

MANAGING THE BURN WOUND

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SECTION I: SKIN (BIOLOGIC PROPERTIES)

WHAT ARE THE PROPERTIES OF NORMAL SKIN

Skin is a bilayer organ whose functions are essential for survival. Although the bilayers works as a unit, each component has specific properties which need to be recognized if one is to duplicate these properties with a skin substitute.

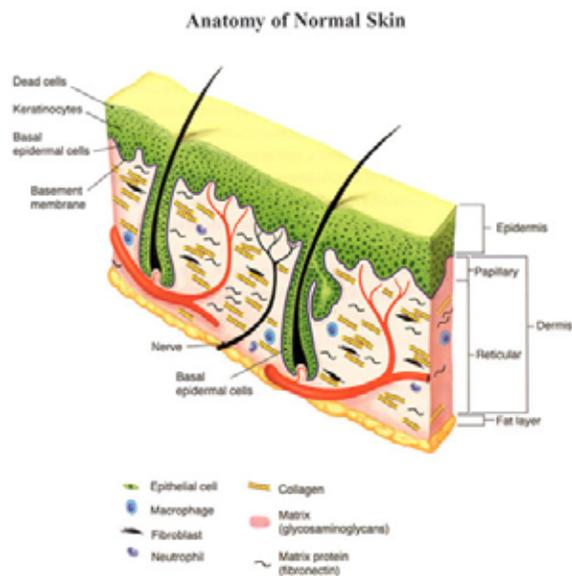
SKIN FUNCTIONS

Epidermis

- Protection from desiccation
- Protection from bacterial entry
- Protection from toxins
- Fluid balance: avoiding excess evaporative loss
- Neurosensory
- Social-interactive

Dermis

- Protection from trauma due to elasticity, durability properties
- Fluid balance thru regulation of skin blood flow
- Thermoregulation through control of skin blood flow
- Growth factors and contact direction for epidermal replication and dermal repair
- contact direction for epidermal replication and dermal repair



FUNCTIONAL COMPONENTS OF SKIN

EPIDERMIS: The outer thinner layer known as the epidermis is composed mainly of epithelial cells. The outermost cells contain the protein keratin and are known as keratinocytes. The basal or deepest epidermal cells are anchored to the basement membrane by adhesion molecules (or glue), namely fibronectin. These immature cells are continually dividing and migrating toward the surface to replace lost surface cells e.g. after an injury. The same type of regenerating epidermal cells are found in hair follicles and other skin appendages which are anchored in the dermis. As the cells mature and migrate to the surface they form keratin which becomes an effective barrier to environmental hazards such as infection and to excess water evaporation.

Replacement of the epidermal layer by this regenerative process takes 2-3 weeks. Cues and biologic stimuli at the wound surface are necessary to direct proper orientation and mitotic response of the epidermal cells. Many of the cues come from dermal elements, especially the matrix proteins and matrix glycosaminoglycan.

Components of Epidermis

- Outer cells: keratinocytes
- Keratin, a tough protein on surface, preventing bacteria or toxin entry
- Inner layer: epidermal cells which are proliferating and migratory to surface and will become keratinocytes
- Innermost layer: basal epidermal cells anchored to basement membrane by adhesion molecules
- Skin appendages anchored in dermis also lined by epidermal cells
- Langerhan's' cells, contain granules, fix antigens (felt to be responsible for antigen-antibody and allergy functions)

Characteristics of Epidermis

- Protection from environmental insults
- Ability to regenerate every 2-3 weeks resulting from biologic cues and contact direction provided by dermis, basement membrane

DERMIS: The **dermis** is a very dynamic layer of thick connective tissue, also in constant turnover. The dermis is divided into a thin superficial layer known as the papillary dermis containing the anchoring epidermal rete pegs and the thicker deeper portion known as the reticular dermis. The papillary dermis is the major factory for the proteins providing direction for epidermal replication. The upper dermis also contains the highest blood flow. The primary cell type is the fibroblast which produces the key structural extra cellular matrix proteins collagen and elastin as well as matrix or ground substance.

In addition these cells produce the key adhesion proteins used to attach epidermal cells to the basement membrane and for used epidermal cell migration and replication. Fibronectin is a key fibroblast derived signal protein for orchestration of healing. The ground substance or matrix is made up of complex polysaccharide - protein complex known as glycosaminoglycan or the GAG component as well as hyaluronic acid. The matrix provides a semi fluid which allows for cell and connective tissue orientation as well as nutrient diffusion to the cells and a scaffolding for cell migration.

COMPONENTS OF DERMIS

- Papillary dermis: upper dermis containing anchoring rete pegs and also is the most biologically active part of the dermis
- Reticular dermis: the thicker deeper portion responsible for durability and anchoring of skin appendages
- Matrix proteins
 - collagen is the predominant protein, mainly collagen Type 1. (besides structure; collagen type 1 provides a contract orientation for dividing and migrating epithelial cells)
 - fibronectin is the primary adhesive protein playing a major role in healing
 - other adhesive proteins
- Ground substance (glycosaminoglycan)
 - carbohydrates protein complexes
 - hyaluronic acid
- Cells
 - fibroblasts
 - macrophages
 - platelets
 - endothelial cells
- Blood vessels (auto regulated flow)

CHARACTERISTICS OF DERMIS

- Provides durability, flexibility of skin
- Factory for all the components required for replication and repair of epidermis and dermis
- Scaffolding for cell migration and the conduit for nutrient delivery

INTERFACE: The interface between the layers or the dermo-epidermal junction is the basement membrane rich in the adhesive protein fibronectin which anchors the epidermal cells from above and dermis from below.

THICKNESS: Average thickness of the bilayer is 1-2 mm being considerably thinner in infants and the elderly especially the dermis which is underdeveloped in infants and atrophic in the elderly.

CELL TYPES
<p>Epidermis</p> <ul style="list-style-type: none"> - Keratinocytes - Epithelial cells - Langerhan's cells <p>Dermis</p> <ul style="list-style-type: none"> - Fibroblasts - Macrophages - Endothelial cells - Platelets

EPITHELIAL CELLS: These cells make up the majority of the epidermis. Immature cells are programmed to divide, migrate and mature to keratin producing cells called keratinocytes. The signals to activate this process come from messenger proteins called growth factors as well as through contact direction from key dermal adhesive proteins, especially collagen.

FIBROBLASTS: The cells of mesenchymal original are normal present in the dermis and produce normal dermal replacement components. After injury these cells migrate into the wound and proliferate; in order to produce increased quantities of these dermal proteins and matrix.

ENDOTHELIAL CELLS: These cells make up the lining of micro and macro vessels and also make up the lining of new capillaries produced after injury. These cells like fibroblasts do differentiate from local mesenchymal cells and are also attracted into the wound by local signals.

MACROPHAGES: These cells of mesenchymal origin are normally present in tissue but increase in number after injury, attracted by chemical messages released by the activation of inflammation. The long-lived cells release the protein chemical messages, growth factors and growth stimulants, which orchestrate healing in an organized fashion.

PLATELETS: The factor-rich particles release a host of growth factors and adherence proteins during the initial post burn period.

<p>FIBROBLAST PRODUCTS</p> <ul style="list-style-type: none"> • Collagen (type one in skin) • Matrix proteins (fibronectin, tenascin, others) • Proteoglycans, glycosaminoglycan, hyaluronic acid, other matrix components • Cytokines and other growth stimulants
<p>MACROPHAGE PRODUCTS</p> <ul style="list-style-type: none"> • Growth factors • Growth stimulants • Opsonins

CHARACTERISTICS OF SKIN COLLAGEN TYPE 1

Function

- Creates adherence to wound surface via fibrin and fibronectin
- Provides surface orientation for epithelial cell migration
- Stimulates dermal cell migration

Structure

- Provides dermal scaffolding and durability
- Complex surface morphology

CHARACTERISTICS OF MATRIX (GAG)

Functions

- Glue or adherence properties in tissue via cell-matrix interaction
- Substrate for migration of nutrients, cells and growth factors
- Deactivator of toxic protease released by neutrophils
- Conduit for living fibrin, fibronectin and growth factors in contact with the wound surface
- Scaffold for surface deposition of fibrin and fibronectin i.e., cell guidance proteins

Structure

- The foundation for deposition of dermal cells, collagen, other proteins
- These compounds also provide the scaffold for the epidermal basement membrane
- Brings critical matrix proteins and growth factors into contact with each other

Dermal Molecules Influencing Burn Wound Closure

Molecule	Source	Location
Collagen type I	Fibroblast	Dermis - Supports epidermal cell attachment and migration
Collagen type IV	Epidermal cell, fibroblast	Lamina densa - Supports epidermal cell attachment
Fibronectin	Fibroblasts, macrophage, serum	Basement membrane - Dermis
Laminin	Epidermal cell	Epidermal cell adherence
Glycosaminoglycans	Fibroblast	Promotes cell adherence, migration, nutrient delivery

FIBRONECTIN: This adhesion protein, produced mainly by fibroblasts and macrophages, is a large glycoprotein found in all tissues and plasma. One of its key functions is as an attachment protein for skin cells via collagen type I. Production is induced with a burn. Fibronectin plays a major role in wound healing.

Fibronectin	
Adherence Function	
<ul style="list-style-type: none"> • Cross linking to fibrin (and collagen) causing adherence of tissues to each • Key adherence molecule attaching epithelial and endothelial cells at cell junctions • Contact orientation for all cells in the healing process 	
Epithelialization	
<ul style="list-style-type: none"> • Cell migration, spreading, and orientation • Cell division • Cell re-adherence to form a layer 	
Chemo Attractant (Fibronectin Fragments)	
<ul style="list-style-type: none"> • For fibroblasts • For macrophages 	

A large variety of "polypeptide growth factors" have been identified and named. Although each has a predominant function on a specific cell, all growth factors have a multitude of actions. Epidermal growth factor (EGF) is a key component for re-epithelialization of a partial-thickness burn, and addition of EGF to the wound surface increases re-epithelialization. Keratinocyte growth factor (KGF) is an important fibroblast derived stimulant for epithelialization. Macrophages are thought to be the main producers of growth factors; however, all skin cells, including fibroblasts and keratinocytes, play an important role in secreting growth factors. The initial stimulus requires the onset of wound inflammation, and once activated, further production continues until the wound is healed.

Once formed the growth factors can be rapidly deactivated by wound proteases, e.g. collagenases, proteases, probably in an attempt to break down surface dead tissue. Surface exudate is a rich source of such proteases, especially the class of metalloproteases.

GROWTH FACTORS INVOLVED IN WOUND HEALING		
MOLECULE	SOURCE	ACTION
Fibroblast Growth Factor	Keratinocytes, macrophages	Stimulates angiogenesis
Epidermal Growth Factor	Platelets	Stimulates epidermal cell proliferation
Keratinocyte Growth Factor	Fibroblasts	Stimulates epidermal cell growth
Interleukin -1	Macrophages, epidermal	Stimulates epidermal growth and motility
Platelet-Derived Growth Factor	Platelets, macrophages	Stimulates epidermal growth, fibroblast proliferation
Transforming Growth Factor-B	Fibroblasts, platelets	Fibrosis and increased tensile strength

FUNCTIONS OF SKIN GROWTH FACTORS

- Cell proliferation: epidermis, fibroblasts, endothelial cells
- Cell migration: white cells, epithelial, endothelial, fibroblast
- Structure formation: capillaries, epidermis
- Cell production of tissue proteins:
collagen, matrix proteins, keratin

As can be seen, skin is a very biologically active organ.

SECTION II: PATHOGENESIS OF BURN INJURY (INITIAL and DELAYED)

The damage to epidermis and dermal elements from a burn is the result of several key insults which can be divided also into initial and delayed insults.

KEY INSULTS

- Heat Induced Injury
- Inflammatory Mediator Injury
- Ischemia Induced Injury

A. INITIAL INJURY

HEAT INJURY

The most immediate and obvious injury is that due to heat. Excess heat causes rapid protein denaturation and cell damage. The depth of heat injury is dependent on the depth of heat penetration. Wet heat (scald) travels more rapidly into tissue than dry heat (flame). A surface CI- temperature of over 60' C produces immediate cell death as well as vessel thrombosis. The dead skin tissue on the surface is known as eschar. The depth of burn is dependent on the temperature of the heat insult, the contact time and the medium (air-water). In addition, the depth of the skin layer is critical as the thinner the skin, the deeper the burn.

INFLAMMATORY MEDIATOR INJURY (First to Third Day)

It is now clear that much of the tissue damage, especially in the perfused subsurface burn, is caused by toxic mediators of inflammation which are activated with the burn. Although onset of inflammation is required for healing, excess production of mediators especially oxidants and proteases will cause more capillary endothelial and skin cell damage. Early release of oxidants and increased proteolytic activity in the burn is now well recognized. Current evidence indicates that the inflammatory response initiated by the heat injury is responsible for further early tissue damage, increased capillary permeability and is in large part responsible for the later wound conversion if inflammation becomes excessive. This now well-established concept allows the care providers to intervene early and control the further mediator injury thereby limiting the final injury. The early use of both mediator inhibitors and skin substitutes is based on this concept. The clinician therefore has the potential of significantly modifying the degree of injury.

INFLAMMATION INDUCED WOUND INJURY

- Protease Release Injuring Healing Tissue and Deactivating Growth Factors
- Oxidants Release Injuring Cells, Denaturing Proteins, and Activating Inflammation
- Consumption of Wound Oxygen by Neutrophils Leading to Tissue Hypoxia
- Increasing Stimulus to Fibrosis

B. DELAYED INJURY

Continued tissue damage can occur in the burn wound after the initial heat and mediator damage. The common element is ongoing inflammation perpetuated by surface eschar, bacterial colonization, mechanical trauma or that caused by topically applied antibacterial agents. Increased neutrophils, especially in exudate on the surface, results in increased damage to viable tissue by both neutrophil proteases and oxidants and neutrophil consumption of oxygen. Wound surface protease activity, particularly metalloprotease, has been noted to be markedly increased on open burn wounds. Of great importance is the fact that there is recent evidence that the proteolytic activity, on the normal partial thickness human burn, is markedly increased. This results in a net ongoing proteolysis in the burn both on the surface and in the matrix. Besides injury to new tissue formation, these proteases deactivate locally released growth factors, further impairing healing. This constant impediment to wound healing will stimulate further collagen synthesis, with subsequent increased scar, even in a mid-dermal burn. Eliminating surface exudate is a major goal of the use of occlusion dressing but active surface inflammation will persist.

However, only a wound closure using an adherent membrane or skin substitute will decrease the degree of surface inflammation as well as prevent surface exudate. Dead tissue or any residual exudate needs to be removed prior to an attempt at wound closure since this approach will not work unless the wound has a viable tissue wound surface to allow adherence.

CONTINUING BURN WOUND INJURY

- Ongoing inflammation caused by
 - neurotic tissue
 - bacteria on surface
 - caustic topical agents
 - surface exudate
- Excess wound proteolytic activity
 - activated by surface insults
 - continued damage to viable cells and new tissue growth
 - damage to wound surface and matrix denaturation of growth factors
- Excess oxidant release
 - injuring viable cells

MID-DERMAL BURN (Admission)



Note red dermal surface free of exudate after initial cleaning prior to placement of topical antibiotic silver sulfadiazine

MID-DERMAL BURN (2 Days later)



Note presence of eschar or pseudo eschar. Surface exudate is adherent and composed of denatured protein, inflammatory cells, and rich in surface metalloproteinase activity which can denature growth factors and increase the degree of injury.

SECTION III: BURN WOUND ASSESSMENT

The burn wound is defined in terms of the evolving injury that occurs. Therefore the histological description is defined in terms of specific areas of pathologic change called zones. Three zones have been classically described. The actual pathophysiology is now recognized to be much more complex than the terms used for defining the zones.

A. Histological Assessment

Histological Assessment of the Burn Wound

- Zone of coagulation (necrosis)
- Zone of stasis (injury)
- Zone of hyperemia

Zone Of Coagulation

This zone is comprised of the surface tissue necrosis of the initial burn eschar. The surface injury is caused mainly by the heat or chemical insult. Obviously this zone has an irreversible injury.



Full thickness burn (admission)

Zone of coagulation is the depth of tissue necrosis which, in this patient, compress both s layers of skin

INSERT IMAGE OF MIXED DEPTH BURN



Zone of coagulation varies markedly from center of burn which is full thickness, to the very periphery where all necrotic tissue has been removed



Zone of necrosis has been removed. Wound bed is viable tissue although injured (zone of injury) beneath the surface

Zone Of Injury (Stasis)

Deep and peripheral to the zone of coagulation, there is a sizable area of tissue injury where cells are viable but can easily be further damaged. The terms "stasis" or "ischemia" were used because the progressive injury in this area was thought to be due to capillary thrombosis from injured endothelium, leading to ischemia-induced cell death. Fibrin deposition, vasoconstriction, and thrombosis indeed do occur, most likely as a result of continued release of mediators. However, early epithelial cell death in this area, unrelated to blood flow, is reported to be quite high, leading to slowing of healing. Epithelial cells are particularly prone to environmental insults such as desiccation- and inflammation-induced injury. This zone is most prominent in mid-to-deep-dermal burns where there is less reserve in the remaining viable cells and less blood flow.

Zone Of Hyperemia

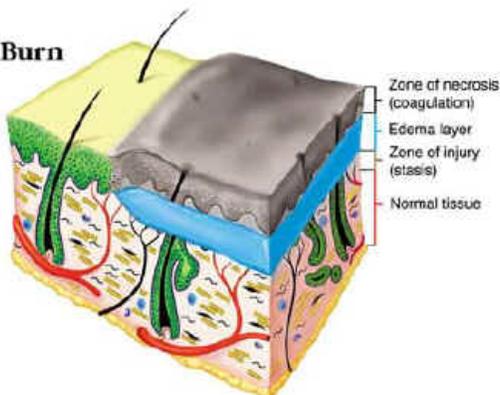
Peripheral to and below the zone of stasis is the zone of hyperemia. The area is characterized by minimal cell injury but with vasodilatation due to neighboring inflammation-induced mediators. Completed recovery of this tissue is expected unless there is an additional severe insult such as an invasive infection or profound tissue inflammation.

ZONES OF INJURY VARIES WITH BURN DEPTH

Superficial Dermal Burn

Characteristics

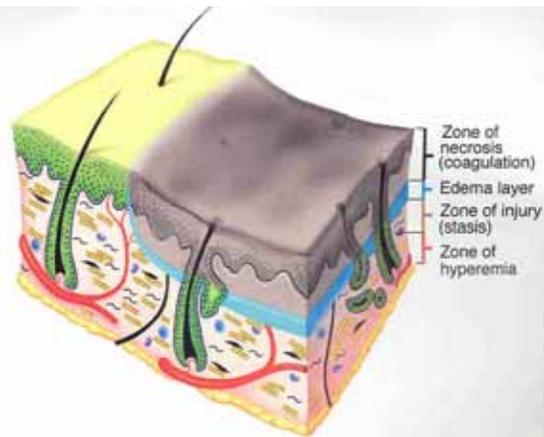
1. Necrosis confined to upper third of dermis
2. Zone of necrosis lifted off viable wound by edema
3. Small zone of injury



Mid-Dermal Burn

Characteristics

1. Necrosis to mid-dermis
2. Large zone of injury (potential conversion)
3. Eschar separated from viable tissue by edema layer



MID DERMAL BURN IMAGE OF HAND

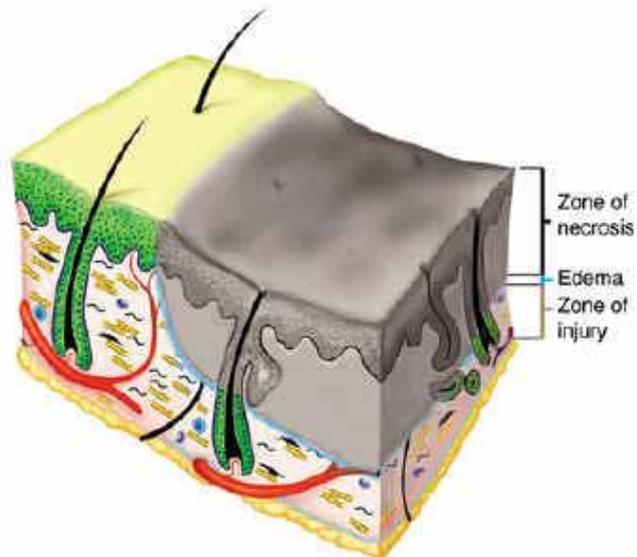


This mid-dermal burn has larger zone of injury than a more superficial burn

Deep Dermal Burn

Characteristics

1. Necrosis involving majority of skin layers
2. Zone of necrosis adherent to zone of injury
3. Smaller edema layer



DEEP DERMAL BACK BURN



Deep dermal back burn (with full thickness on flank) Dry white to dry red appearance reflective of lack of surface blood flow. The zone of injury below the surface is at high risk for conversion to a full thickness wound.

Full Thickness Burn

Characteristic

No remaining viable dermis

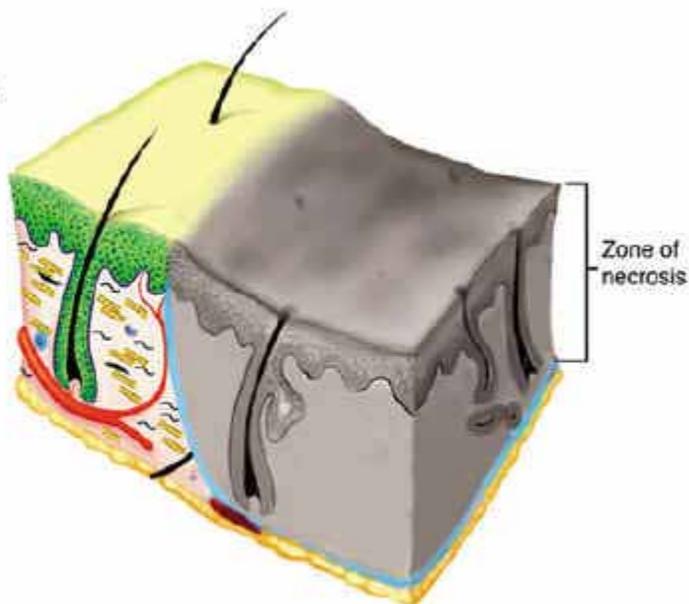


IMAGE OF FULL THICKNESS BURN TO BACK



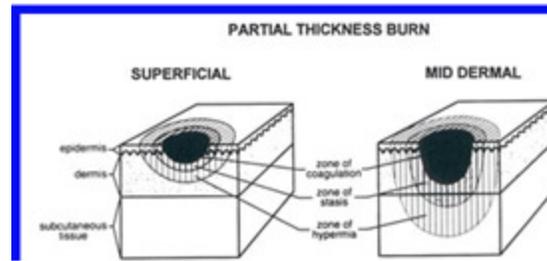
In a full thickness burn, the zone of injury can readily extend below the skin into subcutaneous tissues. The zone of hyperemia develops in the subeschar area being most evident beginning about 7 days post burn.

IMAGE OF ZONE OF HYPEREMIA



Zone of hyperemia is very evident in this wound excised at day 10. The blood flow to this fatty tissue is markedly increased over normal sub-dermis vascularity.

PARTIAL THICKNESS BURN



ZONES OF INJURY IN TWO BURN DEPTHS

THE ZONES OF STASIS OR INJURY AND HYPEREMIA (reaction) are much larger in a mid dermal burn compared to a superficial burn. This larger zone of injury exceeds the increase in size of the zone of coagulation (necrosis) between the two depths.

The reason is that 1) the best blood flow is present in the superficial dermis and ischemia is a greater risk beyond that point, 2) once through the epidermis, the heat transfer increases into the dermis such that a deeper area of heat and inflammatory injury results. A mid (or deep) dermal is much more prone to further injury during the treatment period.

Factors Increasing Zone of Injury

- Lower blood flow with a deeper burn
- Increased risk of infection with deeper burn
- Presence of surface necrotic tissue

Burn Edema

A layer of protein-rich edema fluid develops between the eschar (zone of coagulation) and the perfused, heat-injured micro vessels as a result of increased (heat and mediator-induced) micro vascular permeability. The leak is most prominent in the first 8-12 hours but can persist for days. In superficial burns, the edema actually physically separates viable and non-viable tissue, producing blisters, so that mechanical cleaning can remove the dead tissues. In deep second-degree and third-degree burns, the edema occurs throughout the injured tissue. However, the necrotic dermis remains physically adherent to the sub dermal space and requires sharp dissection (debridement) to remove the dead tissue or the process of necrolysis must occur. This process is deleterious due to the risk of infection and degree of tissue inflammation, as well as absorption of dead tissue. The degree of tissue edema is dependent on the amount of resuscitation fluid given and the vascular pressures perfusing the area.



Note: Edema developing below the zone of coagulation is very prominent in facial burns

B. ANATOMIC ASSESSMENT OF THE BURN WOUND

Burn depth is defined based on the depth of coagulation necrosis into epidermis and dermis (recognizing that the anatomical depth may change with wound conversion).

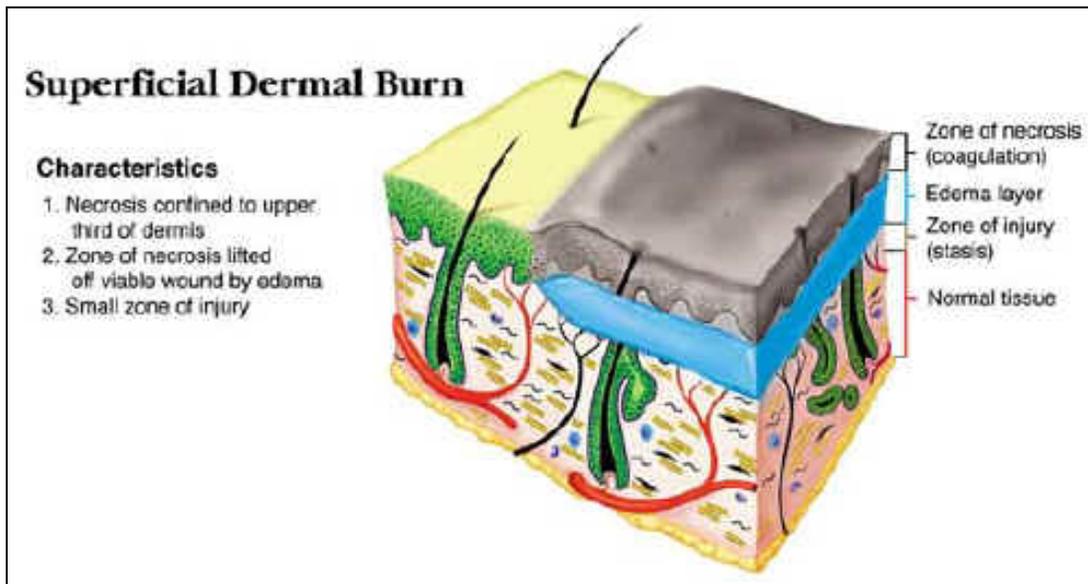
CAUSE	DEPTH DEGREE	APPEARANCE	PAIN
Hot Liquids - Short exposure - Long exposure	Superficial dermal	wet, pink, blisters	severe
	Deep dermal	wet, dark red	minimal
Flames - Flash exposure - Direct contact	partial thickness	severe	wet, pink blisters
	full thickness	minimal	dry, white, waxy or brown, black leathery
Chemicals	usually full thickness	severe	light brown to light gray

PARTIAL THICKNESS BURN

There are three categories of second-degree burn typically used to characterize the depth of injury. Each corresponds with healing time, treatment modalities and outcome.

These are: 1) superficial, 2° or dermal burn, mid 2° or dermal burn, and 3) deep 2° or dermal burn

1) Superficial Dermal Burn



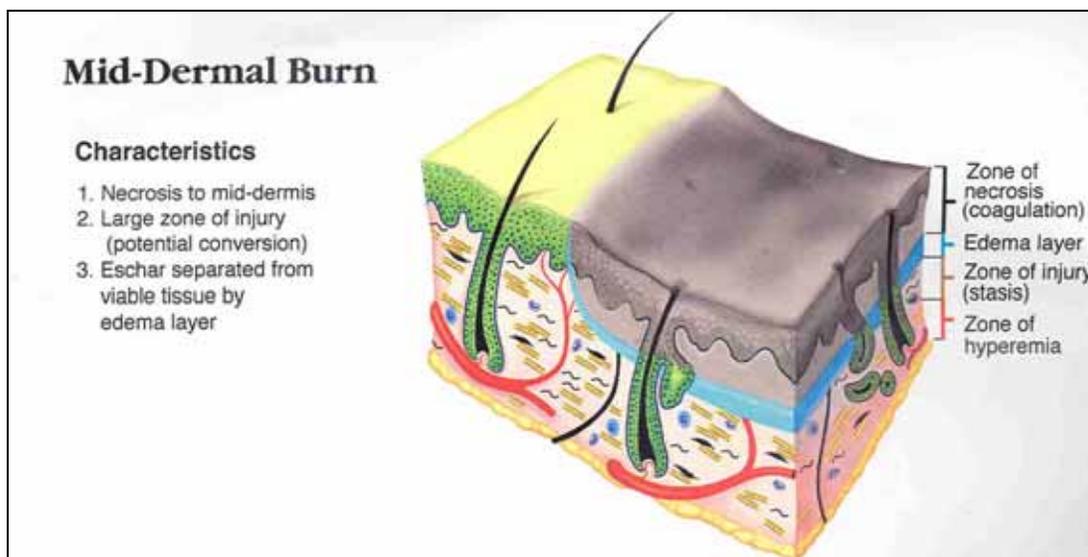
Characteristics:

- Wet blisters
- Pink painful wound
- Good blood supply
- Low risk of infection and scar
- Heals without scar in 10-14 day



A superficial burn is characterized by blisters as the dead epidermis and superficial dermis is lifted off the viable dermis by edema fluid.

2) Mid-Dermal Burn



Characteristics

- wet, red
- painful
- good blood supply
- moderate risk of infection and scar
- heals in less than 21 days

A mid-dermal burn is less wet and less pink as more of the dermis is injured



3) Deep Dermal Burn Characteristics

- white, dry
- little to no pain
- low blood flow
- very high risk of infection or scar if it heals
- heals in greater than 4 weeks

A deep dermal injury involves the majority of the dermis including blood supply and nerves.

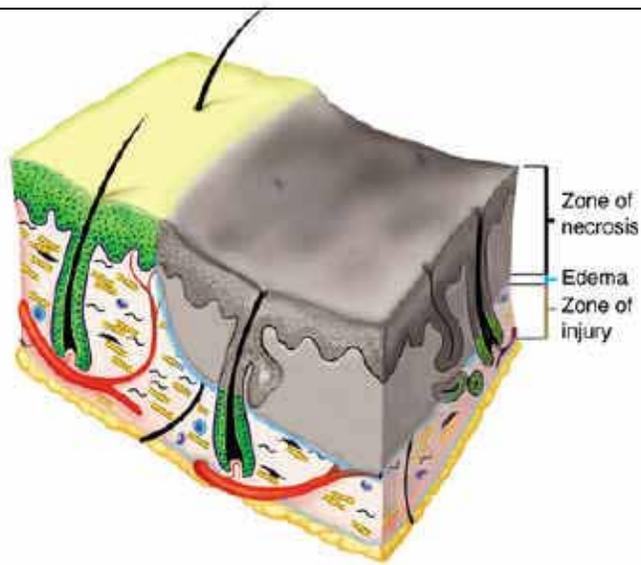


Deep dermal hand burn: note white appearance

Deep Dermal Burn

Characteristics

1. Necrosis involving majority of skin layers
2. Zone of necrosis adherent to zone of injury
3. Smaller edema layer



Indeterminate Dermal Burn

A deep burn that cannot be clinically distinguished as a deep dermal or full thickness
Mixed deep dermal and full thickness compound



(Waxy white in middle is full thickness)



Appearance: white, waxy, but some bleeding from escharotomy. Is it deep dermal or full thickness?

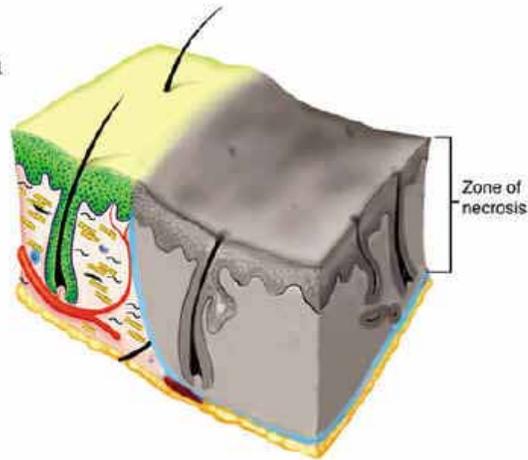
4) Full Thickness Burn

Necrosis of entire dermis including all epidermal elements edema diffuse beneath eschar

Full Thickness Burn

Characteristic

No remaining viable dermis



Characteristics:

- white, dry or char heat
- no pain
- no blood supply
- high risk for infection
- won't heal

In a full thickness burn both layers of skin are destroyed and the wound cannot heal.



Full thickness burns: dry waxy appearance. Note: thrombosed vessels

BURN DEPTH AND OUTCOME

Second Degree	Cause	Appearance	Pain	Healing	Scar
Superficial	hot liquid short exposure	wet, pink, blisters	severe	10-14 days	minimal
Mid-Dermal	hot liquid longer exposure, flash flame	less wet, red ± blisters	moderate	2-4 weeks	moderate
Deep-Dermal	chemicals, flames	dry, white	minimal	3-8 weeks	severe (needs graft)
Indeterminate (2 nd or 3 rd)	chemicals, flames	dry, white	none	—	—
Third Degree (full thickness)	chemicals, flames, explosion, with very high temperature	dry, white or char	none	need graft	mild to severe, depending on timing and type of graft

SECTION IV: INITIAL WOUND MANAGEMENT

Wound assessment as to size, depth and location is necessary before the correct treatment can be initiated. Treatment is in large part based on depth.

However, it is important to recognize which burns should be immediately referred to a burn center as only minimal initial care is needed for those patients who are to be transferred.

The American Burn Association (ABA) has identified those injuries that should be treated in a specialized burn center. Patients with these burns should be treated in a specialized burn facility after initial assessment and treatment at an appropriate hospital emergency department. Sometimes major burns are directly to a burn center from scene if the center is within a safe transport time.

Transfer Criteria to a Burn Center

Burn Injuries that should be referred to a burn unit include the following:

1. Partial thickness burns greater than 10% total body surface area (TBSA)
2. Burns that involve the face, hands, feet, genitalia, perineum or major joints (see High Risk section)
3. Third degree burns in any age group
4. Electrical burns, including lightening injury (see Electrical Burn section)
5. Chemical burns (see Chemical Burn Section)
6. Inhalation injury
7. Children with any of the above burn injuries
8. Burn injury in patients with preexisting medical disorders that could complicate management
9. Any patients with traumatic injury (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient must be initially stabilized in the nearest appropriate facility before being transferred to a burn unit
10. Any burned children if the hospital initially receiving the patient does not have qualified personnel or equipment for children.

A. Superficial Second Degree (Partial Thickness Burn)

This depth of burn is at low risk for infection unless grossly contaminated. Initial cleansing should include removal of dirt, broken blisters and dead epidermis. Large blisters can be debrided off if using a temporary skin substitute or left intact for a few days. Often blisters get larger with time and impede movement at which time they should be removed. Topical antibiotics are not needed, especially cream based agents such as silver sulfadiazine as these agents impede healing and are only used if infection risk is high.

Definition: Second-degree burns are defined as those burns in which the entire epidermis and variable portions of the dermis layer are heat destroyed. A superficial second-degree (partial thickness) burn is characterized by heat injury to the upper third of the dermis leaving a good blood supply.

Cause: Usually hot water.

Appearance: The micro vessels perfusing this area are injured resulting in the leakage of large amounts of plasma, which in turn lifts off the heat-destroyed epidermis, causing blister formation. The blisters will continue to increase in size in the post-burn period as well and protein breakdown occurs. A light pink, wet appearing very painful wound is seen as blisters are disrupted. Frequently, the epidermis does not lift off the dermis for 12 to 24 hours and what appears initially to be a first degree is actually a second degree burn.

Outcomes:

Healing Rate: Despite loss of the entire basal layer of the epidermis, a burn of this depth will heal in seven to fourteen days if non-infected due to repopulation of the epithelial cells that are also present in skin appendages, anchored deep in the dermis. Minimal to no scarring is expected to occur. There is a relatively small zone of injury and conversion is uncommon except at extreme of age or chronically ill. Most antibiotic creams will slow the healing rate.

Treatment:

1. Clean, remove small blisters; apply grease gauze and soft gauze dressing (occlusion, absorbent dressing, changed daily).
2. On face, perineum, apply bacitracin or neomycin ointment, applying several times a day.
3. Excellent alternative is the use of a synthetic adhesive dressing which seals the wound and decreases pain.
4. Use a water-soluble topical antibiotic if the wound is grossly contaminated or if one is unsure if the wound is superficial or deep.
5. Prophylactic systemic antibiotics are not needed.

HOT WATER (SUPERFICIAL DERMAL BURN)



TREATMENT

- 1) Transfer to Burn Center due to size and age
- 2) Gentle wash
- 3) Xeroform- followed by layer of dry gauze and flex net (except face)
- 4) Can use skin substitute
- 5) Bacitracin to face and neck burn

FLASH BURN (SUPERFICIAL 2nd Degree) HOT WATER BURN

TREATMENT



- 1) Wash
- 2) Debrided blisters and loose skin
- 3) Closed dressing with Xeroform followed by gauze
- 4) Can use skin substitute

MID PARTIAL THICKNESS SCALD BURN (Dorsum of Hand)



TREATMENT

- 1) Initially cool water can control pain
- 2) Consider admission for elevation, debridement, pain control
- 3) Admit if both hands involved
- 4) Use xeroform, and closed dressing with compression
- 5) Bacitracin ointment is optional
- 6) Can use skin substitute

B. Mid-Partial Thickness Burn

It is not necessary to distinguish a superficial from a mid-dermal burn on initial assessment as initial management is basically the same.

Definition: A mid second degree extends to the mid portion of the dermis. Longer exposure to hot liquids (5-10 seconds) or flash flames (not direct contact of flames with skin) are the most common causes.

Cause: Brief exposure to flames or flash explosion: hot water in infant or elderly.

Appearance: The burn surface may have blisters but is more red, less wet and only moderately painful.

Outcome: These burns usually heal in about two to four weeks. The exception is the very young and elderly where the dermis is thin and dept of burn is invariable deeper. However, there is a large zone of injury and risk of conversion. If a burn heals in two weeks, then minimal to no scarring is expected. With healing time beyond three weeks scarring will occur, the degree being greater in dark skinned individuals.

Treatment:

1. In patients six years to 60 years, without diabetes, chronic illness, etc., treatment is grease gauze and occlusive dressing. A topical antibiotic ointment such as bacitracin can also be used daily. The depth can be underestimated and a switch to an antibiotic cream may be needed if the wound appears deeper on the first dressing change because of risks of infection.
2. In very young, and very old patients, or those with chronic illness, contaminated wounds or perineal wounds, the traditional choice is a topical antibiotic. First choice is silver sulfadiazine or a silver dressing with closed dressing technique.
3. New Approach: (a temporary skin substitute) which could increase healing and decrease conversion.



TREATMENT

- 1) Transfer to Burn Center due to size i.e., >15% TBS
- 2) Too big to use cold dressings except for a very brief initial period
- 3) Use topical antibiotic in view of age, high risk of conversion, infection
- 4) Alternative: temporary skin substitute to generate wound closure



TREATMENT

- 1) Cold water to control pain
- 2) Gently cleanse
- 3) Grease gauze, plus gauze dressing (closed technique)
- 4) Antibiotic ointment is optional but a silver cream not needed
- 5) Apply dressing to allow for mobility of hand



Closed dressing to hand allowing for mobility of fingers

MID-DERMAL BURN (HOT GREASE)



TREATMENT

- 1) Transfer to burn center due to location (bilateral feet)
- 2) Temporary use of cold to control pain
- 3) Debride loose tissue
- 4) Grease gauze, topical antibiotics on it, ointment or silver dressing with closed dressing
- 5) Consider temporary skin substitute



Skin Substitute at day 1 and day 5

c) Deep Partial Thickness (Deep Second Degree) Burn

A deep dermal burn has not only compromised blood flow but also a layer of adhered dead dermis. The combination increases the risk for infection. A topical antibiotic is required to prevent infection until the area is excised and grafted. Spontaneous healing typically leads to significant scarring. Often, there are patches of deep and less deep burn together. Unless the more superficial burn is covered with a skin substitute, the whole area is often treated with a topical agent. Ointments such as Bacitracin do not adequately penetrate the eschar of a deep burn so a silver release dressing or cream is required.

Prophylactic systemic antibiotics are not needed. Do not apply a cream on to the wound if the patient is to be transferred immediately to a burn center as it interferes with a wound assessment.

Definition: A deep partial thickness or deep second-degree burn extends well into the dermal layer and fewer viable epidermal cells remain. Therefore, re-epithelialization is extremely slow, sometimes requiring months. Grafting is often the preferred treatment for long-term function.

Appearance: In these patients, blister formation does not characteristically occur because the dead tissue layer is sufficiently thick and adherent to underlying viable dermis that it does not readily lift off the surface. The wound surface may be red and dry in appearance with white areas in deeper parts (dry since fewer blood vessels are patent). There is a marked decrease in blood flow making the wound very prone to conversion to a deeper injury and to infection. It is often not possible to distinguish a deep partial from a full thickness burn by initial appearance. Frequently the wound is a mixed second and third degree. Direct contact with flames is a common cause. Most chemical burns are also deep. The appearance of the deep dermal burn changes dramatically over the next several days as the area of dermal necrosis along with surface coagulated protein turns the wound a white to yellow color. The amount of surface coagulum is accentuated with the use of a topical antibiotic, making the deep second degree burn difficult to differentiate from a third degree burn. The presence of some pain can assist in the diagnosis because pain is usually absent in a full thickness injury. Fluid losses and the metabolic effects of deep dermal burns are basically the same as that seen with the third degree burn.

Outcome: A deep dermal burn will require 4-10 weeks or longer to heal. Since the new epidermis is very thin and not adhered well to dermis (no rete pegs), wound breakdown is common. Excision and grafting is the preferred treatment. Dense scarring is usually seen if the wound is allowed to heal primarily.

Treatment: After initial cleaning and removal of dirt and loose dead tissue, a topical antibiotic is required. A silver based dressing or cream is the first choice.

MID TO DEEP DERMAL BURN



TREATMENT

- 1) Transfer to Burn Center due to size, i.e. > 15% TBS
 - 2) Too big for cold dressings (avoid hypothermia)
 - 3) Gently clean, Debride loose tissue
 - 4) Mid dermal areas, use grease gauze, antibiotic ointment. Deeper areas, especially arm, use Silver sulfadiazine or silver dressing
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TREATMENT

- 1) Gently clean with mild soap
- 2) Apply topical silver based antibiotic cream or dressing
- 3) Close using a compression dressing
- 4) Perineum can also be treated open
- 5) Meets criteria for Burn Center due to high risk location

Mid to Deep Dermal Burn



TREATMENT

- 1) Consider admission because of area involved (hand)
- 2) Use topical antibiotics daily
- 3) Closed dressing which allows function
- 4) May require grafting

D) THIRD DEGREE (Full Thickness Burn)

A third degree or full thickness burn is at high risk for infection due to the presence of dead tissues and lack of blood flow. Surgical excision and grafting will be needed. Initial use of a silver dressing or cream is required. Do not apply any agent if patient is to be immediately transferred to a burn center.

Definition: A full thickness or third degree burn occurs with destruction of the entire epidermis and dermis, leaving no residual epidermal cells to repopulate. This wound will therefore not re-epithelialize and whatever area of the wound is not closed by wound contraction will require skin grafting.

Appearance: A characteristic initial appearance of the avascular burn tissue is a waxy white color. If the burn produces char or extends into the fat as with prolonged contact with a flame source, a leathery brown or black appearance can be seen along with surface coagulation veins. Direct exposure with a flame is the usual cause of a third degree burn. However, contact with hot liquids such as hot grease, tar or caustic chemicals will also produce a full thickness burn. The burn wound is also painless and has a coarse non-pliable texture to touch. A major difficulty is distinguishing a deep dermal from a full thickness (third degree) burn that extends just through the dermis. This burn is termed an indeterminate burn.

Outcome: Except for a very small wound, e.g. 2x2 inches, the burn wound will require excision and a skin graft.

Treatment:

1. Gentle wash, removing loose tissue, char.
2. Eschar penetrating antibiotics, silver cream or dressing first choice, using closed dressing.
3. Early surgical excision and grafting.
4. Prophylactic parenteral antibiotics are not indicated.

Deep Burn to Knee



TREATMENT

- 1) Clean. Apply topical antibiotic
- 2) Dry dressing applied with some compression
- 3) Plan early excision and grafting

Deep Burn to Legs



TREATMENT (37)

- 1) Transfer to burn center due to size and depth
- 2) Gently cleanse and debride
- 3) Apply SSD (in view of depth) or silver dressing, plus outer gauze dressing (closed)
- 4) Early excision and grafting

Note the white patches are the deep burn surrounded by a mid-dermal injury.

**Full thickness burn to forearm
with surrounding deep dermal burn
(38)**



- 1) Consider transfer to burn center because of hand burn
- 2) Assess distal perfusion if circumferential
- 3) Gentle cleansing
- 4) Apply silver cream or dressing

E) Chemical Burns

Common strong acids and alkali used in industry cause the majority of injuries. Other less common agents are described in the table. The burn injury is typically caused by coagulation necrosis of tissue rather than by direct heat production. The degree of tissue injury is dependent on the toxicity of the chemical and the exposure time. The burn wound is characteristically gray to brown in color due to the chemically denatured protein. Persistent burning pain is commonly described as the burning in process continuous as long as the chemical is in contact with the skin. Burns are invariably deeper than first appearance indicating ongoing injury. Also the degree of tissue damage takes longer to declare itself such that after 13 to 24 hours the wound is invariably deeper. The specific nature of the chemical injury, its characteristics, diagnosis and treatment are described.

(39)



**Note brownish, gray appearance characteristic
of coagulation necrosis. Wounds usually deeper
in first 24 hours**

Wound Management

- 1) Initial management of the chemical burn has a major impact on outcome
- 2) Continuous water irrigation of the area should be initiated]
 - use of showers in the workplace is optimum
 - use tepid water if possible, to avoid long exposure to cold or hot water
 - irrigation for strong acid or alkali exposure is 30-60 minutes
 - continuous irrigation if eye is exposed to chemicals
 - do not attempt to neutralize acids with alkali or vice versa, just use copious water
- 3) Solid chemicals should be brushed off first prior to irrigation using safety gloves
- 4) Consider transfer
- 5) Will need topical antibiotic as burn is invariably deep