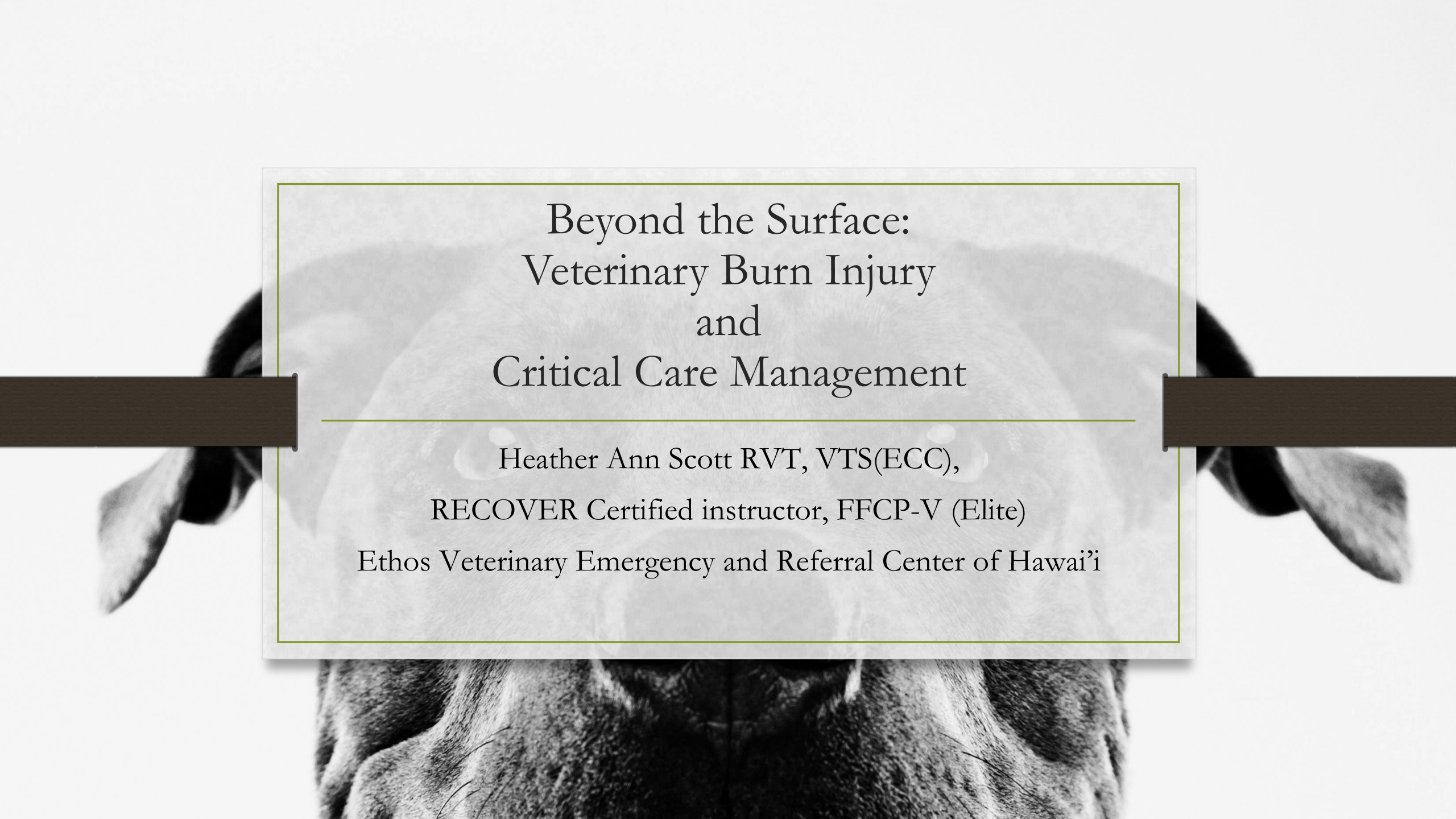


Beyond the Surface: Veterinary Burn Injury and Critical Care Management

Heather Ann Scott RVT, VTS(ECC),

RECOVER Certified instructor, FFCP-V (Elite)

Ethos Veterinary Emergency and Referral Center of Hawai'i

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

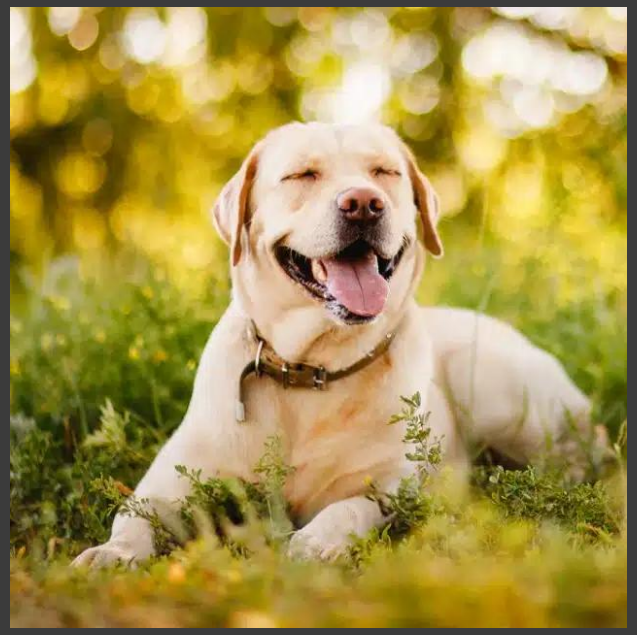
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Audience check-in : When you think of burns in veterinary patients, what comes to mind?

ⓘ The Slido app must be installed on every computer you're presenting from

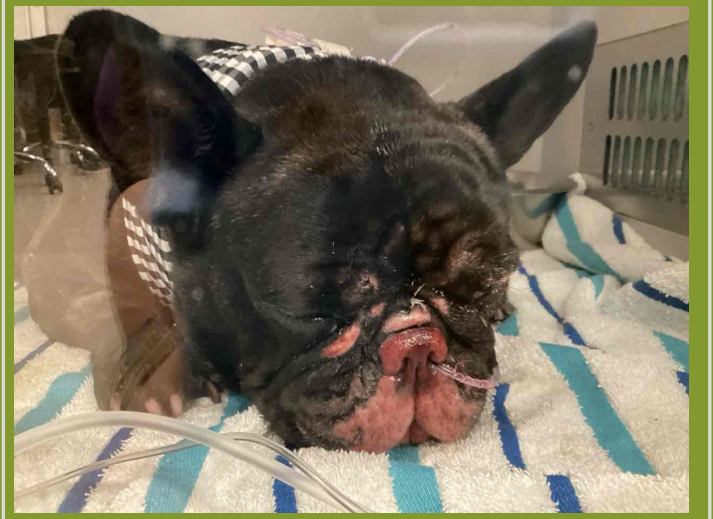
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- **Case Introduction: Loki**
- **Signalment** 4-year-old MN Labrador Retriever
- **History**

Walked on hot pavement during a hot California summer day.

- **Physical Examination:**
- T: 102.1°F HR: 140 bpm RR: Panting
- Mentation: BAR but trembling/shaking , Reluctant to walk , Frequently licking paws. Multifocal paw pad burns with epithelial sloughing
- **Pain Assessment:** MGPS: 11/24 **Burn Assessment** Estimated TBSA: <1%
- **Diagnostics:** Mild CK elevation. Remaining laboratory findings unremarkable, No evidence of systemic heat-related illness
- **Clinical Considerations:**
- Localized thermal and friction injury. Significant pain despite minimal TBSA involvement
- Outpatient versus inpatient management considerations?
- *We will revisit Loki later in the presentation.*



Case Introduction: Ember
Signalment: 6-year-old FS French Bulldog

History: Rescued from a residential house fire approximately 30 minutes prior to presentation.

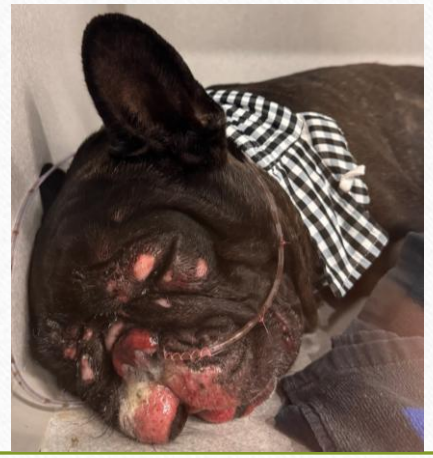
Physical Examination:
T: 103.4°F | **HR:** 180 bpm | **RR:** 60 bpm with increased respiratory effort |
Mentation: Quiet but responsive. Singed facial hair and whiskers. Soot present around nares and oral cavity, facial, cervical, and caudal body burns, corneal/ocular burns were also present
•Mild erythema and edema of the oral mucosa
Pain Assessment: MGPS 14/24
Burn Assessment: Estimated TBSA 18%
Diagnostics:
SpO_2 94% on room air, Lactate 3.8 mmol/L, Mild hemoconcentration
Clinical Considerations:
Thermal injury, Suspected inhalation injury, Potential airway compromise.

We will revisit Ember later in the presentation.

Types of Burn 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, poorly regulated topical flea products	Any exposed area, often paws, face, oral cavity	**Continues to damage tissue until removed**; requires immediate, copious lavage







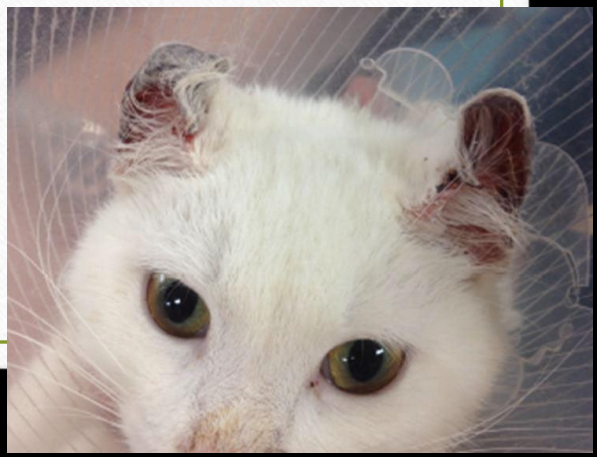
Types of Burn Injury Cont.

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**

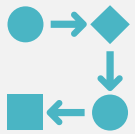








Why are burn injuries different?



MULTI-STAGE AND PROGRESSIVE



SYSTEMIC AS WELL AS LOCAL



TIME SENSITIVE INJURIES

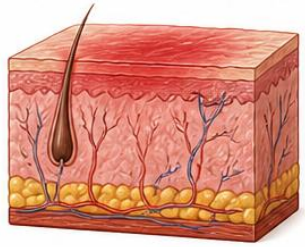
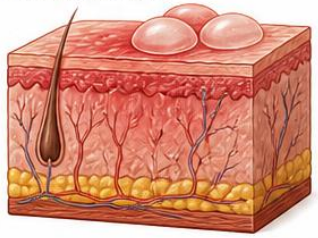
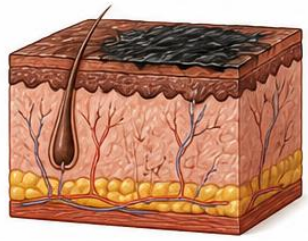
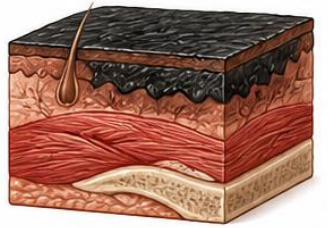


FREQUENTLY UNDERESTIMATED IN EARLY PRESENTATION

Feature	Traumatic Wounds	Severe 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

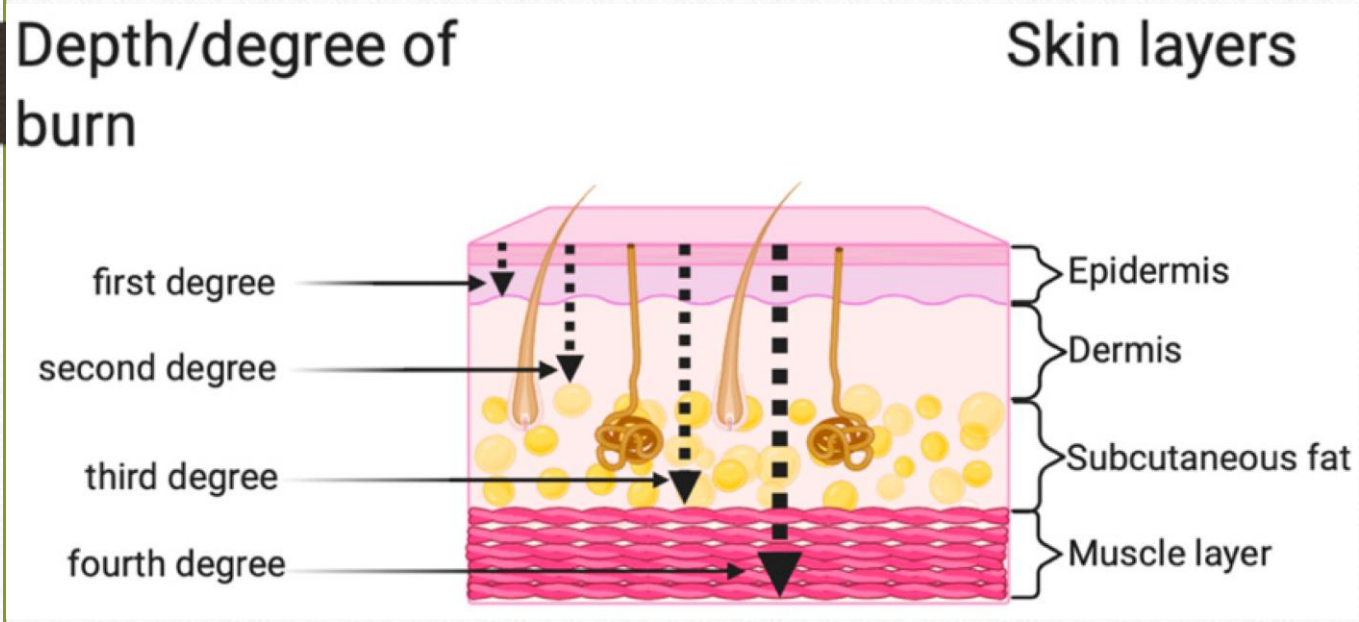
Burn Injury Wound vs Traditional Wound

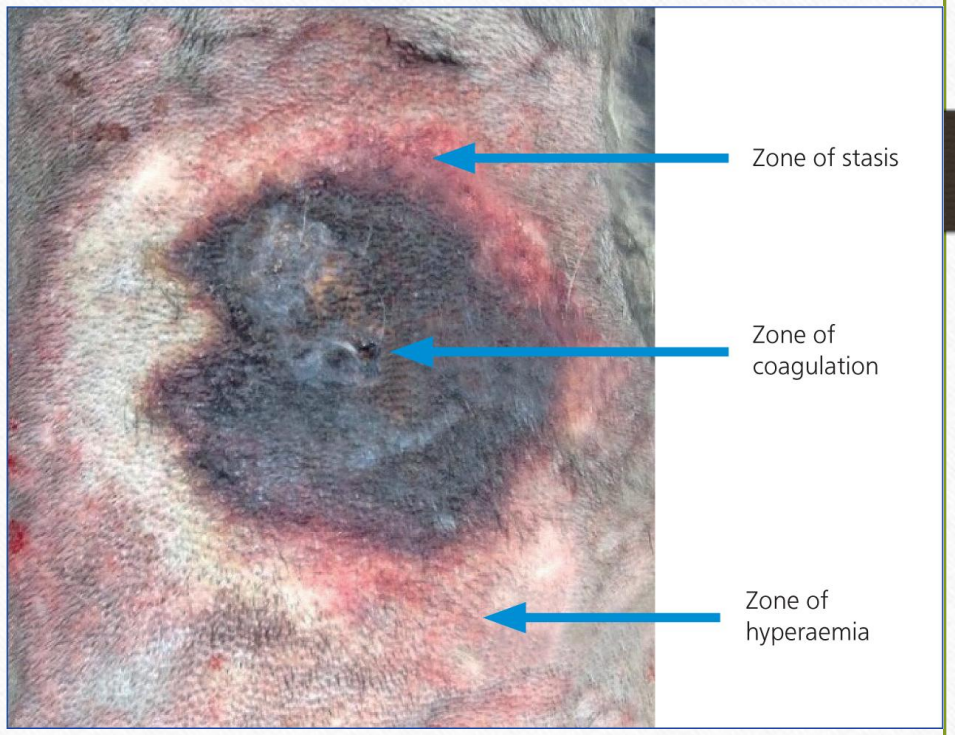
BURN WOUND ASSESSMENT AND HEALING

	FIRST DEGREE	SECOND DEGREE	THIRD DEGREE	FOURTH DEGREE
DEPTH	Superficial (epidermis only)	Epidermis and superficial part of dermis	Full thickness (entire epidermis and dermis)	Full thickness with extension to muscle, tendon, and bone
APPEARANCE	• Erythematous • Painful to touch 	• Epidermis will be charred and sloughs, plasma leakage occurs • Hair follicles spared • Painful to touch 	• Skin is black, leathery; eschar insensitive to touch • Hair follicles destroyed • Decreased pain sensation 	• Skin is black, leathery; eschar insensitive to touch • Extends into subcutaneous tissue (fat, muscle, tendon, bone may be involved) 
HEALING	Healing is rapid; reepithelializes in 1 week with topical wound management **No systemic effects**	Healing by epithelialization from the wound margin with minimal scar in 10 to 21 days **May have systemic effects**	Healing by contraction and epithelialization but scarring is significant without surgical intervention **Significant systemic effects expected**	Healing often requires extensive surgical intervention, possible skin grafts and flaps **Skin grafts usually required to prevent scarring that could restrict joint movements**
SYSTEMIC EFFECTS	No systemic effects	May have systemic effects	Significant systemic effects expected	May have life-threatening systemic effects

IN ALL BURNS: The depth can be difficult to assess early. Reassess within 24–72 hours as wounds can progress.

Jackson's Model



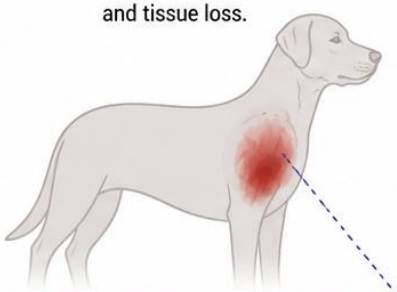


THE ZONE OF STASIS

The tissue we can still save.

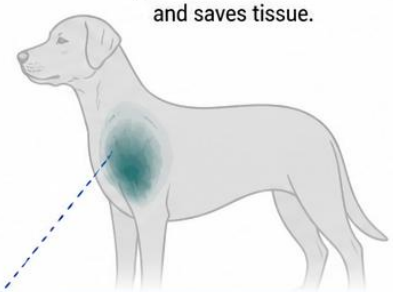
Too much injury

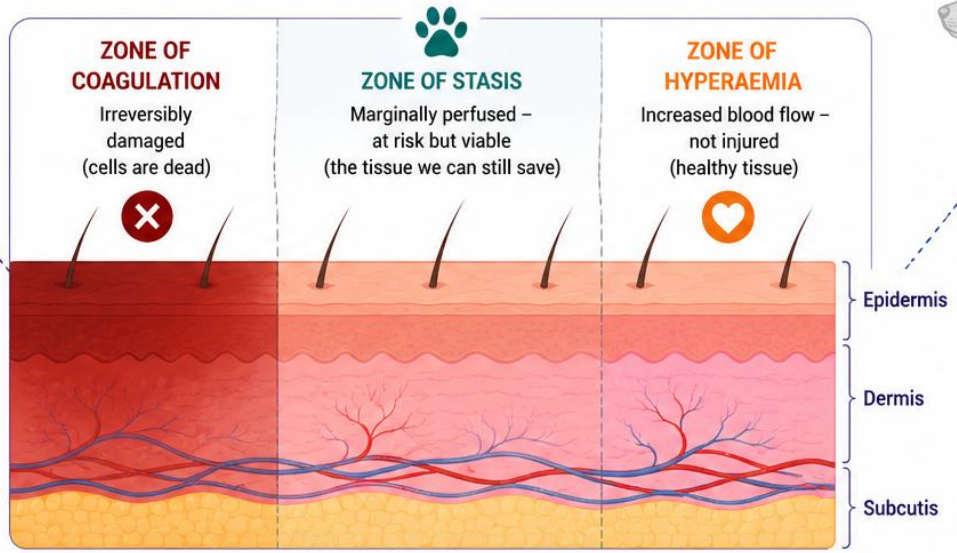
leads to conversion and tissue loss.



Appropriate care

prevents conversion and saves tissue.





WHAT HAPPENS WITHOUT INTERVENTION

INFLAMMATION
Cytokines, PMNL, complement, eicosanoids

↓

OXIDATIVE STRESS
ROS, lipid peroxidation, cellular injury

↓

HYPOPERFUSION
Edema, thrombosis, vasoconstriction

↓

CONVERSION
Stasis → coagulation (tissue is lost)

WHAT'S HAPPENING IN THE ZONE OF STASIS?

- Vasospasm ↓ blood flow
- Endothelial injury & hypercoagulation → microthrombosis
- Inflammatory cascade & ROS → cell stress

Timely, appropriate care can prevent progression and preserve viable tissue.

HOW WE SAVE THE ZONE OF STASIS

RESTORE PERFUSION
Fluids, oxygen delivery, avoid hypotension

REDUCE INFLAMMATION
Limit ongoing injury, control pain, warmth

PROTECT THE WOUND
Topical therapy, gentle cleansing, moisture

PREVENT INFECTION
Asepsis, topical antimicrobials

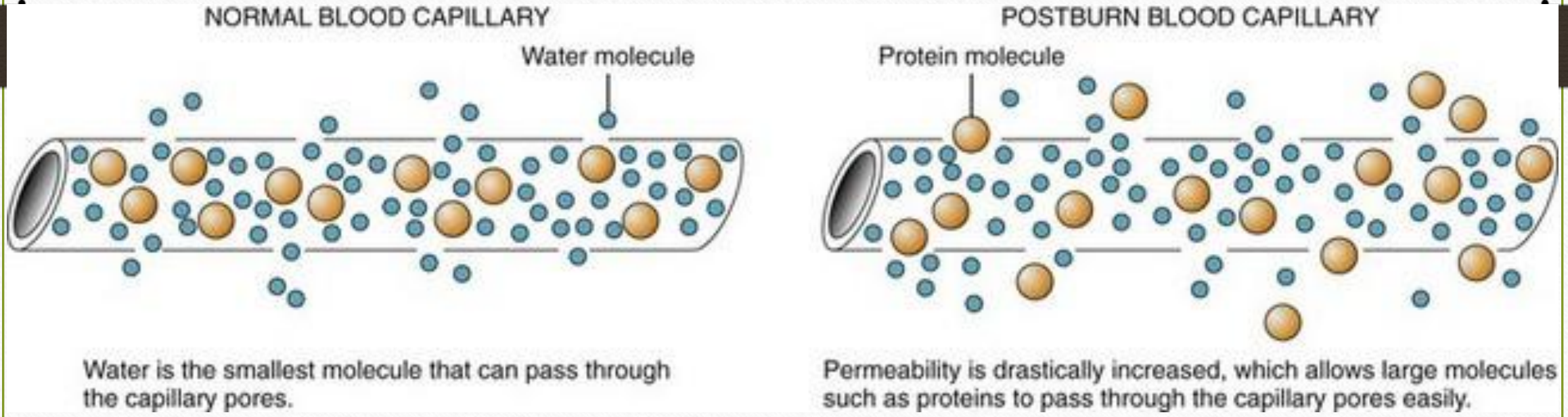
SUPPORT RECOVERY
Nutrition, monitor and reassess

KEY GOALS IN MANAGEMENT

- Early, goal-directed fluid therapy
- Prevent edema & improve perfusion
- Wound protection & infection prevention
- Pain control & stress reduction
- Early nutrition support
- Frequent reassessment – burns evolve

Save the Zone of Stasis today. Save the tissue tomorrow.

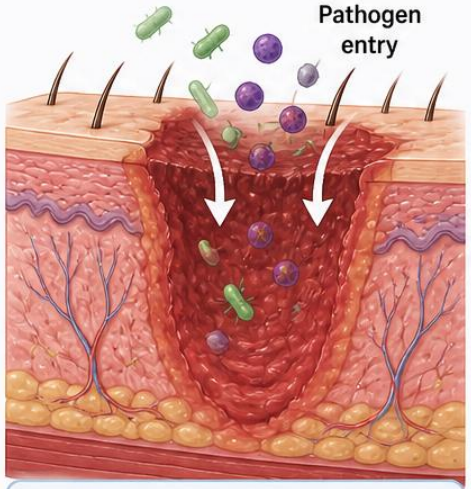
Early Local Pathophysiology



IMMUNE DYSFUNCTION

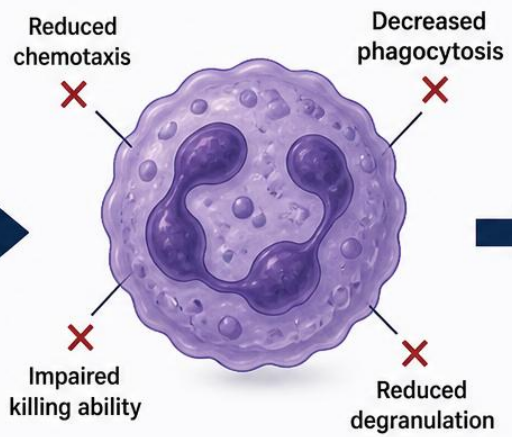
Barrier loss + impaired neutrophil function = **High Sepsis Risk**

1. BARRIER LOSS



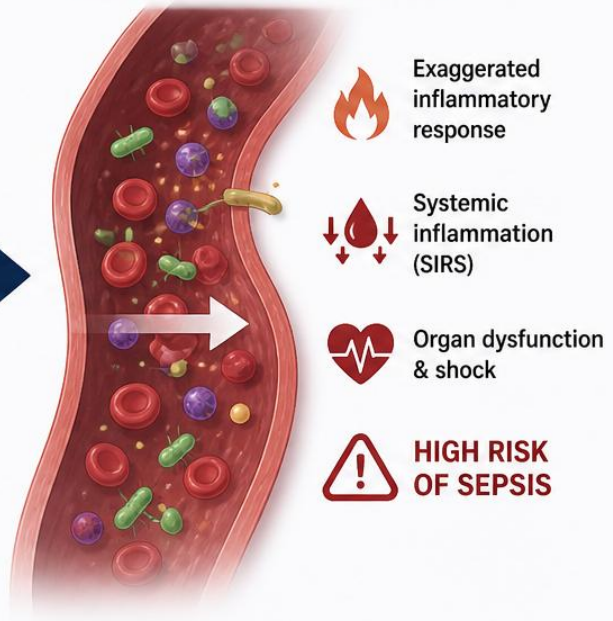
Compromised skin barrier allows bacteria and toxins to invade.

2. IMPAIRED NEUTROPHIL FUNCTION



Neutrophils are essential but become dysfunctional after burn injury.

3. HIGH SEPSIS RISK

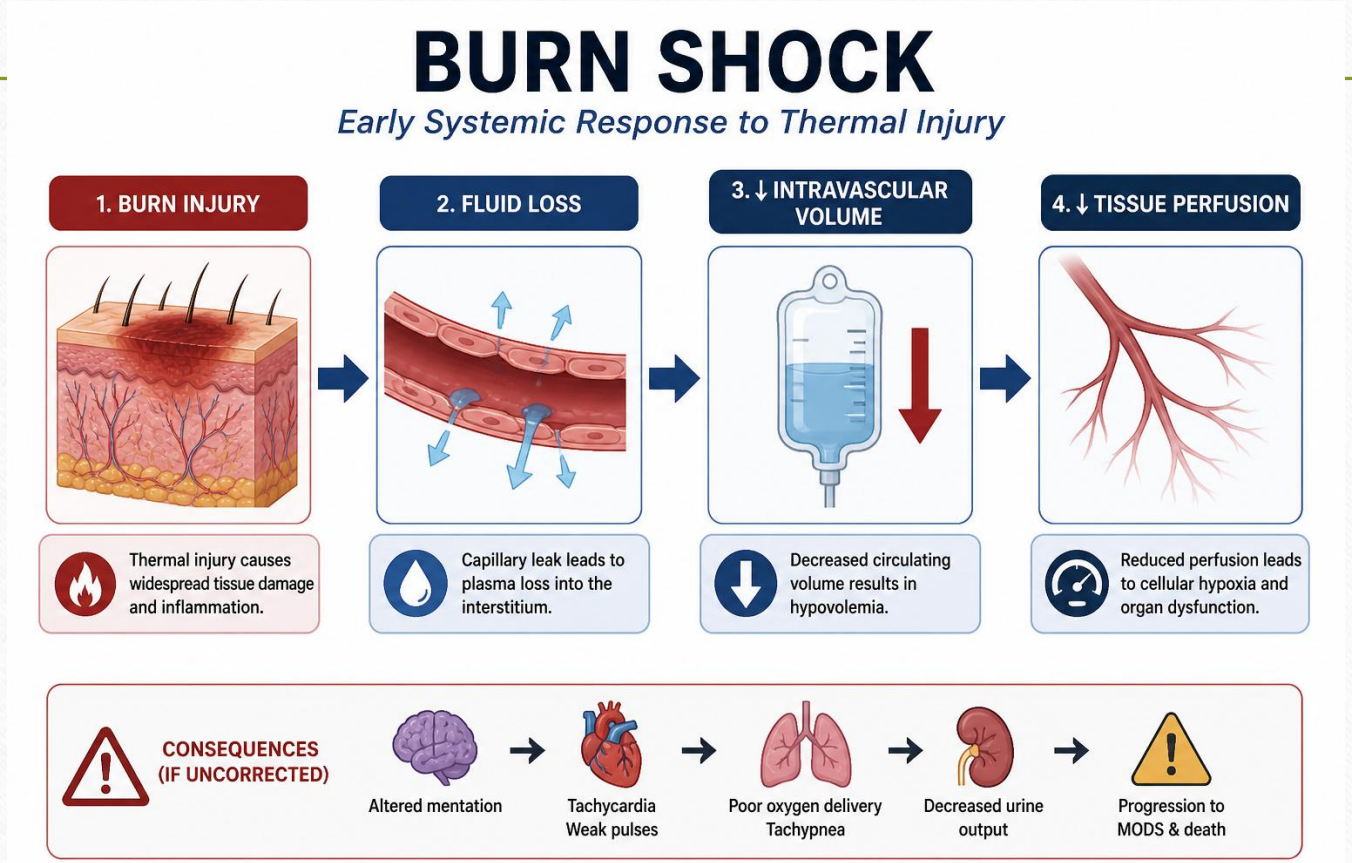


- Exaggerated inflammatory response
- Systemic inflammation (SIRS)
- Organ dysfunction & shock
- **HIGH RISK OF SEPSIS**

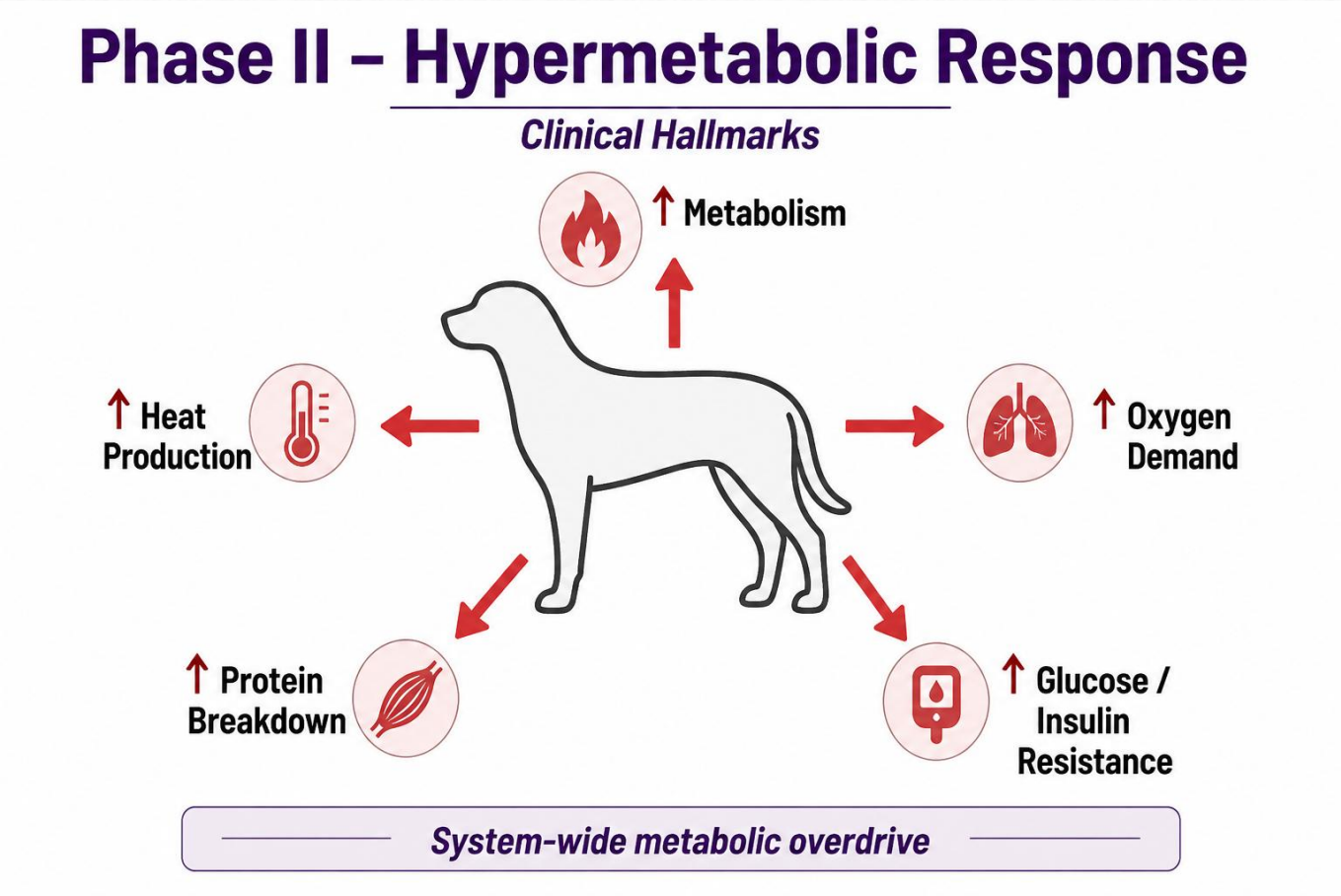
KEY TAKEAWAY:
Burn injury compromises both the barrier and the immune system, leading to a dangerous cycle of infection and systemic inflammation.

Early infection control, immune support, and close monitoring are critical.

Systemic Impact (The Crisis)
Phase I – Burn Shock (The First 36 Hours)



Clinical Hallmarks of Hypermetabolism

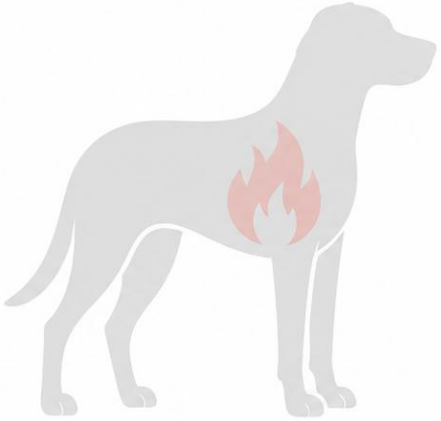


Initial Stabilization (ABCDE)



INITIAL STABILIZATION

ABCDE Approach



A — AIRWAY
Secure and protect the airway

B — BREATHING
Assess ventilation and oxygenation

C — CIRCULATION
Establish IV access, assess perfusion, start fluids

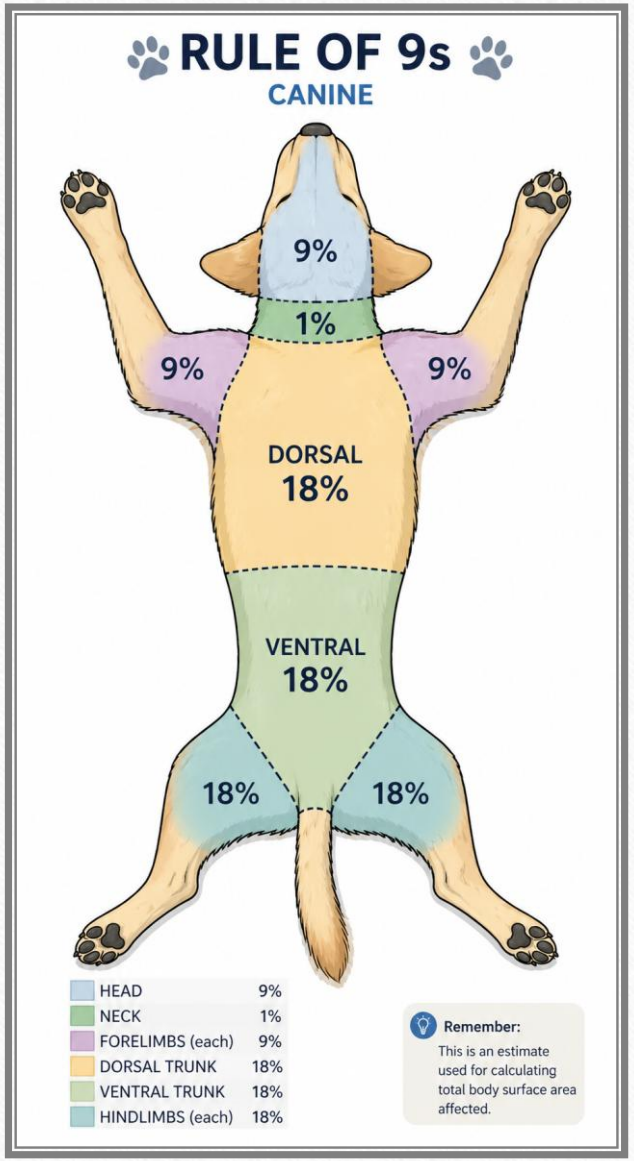
D — DISABILITY (PAIN)
Assess mentation and provide analgesia

E — EXPOSURE
Assess full extent of burns, prevent hypothermia

Treat life threats first, wound care comes later.

“Rule of 9s” veterinary adaptation

- **TBSA + Depth = Treatment Strategy**
- <10%: Local wound care focus
- 10–20%: Moderate risk, monitor closely
- >20%: Systemic disease (SIRS risk)
- >40–50%: Grave prognosis

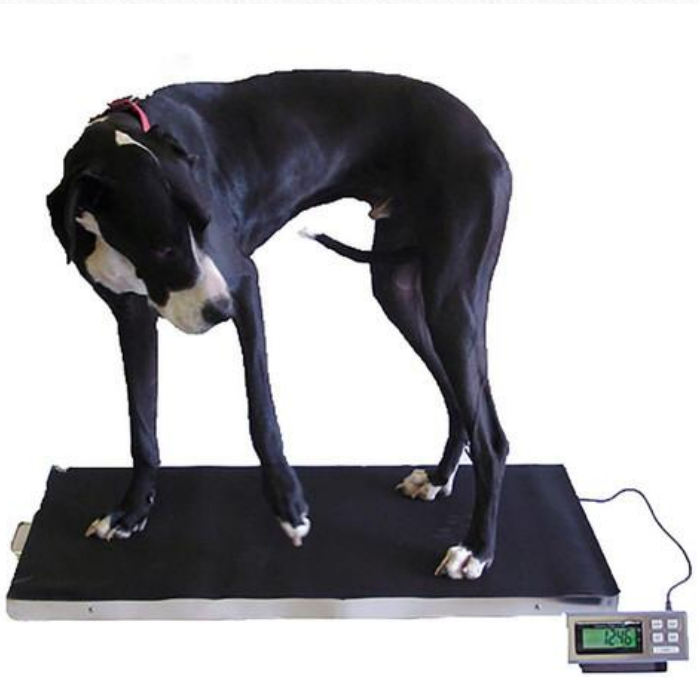


Fluid Resuscitation

⚡ ELECTROLYTES 🩸 PERFUSION ⚠️ OVERLOAD







<u>Fluid Goals:</u>

Balanced Crystalloids ± colloids/blood products as indicated

First 24 hr estimate

1–4 mL/kg × % TBSA burned

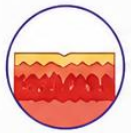
Monitoring goals

Urine output: **1–2 mL/kg/hr**

MULTIMODAL PAIN MANAGEMENT

Target multiple pain pathways. Improve comfort. Support healing.

BURN PAIN IS COMPLEX



NOCICEPTIVE
Tissue injury, inflammation



NEUROPATHIC
Nerve injury, neuropathic pain



CENTRAL SENSITIZATION
Wind-up, hyperalgesia with ongoing pain

Pain is ongoing and dynamic—not episodic.

ANALGESIA LADDER FOR BURN PATIENTS

MORE INTENSE PAIN

4 OPIOID ANALGESIA (CRI PREFERRED)
Fentanyl • Hydromorphone • Methadone
Foundation for moderate to severe pain. Continuous infusion for steady control.

3 ANTI-WINDUP / CRI ADJUNCTS
Ketamine CRI
± Lidocaine CRI (dogs)
Reduces central sensitization (wind-up) and inflammatory response. Opioid-sparing.

2 NEUROPATHIC ADJUNCT
Gabapentin
Targets neuropathic pain and helps reduce chronic pain and central sensitization. *Full benefit develops over days.*

1 NSAIDs (AFTER STABILIZATION ONLY)
Carprofen, Meloxicam, Deracoxib, etc.
Anti-inflammatory and opioid-sparing. Use ONLY after adequate perfusion, renal function and hydration are stable.

FOUNDATION: WOUND & SUPPORTIVE CARE
Gentle handling • Topical therapies • Environmental comfort
Nutrition • Adequate hydration
Treat the source of pain. Handle gently. Keep warm. Minimize stress.

LESS INTENSE PAIN

LIDOCAINE CRI (DOGS)

- Loading dose: 1–2 mg/kg IV
- CRI: 25–50 mcg/kg/min
- Adjunct for analgesia, anti-hyperalgesia, and anti-inflammatory benefits.

USE CAUTION

- Monitor for CNS signs (tremors, seizures)
- Avoid or use with extreme caution in cats (higher risk of toxicity).

NSAIDs: TIMING MATTERS

- Avoid in initial 24–72 hr (hypovolemia, risk of AKI, GI ischemia).
- Introduce later in stable patients only.

CORE PRINCIPLES

PRE-EMPTIVE ANALGESIA
Treat before procedures.

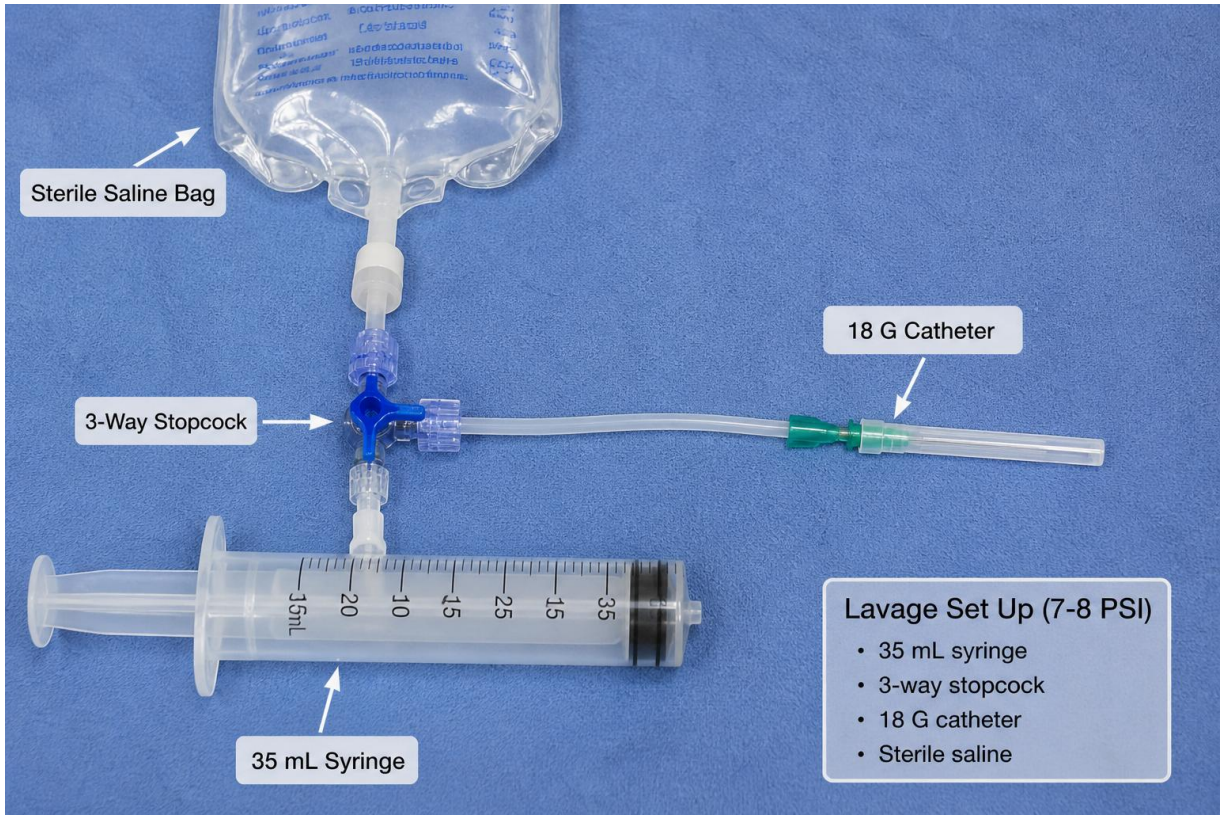
CONTINUOUS ANALGESIA
CRI preferred over intermittent dosing.

MULTIMODAL APPROACH
Target different pain pathways.

FREQUENT REASSESSMENT
Pain changes over time and with care.

GOOD PAIN CONTROL IMPROVES OUTCOMES
Less stress, better perfusion, faster healing.

Effective pain management is part of resuscitation. | *If we under-treat pain, we worsen perfusion—and we worsen the burn.*



Initial wound care

Cooling → Clipping → Lavage → Protect

DEBRIDEMENT STRATEGIES

Goal: Remove nonviable tissue while preserving viable tissue to promote healing and prevent infection.

1. CONSERVATIVE (EARLY, 3–7 DAYS)

- Gradual, piecemeal removal of necrotic tissue
- Use sterile gauze, instruments, and hydrotherapy
- Preserve viable tissue (especially over tendons, ligaments, bone)
- Wound is "declaring" itself



2. ENZYMATIC (SELECT CASES)

- Topical enzymatic agents soften and loosen necrotic tissue
- Facilitates removal with gentle lavage
- Spares healthy tissue
- Best for smaller or more delicate wounds



3. SURGICAL (AFTER WOUND DECLARATION)

- Aggressive removal of nonviable tissue
- Performed once viable vs. nonviable tissue is clear
- Indicated for deep partial-thickness and full-thickness burns
- Often under general anesthesia



4. SERIAL, PROGRESSIVE REMOVAL

- Debridement is not one-time
- Reassess and repeat as the wound evolves
- Decreases bacterial burden
- Improves topical penetration and granulation tissue formation



BOTTOM LINE: Debridement is a serial process that evolves with the wound.

Reduces bacterial burden | Improves topical penetration | Promotes healthy granulation tissue | Prepares wound for closure

Based on wound progression and patient stability

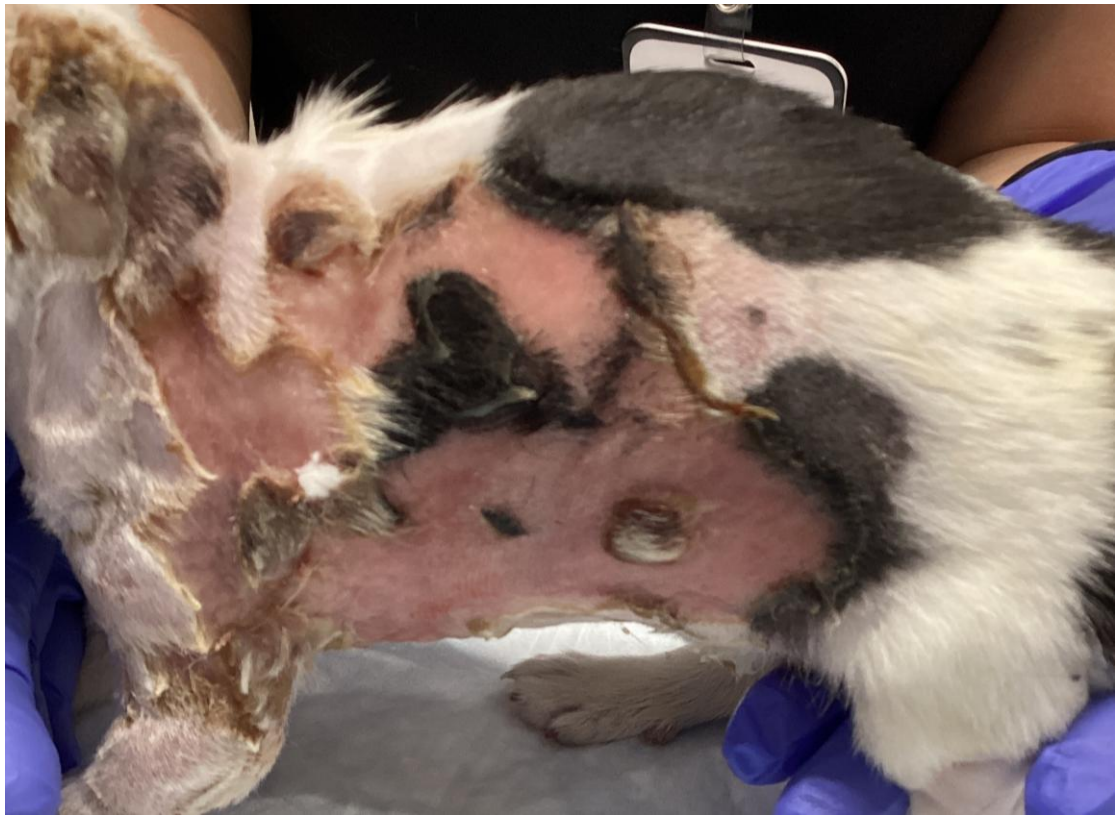
BURN WOUND INFECTION TIMELINE

Infection risk increases over time. Early recognition and intervention are key.



TIME	1. EARLY CONTAMINATION	2. EARLY COLONIZATION	3. CRITICAL WINDOW	4. LATE COLONIZATION / INFECTION	5. CHRONIC / DEEP INFECTION
	0–24 HOURS	1–3 DAYS	3–7 DAYS	7–14 DAYS	>14 DAYS
INFECTION RISK	LOW	LOW–MODERATE	HIGHEST	MODERATE	VARIABLE
COMMON PATHOGENS	Environmental organisms (transient) *e.g., Pseudomonas spp., Staphylococcus spp.*	Gram-positive cocci *Staphylococcus pseudintermedius* *Streptococcus spp.*	Mixed flora *Staphylococcus spp.* *Pseudomonas aeruginosa* *Enterobacteriaceae*	Gram-negative rods more prevalent *Pseudomonas aeruginosa* *Enterobacteriaceae* *± Staphylococcus spp.*	Polymicrobial / biofilm formation *Pseudomonas spp.* *Anaerobes* (in deep wounds) *Resistant organisms possible*
TYPICAL CLINICAL SIGNS	• Minimal to none • Mild redness or sensitivity expected	• Increasing redness • Serous to serosanguinous exudate • Mild swelling	• Purulent or malodorous exudate • Increasing pain, swelling, heat • Delayed healing • Systemic signs may appear	• Persistent or worsening discharge • Granulation tissue changes • Persistent inflammation	• Chronic drainage • Non-healing wound • Sinus tracts / tunneling • Systemic illness possible
MANAGEMENT PRIORITIES	• Thorough cleaning • Debridement of devitalized tissue • Topical therapy • No systemic antibiotics indicated	• Continue aggressive wound care • Topical antimicrobials • Monitor closely • Systemic antibiotics if clinical signs present	• Culture & sensitivity • Systemic antibiotics indicated • Debridement as needed • Optimize perfusion & nutrition • Pain management	• Reassess & culture if not improving • Adjust antibiotics as needed • Consider advanced wound care (e.g., negative pressure therapy)	• Advanced diagnostics (culture, imaging if needed) • Targeted, prolonged antibiotics • Surgical intervention may be required

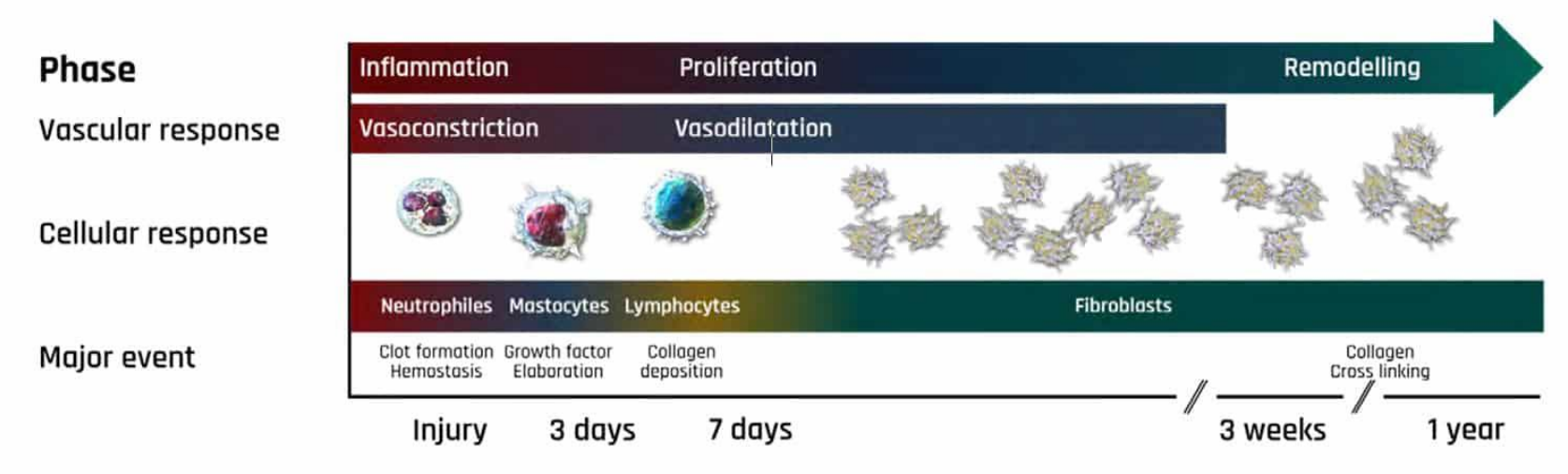
KEY POINT: Infection risk peaks between days 3–7. Vigilant monitoring, early culture, and appropriate intervention improve outcomes.



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

Burn Wound Phases



Traditional Wound

Brief inflammation

Minimal tissue progression

Lower infection risk

Normal metabolic demand

Predictable healing

Limited scarring

Burn Wound

Prolonged inflammation

Burn wound conversion

High infection risk

Hypermetabolic state

Delayed healing

Significant scar/contracture risk

The "Eschar" Challenge



Topicals





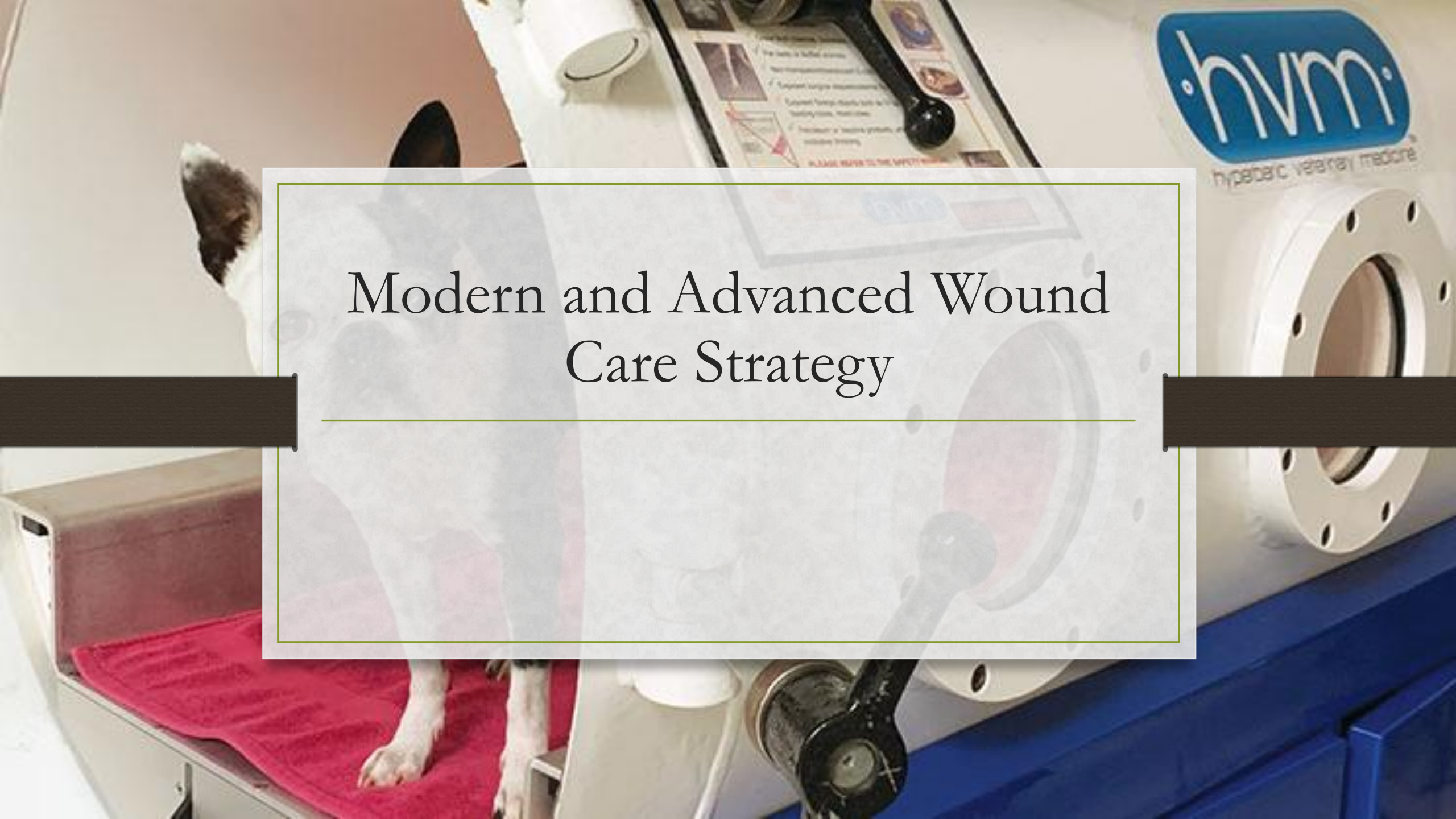








Agent	Feature	Best Use	Clinical Notes:
SSD	Broad antimicrobial	Early superficial/partial burns	May delay epithelialization
Mafenide	Penetrates eschar	Deep burns; eschar	Painful; acidosis risk
Medical Honey	Antimicrobial	Partial burns; contamination	Supports autolytic debridement
Hydrogel (aloe vera containing)	Anti-inflammatory, Cooling; moisture,	Early in thermal burns, dry or superficial burns	Improves comfort, May help preserve tissue within the zone of stasis
Triple Antibiotic	Surface coverage	Minor burns	Limited penetration
Calcium Alginate	Absorbent	Heavy exudate	Not for dry wounds
Silver Alginate	Absorbent + antimicrobial	Exudative/colonized wounds	Local bacterial control



Modern and Advanced Wound Care Strategy

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





Negative Pressure Wound Therapy (NPWT)









Fish Skin/Xenografts
(Acellular Cod Matrix or Tilapia Skin Options)
Biologic Scaffold

Technical Application of Fish Skin

Debride
- Debride to clean, viable wound bed

Graft
- Trim graft to size

Apply
- Apply directly to wound surface and suture

Ensure
- Ensure full contact (no air pockets)

Cover
- Cover with non-adherent secondary dressing where possible

Systemic Support & Recovery





Nutritional Support As A Primary Therapy

Hypermetabolic state →
↑ caloric needs

High protein requirements

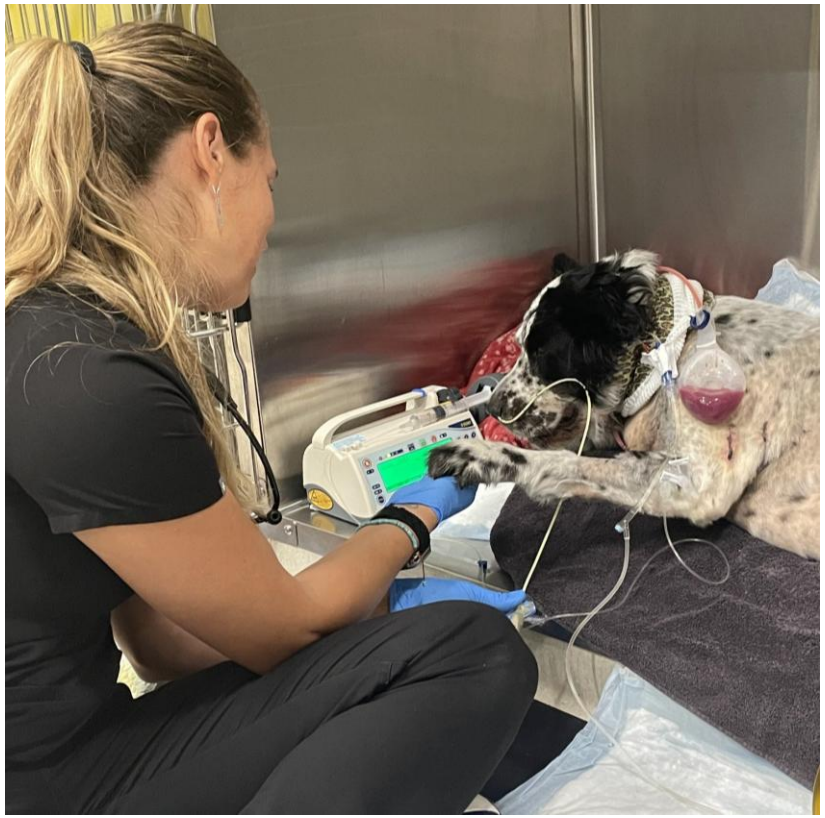
Early enteral nutrition preferred

Feeding tubes if needed
Start @ RER:
RER = 70 × BWkg$^{0.75}$

Burn patient estimate:
MER ≈ RER × 1.5–2.0
Higher end for large TBSA, deep burns, infection, hypermetabolism.

Example:
20 kg dog:
RER = 70 × 20$^{0.75}$ ≈ **660 kcal/day**
Burn estimate = **990–1320 kcal/day**

Protein:
High-protein diet or enteral nutrition support

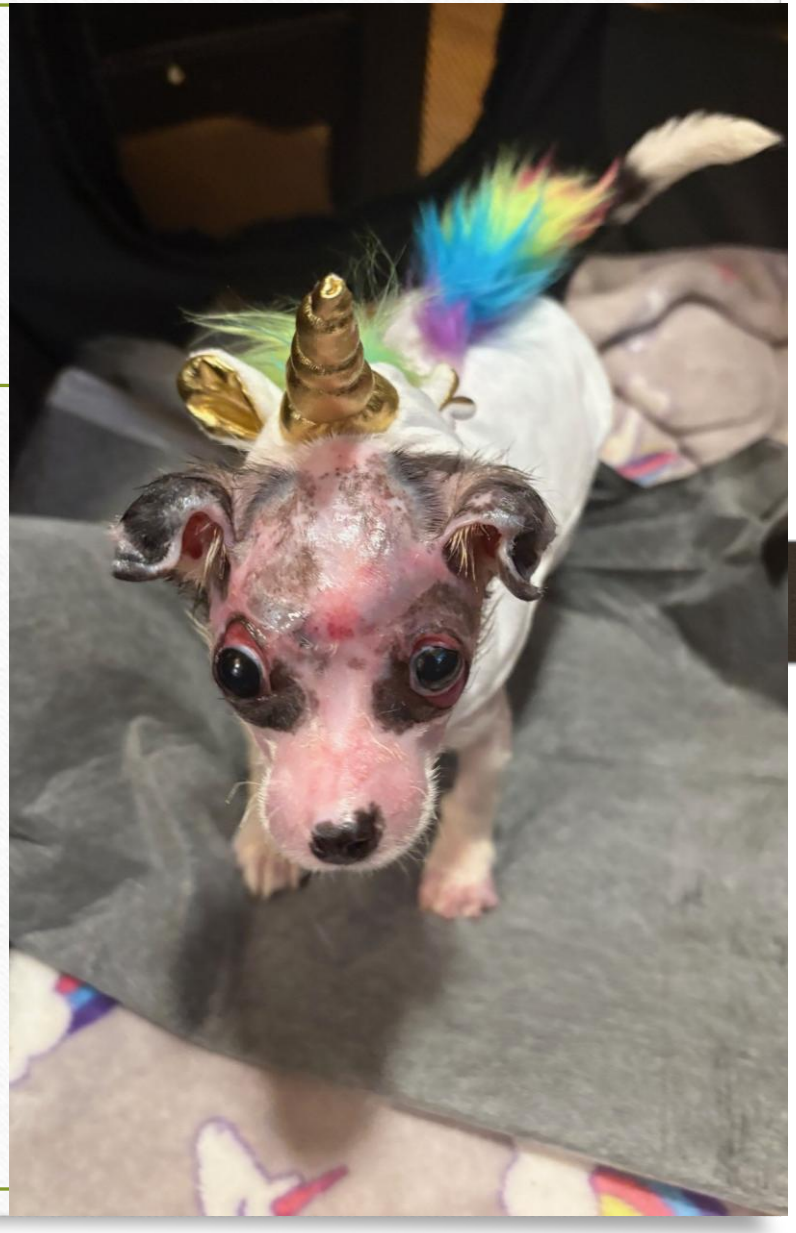


Nursing Care and Interventions

- 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”

RECOVERY AND HEALING CONSIDERATIONS

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







Loki The Lab : Burned Paw Pad Injury

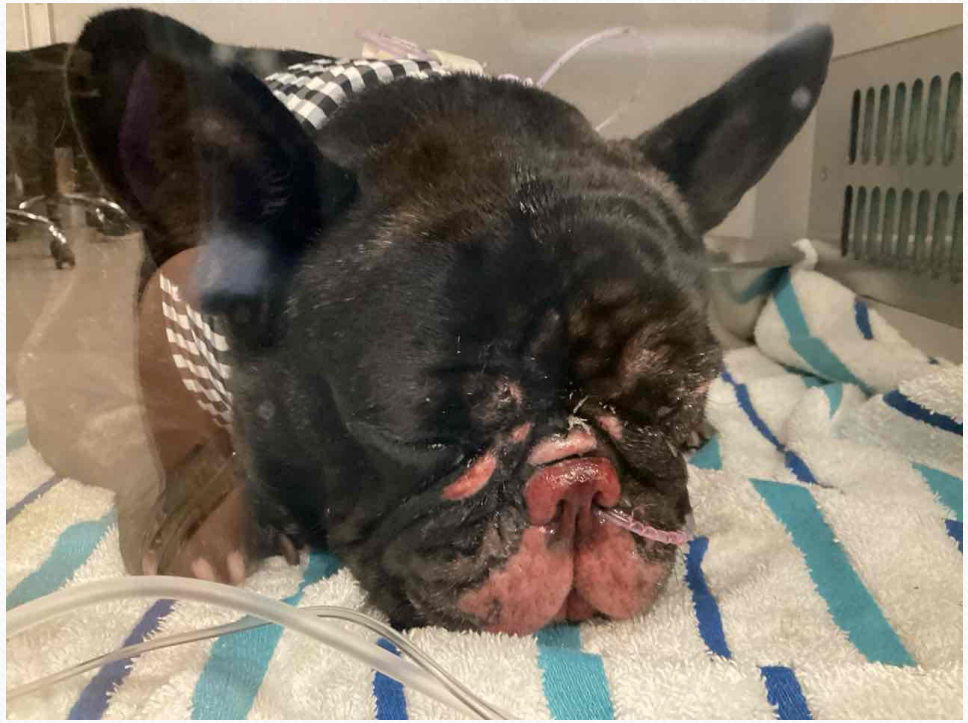
Do not edit
How to change the design



Given what we discussed today, what would your treatment plan look like for Loki? Techs what would you have ready, anticipate, and or advocate for?

ⓘ The Slido app must be installed on every computer you're presenting from slido

Ember the housefire French Bulldog



Do not edit
How to change the design



Given what we discussed today, what would your initial treatment plan look like for Ember? Techs: What would you be vigilant of, what would you advocate for?

ⓘ The Slido app must be installed on every computer you're presenting from slido

Key Clinical Takeaways

- Burns are **progressive injuries** → Protect the Zone of Stasis
- Treat the **whole patient**, not just the wound
- **Debridement + Aseptic diligent wound care drive outcomes**
- Topicals > Routine systemic antibiotics
- Pain control impacts **healing and recovery**
- Reassess daily—burn care is dynamic



Questions?

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ADDITIONAL CLINICAL RESOURCES

VIN. Burn management overview.
https://www.vin.com/apputil/content/defaultadv1.aspx?id=3866679&pid=11268

VetFolio. Thermal injury.
https://www.vetfolio.com/learn/article/thermal-injury

Vet Times. Burns: first aid, triage, wound care, and healing.
https://www.vettimes.com/clinical/small-animal/burns-first-aid-triage-wound-care-and-secondary-healing

Veterinary Nurse. Initial management of the burn wound patient.
https://www.theveterinarynurse.com/content/clinical/initial-management-of-the-burn-wound-patient

Veterian Key. Burns overview.
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