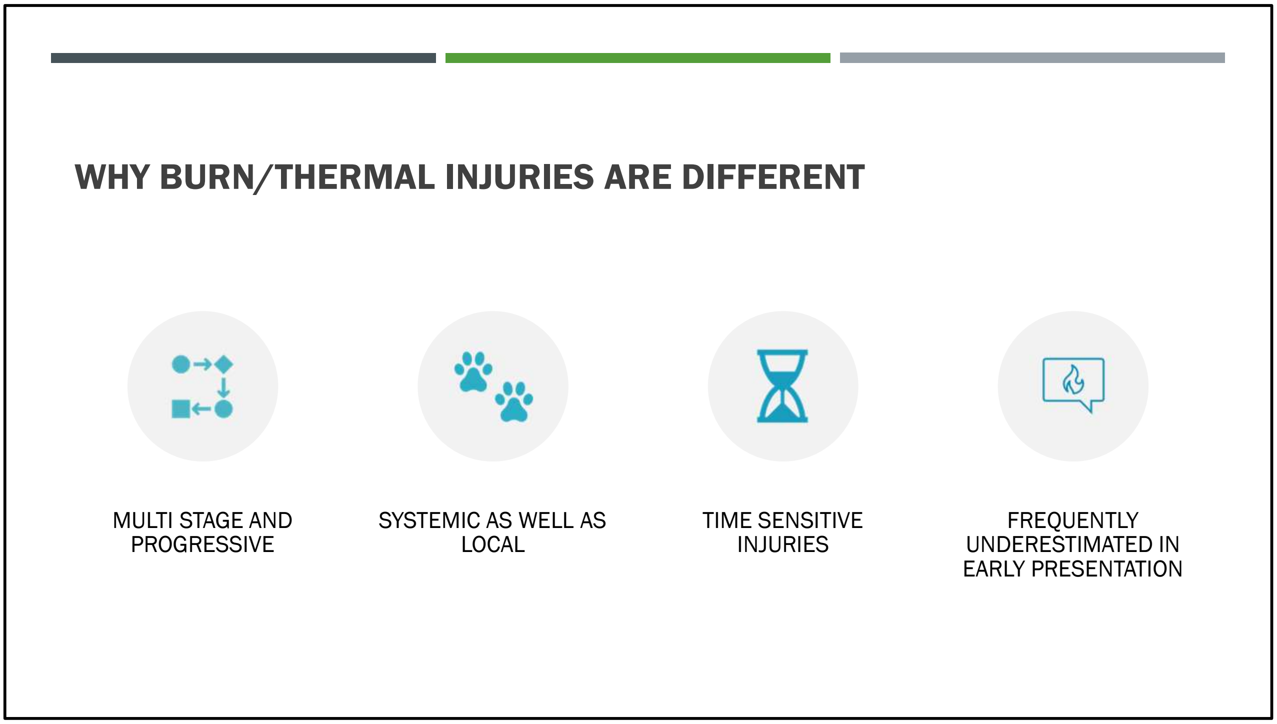
Burn injuries represent a unique and complex form of trauma that extend far beyond the skin. Unlike traditional wounds, burns initiate a dynamic and evolving process that impacts multiple organ systems. The focus of this lecture is to bridge the gap between local wound care and systemic critical care, emphasizing how early intervention, stabilization, and targeted therapies directly influence patient outcomes.

PRESENTATION OBJECTIVES

Differentiate	Understand	Apply	Implement	Evaluate
Differentiate burns from traumatic wounds	Understand burn pathophysiology and progression	Apply burn-specific stabilization and critical care principles	Implement infection prevention and wound management strategies	Evaluate advanced therapies and clinical decision-making

Burn care is not just about the skin. These patients require full critical care management including fluid resuscitation, perfusion support, pain control, and metabolic management. The success of the wound is directly tied to how well. The goal of this presentation is to build a comprehensive understanding of burn injury as both a local and systemic disease process. We will differentiate burns from traditional traumatic wounds, review the underlying pathophysiology that drives progression, and apply burn-specific stabilization strategies. A strong emphasis will be placed on infection prevention, particularly within the first 72 hours, as well as multimodal pain management and advanced wound care techniques. Ultimately, the objective is to integrate clinical reasoning with practical application to improve patient outcomes. the entire patient is stabilized. Integrating systemic stabilization with local wound management optimizes outcomes



This is not a “treat the wound first” scenario. If perfusion is not restored, the wound will worsen regardless of what you put on it.

Burn injuries are fundamentally different from other forms of trauma due to their dynamic nature. The full extent of tissue damage is often not immediately apparent and can worsen significantly over the first 24 to 72 hours through a process known as burn wound conversion. While these wounds are initially sterile, they rapidly become colonized due to loss of the skin barrier and immune dysfunction. Additionally, burns trigger a systemic inflammatory response that affects cardiovascular, respiratory, and metabolic function. Successful management requires simultaneous attention to systemic stabilization and local wound care, as inadequate perfusion or oxygen delivery will directly contribute to progression of tissue injury.

Burn injuries are:

Multi stage and progressive

Systemic as well as local

Time sensitive injuries

Frequently underestimated in early presentation

Key risk:

→ clinical progression continues after the initial insult

Burn injuries differ fundamentally from standard traumatic wounds in both their pathophysiology and clinical progression. While standard wounds typically have a defined

depth at the time of injury and follow a predictable healing trajectory, burn wounds are dynamic and may continue to evolve over the first 24 to 72 hours following injury.
This progression is primarily due to ongoing microvascular damage, capillary leak, and tissue ischemia within the surrounding zone of injury. As a result, the apparent depth of a burn at initial presentation may underestimate the eventual extent of tissue damage.
In addition, burn wounds differ from standard wounds in their systemic impact. Whereas most traumatic wounds remain localized unless complicated by infection, moderate to severe burn injuries can result in systemic inflammatory response, hypovolemia due to plasma loss, and metabolic dysregulation.
Another key distinction is infection risk. Standard wounds are typically contaminated at the time of injury, whereas burn wounds are often initially sterile but become increasingly susceptible to infection over time due to impaired immune function and loss of the protective skin barrier.
Finally, burn injuries are associated with a significant hypermetabolic response that increases energy expenditure and contributes to protein catabolism, further differentiating them from standard wound healing processes.

The Core Problem in Burn Injury: Unlike standard wounds, burns are not a static injury. They evolve in depth, systemic impact, and metabolic demand, which is why they require a fundamentally different treatment strategy
They evolve through:
Progressive tissue destruction (24–72 hours)
Expanding zones of injury
Systemic inflammatory response
Metabolic and immune dysfunction
Key concept:
→ what you see initially is not the final injury

THERMAL INJURY WOUND VS TRADITIONAL WOUND

Feature	Traumatic Wounds	Burn Wounds
Injury Pattern	Immediate, fixed at time of injury	Progressive over 24–72 hours
Depth	Defined at presentation	Evolves and may deepen
Primary Mechanism	Mechanical disruption	Thermal/chemical cellular injury + microvascular damage
Tissue Perfusion	Usually preserved at margins	Zone of stasis with evolving ischemia
Fluid Loss	Whole blood (if present)	Plasma loss from capillary leak
Inflammation	Localized and proportional	Exaggerated and systemic (SIRS)
Systemic Impact	Rare	Common in moderate to severe burns
Immune Function	Generally intact	Immunosuppressed over time
Infection Pattern	Contaminated at onset	Initially sterile → colonization → infection
Pain Profile	Proportional to injury	Severe early (partial thickness), minimal in full thickness
Unique Features	No eschar formation	Eschar, burn wound conversion, hypermetabolism
Healing	Predictable phases	Delayed, dysregulated, prolonged inflammation
Treatment Approach	Local wound care ± antibiotics	ICU-level care, fluid therapy, multimodal management

Lets compare !
While standard wounds typically have a defined depth at the time of injury and follow a predictable healing trajectory, burn wounds are dynamic and may continue to evolve over the first 24 to 72 hours following injury. This progression is primarily due to ongoing microvascular damage, capillary leak, and tissue ischemia within the surrounding zone of injury. As a result, the apparent depth of a burn at initial presentation may underestimate the eventual extent of tissue damage.

In addition, burn wounds differ from standard wounds in their systemic impact. Whereas most traumatic wounds remain localized unless complicated by infection, moderate to severe burn injuries can result in systemic inflammatory response, hypovolemia due to plasma loss, and metabolic dysregulation.

Standard wounds, fluid loss is typically related to hemorrhage when present, and systemic effects are generally limited unless secondary infection develops. In contrast, burn injuries are characterized by substantial plasma loss secondary to increased capillary permeability, which can contribute to hypovolemic shock even in the absence of external blood loss.

Another key distinction is infection risk. Standard wounds are typically contaminated at the time of injury, whereas burn wounds are often initially sterile but become increasingly susceptible to infection over time due to impaired immune function and loss of the protective skin barrier. Infection patterns also differ. Standard wounds are contaminated at the time of injury but are often manageable with routine wound care and, when necessary, antimicrobial therapy. Burn wounds, however, become increasingly susceptible to infection over time due to immune suppression, decreased neutrophil function, and the loss of the skin barrier, making sepsis a major risk factor in severe cases.

TYPES OF THERMAL INJURY

Burn Type	Common Causes	Typical Locations / Presentation	Key Clinical Considerations
Thermal – Wet (Scalds)	Hot water, steam, grease, bathing accidents	Ventral abdomen, limbs, dorsum depending on spill pattern	Often **more diffuse**, can be underestimated; rapid heat transfer → deeper injury
Thermal – Dry (Contact/Flame)	Fire, hot surfaces (stoves, mufflers, heating pads)	Paw pads, lateral body, focal contact points	Often **well-demarcated**, may have deeper focal injury
Chemical Burns	Acids, alkalis (lye), cleaners, gasoline	Any exposed area, often paws, face, oral cavity	**Continues to damage tissue until removed**; requires immediate, copious lavage
Electrical Burns	Chewing cords, electrical exposure	Oral cavity, lips, tongue; possible entry/exit wounds	Can cause **severe internal injury**, arrhythmias, pulmonary edema
Mechanical / Friction Burns	Rope, carpet, restraint injuries	Limbs, digits, pressure points	Often superficial to partial thickness but **painful and contaminated**
Radiation Burns	Sun exposure, radiation therapy	Non-pigmented skin (ears, nose, abdomen)	May be **delayed onset**, cumulative damage
Cold Injury (Frostbite)	Prolonged cold exposure	Extremities: ears, tail, paws	Tissue ischemia → necrosis; **rewarming injury possible**

Burn injuries can result from a variety of mechanisms, each with unique clinical considerations. Thermal burns are the most common and may be caused by direct flame, hot liquids, or contact with heated surfaces. Chemical burns result from exposure to corrosive substances and may continue to cause damage until neutralized.

Electrical burns often cause severe internal injury that is not immediately visible on the surface. Friction burns occur from mechanical abrasion, while radiation burns may result from sun exposure or therapeutic treatments.

Cold injuries, such as frostbite, represent the opposite end of the spectrum but similarly result in tissue necrosis and vascular compromise.

Burn injuries in veterinary patients can result from several different mechanisms, and the source of the burn often influences both the severity and the complications we need to anticipate.

Thermal burns are the most common and include scalds, flames, and contact injuries. Scald burns are particularly important in veterinary patients because liquid can penetrate the fur and remain in contact with the skin longer, often resulting in deeper injuries than initially appreciated. Contact burns, such as those from heating pads or hot surfaces, are often more localized but can be full thickness.

Flame burns, such as those seen in house fires, are especially concerning because they are frequently associated with inhalation injury. Even if the cutaneous burns appear mild, these patients may develop airway edema, carbon monoxide toxicity, or chemical pneumonitis.

These cases should always raise concern for respiratory compromise early in presentation.

Electrical burns most commonly affect the oral cavity in young animals that chew on cords. These injuries can appear deceptively mild externally but often involve deeper tissue damage. A key complication to be aware of is delayed hemorrhage, typically occurring several days after injury when eschar sloughs from underlying vessels. These patients also require close monitoring for airway swelling and often need aggressive pain management and nutritional support.

Chemical burns result from exposure to caustic substances and can continue to cause tissue damage until the agent is fully removed. Immediate and thorough decontamination is critical in these cases, and these burns may penetrate deeper than expected depending on the chemical involved.

Frostbite, or cold injury, represents the opposite end of the spectrum but results in similar tissue necrosis. These injuries often affect distal extremities and may initially appear mild before progressing as perfusion is compromised. Rewarming must be controlled, as rapid or improper warming can worsen tissue damage.

Overall, the key concept is that the **type of burn provides important insight into both the expected progression of injury and the complications we need to monitor for**, even before we fully assess depth and total body surface area.

Types of Thermal Injury

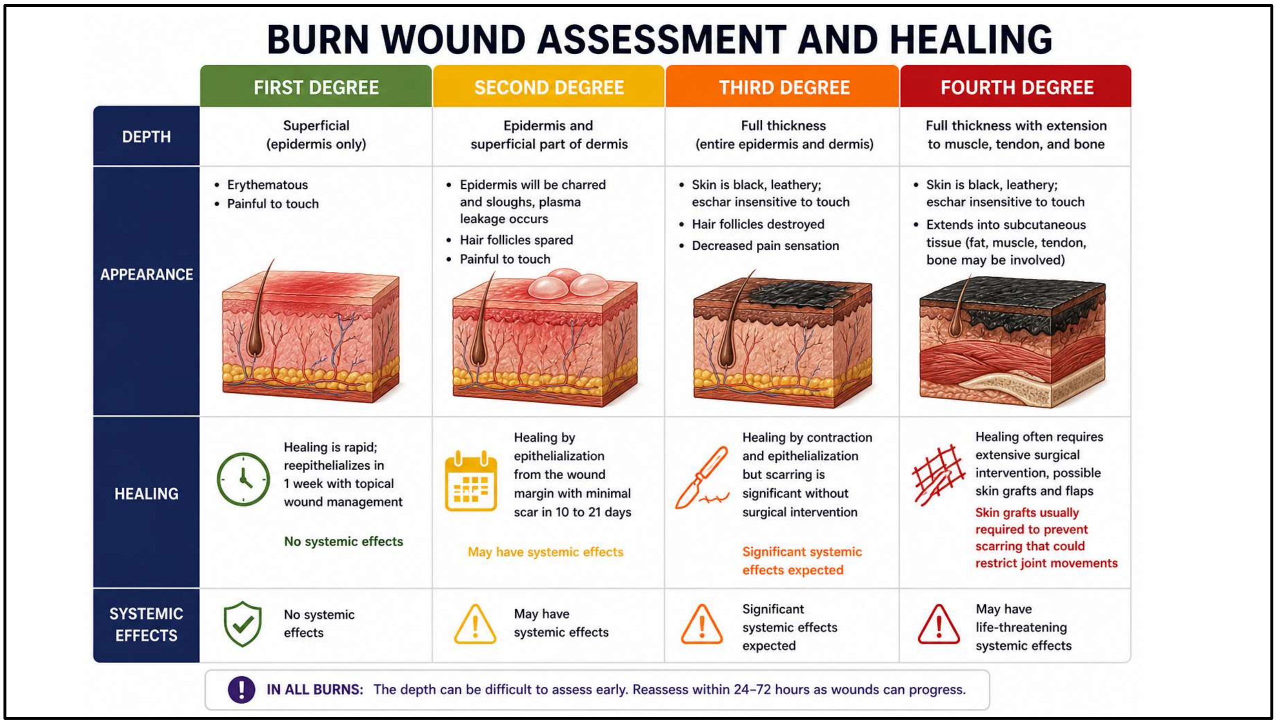
Scald: Most common; liquids "creep" under the coat.

Flame: High risk for deep penetration and inhalation trauma.

Contact: e.g., heating pads; often full-thickness but localized.

Chemical/Electrical: Deep tissue destruction often underestimated externally.

Burn injuries in veterinary patients can result from a variety of thermal, chemical, electrical, and environmental sources, each with distinct mechanisms of injury and clinical implications.



Burn depth classification is critical for determining prognosis and guiding treatment. Superficial burns involve only the epidermis and are typically painful due to intact nerve endings. Partial-thickness burns extend into the dermis and are often characterized by blistering and significant pain. Full-thickness burns destroy all dermal structures, resulting in a dry, leathery appearance and reduced sensation due to nerve damage. Fourth-degree burns extend into deeper tissues such as muscle and bone and are associated with severe functional impairment. It is important to recognize that burn depth may evolve over time, particularly in the zone of stasis.

Burn Depth Classification
Superficial (1st): Epidermis only; painful, red, dry.
Partial Thickness (2nd): Dermis involved; blisters, extreme pain.
Full Thickness (3rd): Entire dermis destroyed; leathery, "Eschar," **painless** (nerves destroyed).
Fourth Degree: Muscle and bone involvement.
First Degree Burns (Superficial)
First degree burns involve only the epidermis. Clinically, these present as erythema, warmth, and mild edema without loss of skin integrity. The skin surface remains intact and there is no blistering.
Pain is typically **mild to moderate** due to inflammatory stimulation of intact superficial

nerve endings. Discomfort is usually transient and improves as inflammation resolves. These injuries generally heal without complications or scarring.

Second Degree Burns (Partial Thickness)

Superficial second degree burns involve the epidermis and superficial dermis. These lesions are characterized by erythema, moisture, blister formation, and significant pain due to exposed dermal nerve endings. Capillary refill may be increased in the affected area. Healing potential is good with appropriate wound care, typically within 2 to 3 weeks.

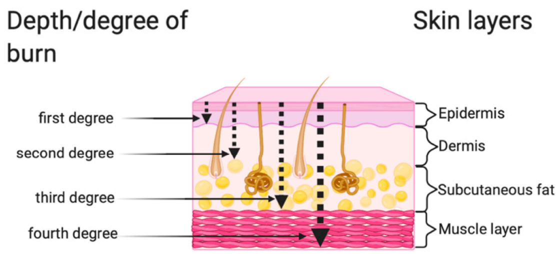
Deep second degree burns extend deeper into the dermis with partial destruction of dermal structures. Tissue may appear pale, mottled, or waxy, and sensation may be decreased compared to superficial partial thickness burns. Pain can be variable due to partial nerve damage. These wounds have delayed healing, higher risk of infection, and may require surgical intervention depending on progression.

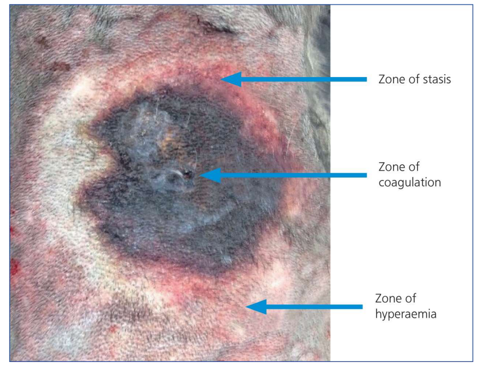
Third Degree Burns (Full Thickness)

Third degree burns involve complete destruction of the epidermis and dermis, often extending into subcutaneous tissue. The tissue is typically dry, leathery, or charred, and may appear white, gray, or black.

These wounds are generally **insensate** due to complete nerve destruction. They do not heal by re-epithelialization and require surgical management such as debridement and skin grafting. Systemic complications, including fluid loss and infection risk, are significant concerns.

Burn depth is often underestimated in the initial 24 to 48 hours. Progressive tissue necrosis can result in apparent worsening of burn classification. Serial reassessment is essential for accurate staging and treatment planning.





JACKSON MODEL

Jackson's model describes three concentric zones of injury within a burn wound. The central zone of coagulation represents irreversible tissue necrosis due to direct thermal damage. Surrounding this is the zone of stasis, which is characterized by decreased perfusion and is at high risk for progression to necrosis if not properly managed. The outermost zone of hyperemia contains viable tissue with increased blood flow and is expected to recover. The primary focus of early burn management is to preserve the zone of stasis by optimizing perfusion and minimizing secondary injury. **Zones of Injury: The Jackson Model**

Zone of Coagulation: Central irreversible necrosis.

Zone of Stasis: The **Target Zone**. Ischemic but salvageable with aggressive care.

Zone of Hyperemia: Outer ring; viable inflamed tissue.

Jackson's burn wound model is a foundational concept in thermal injury pathophysiology and is especially important in veterinary patients where burn depth is not static in the acute phase.

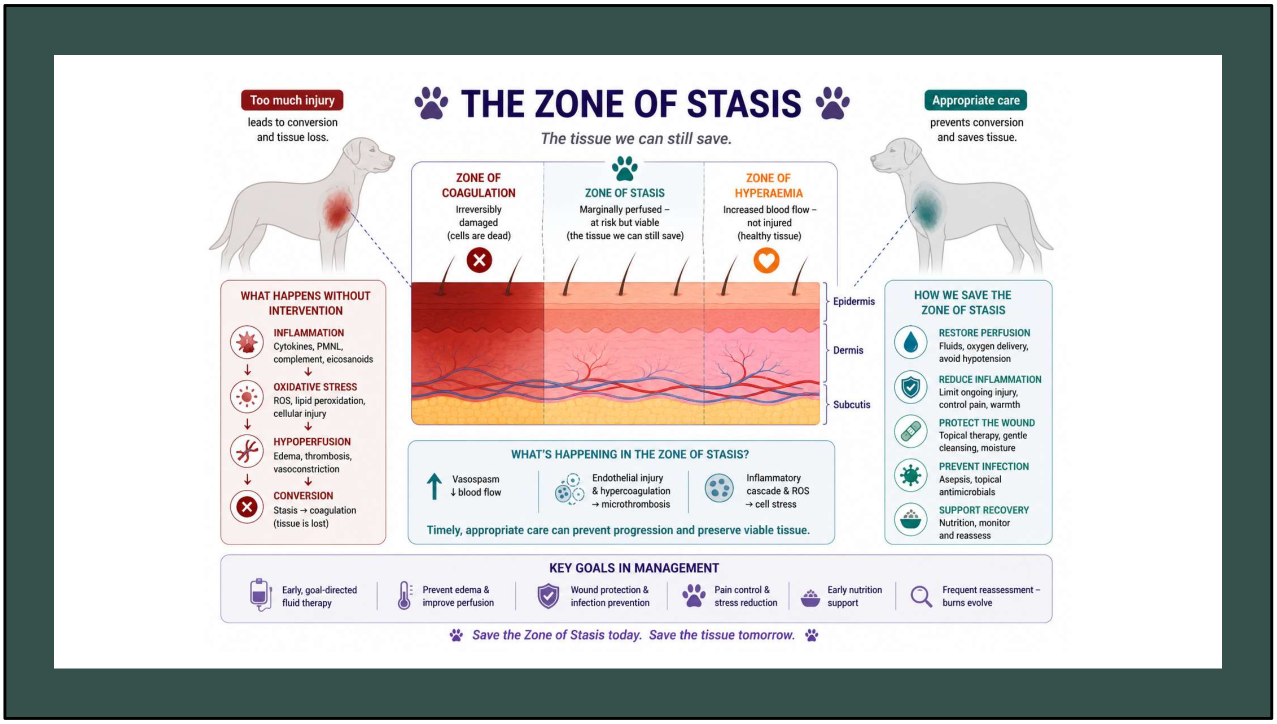
The model describes three distinct but interrelated zones of tissue injury surrounding the point of thermal contact.

The innermost region is the **zone of coagulation**. This represents the area of maximal injury where irreversible cellular death has occurred at the time of insult. Protein denaturation and coagulative necrosis are present, and tissue in this zone is nonviable. This area corresponds most closely to full thickness injury.

Surrounding this is the **zone of stasis**, which is critically important in clinical management. Tissue here is initially viable but severely compromised. Perfusion is reduced, capillary integrity is disrupted, and microvascular thrombosis may develop. This zone is potentially salvageable but highly vulnerable to secondary injury such as hypovolemia, hypotension, infection, or inappropriate fluid resuscitation. With proper perfusion support and wound management, this zone may recover, but if compromised, it will progress to necrosis and effectively deepen the burn.

The outermost region is the **zone of hyperemia**, where tissue perfusion is increased due to inflammatory vasodilation. Cellular injury is minimal and this zone typically recovers fully without intervention unless secondary insults occur.

In veterinary ECC patients, understanding this model is essential because burn wounds evolve. What appears as partial thickness injury on presentation may progress in depth as the zone of stasis deteriorates over the first 24 to 72 hours. This is why aggressive early stabilization, perfusion support, and infection control are critical to limiting burn progression.



Mechanism of the Zone of Stasis

The zone of stasis is the most clinically important area in a burn wound. Unlike the zone of coagulation, which is already irreversibly damaged, the zone of stasis contains tissue that is ischemic but still viable. However, this tissue is highly vulnerable and can either recover or progress to necrosis depending on how it is managed.

Microvascular thrombosis, increased interstitial pressure from edema, and ongoing inflammation all contribute to reduced perfusion in this zone. Without intervention, this leads to tissue hypoxia and eventual cell death.

This process occurs over the first 24 to 72 hours, making this time period critical for intervention.

Every major component of burn management is aimed at preserving this tissue. Adequate fluid resuscitation restores perfusion, oxygen delivery supports cellular metabolism, and gentle wound care minimizes further injury.

Clinically, the zone of stasis determines the final depth and severity of the burn, which is why early management decisions have such a profound impact on outcome.

Heat-damaged proteins → Reduced erythrocyte deformability.

Microvascular thrombosis and increased interstitial pressure.

Key Concept: The management of this zone determines the final wound size.

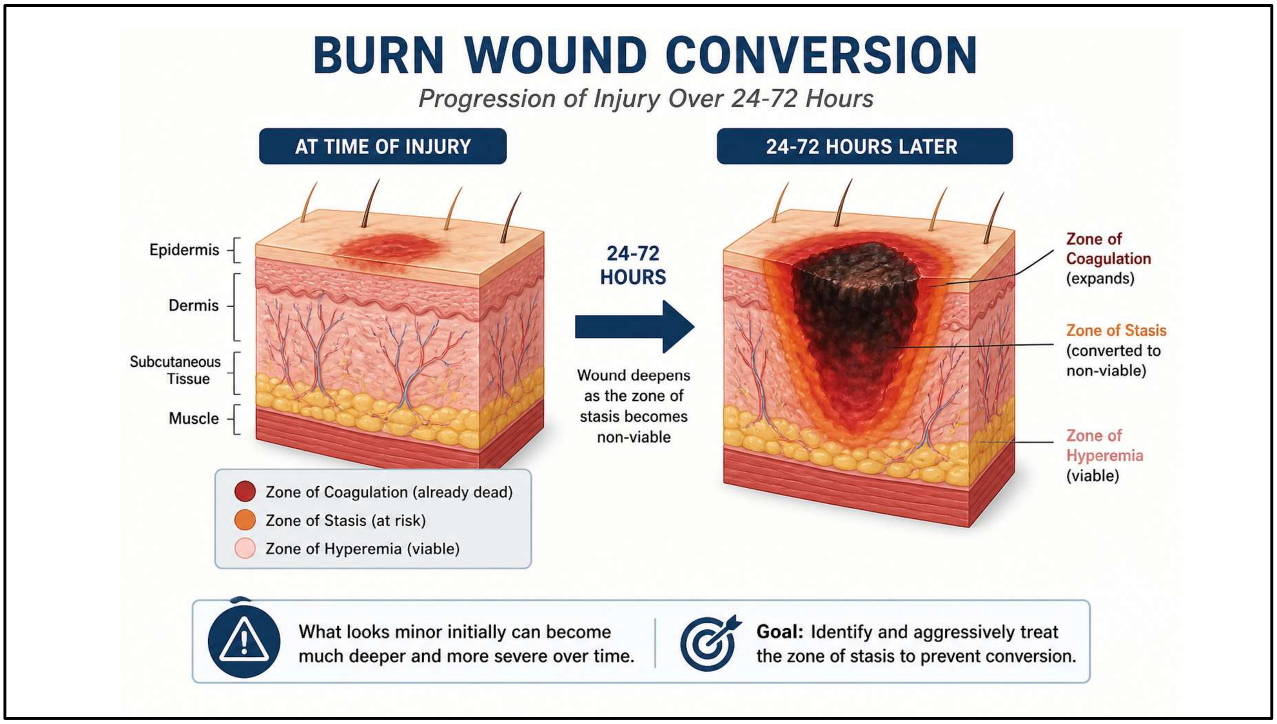
The zone of stasis represents the intermediate area of injury surrounding the central zone of coagulation in a burn wound. While tissue in this region is initially viable, it exists in a state of significant physiologic instability and is highly susceptible to progression toward irreversible injury.

At the cellular level, heat damage results in protein denaturation and structural compromise of endothelial cells and surrounding tissues. This contributes to reduced erythrocyte deformability, which impairs microcirculatory flow and further compromises oxygen delivery to already stressed tissues.

As endothelial injury progresses, the coagulation cascade is activated locally, leading to microvascular thrombosis. This, combined with increased capillary permeability, results in fluid extravasation into the interstitial space. The subsequent rise in interstitial pressure further compresses capillaries, creating a cycle of worsening hypoperfusion and tissue ischemia.

Clinically, the zone of stasis is dynamic and potentially salvageable. However, it is extremely sensitive to secondary insults such as hypotension, hypovolemia, hypothermia, infection, or excessive vasoconstriction. Any of these factors can convert potentially viable tissue into necrosis, effectively deepening the burn over time.

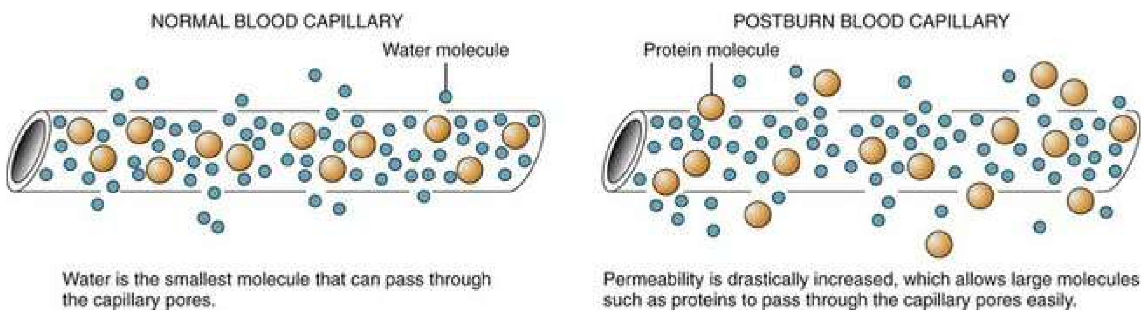
A key clinical concept is that the ultimate size and severity of the burn wound is not determined solely at the time of injury, but rather by what happens to the zone of stasis in the hours to days following the insult. Preservation of perfusion, prevention of microvascular thrombosis, and meticulous supportive care are central to limiting burn progression in this region.



Burn Wound Conversion

Burn wound conversion refers to the progression of initially viable tissue into necrotic tissue over time. This process is driven by microvascular thrombosis, increased interstitial pressure from edema, and ongoing inflammation. Reduced blood flow leads to tissue hypoxia, while the accumulation of reactive oxygen species and inflammatory mediators further exacerbates cellular damage. Infection and inadequate resuscitation significantly accelerate this process. Preventing burn wound conversion is a key goal during the early management phase.

EARLY LOCAL PATHOPHYSIOLOGY



Early Local Pathophysiology

Burn injury increases vascular permeability, allowing fluid and proteins to leak into the interstitial space. This results in edema, hypovolemia, and decreased tissue perfusion. This process is the foundation of burn shock and contributes to progression of the zone of stasis.

Capillary leak syndrome.

Plasma loss into the interstitial → Progressive edema

After a burn injury, one of the first changes happens in the blood vessels at the site of injury. These vessels become damaged and lose their normal ability to hold fluid inside the bloodstream.

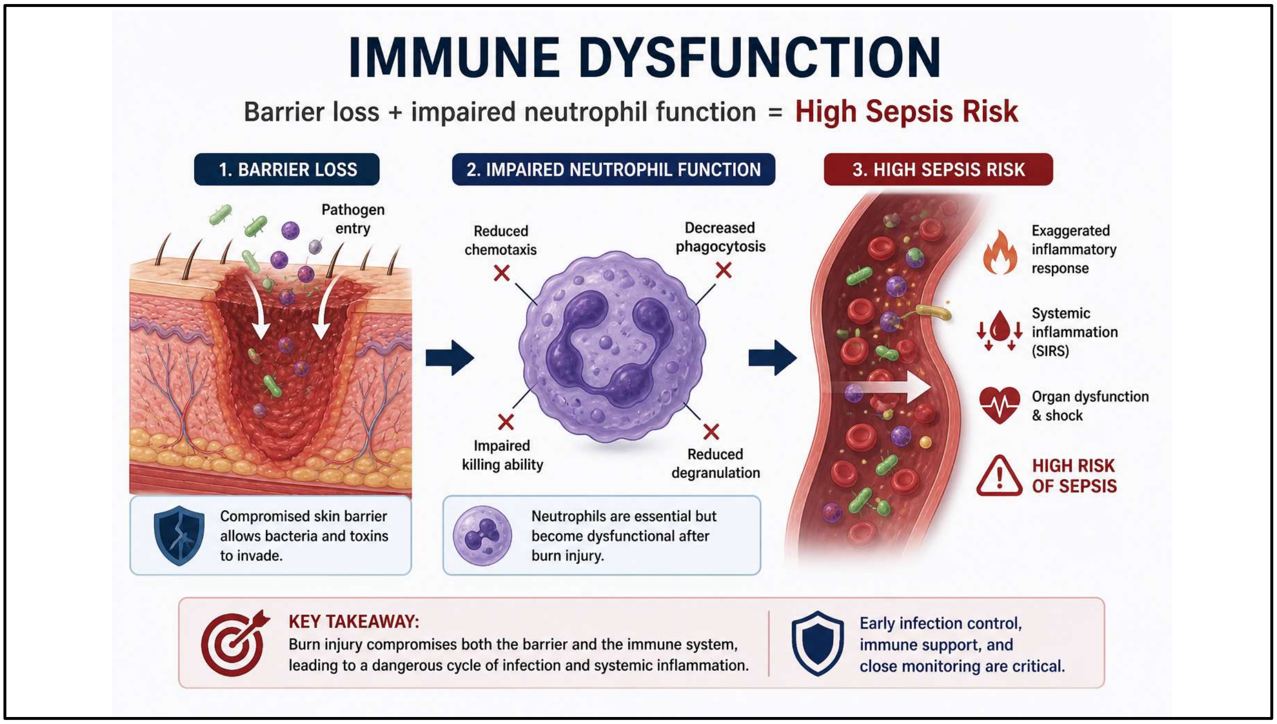
Right after injury, there may be brief vasoconstriction, but this is quickly followed by vasodilation and increasing vessel wall injury from inflammation. The lining of the capillaries (the endothelium) becomes “leaky.”

This leads to capillary leak syndrome, where fluid, proteins, and electrolytes move out of the blood vessels and into the surrounding tissues. This is also called third spacing.

As fluid leaves the vascular space, swelling (edema) develops. This swelling can extend beyond the burn itself because the inflammatory response is systemic, not just local.

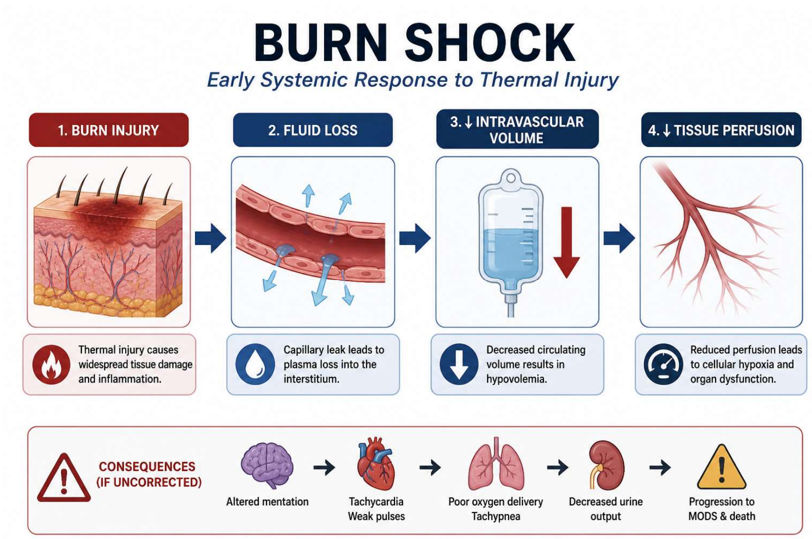
At the same time, the loss of fluid from the bloodstream leads to decreased circulating blood volume, which can contribute to poor perfusion and burn shock in more severe cases. Locally, the swelling increases pressure in the tissues, which further reduces blood

flow and can worsen tissue injury.
These changes are most significant in the first 12 hours and can continue for up to 24 to 36 hours after the burn.
The image showing intact versus damaged capillaries helps illustrate this process: a normal vessel keeps fluid inside, while a burn-injured vessel allows fluid to leak out into the tissues.



Immune Dysfunction
Barrier loss + impaired neutrophil function = High Sepsis Risk.
Burn patients experience both inflammation and immune suppression. Loss of the skin barrier and impaired neutrophil function increase susceptibility to infection. This creates a high risk for sepsis, particularly as the wound becomes colonized.

SYSTEMIC IMPACT (THE CRISIS)
PHASE I – BURN SHOCK (THE FIRST 36 HOURS)



Systemic Impact (The Crisis)

Burn shock is primarily a hypovolemic state caused by massive plasma loss due to increased capillary permeability rather than hemorrhage. Fluid shifts from the intravascular space into the interstitial space result in decreased circulating volume, reduced cardiac output, and impaired tissue perfusion. Even in the presence of normal blood pressure, patients may be in a compensated shock state. Early and appropriate fluid resuscitation is essential to restore perfusion and prevent progression of tissue injury.

Phase I – Burn Shock (The First 36 Hours)

Hypovolemic crisis from plasma loss (not blood loss).

Decreased cardiac output and tissue hypoperfusion.

Phase I of systemic burn response is commonly referred to as burn shock, and it represents a hypovolemic state that develops from fluid and plasma loss rather than blood loss.

The primary problem is widespread capillary leak caused by endothelial damage and inflammatory mediators. Fluid, electrolytes, and proteins move out of the intravascular space into the interstitium, significantly reducing circulating blood volume.

As intravascular volume decreases, preload to the heart drops, which leads to a reduction in cardiac output. Despite the heart itself being structurally normal, it cannot maintain adequate circulation due to lack of volume.

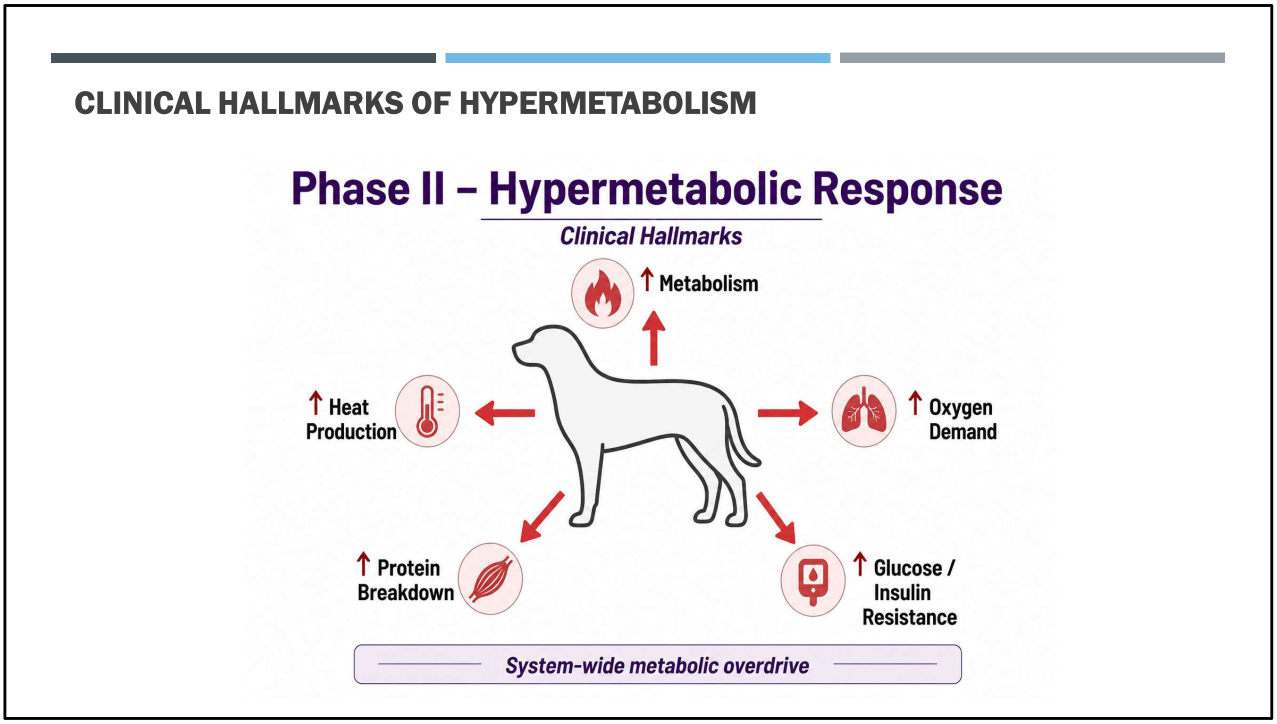
This results in systemic tissue hypoperfusion. Organs and tissues receive less oxygen delivery, leading to cellular hypoxia and the early metabolic consequences of shock.
Clinically, this phase is most critical within the first 24 to 36 hours after injury. Without appropriate fluid resuscitation and monitoring, patients can rapidly deteriorate into multi system dysfunction.

Cardiovascular & Renal Compromise

Myocardial depression from inflammatory mediators.
Acute Renal Failure (ARF) risk due to profound hypotension.

Respiratory & Inhalation Injury

Upper airway obstruction (edema) and chemical pneumonia.
Nursing Note: Intubate early if facial burns are present.

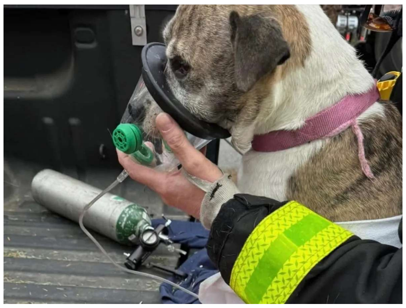


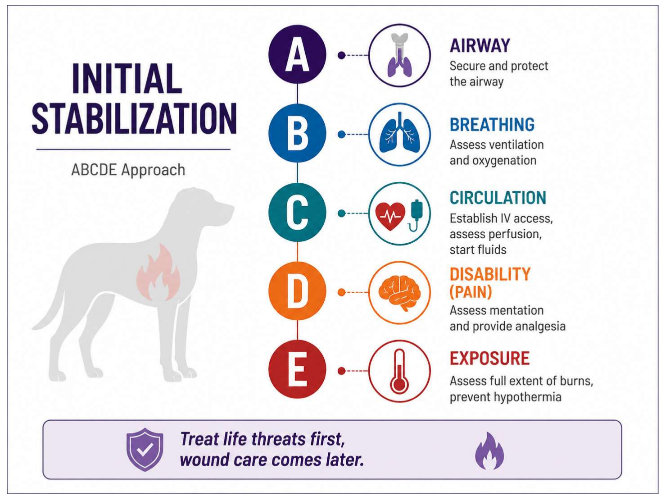
Phase II – The Hypermetabolic Response
Clinical Hallmarks of Hypermetabolism
Following the shock phase, burn patients enter a hypermetabolic state characterized by increased energy demand, protein breakdown, and insulin resistance. Without adequate nutritional support, this leads to muscle wasting and delayed healing.
Insulin resistance and stress hyperglycemia.
Rapid protein catabolism and muscle wasting.

Catecholamine/Cytokine driven surge.
Metabolic rate can increase by **100%+**.
Hypermetabolic Response (CRITICAL SYSTEMIC EFFECT)
Catecholamine driven state
Increased metabolic rate
Protein catabolism and muscle wasting
Insulin resistance
Immune dysfunction
Clinical consequences:
Delayed wound healing
Increased infection risk
High nutritional demand

Clinical Implications of Hypermetabolism
Early enteral nutrition required
High protein support
Glycemic monitoring
Energy demand significantly increased

INITIAL STABILIZATION (ABCDE)

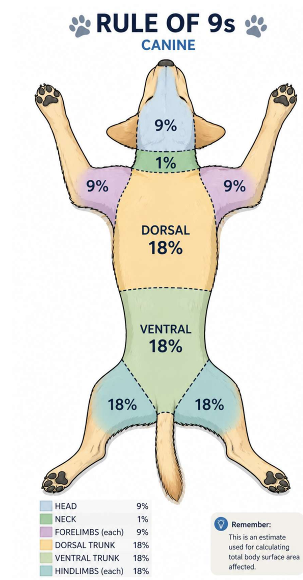




Clinical Management (The "How-To")
Slide 18: Initial Stabilization (ABCDE)
Burn patients are stabilized using the ABCDE approach. Airway management is critical, especially with suspected inhalation injury. Circulation is supported through aggressive fluid therapy. Pain control is essential and should be addressed early. Exposure includes assessing burn extent while preventing hypothermia.

Airway, Breathing, Circulation, Disability (Pain), Exposure (TBSA).
Airway management (inhalation injury consideration) OXYGEN IS CRITICAL with patients experiencing smoke inhalation!! Even if they don't seem dependent ! They need it!
Circulation and vascular access
Fluid resuscitation
Temperature control
Early analgesia

"RULE OF 9S" VETERINARY ADAPTATION



The Rule of 9s is a rapid clinical tool used to estimate the percentage of total body surface area affected by burns. In emergency and critical care, this estimate is essential for guiding early fluid resuscitation, determining prognosis, and anticipating systemic complications.

In veterinary medicine, the classic human Rule of 9s is adapted due to species differences in body conformation. While the human model assigns fixed percentages to anatomical regions such as the head, each upper limb, each lower limb, anterior trunk, posterior trunk, and perineum, this distribution does not directly translate to dogs and cats without adjustment.

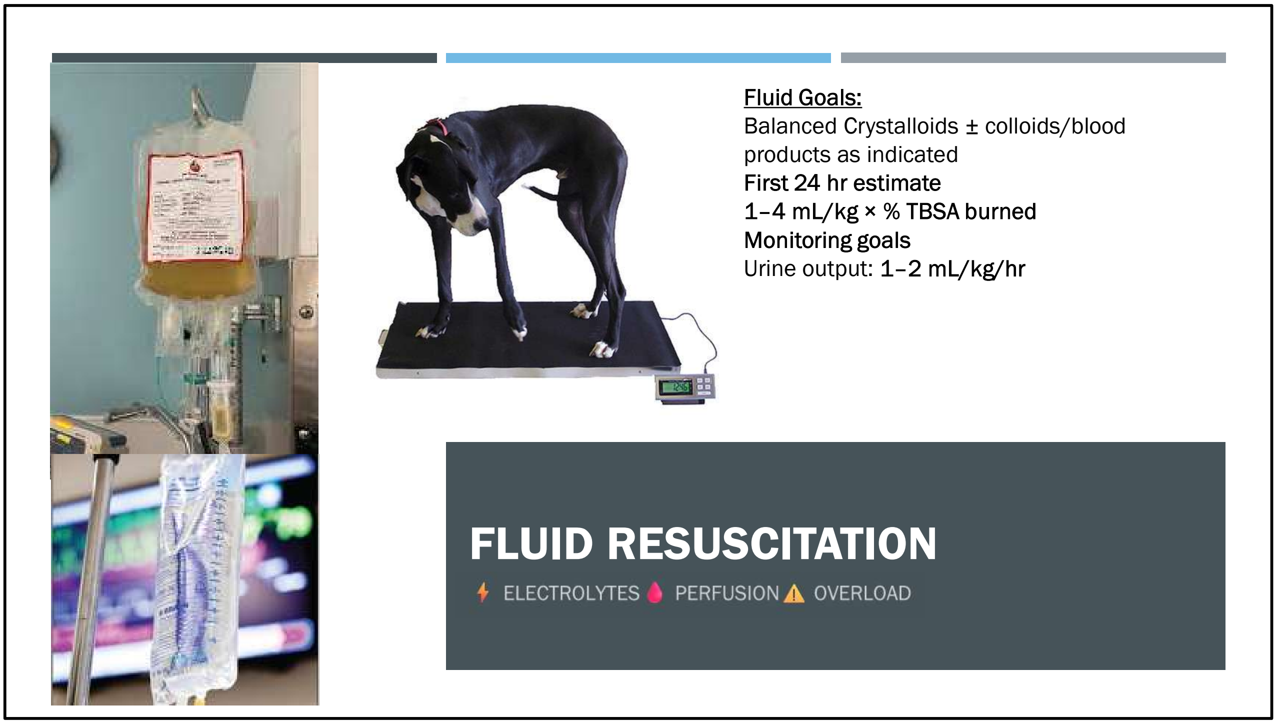
In general veterinary application, the head and neck represent a larger proportion of surface area relative to humans, while limb proportions vary significantly depending on breed conformation, particularly in dogs. Cats and brachycephalic or chondrodystrophic breeds further deviate from standard estimates.

Despite these limitations, the Rule of 9s remains a useful **initial rapid assessment tool in the triage setting**, especially when more precise methods such as the Lund and Browder chart are not immediately available. It is important to recognize that it tends to **overestimate or underestimate TBSA in veterinary patients**, and should always be refined with more detailed assessment once the patient is stabilized.

Clinically, TBSA estimation is most important when burns exceed approximately 10 percent in dogs or 5 percent in cats, as systemic inflammatory response, capillary leak, and fluid shifts become clinically significant at these thresholds.

Accurate TBSA estimation directly impacts fluid resuscitation calculations, monitoring requirements, and the level of intensive care needed in the acute phase of burn management.
stimating total body surface area burned is one of the most clinically important steps early on because it directly influences fluid therapy, monitoring intensity, and prognosis.
In veterinary medicine, we commonly adapt the human Rule of 9s to dogs. While it is not perfect due to breed variation, it is fast, practical, and appropriate for emergency use.
The head and neck account for approximately 9 percent. Each front limb is 9 percent. Each hind limb is larger at about 18 percent. The dorsal and ventral trunk are each 18 percent, and the perineum and tail are about 1 percent.
This gives us a rapid estimate at the bedside without needing advanced tools.
Clinically, once we reach about 20 percent TBSA involvement, the patient should be treated as a systemic burn patient. That means aggressive fluid resuscitation, intensive monitoring, and anticipation of complications like SIRS and organ dysfunction.
For smaller or patchy burns, you can also use the 'palm method,' where the patient's paw pad approximates about 1 percent TBSA, which is helpful for irregular wounds.
The key takeaway is that TBSA is not just documentation. It drives your entire treatment plan."

: TBSA – Clinical Relevance
On Slide:
<10%: Local wound care focus
10–20%: Moderate risk, monitor closely
>20%: Systemic disease (SIRS risk)
>40–50%: Grave prognosis
Small footer:
"TBSA + Depth = Treatment Strategy"
Speaker Notes:
"TBSA does not exist in isolation. It must always be interpreted alongside burn depth.
Small surface area burns can still be devastating if they are full thickness, especially over critical structures.
However, as TBSA increases, the risk shifts from a local wound problem to a systemic inflammatory disease.
Above 20 percent, we anticipate capillary leak, fluid shifts, and immune dysfunction. These patients require ICU-level care.
When burns exceed 40 to 50 percent TBSA, prognosis becomes guarded to poor in veterinary patients, and this is where early, honest client communication is critical.
So again, TBSA is not just a number. It is a decision-making tool."



When managing burn patients, fluid therapy occurs in two distinct phases.
First, if the patient is in hypovolemic shock, we treat shock just like any other critical patient. In dogs, this can be up to 80 to 90 mL per kilogram of Balanced crystalloids, such as Lactated Ringer’s Solution or Plasma-Lyte (preferred for burn resuscitation), and in cats up to 40 to 50 mL per kilogram, NaCl 0.9% is not typically recommended as this can worsen acidemia . This is not given all at once, but rather in incremental boluses with reassessment after each one. The goal here is to restore perfusion, not to complete burn resuscitation.
Once the patient is stabilized, we transition to burn-specific fluid therapy.
The standard starting point is calculating fluid needs using 1 to 4 milliliters per kilogram multiplied by the percent total body surface area burned. It is important to understand that this number represents the total volume of fluids to be given over the first 24 hours, not an hourly rate.
Typically, about half of that calculated volume is administered within the first 8 hours from the time of injury, and the remaining half is given over the following 16 hours.
However, this calculation is only a starting point. Burn patients are dynamic, and their fluid needs can change rapidly due to ongoing capillary leak and fluid shifts. Because of this, fluid therapy must be continuously adjusted based on patient response. WEIGH THESE PATIENTS EVERY 4-6 HOURS for the first 24 hours
Key parameters to monitor include urine output, with a goal of approximately 1 to 2 milliliters per kilogram per hour in dogs and around 1 milliliter per kilogram per hour in

cats, as well as lactate, perfusion parameters, and overall clinical status.
The most important takeaway is that burn fluid calculations provide a framework, but effective resuscitation is guided by ongoing reassessment and the patient's response to therapy.

Fluid Resuscitation

Crystalloids (LRS); Goal-directed (Monitor urine output 1-2ml/kg/hr).
Avoid "over-resuscitation" which worsens tissue edema.

Fluid Resuscitation Goals

Initial shock resuscitation

Crystalloids ± colloids/blood products as indicated

First 24 hr estimate

1–4 mL/kg × % TBSA burned

Monitoring goals

Urine output: **1–2 mL/kg/hr**
Perfusion: HR, pulse quality, MM, CRT, lactate
Watch for overload after **24–48 hr**

Consider oncotic support

Plasma/colloids if hypoproteinemic, coagulopathic, or poor oncotic pressure

Plasma for oncotic support

Dose: **10–20 mL/kg**
Expected albumin increase: often only **~0.3–0.5 g/dL**
Best used when hypoproteinemia is combined with:

- coagulopathy
- ongoing plasma loss
- need for volume/oncotic support

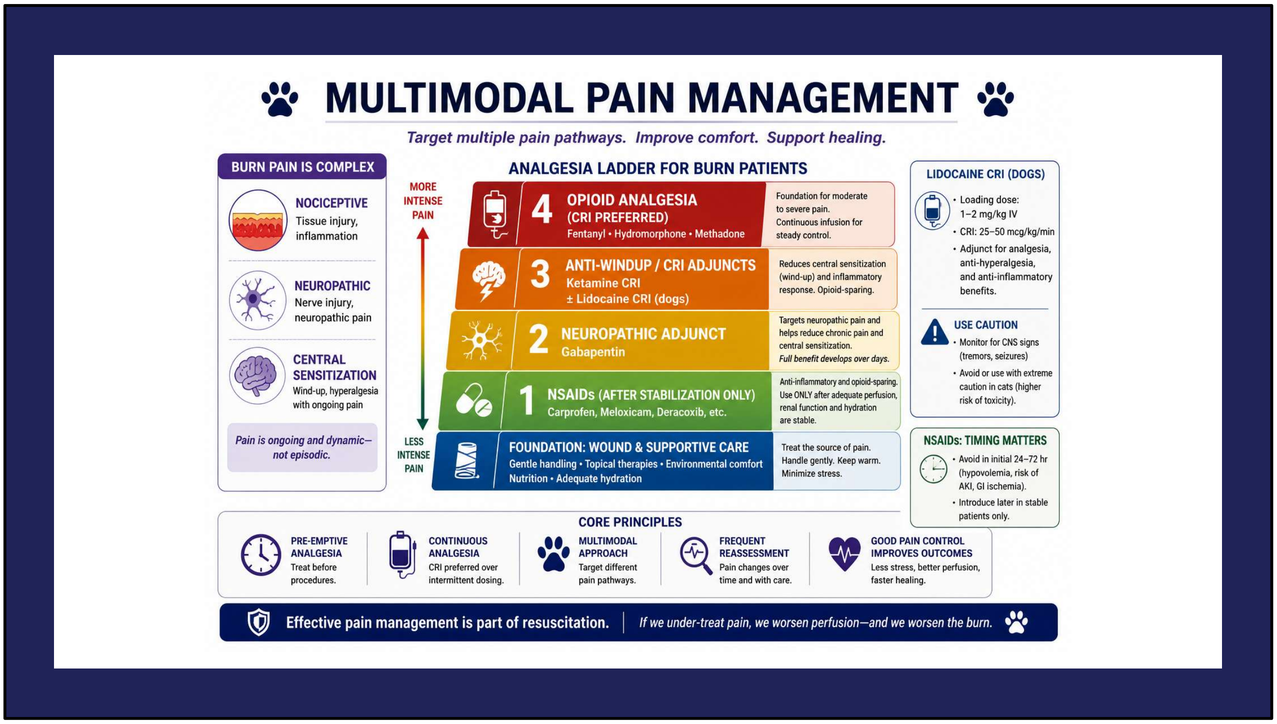
Not an efficient way to "normalize" albumin alone

Speaker note line

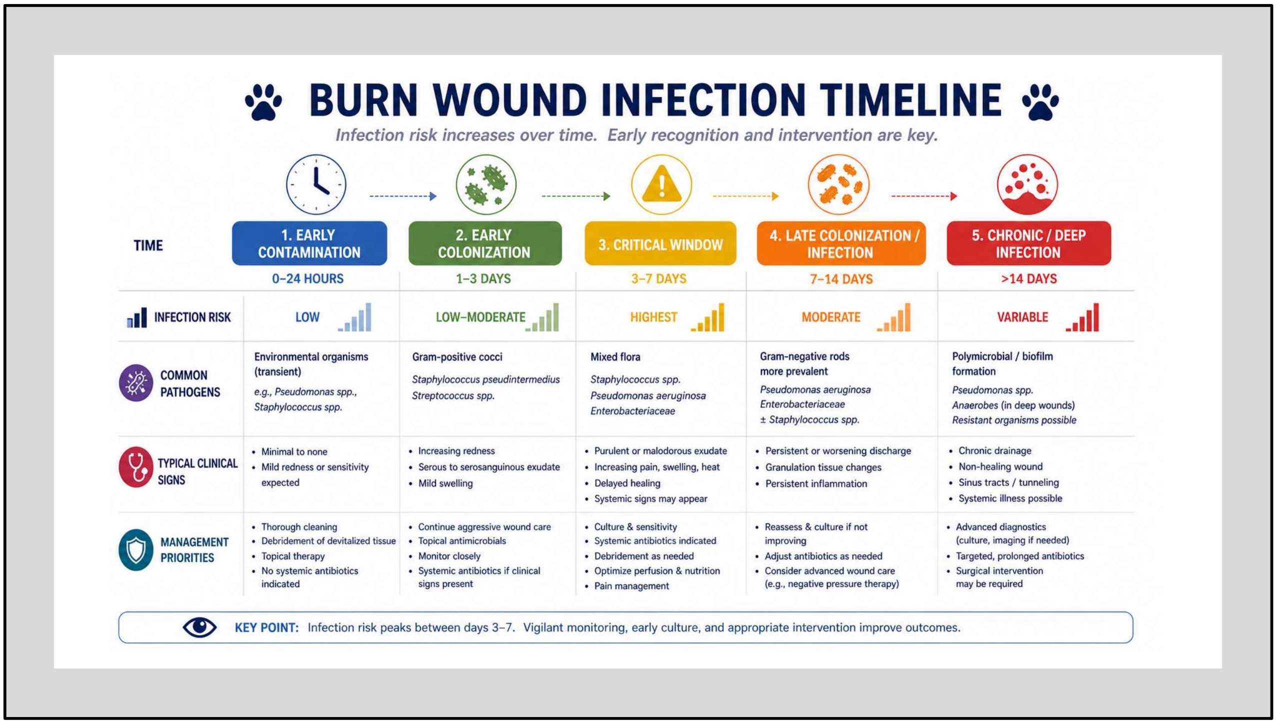
Plasma can support oncotic pressure and coagulation factors, but it takes a large volume to meaningfully increase albumin. In burn patients with ongoing protein loss, plasma may be helpful, but it should not be viewed as a rapid or efficient way to correct hypoalbuminemia by itself.

FLUID AND ELECTROLYTE QUICK REFERENCE

Category	Key Points	Clinical Notes
Fluids	1–4 mL/kg × % TBSA (first 24 hr)	After shock resuscitation (dogs up to ~90 mL/kg, cats ~50 mL/kg)
Urine Output	Dog: 1–2 mL/kg/hrCat: ~1 mL/kg/hr	Oliguria = reassess perfusion
Electrolytes & Acid–Base	**Na^+:** hyper or hypo**K^+:** hyper or hypo**Acidosis:** metabolic ± respiratory	Electrolytes shift over time → monitor frequently
Protein / Oncotic Support	Hypoproteinemia common	**Colloids:** 20–40 mL/kg/day CRI**Plasma / Cryopoor plasma:** 10–20 mL/kg
Albumin Impact	Plasma products have **limited effect**	↑ albumin typically **~0.3–0.5 g/dL** → often transient
Monitoring	Lactate, TS/TP, electrolytes, UOP	Watch for **DIC, SIRS, MODS, fluid overload (after ~48 hr)**



Slide 20: Multimodal Pain Management
CRIs are Gold Standard: Fentanyl/Ketamine.
Pre-emptive Sedation: Required for *every* bandage change.
Avoid: Phenothiazines (Acepromazine).



Burn wounds are initially sterile but become colonized within 24 to 72 hours. Infection risk increases significantly after this period, making early intervention critical.

INFECTION & ANTIMICROBIAL STEWARDSHIP

Protect from contamination

Serial debridement prevents infection

Topicals = primary antimicrobial therapy

Avoid routine systemic antibiotics unless indicated

Treat gram negative empirically until culture-directed therapy can be achieved

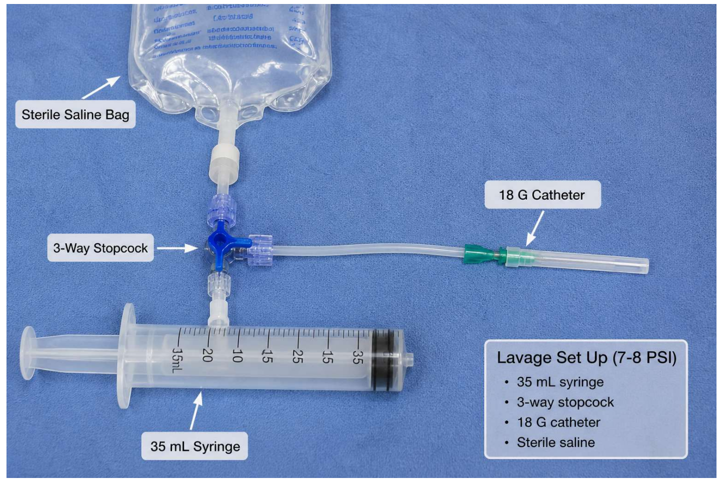
Slide 30: Infection & Antimicrobial Stewardship
Avoid routine systemic antibiotics; use culture-directed therapy.

Sepsis is one of the greatest threats to burn patients, particularly those with large total body surface area involvement. The loss of the skin barrier allows bacteria to colonize and proliferate within the wound.
One of the most important steps in preventing infection is protecting the wound from contamination in the hospital environment. This includes strict aseptic technique, minimizing unnecessary exposure, and maintaining a clean, low-fomite environment during wound care.
Serial debridement plays a critical role in infection prevention by removing necrotic tissue and exudate that support bacterial growth.
Topical antimicrobials are the primary method of infection control in burn wounds because they provide high local concentrations directly at the wound surface, where systemic antibiotics often have limited penetration due to avascular tissue.
Systemic antibiotics are not used routinely and should be reserved for patients with suspected infection, sepsis, pneumonia, or significant immunocompromise.
When infection *is* suspected, empiric antimicrobial therapy should include coverage for gram-negative organisms, particularly *Pseudomonas*, as these are among the most common pathogens associated with invasive burn wound infections. Therapy should then

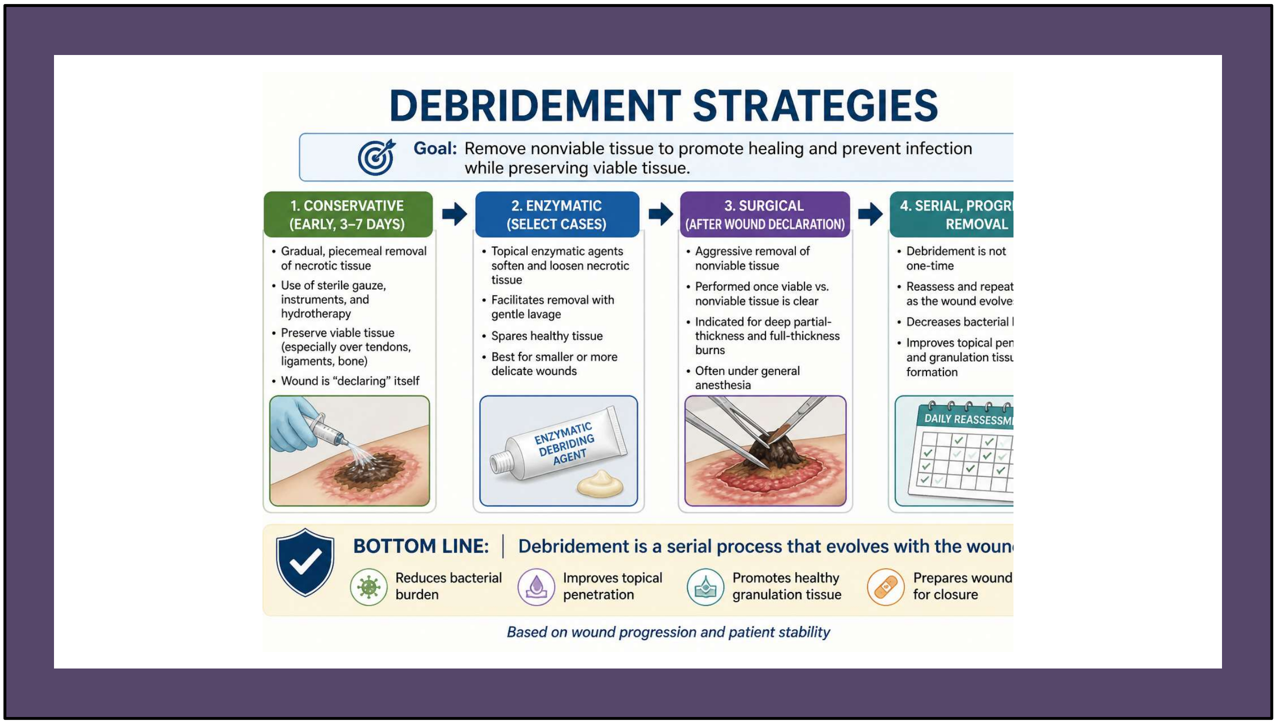
be adjusted based on culture and sensitivity results.
Overall, effective infection control in burn patients relies more on proper wound management and debridement than on routine use of systemic antibiotics

INITIAL WOUND CARE

- Cooling → Clipping → Lavage → Protect



Initial wound care begins only after the patient has been stabilized, as burn management is first and foremost a systemic priority.
If the patient presents within approximately two hours of injury, cooling the affected area with cool water or saline for up to 20 to 30 minutes can help limit the depth of injury by dissipating heat. It is important to avoid ice or excessively cold water, as this can cause vasoconstriction and worsen tissue damage. In patients with large surface area burns, care must also be taken to prevent hypothermia.
Once stable, the wound is gently prepared. The surrounding fur is widely clipped to allow full visualization. If hair pulls easily from the skin, this often indicates a deeper burn injury.
Hydrotherapy is a cornerstone of early wound care. This involves gentle lavage using room temperature sterile saline or lactated Ringer's solution. The goal is to remove debris and begin loosening necrotic tissue without damaging viable tissue.
Lavage should be performed at approximately 7 to 8 psi, which can be achieved using a 35 milliliter syringe with an 18 to 19 gauge catheter. Higher pressures should be avoided, as they can drive bacteria deeper into tissue and worsen injury.
At this stage, the goal is gentle cleaning and preservation of viable tissue—not aggressive debridement.



Slide 22: Debridement Strategies

Sharp, Mechanical, Enzymatic, and Hydrosurgical.

Debridement is the process of removing nonviable tissue and is a critical component of burn wound management.

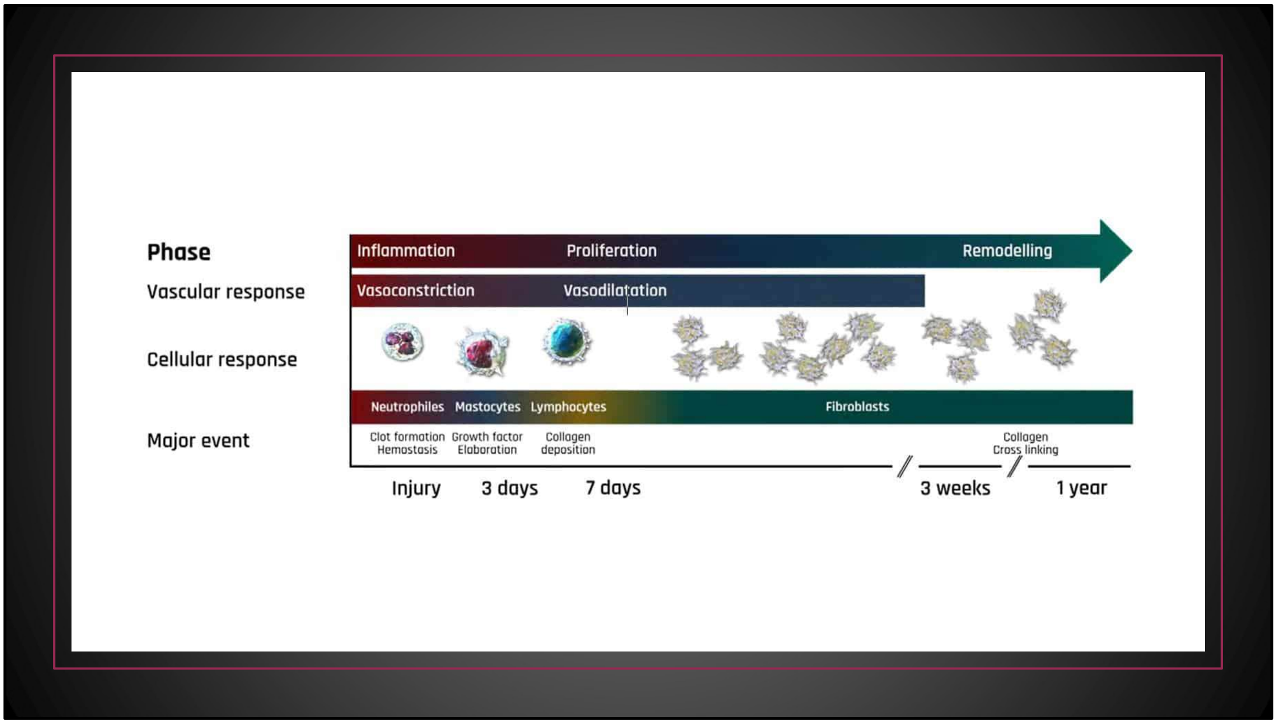
In the early phase, debridement is typically conservative and performed over several days as the wound declares itself. This involves gradual, piecemeal removal of necrotic tissue using gauze or instruments.

This approach helps preserve viable tissue and is especially important in areas overlying tendons, joints, or bone.

Enzymatic debridement can be used in select cases to help soften and loosen necrotic tissue, allowing for easier removal.

Once the wound has declared and margins are clear, more aggressive surgical debridement may be performed, particularly in deeper burns.

Debridement not only facilitates healing but is also one of the most important methods of infection control. Depending on the severity and extent of the burns the patient may need to be fully sedated to perform this comfortably and humanely.



Wound healing occurs in overlapping phases, but in burn patients these phases are often disrupted or prolonged.
The inflammatory phase begins immediately after injury. In burn patients, this phase is exaggerated and prolonged due to extensive tissue damage and ongoing cytokine release. This contributes to edema, pain, and can drive further tissue injury, especially in the zone of stasis.
The proliferative phase involves formation of granulation tissue, angiogenesis, and epithelialization. This phase can be delayed in burns, particularly if there is ongoing necrosis, infection, or poor perfusion.
The remodeling phase involves collagen reorganization and scar formation. In burn patients, this phase is often associated with excessive fibrosis and contracture, especially in areas over joints.
Understanding these phases is important because our interventions aim to control inflammation, remove nonviable tissue, and support progression into the proliferative phase as efficiently as possible.

THE "ESCHAR" CHALLENGE

Slide 21: The "Eschar" Challenge
Avascular leathery layer that harbors bacteria.
Requires debridement to allow topical/biological penetration.
Eschar is the devitalized, avascular tissue that forms in full-thickness burns.
Because it lacks blood supply, it prevents delivery of both systemic antibiotics and topical therapies to the underlying tissue. It also provides an ideal environment for bacterial growth, increasing the risk of infection and sepsis.
In addition, eschar acts as a physical barrier that delays wound healing and prevents formation of healthy granulation tissue.
For these reasons, eschar must ultimately be removed. However, timing is critical. Early in the course of injury, while the wound is still declaring itself, removal is typically conservative. More aggressive debridement is performed once viable and nonviable tissue can be clearly distinguished.

ANTIMICROBIAL AND TOPICALS USE

Agent	Key Feature	Best Use	Clinical Notes
Silver Sulfadiazine (SSD)	Broad-spectrum topical	Superficial & partial burns	May delay epithelialization
Mafenide Acetate (Sulfamylon)	Penetrates eschar	Deep burns, necrotic tissue	Painful, risk metabolic acidosis
Medical-Grade Honey (Manuka)	Antimicrobial + anti-inflammatory	Partial burns, contaminated wounds	Promotes autolytic debridement
Hydrogels	Moisture + cooling	Superficial burns	Improves comfort, not strongly antimicrobial
Triple antibiotic (bacitracin/neomycin/polymyxin)	Surface-level coverage	Minor burns	Limited depth penetration

Topical therapy plays a central role in burn wound management, particularly in the early phase while the wound is still declaring itself.

Burn wounds are initially sterile, but they rapidly become colonized due to loss of the skin barrier. Because of this, topical antimicrobials are the first line of infection prevention. They allow for high local antimicrobial activity directly at the wound surface, where systemic antibiotics are often ineffective due to poor penetration into avascular tissue.

Common topical options include silver-based products, mafenide acetate, and medical honey. Silver sulfadiazine has broad antimicrobial coverage and is commonly used early, although it may delay epithelialization with prolonged use. Mafenide penetrates eschar more effectively and is particularly useful when deeper tissue involvement is a concern.

Medical honey is a valuable option because it provides antimicrobial effects while also supporting a moist wound environment and promoting autolytic debridement. It is often well tolerated and can be especially useful as the wound transitions out of the acute phase.

These therapies are not used in isolation. Their role is to reduce bacterial burden and protect the wound while we address the underlying drivers of burn progression, including perfusion, edema, and inflammation.

As the wound evolves, our approach shifts toward moisture-balanced healing. Dressings such as hydrogels and calcium alginates may be introduced depending on wound characteristics. Hydrogels are useful for dry wounds, while alginates are better suited for

supportive dressings and do not provide antimicrobial activity on their own.
Equally important is proper wound preparation. Gentle cleansing, serial debridement of necrotic tissue, and strict aseptic technique are critical components of infection control. Without removal of devitalized tissue, even the best topical agents will be ineffective.
The overall goal of topical therapy is not to sterilize the wound, but to create a controlled environment that limits bacterial proliferation, supports viable tissue, and prevents burn wound conversion.

HEAVILY EXUDATIVE WOUNDS

- After debridement
- Highly absorbent
- Maintains moist healing environment
- Supportive dressing
- Not for infected wounds

Calcium alginate dressings are used as part of moisture-balanced wound care, particularly once the wound has been debrided and is producing moderate to heavy exudate.
These dressings are highly absorbent and form a gel when they come into contact with wound fluid. This helps maintain a moist environment, which is important for healing and supports autolytic debridement.
However, it is important to recognize that alginates do not actively debride the wound. They do not replace enzymatic or surgical debridement, which is often required in burn patients with necrotic tissue.
Instead, they function as a supportive dressing that manages exudate and helps maintain an optimal wound environment.
They are best used after the wound is clean and viable, and are not appropriate for dry wounds or those with intact eschar.

Up to this point, management has focused on stabilization, infection prevention, and preserving viable tissue while the wound declares itself.

As the wound evolves, typically over the first 3 to 7 days, the focus begins to shift. Once viable and nonviable tissue can be clearly identified and the patient is stable, management transitions from preventing progression to actively promoting healing.

At this stage, the goal becomes preparation of the wound bed, promotion of granulation tissue, and progression toward closure.

Advanced wound care techniques are introduced based on wound condition and patient stability rather than a strict timeline.

Shift toward moist wound healing and reduced disruption.

HYPERBARIC OXYGEN THERAPY (HBOT)

- Increases amount of dissolved oxygen in the blood, improving oxygen delivery to hypoxic tissues.
- Supports cellular function, enhances healing, and may help limit burn progression, particularly in the zone of stasis.
- Reduces edema and may improve microvascular perfusion.
- Can be used at all phases of healing

Section 5: Advanced Care Bundles
Hyperbaric Oxygen Therapy (HBOT)
Reduces edema while hyper-oxygenating tissues.
Inhibits neutrophil adhesion in the Zone of Stasis.

NEGATIVE PRESSURE WOUND THERAPY (NPWT)

Slide 26: Negative Pressure Wound Therapy (NPWT)
"Wound Vac" – removes exudate and stimulates angiogenesis.
Excellent for preparing the wound bed for a graft.

FISH SKIN GRAFTS (ACELLULAR COD MATRIX) BIOLOGIC SCAFFOLD

Fish Skin Grafts (Acellular Cod Matrix)
Biologic Scaffold: Framework for cellular infiltration.
Omega-3 rich; modulates local inflammation.
Clinical Value of Fish Skin
Reduced bandage frequency = Reduced pain.
Fewer anesthesia events for the patient
Intended Use
Kerecis® VET is indicated for the management of wounds including:
Partial and full-thickness wounds
Traumatic wounds (abrasions, lacerations, skin tears)
Superficial and partial thickness burns

TECHNICAL APPLICATION OF FISH SKIN

Debride	Debride to clean, viable wound bed
Graft	Trim graft to size
Apply	Apply directly to wound surface
Ensure	Ensure full contact (no air pockets)
Cover	Cover with non-adherent secondary dressing

Slide 28: Technical Application of Fish Skin
Hydration: Must be pre-hydrated in sterile saline.
Fixation: Suture into place for 100% contact.
Fish skin grafts should only be applied after the wound has been adequately debrided and a clean, viable wound bed is present.
The graft is trimmed to fit the wound and applied directly to the surface, ensuring full contact with the underlying tissue. Avoiding air pockets is critical, as these can interfere with integration.
A non-adherent secondary dressing is applied to maintain positioning and protect the graft.
These grafts integrate over time and reduce the need for frequent bandage changes, improving patient comfort and decreasing repeated sedation.

Up to this point, we have focused on stabilization, wound management, and preventing complications.
Now we transition into recovery, where the focus shifts from survival to long-term function and quality of life.
This includes nutrition, nursing care, and managing complications such as contracture and scarring.

NUTRITIONAL SUPPORT AS A PRIMARY THERAPY

•Hypermetabolic state → ↑ caloric needs
•High protein requirements
•Early enteral nutrition preferred

- Feeding tubes if needed
  Start with RER:
  RER = 70 × BWkg$^{0.75}$
- Burn patient estimate:
  MER ≈ RER × 1.5–2.0
  Higher end for large TBSA, deep burns, infection, or severe hypermetabolism.
- Protein:
  High-protein diet or enteral nutrition support
- Example:
  20 kg dog:
  RER = 70 × 20$^{0.75}$ ≈ 660 kcal/day
  Burn estimate = 990–1320 kcal/day

Nutritional Support as Primary Therapy
Early and often! Enteral feeding to meet doubled caloric demands.
High protein to replace losses through the wound.
✅ **It supports:**
Ongoing protein synthesis
Wound healing
Immune function
Again, burn patients enter a hypermetabolic state, significantly increasing their caloric and protein requirements.
Adequate nutrition is critical for wound healing, immune function, and overall recovery.
Whenever possible, voluntary intake is preferred, but if patients are not meeting their needs, enteral feeding should be initiated early.
In some cases, feeding tubes or parenteral nutrition may be required.

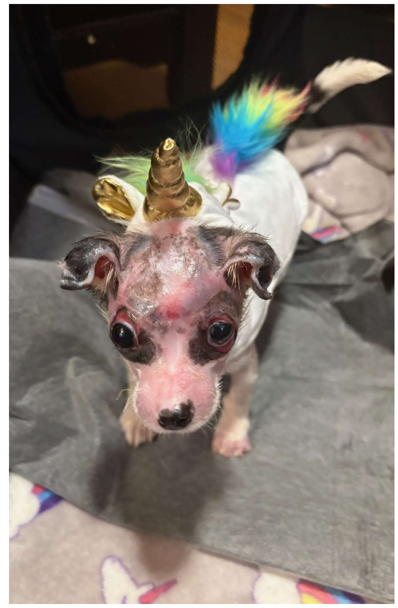
NURSING CARE AND INTERVENTIONS

- Frequent repositioning
- Wound Care and monitoring
- Pain management
- Monitor fluid balance & urine output
- Frequent vitals, mentation, and weight checks
- Nutrition administration
- Maintain clean environment
- Passive range of motion
- Tender loving care
- "Nursing care drives outcomes"

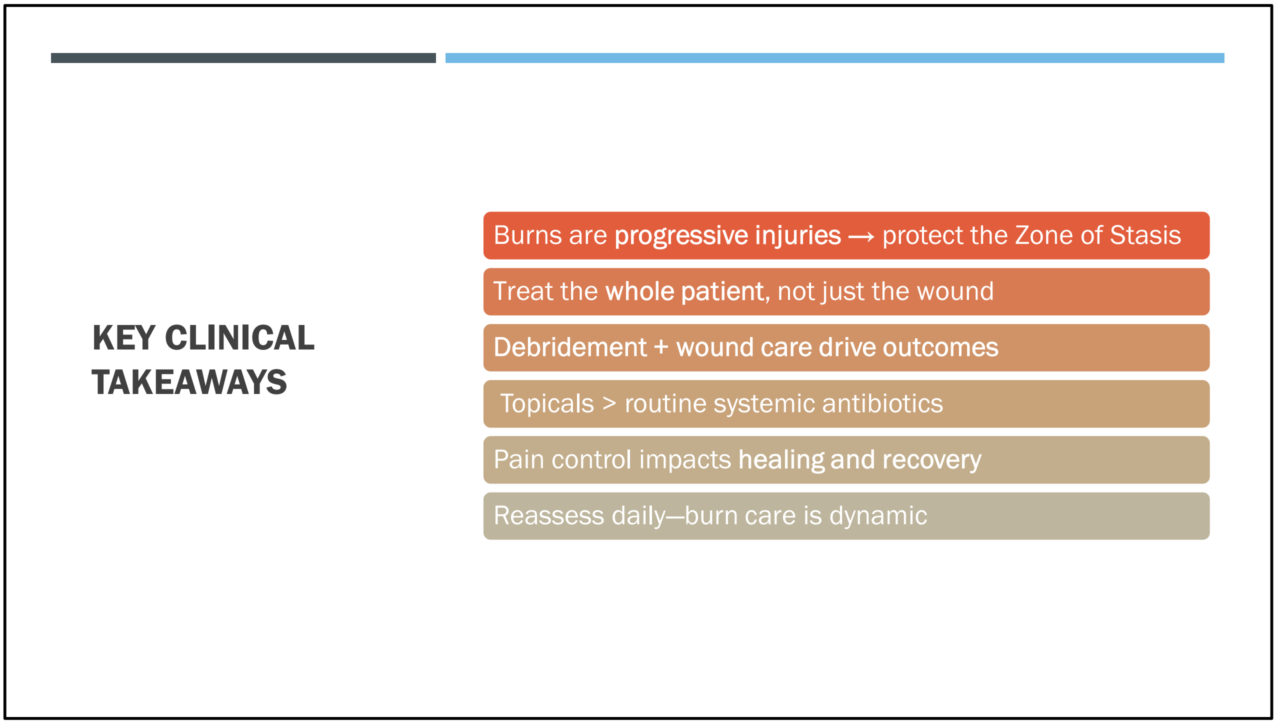
The "Burn Nursing Bundle"
Temperature regulation (Active warming).
Recumbency care (Pressure sore prevention).
Elevation of burned limbs/head to reduce edema.
Nursing care is one of the most important components of burn patient management.
Patients should be repositioned regularly to prevent pressure injuries. Pain management must be proactive, as these patients experience significant discomfort.
Monitoring fluid balance and urine output is critical, especially early in treatment.
Mentation, vitals signs, nutrition intake, weight checks, invasive device maintenance
Passive range of motion exercises help prevent stiffness and contracture, particularly in patients with limb involvement.

RECOVERY AND HEALING CONSIDERATIONS

- Risk of contracture
- Scar formation
- Decreased mobility
- Pain sensitization (acute → chronic)
- May require surgical intervention



Even after the wound has closed, burn patients remain at risk for significant long-term complications.
Contracture is one of the most important concerns, especially around joints, and can lead to decreased mobility and long-term functional impairment. Scar formation can also be extensive and impact both comfort and movement.
Another important consideration is pain sensitization. Due to ongoing inflammation and repeated painful procedures during treatment, burn patients are at risk for both peripheral and central sensitization.
This means that pain can become amplified over time and may persist even after the wound has healed, contributing to chronic pain states and making rehabilitation more difficult.
Because of this, early and effective pain management is critical not just for comfort, but for long-term outcomes.
Some patients may ultimately require surgical intervention, such as grafting or reconstructive procedures, to restore function.



Slide 33: Key Clinical Takeaways
Burns are progressive; target the Zone of Stasis.
Manage the "Whole Patient" (Nutrition/Pain).
Biologics (Fish Skin) optimize comfort and outcome.

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ADDITIONAL CLINICAL RESOURCES

VIN. Burn management overview.
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VetFolio. Thermal injury.
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