# **Skin Tears**

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### Skin Tear

Skin tears are small avulsion injuries. They occur most commonly in older adults and are the result of friction and shear. The pretibial area is most commonly involved, but the lesions also occur on other anatomical areas with thin or fragile skin, such as the dorsum of the hand or the elbows. Skin tears may be caused by an accident, but in the frail skin of older adults they may even be caused by the removal of an adhesive dressing. The exact incidence of skin tears is not known, but data gathered in a long-term care institution indicate that more than 95% of all injuries not related to falls were actually skin tears and bruises<sup>(2)</sup>.

Skin tears may involve not only the epidermis but can also be full thickness. Usually, the lesion retracts somewhat and part of the underlying tissues is visible. The diagnosis is straightforward<sup>(2)</sup>.

Although a small percentage of patients will develop wound healing problems, most lesions will heal fairly quickly with proper wound care, including moisture retentive dressings<sup>(2)</sup>.

# **Aging Skin**

As we get older we undergo changes that make our skin more susceptible to damage such as skin tears and bruising. 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<sup>(3, 4)</sup>. 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<sup>(5, 6)</sup>. 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<sup>(7, 8)</sup>. 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<sup>(9)</sup>
- 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<sup>(9-11)</sup>
- loss of hypodermis (~50% by age 80) reduces cushioning and temperature regulation<sup>(12)</sup>
- decrease in vascularity and structures supporting the vasculature lead to bruising and bleeding<sup>(9)</sup>
- loss of collagen and elastin fibres in the dermis means lower tensile strength and less structural support
- immune changes<sup>(9)</sup>

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 prutitis, and eczema being widespread in the older population<sup>(9)</sup>. 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<sup>(9)</sup>. With all this, do we really need a skin tear?

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

- Centre of balance and gate changes
- · Peripheral neuropathy
- Altered vision
- Incontinence

So let's say we have a skin tear, 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<sup>(13)</sup> 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<sup>(14)</sup>. Table 1 lists how the various stages of healing may be affected by aging. So when we do our assessment of systemic features that may impair healing, while we may list healing 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.

So what's the plan?

# Table 1. Changes in the older population specifically related to healing

#### **Haemostasis**

- Increased platelet aggregation
- Increased release of alphagranules

#### Inflammation

- Decreased vascular permeability
- Increased secretion of inflammatory mediators Delayed infiltration of macrophages and lymphocytes Impaired macrophage function
- Decreased expression of growth factors

#### **Proliferation**

- Delayed re-epithelialisation
- Delayed angiogenesis
- Delayed collagen deposition

#### Remodelling

- Reduced collagen turnover and remodeling
- Delayed tensile strength
- Decreased tensile strength

Table copied from Toy (2005)<sup>(1)</sup>

# **Management**

Management of the skin tear is straightforward and follows the basic wound bed preparation principles. There is a simple flow chart that can be incorporated into a skin tear package in Appendix C if you feel this would be beneficial in your area.

#### Stop the bleeding

The use of anticoagulants and antiplatelet medications may inhibit the normal clotting progress and slow healing by delaying the healing process<sup>(15)</sup>. Management of the bleeding patient involves both the immediate management of the bleed and the post bleed assessment. If there is very little bleeding, clean first (see below), then manage the bleed.

If you do not expect the patient to have any coagulation problems the basic bleeding management involves pressure and elevation<sup>(16)</sup>. Lay the patient down, elevate the bleeding area, cover with an absorbent pad and apply pressure. Allow the patient to sit like this for 5 to 10 minutes. Watch for strikethrough and adjust as needed.

For the patient who may have a reduced ability to clot, calcium alginates have been reported as having haemostatic properties. Do the same as above but with a calcium alginate dressing (such as Kaltostat) underneath the absorbent pad. For more severe bleeding you can use adrenaline soaked gauze if you have a medical officer present (beware of rebound bleeding and ischaemic necrosis)<sup>(17)</sup>.

Special training is recommended for nurses in palliative care wards that look after cancer patients. Patients with fungating tumours are at a high risk of a catastrophic bleed from these tumour sites<sup>(17)</sup>, which may be disturbed by the skin tear or whatever caused the skin tear.

Assessment of your patient needs to include circulation obs below the site of the bleed, standard obs to watch for hypovolaemic shock, and should include a review of pathology results and medications to ensure clotting profiles are within acceptable limits.

#### **Assess**

While you're waiting for the bleeding to stop, start running through your assessment. Consider items in the patient's history as well as regional and local observations that would impair healing and devise a plan to manage these risks. I find using a combination of assessment tools such as HEIDI<sup>(18)</sup> and TIME<sup>(19)</sup> help to collect the most relevant information. An example of a tool that combines these to develop a wound management plan can be seen in Appendix D. For documentation purposes it is good to be able to describe the extent of the skin tear. Silver Chain Nursing Association and Curtin University designed a skin tear classification chart that is very helpful in recording skin tear type. The system can be seen in Appendix E. You will also need to address the findings of your pain assessment at this point.

#### Clean

Cleaning should be done in accordance with your local wound cleaning policy. You may use normal saline, potable water, or a specialized cleaning solution such as Prontosan; different solutions have their merits<sup>(20)</sup>. Routine cleaning with topical antimicrobial solutions, while reducing bacterial load may actually inhibit wound healing, possibly even contributing to a wound getting stuck in the inflammatory phase<sup>(21)</sup>. More evidence is needed to determine a specific set of solutions, pressures and techniques to determine optimal cleaning regimes<sup>(22)</sup>.

The aim of cleaning is the same, regardless of your technique. We want to ensure the wound bed is free of any contaminants that may hinder the healing process. This can be

foreign matter, clots or obviously non-viable skin. You may need to incorporate soaking or the use of cotton-tipped applicators, forceps and iris scissors during the cleaning process. Don't just clean around the skin tear, fold the flap back and clean out anything that has become trapped under it.

#### Re-approximate

Once the wound and flap is cleaned gently roll it out to its original position as best you can. However, too much tension has to be avoided



because this may result in flap necrosis<sup>(2)</sup>. Steristrips are often used to pull shut, and hold shut a skin tear. It is true, they do hold the flap in place, but the downside is that this may create too much tension and result in flap hypoxia. Also, the steristrip itself is a foreign body. Does it need to be there? What happens if there is subsequent swelling? Is it a good idea to put something sticky on fragile skin? I, personally, do not recommend the use of steristrips.

#### Moisturize/barrier

If you can moisturize the surrounding skin without disturbing the wound and flap, do so. Also, there are a number of products that you can use to leave a thin film barrier on the skin. This can be used on the periwound skin to help protect it from exudate. Also, if you do need to use something sticky then these barrier films can help reduce skin stripping<sup>(23)</sup>. But be aware that it does increase how well the adhesive will stick to the skin.



#### **Dress**

Secure the flap in place with a simple dressing that will maintain a warm, moist wound environment and not cause any further trauma upon removal. Foams and hydrofibres are a good choice, as they will absorb quite a lot of exudate if the wound is weeping. They keep the area warm and provide a certain amount of cushioning from further damage. Hydrocolloids can be used if there is very low exudate, they will also protect the wound from frictional damage<sup>(24)</sup>. If there are any signs of infection, or if the wound was highly contaminated, hydrocolloids are not recommended. There is also an acrylic dressing that is completely see through. This dressing has an acrylic absorbent pad and a film backing, it has the same benefits and drawbacks of the hydrocolloid with the added benefit of being able to see through it<sup>(25)</sup>. Consider using an antimicrobial if the wound is highly contaminated, covers a large area, or if the patient is immunocompromised.

Put the date on the dressing and draw an arrow showing the direction of the flap (which is also the direction for removal).

#### Leave it

As mentioned earlier, the healing process in the older person still happens as it does in the younger person, it might just take a bit longer. If we do frequent dressing changes we risk disturbing the new tissue because the tensile



strength of wounds in older people is less, therefore it takes less force to interrupt wound healing<sup>(1, 13)</sup>. By putting on a dressing we can leave in place for 3 days or more we give the wound a good chance to start healing and for the flap to stabilize if it is going to survive.

If the area is suitable for compression then a good firm tubular bandage or two not only keeps the dressing in place but also helps to reduce oedema<sup>(26)</sup>.

# **Be Prepared**

For wards/areas where there are a high number of skin tears it might be an idea to have skin tear packets which have everything you need and a quick overview of what to do. This kind of "just in time" education has been shown to improve nurse confidence and ability to apply wound dressings<sup>(27)</sup>. But you would need to weigh up the potential benefits against the cost of making up the single-use kits.

Know what to do! Keep up to date with wound care principles; this can either be through face-to-face training, on-line training or just reading journal articles<sup>(28, 29)</sup>. Trying to keep up with every dressing company's products would consume all your waking hours, so don't try! Get an idea of the generic categories of dressings and what that category does. Stick to the principles of wound bed preparation, then match your needs to the dressing category.

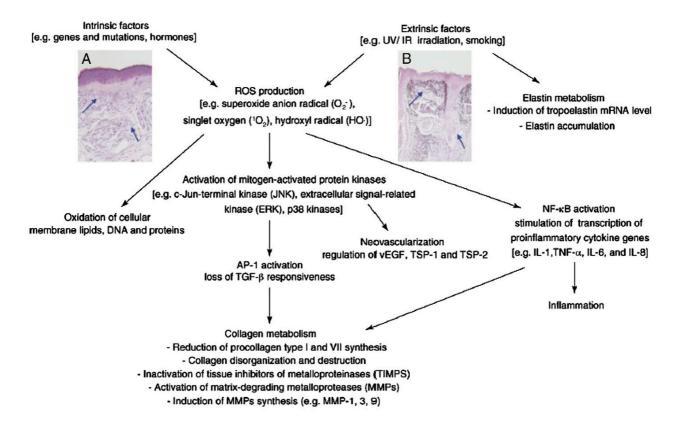
## **Prevention**

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<sup>(12)</sup>. 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<sup>(30)</sup>.
- 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<sup>(12)</sup>.
- No prolonged soaks in the bath that cause maceration<sup>(12)</sup>
- Pat the skin dry, don't rub it<sup>(12)</sup>
- Good lighting<sup>(31)</sup>
- Appropriate furniture (nothing too pointy!)
- Full length sleeves and pants, skin protection devices for vulnerable areas<sup>(31)</sup>
- Maintain adequate fluid intake and nutrition<sup>(31)</sup>
- Good shoes (all over foot protection)
- Good falls prevention practices<sup>(31)</sup>
- Use slide sheets at all times when repositioning patients
- Avoid the use of adhesives, consider alternative options<sup>(31)</sup>
- 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 length-wise 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.

# 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 75year-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- $\beta$  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)<sup>(7)</sup>

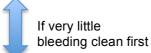
**Appendix B – Clinical Implications of Aging Skin** 

Physiologic change	Pathologic change	Clinical significance
Thinning of epidermis and dermis	Increased vulnerability to	Increased incidence of skin tears
	mechanical trauma, especially shearing and friction	
Flattening of dermal papillae	Increased risk of blister formation	Increased susceptibility to
r latterning or dermal papinae	moreagea new er sneter remnation	infection
Slowdown in turnover rate of	Delayed cellular migration and	Increased time to re-
epidermis; decrease in ratio of	proliferation.	epithelialization.
proliferative-to-differentiated		
keratinocytes	Decreased wound contraction	Longer healing times after injury
	Decreased would contraction	or surgery
Decrease in elastin fibres	Loss of elasticity	Lax skin and wrinkling, with loss of
	,	self-esteem and/or depression
Decrease in vascularity and	Fragile, easily broken blood	Skin easily bruised (senile
supporting structures in dermis	vessels.	purpura)
	Decreased wound capillary growth	Increased risk of wound
Decrease in vascular plexus,	Loss of thermoregulatory ability	dehiscence Hypothermia, heat stroke
blunted capillary loops	Loss of thermoregulatory ability	Trypourennia, neat stroke
Changes in and loss of collagen	Decreased tensile strength, lower	Increased risk of pressure
and elastin fibres	layers more susceptible to injury	damage to elderly skin, decubitus
		ulcers
	Delayed collagen remodeling	Longer healing times after injury
Impaired immune reasons	Impaired inflammatory response	or surgery
Impaired immune response	Impaired inflammatory response Impaired delayed hypersensitivity	Impaired wound healing Increased risk of severe injury
	reaction	from irritants
	Decreased production of	Impaired immune function
	cytokines	•
	Decrease in number of	Increased susceptibility to
	Langerhans cells	photocarcinogenesis, false-
		negative delayed hypersensitivity tests
Impaired neurologic responses	Reduced sensation	Increased risk of thermal or other
,		accidental injury
Decreased skin thickness	Loss of cushioning and support	Increased risk of pressure
		damage, decubitus ulcers
		Increased susceptibility to skin
	Decreased vitamin D precursor	tears, bruising Osteoperosis and bone fractures
	production	Ostcoperosis and bone nactures
Atrophy of sweat glands	Decreased sweating	Less ability to thrermoregulate,
		hypothermia
	_	Dry skin, xerosis
Reduced stratum corneum lipids	Decreased ability to retain water	Variable response to topical
		medications, altered sensitivity to irritants
Structural changes in stratum	Altered barrier function	Variable response to topical
corneum		medications, altered sensitivity to
		irritants
Reduced movement of water from	Reduced epidermal hydration	Dry skin, xerosis
dermis to epidermis		
Decrease in melanocytes	Loss of ability to tan, greater	Cutaneous neoplasms
	susceptibility to solar radiation Greying hair	Loss of self-esteem
	Oleying hair	LU33 UI 3EII-E3IEEIII

Copied from Farage et al (2009)<sup>(9)</sup>

# Appendix C - Skin Tear Flowchart

# CONTROL BLEEDING Pressure and Elevation Consider Kaltostat



# Consider using HEIDI and TIME Identify risks for impaired healing Document assessment findings and STAR Classification Manage pain

#### CLEAN

Soak with Prontosan
Remove debris from wound bed
and under flap
Remove non-viable tissue



RE-APPROXIMATE Do not apply tension



#### DRESS

Protect periwound skin
Apply dressing
Secure with undercast padding
and tubular bandage



#### DOCUMENT

Document care given
Write date on dressing
Draw arrow on dressing showing
the direction of the skin flap

# Package contains:

- 1 x Blue Tray
- 1 x Bluey
- 1 x disposable ruler
- 1 x Prontosan
- 1 x Cavilon Barrier Wipe
- 1 x Mepilex Ag
- 1 x Webril

You will also need to collect: Gloves Appropriate Tubigrip or Tubifast

Optional: Iris scissors Hospital Camera

MANAGEMENT PLAN
Address risk factors identified
during the assessment
Document plan

# Appendix D - Student tool

History	
What is the complaint?	
How long has it existed?	
What has been done about the complaint so far?	
Medical History	
Surgical History	
Medications	
Social History	
Ever Smoked	
Alcohol Intake	
Mobility	
Allergies	
Examination	
Systemic	
Regional	
Local	Location Size
o Tissue	Size
<ul> <li>Inflammation</li> </ul>	
o Moisture	
o Edges	

Investigations	
Diagnosis	
Intervention	Olegania
	Cleansing
	Emollient/Barrier
	Primary Dressing
	Secondary Dressing
	Secondary Dressing
	Retention/Compression

Review:

# **Appendix E – STAR Classification**



# **STAR Skin Tear Classification System**



### **STAR Skin Tear Classification System Guidelines**

- 1. Control bleeding and clean the wound according to protocol.
- 2. Realign (if possible) any skin or flap.
- 3. Assess degree of tissue loss and skin or flap colour using the STAR Classification System.
- 4. Assess the surrounding skin condition for fragility, swelling, discolouration or bruising.
- 5. Assess the person, their wound and their healing environment as per protocol.
- 6. If skin or flap colour is pale, dusky or darkened reassess in 24-48 hours or at the first dressing change.

## **STAR Classification System**



Category 1a
A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is not pale, dusky or darkened.



Category 1b
A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is pale, dusky or darkened.



Category 2a
A skin tear where the edges cannot be realigned to the normal anatomical position and the skin or flap colour is not pale, dusky or darkened.



Category 2b
A skin tear where the edges cannot be realigned to the normal anatomical position and the skin or flap colour is pale, dusky or darkened.



Category 3
A skin tear where the skin flap is completely absent.

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