully monitored so that the minimal effective dose is used. Vasoconstrictors are used when there is a need to increase blood pressure, however at the same time they are diverting blood from the small blood vessels. This can result in damage to structures fed by these smaller vessels, including the skin. Adrenaline (or epinephrine) is used in advanced life support. Again, the balance between the need to preserve life and the need to preserve specific organs must be very carefully balanced.

Pretty much every co-morbidity experienced by the older person will have an impact on skin in some way, either directly or indirectly. This can be by restricting blood and oxygen, reducing nutrient availability, causing oedema, causing a build up of toxins in the skin or conditions where the body’s own natural defenses attack (auto-immune conditions). The use of medications must be balanced with the need to manage the co-morbidity.

**Factors that cause the skin to breakdown**

**Friction**

The physiological changes in aging skin that we have discussed so far, reduce the skin’s ability to tolerate friction. Sources of friction can be:

<table>
<thead>
<tr>
<th>Source</th>
<th>What happens</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>The way in which a person moves</td>
<td>The person may be unable to lift limbs fully off</td>
<td>Protect limbs, hands and feet with appropriate sleeves/gloves/shoes</td>
</tr>
<tr>
<td></td>
<td>surfaces when repositioning or walking</td>
<td></td>
</tr>
</tbody>
</table>

*Produced for Ausmed by Kim Kaim, 2017*
The way in which a person is assisted to move

<table>
<thead>
<tr>
<th>The way in which a person is assisted to move</th>
<th>Sliding or dragging when repositioning in bed or chair</th>
<th>Correct use slide sheets whenever assisting a patient to reposition (24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unintentional movement</td>
<td>Sliding down a bed or chair</td>
<td>Correct positioning in bed/chair and use bed/chair settings to minimize movement (ie bend at the knee) (24)</td>
</tr>
<tr>
<td>Adhesives</td>
<td>Frequent removal of adhesives that hold dressings or devices in place.</td>
<td>Avoid adhesives if possible. Otherwise, use barriers on the skin under the adhesive and use correct removal techniques (25).</td>
</tr>
</tbody>
</table>

**Moisture**

Moisture can come from a number of sources; sweat, bathing, urine/faeces, wound fluid, lymphorrhoea, spillage of water/food, etc... Excess moisture on the skin surface can collect in skin folds, threatening skin integrity (excoriation) and encouraging opportunistic infections. Bacteria, fungi, and viruses all thrive in a warm, moist environment. Excess fluid on the skin may also lead to itching and burning, which results in pruritis; the scratching of which leads to further loss of skin integrity due to superficial trauma. Any breaks in the skin barrier can allow microorganisms to enter, potentially leading to cellulitis (26).

The image here is of incontinence associated dermatitis. Components contributing to IAD are (27):

| Impairments in tissue tolerance | This includes but is not limited to aging skin, reduced skin perfusion (as might be related to oedema secondary to congestive cardiac failure), and presence of previous scar tissue. |
| Problems of the perineal environment | Incontinence, excessive perspiration |
| Altered toileting ability | Cognitive or physical ability, wheelchair bound, restraints |

Prolonged exposure of the skin to moisture leads to maceration, which weakens the epidermis. Friction (such as that from pulling against bedding, clothing or incontinence aids during repositioning) will have a greater impact on this weakened skin allowing it to shear away (28).

The protective barrier of the epidermis relies on its acidic nature. Ammonia (formed when urease-producing bacteria in urine or faeces split urea) is alkaline. When urine or faeces is in prolonged contact with the skin, the pH of the skin increases, reducing its ability to combat infection (28). In addition, faeces contain coliform bacteria and digestive enzymes that encourage greater skin irritation (29).

In the hospital/aged care environment, gentle cleansing after every toileting or pad change is recommended. Barrier creams can also be used to protect the skin (30, 31). Selection of the most appropriate barrier cream should be done in conjunction with the patient and also the incontinence aid manufacturer as some may interfere with the function of the incontinence aid. Always apply thinly.
Ischaemia

There are a number of reasons why the skin might not be adequately perfused (in your own time, see if you can think of 2 more!), but the one we will discuss today is Pressure.

Tissue loading is the defining characteristic of pressure injury formation. Pressure is increased in tissues that are positioned between a bony prominence and a support surface. Research has demonstrated that both magnitude of pressure and duration impact on pressure injury formation\(^\text{32}\). Unrelieved pressure disrupts blood supply to the capillary network, impeding blood flow and depriving tissues of oxygen and nutrients. The most common sites for pressure injuries are the sacrum, heels, ischial tuberosities, greater trochanters, and lateral malleoli\(^\text{33}\). Pressure is relieved and circulation restored by frequently turning and shifting weight distribution as well as with the use of dynamic surfaces that actively redistribute pressure on the body surfaces\(^\text{32}\).

How do I assess an older adults skin integrity?

Now that you have an idea of age-related changes to skin and the diversity of intrinsic and extrinsic forces which impact on the skin, you can start to develop a framework to determine how At Risk your patient is for impaired skin integrity.

There are a range of tools commonly used to measure a person’s level of risk of obtaining a pressure injury (ie Waterlow). Most facilities will have something. Skin tear risk assessments are less common but still exist. Each of these tools look for a specific set of items which are known to be associated with increasing risk. For example, with skin tears risks are related to\(^\text{34}\):

- Age and gender
- History of previous skin tears
- Dry, fragile skin
- Medications that thin the skin such as steroids
- Echymoses (bruising / discolouration of the skin caused by leakage of blood into the subcutaneous tissue as a result of trauma to the underlying blood vessels)
- Impaired mobility or vision
- Poor nutrition and hydration
- Cognitive or sensory impairment
- Comorbidities that compromise vascularity and skin status, including chronic heart disease, renal failure, cerebral vascular accident
- Dependence on others for showering, dressing or transferring

If you do not have a set tool to use, the mnemonic HEIDI can help you to remember some of the things you need to consider. HEIDI stands for History, Examination, Investigation, Diagnosis and Implementation (HEIDI)\(^\text{35}\). The rest of this section will go into more detail about each of these.

History

Start by considering what systemic and environmental factors might impact on wound healing or impact on your plan.

- History of skin tears
- Decision making skills impaired
- Vision impaired
- Extensive assistance for ADLs
• Bed or chair-bound
• Impaired balance/Unsteady gait
• Physically abusive or Agitated
• Resists ADL care
• Hearing impaired
• Decreased tactile stimulation
• Comorbidities that compromise vascularity and skin status, including chronic heart disease, renal failure, cerebral vascular accident
• Medications that thin the skin such as steroids

We need to plan to mitigate the impact of as many factors as we can, also, we need to take into account the impact of these factors on any plans we make. For example, it’s all well-and-good to put bed rails up and ask the patient to buzz for assistance to the toilet, but the confused patient will not be able to recall these instructions and will only try to climb over the rails.

Examination
This is where you look at your patient. What does their skin look like? Do you see outwards signs of those physiological changes?
• Current skin tears
• Bruises
• Contractures of arms, legs, shoulders, hands
• Hemiplegia, hemiparesis
• Pitting oedema of legs
• Open lesions on extremities
• Senile purpura on extremities
• Dry, scaly skin

Again, you can look at each of these factors and put in place a plan to minimize them....

Dry skin? - Moisturise.
Bruises? - Why? - From falls. - Why are we falling? We're in a hurry to get to the toilet. - Can we put in place a toileting plan and/or investigate the reason for the urgency?

Investigation
The toilet example above leads us nicely into this section. Do we have enough information to understand the underlying problem and therefore create a comprehensive management plan, or do we need more? In the example above, we may need to do a urine test to see if there is an infection, which may be causing the urgency. Or, there could be a medical condition that we need a Doctor to investigate.

Other examples of investigations we can do at the beside are things like checking for the presence of pulses and sensation. If we do not have pulses (ie poor circulation to the feet) this area is at greater risk of ischaemia. If we do not have sensation (ie can't feel our feet) this area is at greater risk of trauma. Get into the practice (on healthy patients) of finding brachial, dorsalis pedis, posterior tibial and popliteal pulses. We don’t tend to use these often so don’t get much practice. Also, knowing how to determine sensation perception is very useful (ie. Ipswich Touch Test, Monofilament testing, Vibration testing).
Other investigations undertaken by the doctors include pathology and medical imaging. You can always recommend investigations be done when discussing your patient with the doctor.

The doctor may or may not wish to include this in their plan, but at least you have brought attention to a potential problem. Remember to use your SBAR communication skills when making these recommendations.

**Diagnosis**
We understand a diagnosis to be the identification of a disease or medical condition by examination of the signs, symptoms and any tests performed\(^{[36]}\). Where there is an actual wound it is important to know the diagnosis behind the wound. For example, Skin Tear and Pressure Injury are two different diagnoses. Also, these wounds are treated differently based on their diagnosis.

For general skin assessment though, while I am still assessing signs and symptoms and considering the results of any tests performed, I use the D more for the Determination of risks. I make a list of all the risks, based on what has been learned so far, to be able to create the most appropriate care plan. Failure to identify risks may result in damage to skin, however, interventions based on accurate assessment delivers benefits to patients, healthcare systems and society\(^{[37]}\).

**Implementation**
Once you have collected your history, done your examination, completed any other investigations and determined your risks, you will be ready to put together your comprehensive care plan. For example:

| **History** | **Smoking**<br>• Heart failure with fluid overload<br>• Loss of 10 kg in the last 2 months | **Education to quit smoking**<br>• The treating team will be managing the fluid overload, but the resulting oedema, in the meantime, will increase the risk of ischaemia to affected skin – off-load, reduce oedema if possible.<br>• Refer to dietician for nutritional support |
| **Examination** | **Oedema**<br>• Lymphorrhoea<br>• Altered Gait | **Manage oedema**<br>• Lymphorrhoea sitting on the skin could cause that skin to breakdown – keep dry, use barrier creams<br>• Encourage use of appropriate walking aid, consider physiotherapy involvement |
| **Investigation** | **Poor pedal pulses** | **Risk of ischaemia – off-load** |

**Collaboration**
Communication and collaboration is essential\(^{[38]}\). Who are the collaborators that make up these teams? What about the patient and their family/carers or other support in the home? Are they in a nursing home? For the complex, chronic wound, evidence tells us that we need a coordinated, multidisciplinary care team, including participation from at-home caregivers and the patient, for optimum results. Specialist-lead advanced care (ie. surgeons) is needed when there is evidence of ischemia, suspected malignancy, and peripheral arterial disease\(^{[39]}\). This is best supplemented with a member of the allied health care team (for example, occupational therapist, physical therapist, podiatrist, dietitian, social worker and so on)\(^{[40]}\).

How do you know when to refer? Whenever you are in doubt, if you suspected malignancy, when there is evidence of ischemia or wounds that do not demonstrate an adequate response...
to treatment (39). By getting the right people involved we can allow for earlier diagnosis, better management, and may reduce the cost of treating wounds (39). With the level of complexity in the patients we see, accurate assessment and development of successful care plans can be quite challenging, especially if we do have a wound (39). But it can be equally rewarding.

Strategies to prevent skin tears

With all of our systemic risk factors as we age (such as co-morbidities, polypharmacy, poor nutrition and hydration, slower immune response, etc) our chances of healing in a timely fashion and with no complications are somewhat reduced. So in the case of skin tears and the older population, prevention really is better than cure. Some things that help prevent skin tears are:

- Prevent dry skin, moisturize twice a day (20). Choice of emollient is subject to personal preference and needs of the skin. Occlusives put a film on the skin, humectants go into the stratum corneum and attract water. Some products include a barrier film, anti-pruritic or anaesthetizing agent. Emollients have been shown to be anti-inflammatory when used consistently and appropriately (8).
- Clean and protect skin, use pH neutral products to cleanse excreta and body fluids and use barrier wipes or creams to protect areas of high moisture. Non-soap cleansers prevent acid-mantel stripping and drying of the skin (20).
- No prolonged soaks in the bath that cause maceration (20)
- Pat the skin dry, don’t rub it (20)
- Good lighting (41)
- Appropriate furniture (nothing too pointy!)
- Full length sleeves and pants, skin protection devices for vulnerable areas (41)
- Maintain adequate fluid intake and nutrition (41)
- Good shoes (all over foot protection)
- Good falls prevention practices (41)
- Use slide sheets at all times when repositioning patients
- Avoid the use of adhesives, consider alternative options (41)
- The use of Pool Noodles to cover potential problem areas on wheelchairs, wheelie walkers, corners of coffee tables, etc... (you cut the pool noodle down the side lengthwise and wrap it around the potential problem area)

Skin tears, especially those on the lower leg, carry with them the risk of evolving into chronic ulcers. Hopefully, by following good wound bed preparation practices, moist wound healing principles and addressing the underlying risk factors which impair healing we can assist these wounds to follow a normal wound healing pathway. However, for some patients we will not be able to help them to heal. So it’s better to prevent the injuries we can.

Practical guide to choosing correct products for prevention and treatment of skin tears

There are hundreds of dressings on the market, each company informing you that their product is the best. Unfortunately, there is no single dressing for all wounds, and the selection
of dressing comes down to what your wound is like, what your patient prefers/can tolerate and what you have access to. Considerations for dressings (for skin tears) are:

- Easy to apply
- Provides a protective anti-shear barrier
- Optimises the physiological healing environment (eg moisture and bacterial balance, temperature and pH maintenance)
- Is flexible and molds to contours
- Secure, but does not cause trauma on removal
- Allows extended wear time
- Optimises quality of life and cosmesis
- Is cost-effective

Different dressing types have different strengths and weaknesses. The table on the next page is modified from the Royal Children’s Hospital Melbourne. It does not tell you what to use, but it does give you the strengths and weaknesses of the dressings you may have to choose from.

Also, there is usually a lot to consider when you are changing a dressing. You need to make sure your work area is clean so that you're not introducing new bugs to the wound. What supplies will you need? How will you keep your work area as clean as possible? PLUS, you need to know what to do with the dressing. I like to use the following framework for recording what needs to be done for a specific dressing change. It is a great hand-over tool.

<table>
<thead>
<tr>
<th>Cleaning</th>
<th>Does it need cleaning? Should it get wet? How should it be cleaned and with what solutions?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emollient</td>
<td>It has been shown that moisturizing improves the barrier function of the skin and skin resilience. The use of barrier creams or sprays can also protect periwound skin from damage caused by exudate, other body fluids or adhesives.</td>
</tr>
<tr>
<td>Primary dressing</td>
<td>This is the dressing that manages the moisture and microbial balance at the wound surface.</td>
</tr>
<tr>
<td>Secondary dressing</td>
<td>This may be needed to hold the dressing in place and/or for excess exudate management. Consider the effect of adhesives on the skin and any skin allergies.</td>
</tr>
<tr>
<td>Retention/compression</td>
<td>Retention dressings tend to be used in preference to adhesives on vulnerable skin. Compression is used primarily for oedema and scar management.</td>
</tr>
<tr>
<td>Frequency</td>
<td>Based on the recommendations of the manufacturer, however clinical judgment must be applied to each individual’s circumstance.</td>
</tr>
<tr>
<td>Types</td>
<td>Examples</td>
</tr>
<tr>
<td>--------------------------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Synthetic fibre gauze</td>
<td>Topper</td>
</tr>
<tr>
<td>Island dressings – slightly absorbant non-</td>
<td>Primapore Mepore</td>
</tr>
<tr>
<td>adherent pad with an adhesive cover</td>
<td>Opsite post op Compose</td>
</tr>
<tr>
<td>Semi- permeable – thin, adhesive, transparent polyurethane film</td>
<td>OpSite, Tegaderm</td>
</tr>
<tr>
<td>Non adherent Moist (Tulle Gras Dressing) –</td>
<td>Jelonet, Interpose</td>
</tr>
<tr>
<td>Gauze impregnated with paraffin or similar.</td>
<td>Unitulle Cuticern Mepitel (silicone)</td>
</tr>
<tr>
<td>Non adherent Moist for contaminated wounds</td>
<td>Bactigras Xeroform</td>
</tr>
<tr>
<td>Types</td>
<td>Examples</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Non adherent Dry Thin perforated plastic film coating attached to absorbent pad</td>
<td>Melolin Melolite Tricose Exu-dry Mesorb</td>
</tr>
<tr>
<td>Foam - Polyurethane foam dressing, some with adhesive layer incorporated</td>
<td>PolyMem Allevyn Hydrosorb Mepilex Mepilex border Allevyn cavity</td>
</tr>
<tr>
<td>Hydrocolloid - Polyurethane film coated with adhesive mass</td>
<td>Duoderm Comfeel Coloplast sheet</td>
</tr>
<tr>
<td>Types</td>
<td>Examples</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Hydrogel – Composed mainly of water in a complex network or fibres that keep the polymer gel intact. Water is released to keep the wound moist</td>
<td>Introsate gel Introsate conformable Solosite Solugel</td>
</tr>
<tr>
<td>Hydrofibre – Soft non-woven pad or ribbon dressing made from sodium carboxymethylcellulose fibres</td>
<td>Aquacel</td>
</tr>
<tr>
<td>Multilayer absorbent dressings – either semi-adherent or non-adherent layer, combined with highly absorptive layers of fibers</td>
<td>CombiDerm</td>
</tr>
<tr>
<td>Odour absorbing</td>
<td>Actisorb plus CarboFlex</td>
</tr>
<tr>
<td>Hypertonic saline impregnated - infused with sodium chloride</td>
<td>Hypergel Mesalt</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Types</th>
<th>Examples</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silver dressings</td>
<td>Acticoat Acticoat 7</td>
<td>Infected wounds</td>
<td>Bacteriocidal – kills pathogens such as</td>
<td>Questions remain regarding accumulation toxicity and</td>
<td>Allergy.</td>
</tr>
<tr>
<td>Dressings containing various silver content</td>
<td>Aquacel AG</td>
<td>Burns</td>
<td>MRSA and VRE</td>
<td>resistance. Should be used with care.</td>
<td>Some can't be used with oil based products or topical</td>
</tr>
<tr>
<td></td>
<td>Atrauman AG</td>
<td></td>
<td></td>
<td></td>
<td>antimicrobial</td>
</tr>
<tr>
<td></td>
<td>Mepilex AG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paper adhesive tape - adhesive tape may be</td>
<td>Micropore</td>
<td>Small wounds</td>
<td>Non allergenic. Provides wound support</td>
<td>Non absorbent</td>
<td>Exudative or large wounds.</td>
</tr>
<tr>
<td>applied directly to healing laceration</td>
<td></td>
<td>Dressing retention</td>
<td>water and water vapour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fixation Sheet - Porous polyester fabric</td>
<td>Fixomull Hypafix</td>
<td>Superficial wounds</td>
<td>Conforms to body contours hypoallergenic</td>
<td>Requires adhesive remover to remove</td>
<td>Infected wounds allergy to adhesives</td>
</tr>
<tr>
<td>with adhesive backing</td>
<td>Mefix</td>
<td>To secure dressings</td>
<td>Can be sterilised without reducing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>adhesiveness</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix A – Biochemical changes related to Intrinsic and Extrinsic factors

A schematic overview of major biochemical changes and signaling pathways involved in the generation of intrinsically and extrinsically aged skin. (A) Intrinsically and (B) extrinsically aged skin obtained from the (A) inner side of the upper arm of an 83-year-old and (B) the face of a 75-year-old woman. In the (B) sun-exposed skin sample, the typical histologic characteristics with accumulation of disoriented elastic tissue (blue arrows) in the dermis can be visualized after elastica staining. By contrast, (A) sun-protected skin shows only moderate histologic changes. In aged skin, mitogen-activated protein (MAP) kinase signal transduction pathways are important in regulating a variety of cellular functions. Downstream effectors of the MAP kinases include several transcription factors, including the c-Jun and c-Fos, which heterodimerize to form the activator protein 1 (AP-1) complex. AP-1 is a key regulator of skin aging, because it induces the expression of the MMP family and inhibits type I procollagen gene expression through interference with TGF-β signaling pathway. It has been postulated that MAP kinases may be activated by excess production of reactive oxygen species (ROS) that occurs with advanced age and may be superimposed by extrinsic factors such as ultraviolet irradiation. Excess ROS production also leads to accumulation of cellular damage, which includes oxidation of DNA resulting in mutations, oxidation of proteins leading to reduced function, and oxidation of membrane lipids resulting in reduced transport efficiency and altered transmembrane signalling. IL, interleukin; NF-κB, nuclear factor-κB; TGF-β, transforming growth factor-β; TSP-1, thrombospondin-1; TSP-2, thrombospondin-2; VEGF, vascular endothelial growth factor.

Copied from Zouboulis (2011, p 10)\(^{15}\)
## Appendix B – Clinical Implications of Aging Skin

<table>
<thead>
<tr>
<th>Physiologic change</th>
<th>Pathologic change</th>
<th>Clinical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thinning of epidermis and dermis</td>
<td>Increased vulnerability to mechanical trauma, especially shearing and friction</td>
<td>Increased incidence of skin tears</td>
</tr>
<tr>
<td>Flattening of dermal papillae</td>
<td>Increased risk of blister formation</td>
<td>Increased susceptibility to infection</td>
</tr>
<tr>
<td>Slowdown in turnover rate of epidermis; decrease in ratio of proliferative-to-differentiated keratinocytes</td>
<td>Delayed cellular migration and proliferation.</td>
<td>Increased time to re-epithelialization.</td>
</tr>
<tr>
<td></td>
<td>Decreased wound contraction</td>
<td>Longer healing times after injury or surgery</td>
</tr>
<tr>
<td>Decrease in elastin fibres</td>
<td>Loss of elasticity</td>
<td>Lax skin and wrinkling, with loss of self-esteem and/or depression</td>
</tr>
<tr>
<td>Decrease in vascularity and supporting structures in dermis</td>
<td>Fragile, easily broken blood vessels.</td>
<td>Skin easily bruised (senile purpura)</td>
</tr>
<tr>
<td></td>
<td>Decreased wound capillary growth</td>
<td>Increased risk of wound dehiscence</td>
</tr>
<tr>
<td>Decrease in vascular plexus, blunted capillary loops</td>
<td>Loss of thermoregulatory ability</td>
<td>Hypothermia, heat stroke</td>
</tr>
<tr>
<td>Changes in and loss of collagen and elastin fibres</td>
<td>Decreased tensile strength, lower layers more susceptible to injury</td>
<td>Increased risk of pressure damage to elderly skin, decubitus ulcers</td>
</tr>
<tr>
<td></td>
<td>Decreased collagen remodeling</td>
<td>Longer healing times after injury or surgery</td>
</tr>
<tr>
<td>Impaired immune response</td>
<td>Impaired inflammatory response</td>
<td>Impaired wound healing</td>
</tr>
<tr>
<td></td>
<td>Impaired delayed hypersensitivity reaction</td>
<td>Increased risk of severe injury from irritants</td>
</tr>
<tr>
<td></td>
<td>Decreased production of cytokines</td>
<td>Impaired immune function</td>
</tr>
<tr>
<td></td>
<td>Decrease in number of Langerhans cells</td>
<td>Increased susceptibility to photocarcinogenesis, false-negative delayed hypersensitivity tests</td>
</tr>
<tr>
<td>Impaired neurologic responses</td>
<td>Reduced sensation</td>
<td>Increased risk of thermal or other accidental injury</td>
</tr>
<tr>
<td>Decreased skin thickness</td>
<td>Loss of cushioning and support</td>
<td>Increased risk of pressure damage, decubitus ulcers Increased susceptibility to skin tears, bruising</td>
</tr>
<tr>
<td></td>
<td>Decreased vitamin D precursor production</td>
<td>Osteoporosis and bone fractures</td>
</tr>
<tr>
<td>Atrophy of sweat glands</td>
<td>Decreased sweating</td>
<td>Less ability to thermoregulate, hypothermia Dry skin, xerosis</td>
</tr>
<tr>
<td>Reduced stratum corneum lipids</td>
<td>Decreased ability to retain water</td>
<td>Variable response to topical medications, altered sensitivity to irritants</td>
</tr>
<tr>
<td>Structural changes in stratum corneum</td>
<td>Altered barrier function</td>
<td>Variable response to topical medications, altered sensitivity to irritants</td>
</tr>
<tr>
<td>Reduced movement of water from dermis to epidermis</td>
<td>Reduced epidermal hydration</td>
<td>Dry skin, xerosis</td>
</tr>
<tr>
<td>Decrease in melanocytes</td>
<td>Loss of ability to tan, greater susceptibility to solar radiation</td>
<td>Cutaneous neoplasms</td>
</tr>
<tr>
<td></td>
<td>Greying hair</td>
<td>Loss of self-esteem</td>
</tr>
</tbody>
</table>

Copied from Farage et al (2009)\(^{(17)}\)
References


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