Rapid Fire: Management of burns and carbon monoxide toxicity for hospitalists

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Management of burns and CO toxicity

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Speakers report No Financial Conflicts of Interest

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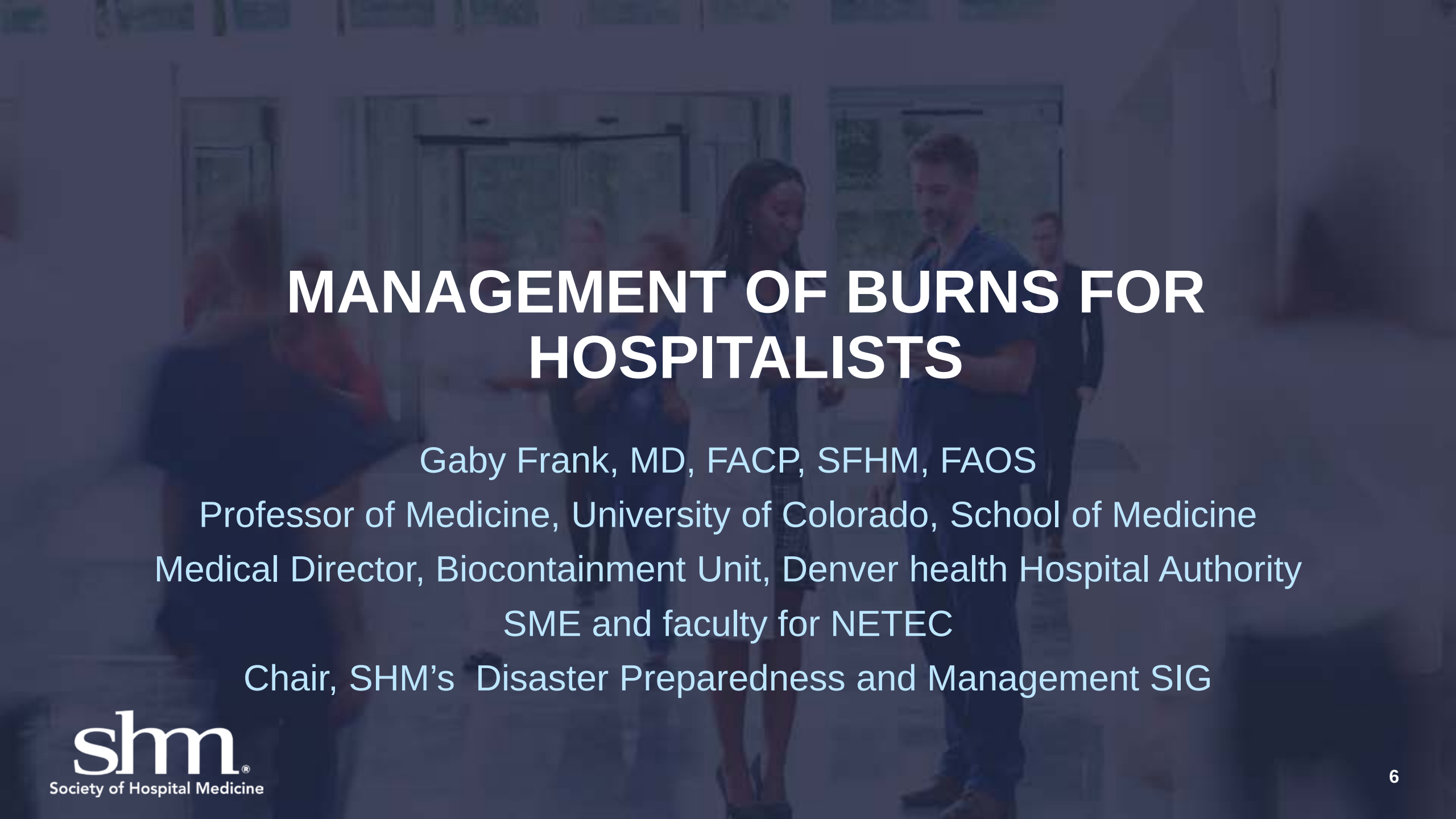

MANAGEMENT OF BURNS FOR HOSPITALISTS

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Learning Objectives

- Recognize need for hospitalists to become familiar with burn injuries classification and its implication to management
- Explore non-surgical management of burns.
- Identify criteria to transfer burn victims to a specialized burn unit/ facility (and ideal timing to pursue it).

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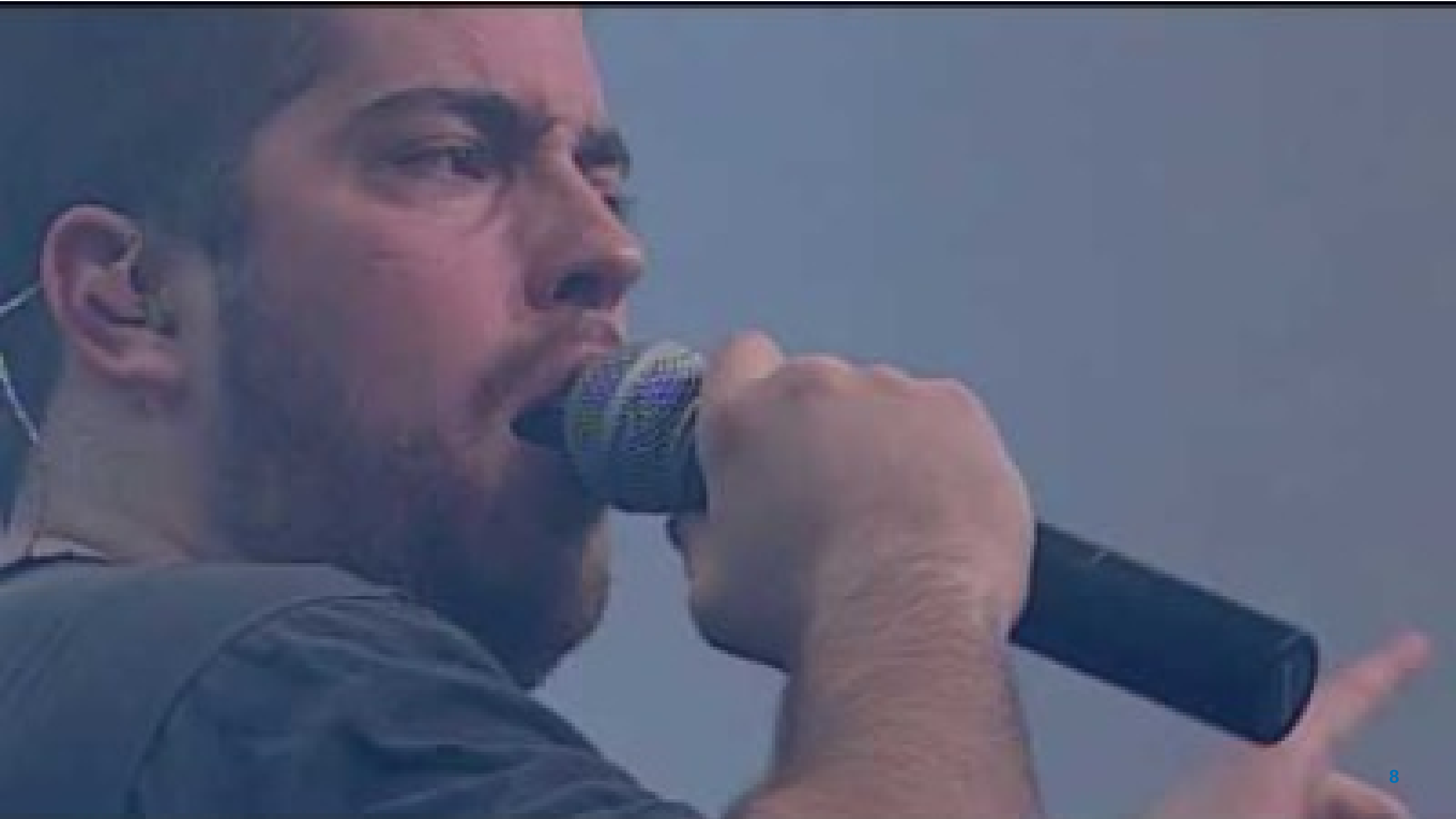

December 30th , 2004

- Callejeros
- República Cromañón
- ~ 3000
- Flare >> Ceiling Foam
- 4/6 doors
- 1492 injured
- 194 fatalities
- 225 ppm cyanide (lethal dose in rats 150-220 ppm)
- 74 patients/ 2 hours

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Ramos G, Flageat G, Queiroz G, et.al.. Massive hospital admission of patients with respiratory failure resulting from smoke inhalation injury: the Cromagnon Republic Tragedy. J Burn Care Res. 2006 Nov-Dec;27(6):842-7. doi: 10.1097/01.BCR.0000245412.23015.FE. PMID: 17091080.

Clinical Scenario

24 yo patient with wheezing and the following (see image) burn affecting left upper extremity. Other than applying Oxygen and managing the likely inhalation injury, what is your assessment of the burn?

a) It is a superficial burn, and I am not concerned

b) It is a partial thickness burn and I will start fluid resuscitation

c) I believe I will need to transfer patient to a specialized center

d) I don’t know, That is why I here today

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Introduction

- Approximately 1.25 million (80% minor)
- Approximately 486K seek treatment
- Admissions to burn centers
  - 43% fire/ flame
  - 34% scald
- 3rd pediatric injury leading to death
- 75 % burn victims die at the scene
- 96.8% US Burn center survival rate
- Always assume CO exposure
- Smoke inhalation increases mortality by 20% (responsible for 80% fire-related deaths)

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Introduction (cont.)

- **Thermal**—Heat or direct fire
- **Chemical**—contact, ingestion, inhalation, or injection of acids, alkalis, or vesicants
- **Electrical**—Low voltage or high-voltage power lines
- **Friction or abrasion**
- **Ultraviolet radiation**—sunburn.

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Management of Burns: Basic Concepts

Total Body Surface Area (TBSA)

Depth of Burn

Parkland's Formula

Transfer Criteria

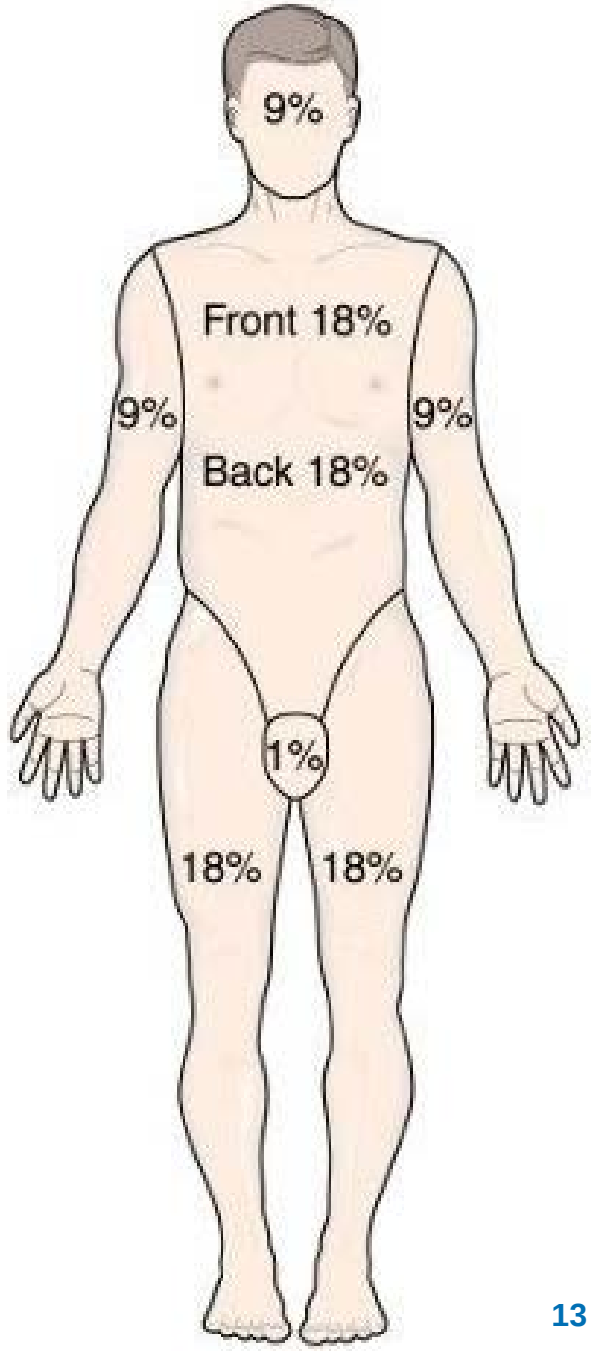
Golden Day

Airway Edema

1 MICU day/ % of TBSA

Circumferential Burns

Coexisting lesions/ injuries



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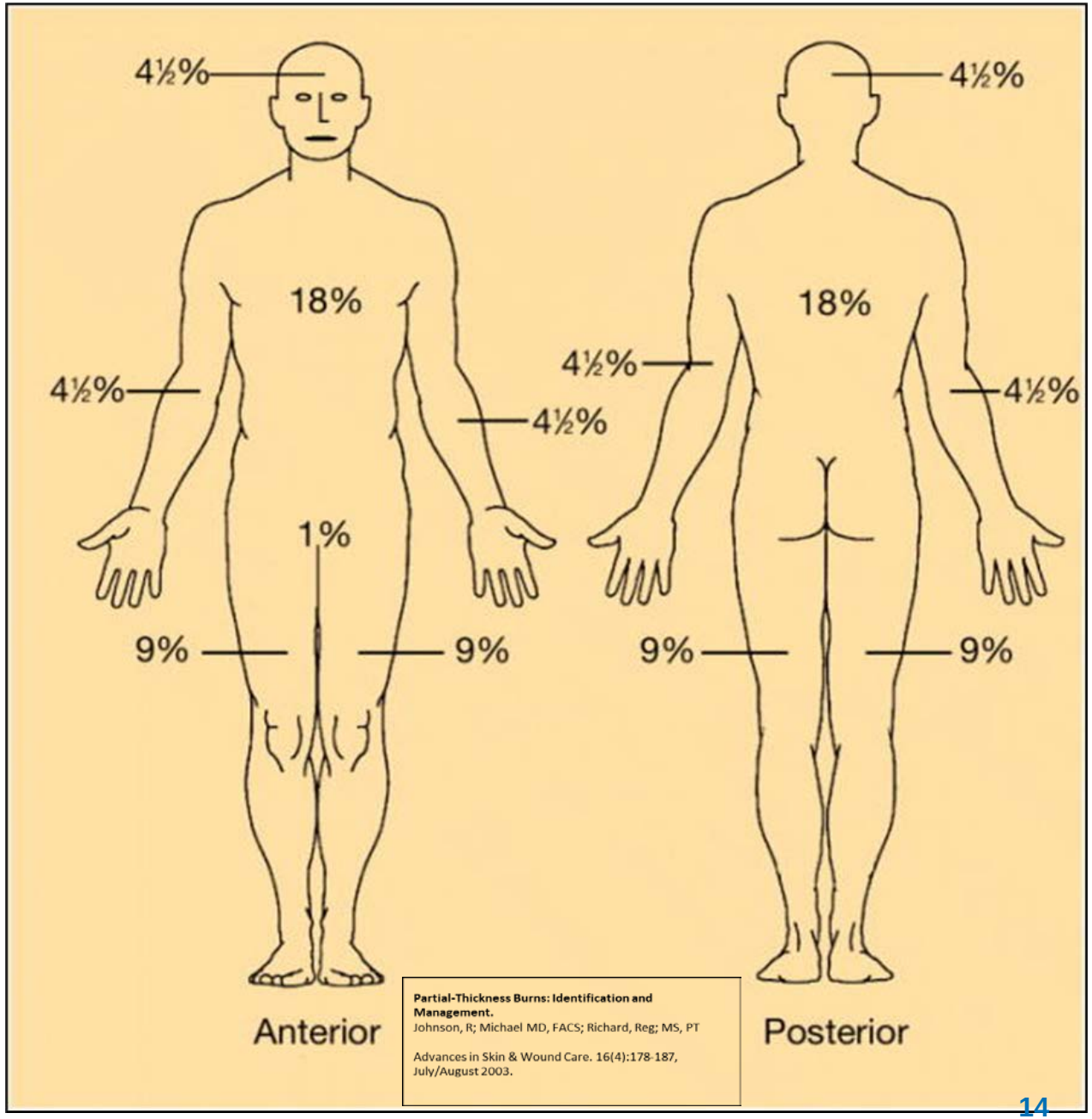
TBSA Assessment

- Rule of 9 (adults and adult-size kids)
- Palm of hand = 1% (think non-traditional body sizes/ shapes)
- Lund and Browder Chart (most accurate)



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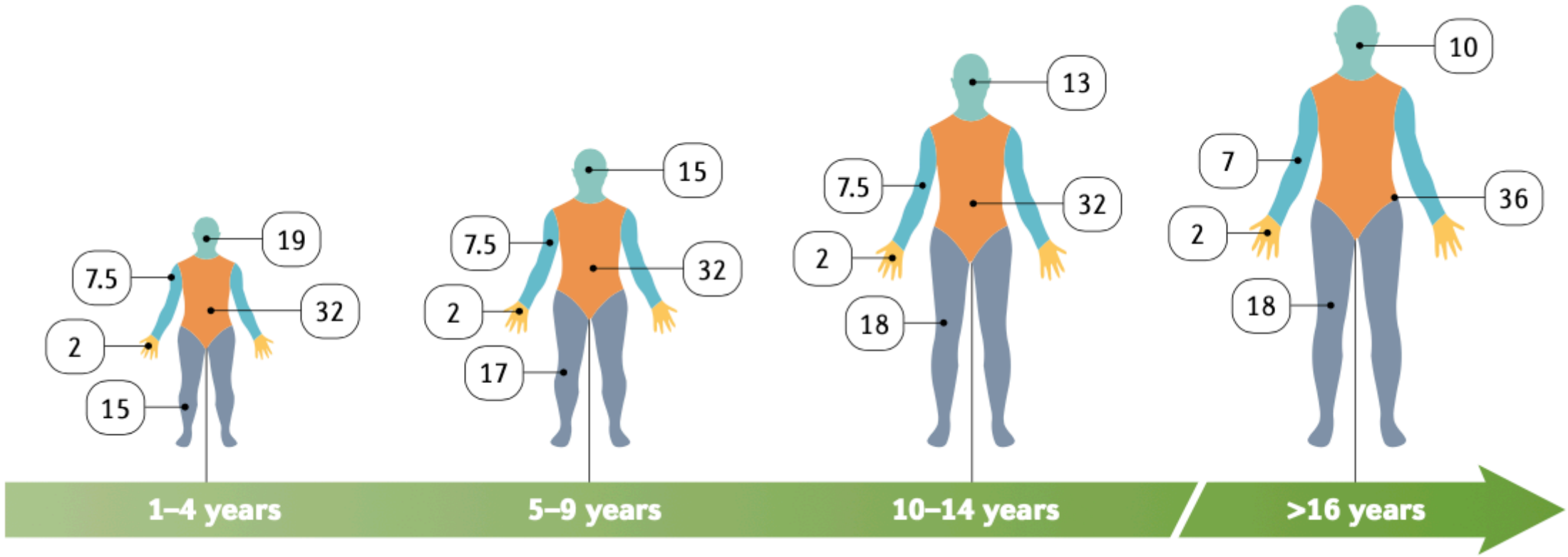
TBSA and age



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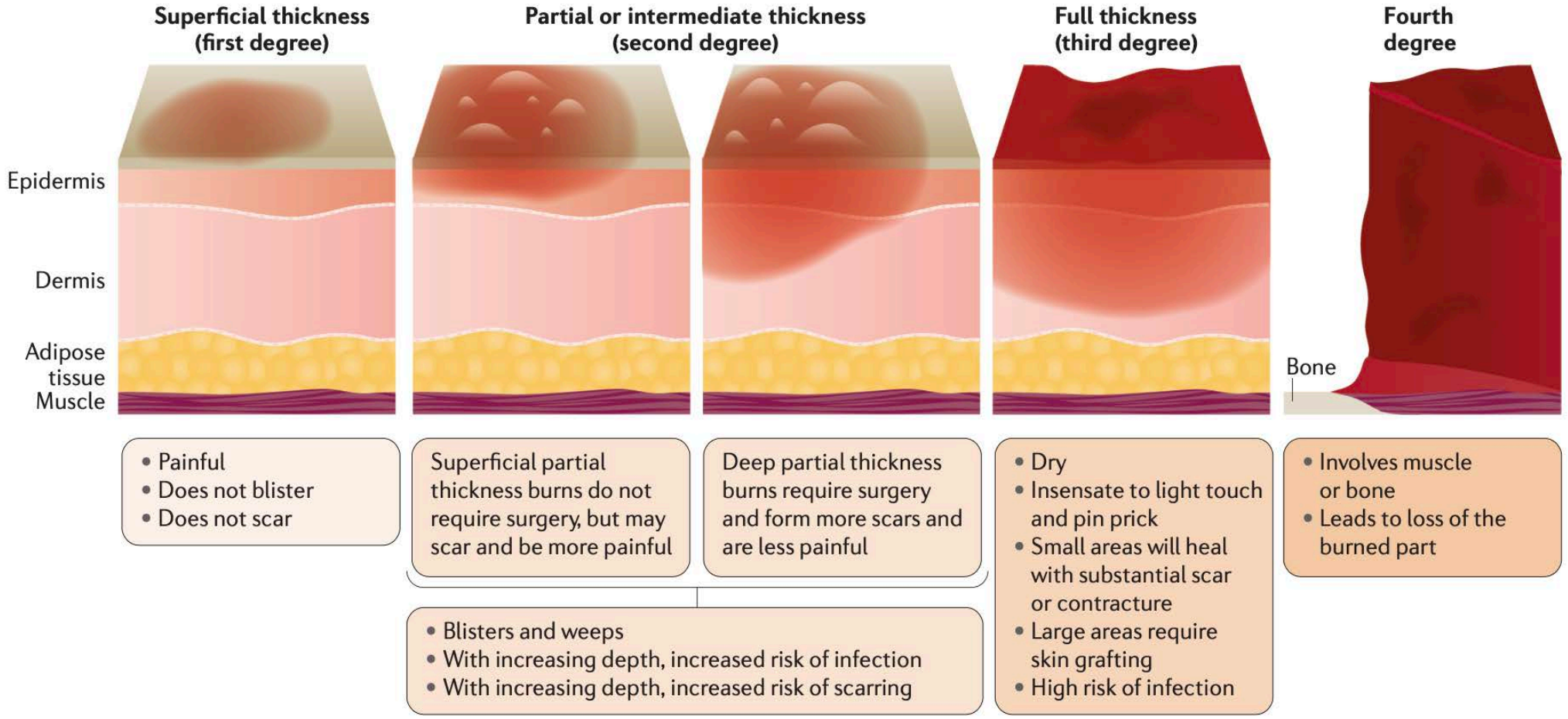
Depth Assessment



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Superficial Thickness Burns – First degree

- **Painful**
- **Do not blister**
- **Do not scar**
- **Appears pink**
- **Not included in TBSA calculation**

- **Think:**
  - Sunburn
  - Mild scalding burns

phil.cdc.gov

Partial or intermediate Thickness Burns- Second Degree

- **Blister and weep**
- **Painful**
- **Increased risk of infection and scarring with increased depth**
- **Usually heals within 2 weeks**
- Superficial partial thickness:
  - Don’t require surgery
  - May scar
  - More painful than deep partial thickness
- Deep partial thickness:
  - Require surgery
  - More scarring
  - Less painful than superficial partial thickness

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Full Thickness Burns- Third degree

- Dry, tough, may appear leathery
- White, brown, black or red and mottled
- Skin doesn’t blanch
- Decreased sensation to light touch and pin prick
- Small areas will heal with significant scarring and /or contractures
- Large areas require grafting
- High risk of infection

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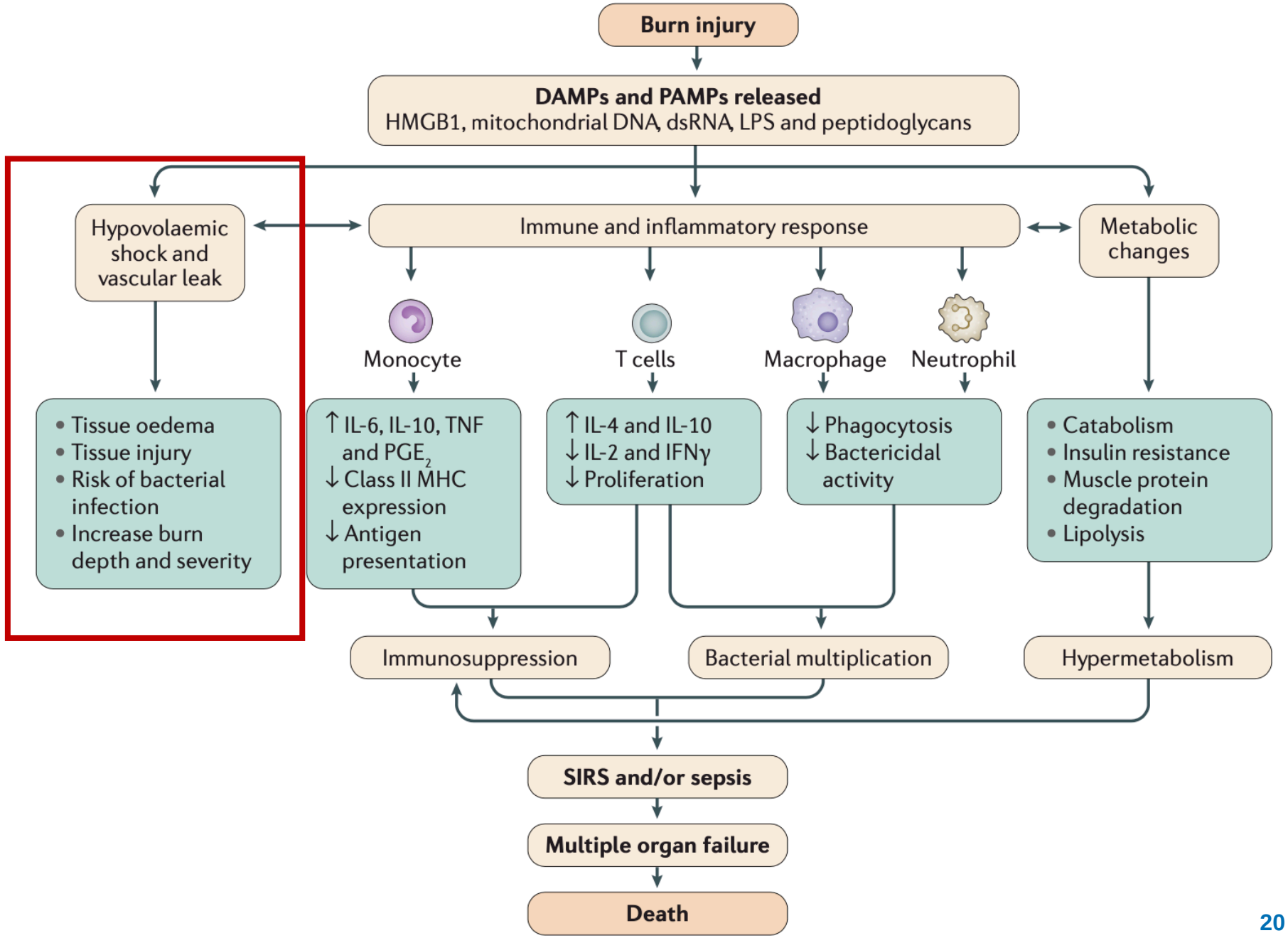
- **SHOCK**
- **HYPERMETABOLIC STATE**
- **IMMUNE DYSREGULATION AND INFECTION**



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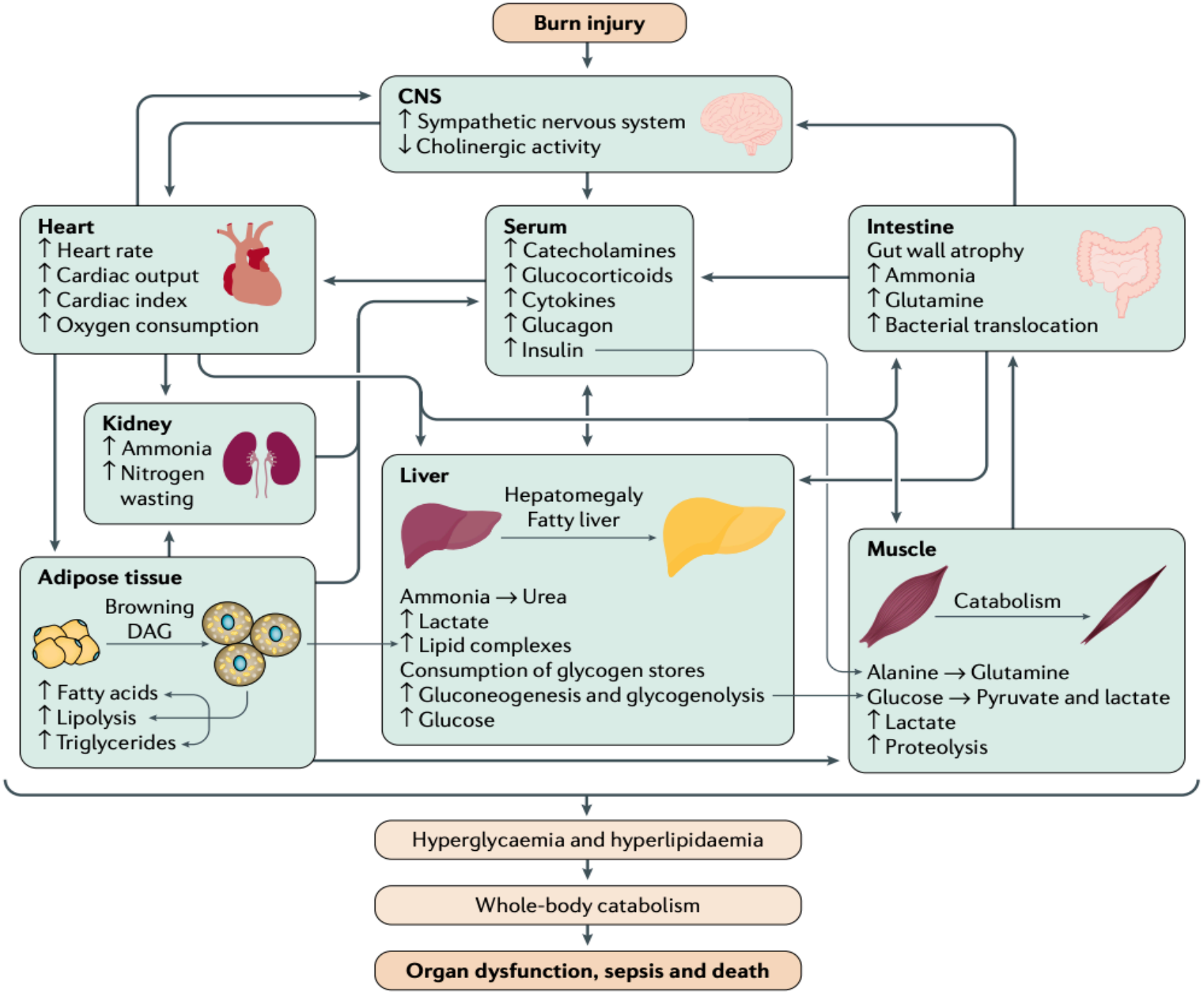

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Clinical Management of Burns

- **Stop the burn (Implement ATLS)**
- **Keep Warm and protect HCW**
- **Search for associated injuries**
- **Start O2 @ 100%**
- **ABLS Burn Resuscitation> Foley in all patients**
- LR 500 ml/h (>15 yo); 250 ml/h (6-14 yo); 125 ml/h (<6 yo).
- D5LR for children </= 5 yo (add'l maint.)
- No Fluid boluses
- Avoid Delay and over-resuscitation
- **Wound care, tetanus ppx**

Parkland Formula:

4 ml x TBSA (partial + full thickness) x weight (Kg)

Half in first 8 hours

2nd half in remaining 16 hours

Start for TBSA of PTB/ FTB > 20%

(adults); >15% (children); inhalation;

and electrical injury

Goal UOP >.5 ml/Kg/H (adults); 1

ml/Kg/H (children); 2 ml/Kg/H (infants)

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Burn Wound Healing Stages:

- Hemostasis- immediate
  - Vasoconstriction
  - PLT activation
  - Release of clotting and growth factors
- Inflammation- 24 hours- weeks/months
  - Monocytes/macrophages
  - Neutrophils
- Proliferation
  - Granulation
  - Angiogenesis
  - epithelialization
- Remodeling
  - Maturation of granulation tissue

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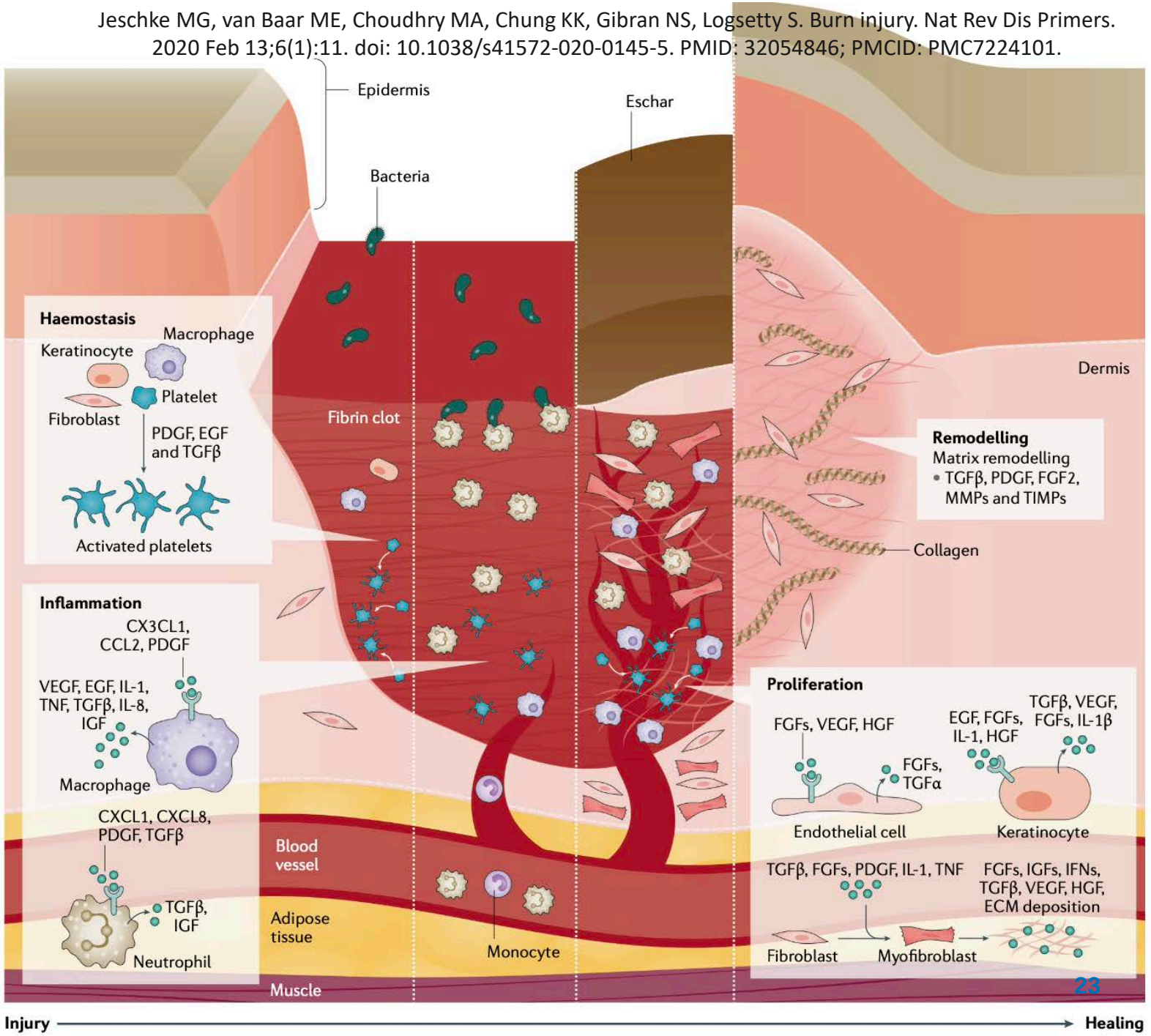

Wound care

- **Local wound care**
- **Topical antibiotics**
  - Silver sulfadiazine (Silvadene) and Mafenide acetate solution (Sulfamylon) **WORSE OUTCOMES**
  - Polymyxin B sulfate and bacitracin zinc (Polysporin)
  - Acetic acid (¼% solution). **Useless.**
  - Antibiotic gauze dressing coated with silver (Acticoat) can be left on for 7 days
- **Biological dressings (Brun centers)**
- **Non-biological dressings (i.e: Xeroform)**
- **Enzymatic debridement (Collagenase)**
- **Growth factors**
- **Surgical**

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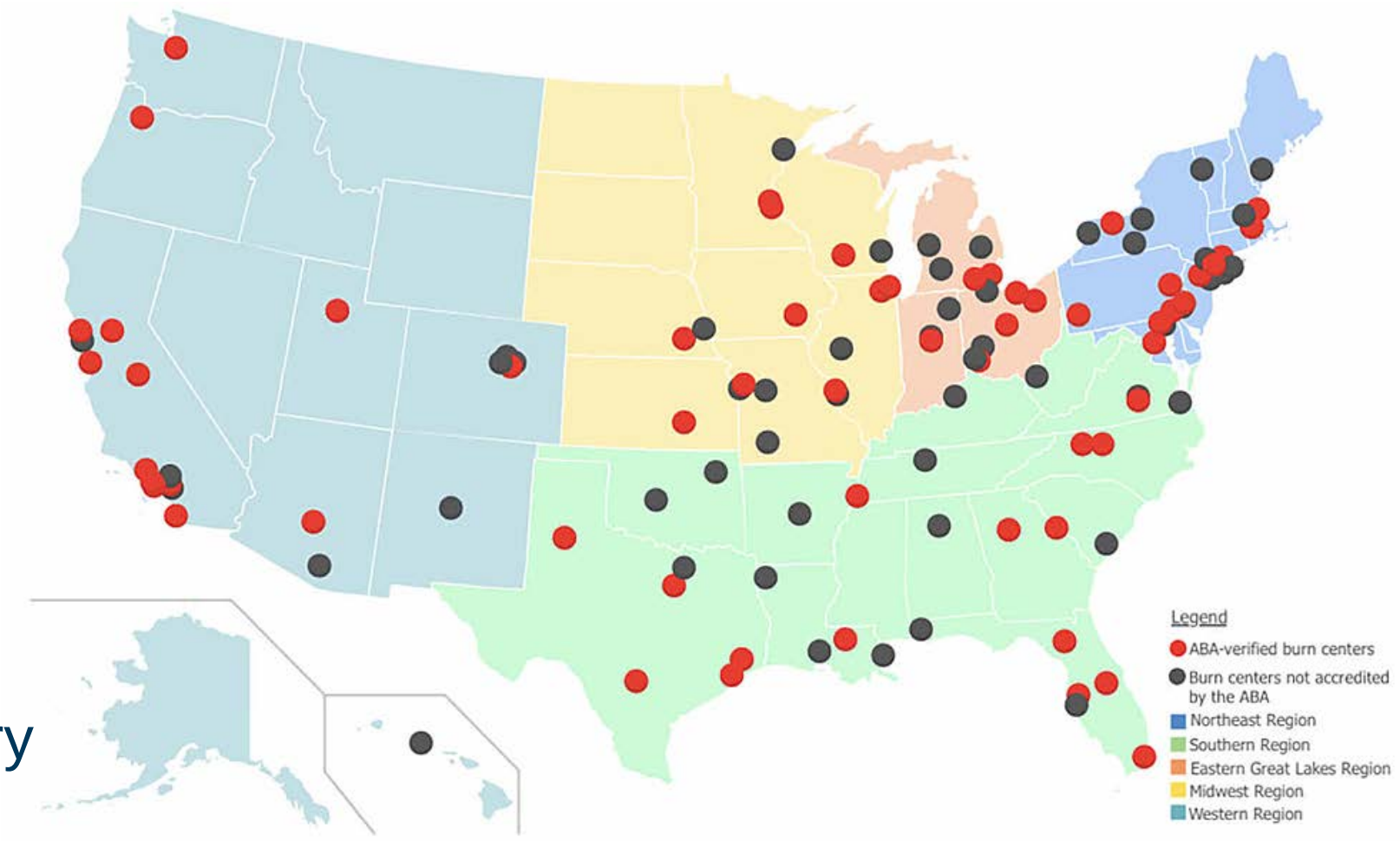
Transfer Criteria

- 2nd degree > 10% TBSA
- Involves:
  - face
  - hands
  - feet
  - genitalia, perineum
  - large joints
- 3rd degree (any TBSA)
- Electric burns
- Chemical burns
- Associated inhalation injury
- Patients with pre-existing conditions

Find your closest Burn center (127) at www.ameriburn.org



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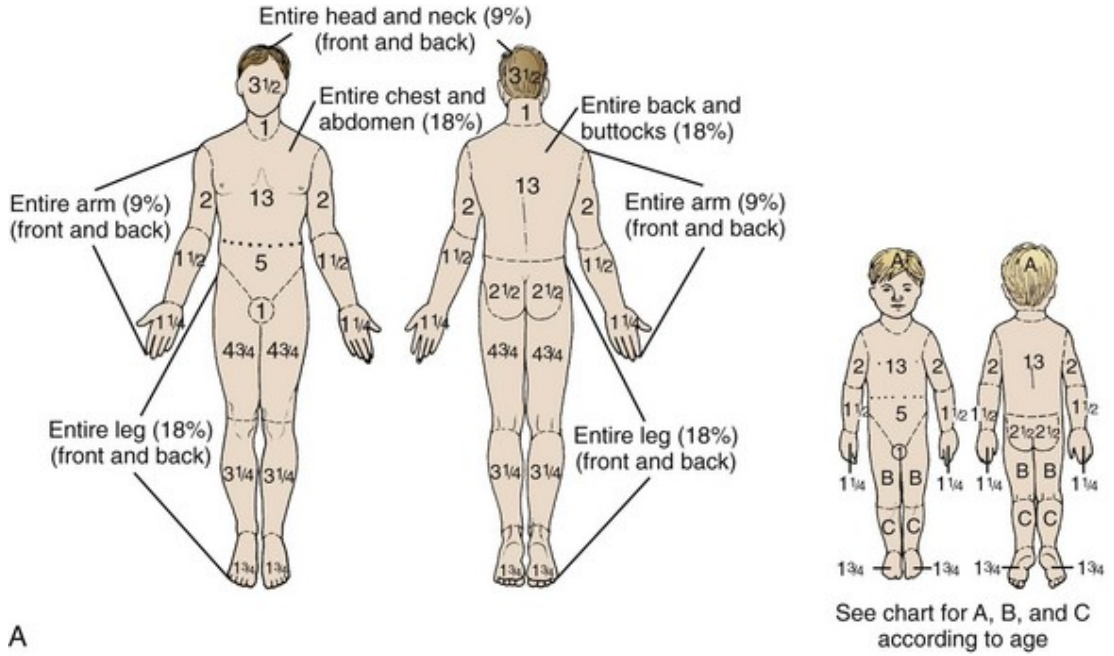
Asif M, Chin A, Lagziel T, et al. (June 22, 2020) The Added Benefit of Combining Laser Doppler Imaging With Clinical Evaluation in Determining the Need for Excision of Indeterminate-Depth Burn Wounds. Cureus 12(6): e8774. doi:10.7759/cureus.8774

Take Home Points

- Most hospitalized burn victims will survive.
- History is key
- Stop the burn, keep warm and clean
- O2, Foley, Resuscitation
- Avoid delay, over- and under-resuscitation
- Assess depth, TBSA, associated injuries
- Identify patients that will require transfer



AGE	Birth–1 yr	1–4 yr	5–9 yr	10–14 yr	15 yr	Adult
Head	19	17	13	11	9	7
Neck	2					
Ant trunk	13					
Post trunk	13					
R buttock	2½					
L buttock	2½					
Genitalia	1					
R U arm	4					
L U arm	4					
R L arm	3					
L L arm	3					
R hand	2½	6½	8	8½	9	9½
L hand	2½	6½	8	8½	9	9½
R thigh	5½	5	5½	6	6½	7
L thigh	5½	5	5½	6	6½	7
R leg	5					
L leg	5					
R foot	3½					
L foot	3½					

B BODY AREA

Modified Lund and Browder chart

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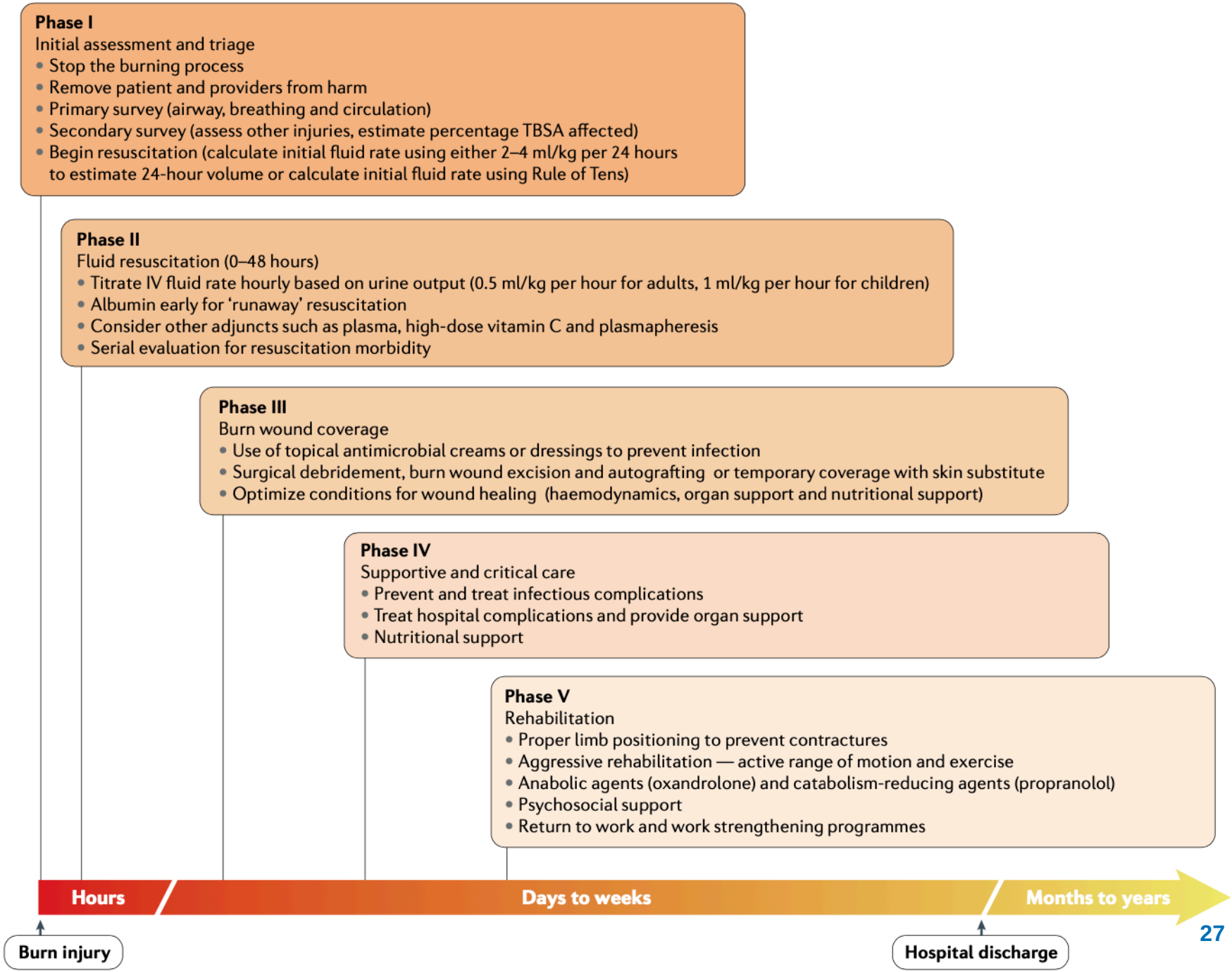
Phases in the Management of Burns



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Resources- Burn Management

- https://ameriburn.org/
- Disaster Management and Emergency Preparedness Course Student Manual. Chapter 8. Pathophysiology and patterns of Injury: Burns. American College of Surgeons. Second Edition. 2018
- https://mountainplainsrdhrs.org/specialty-care-videos/
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Thank you

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Rapid Fire: Management of burns and carbon monoxide toxicity for hospitalists:

Smoke Inhalation and Carbon Monoxide Toxicology

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Executive & Medical Director

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Rocky Mountain Poison Drug Safety, University of Colorado, Dept. of Medicine, Denver, CO

Objectives

01

The attendee will understand the various components of smoke inhalation.

02

The attendee will become familiar with pyrolysis toxicants local and systemic effects.

03

The attendee will become familiar with CO exposure and kinetics.

04

The attendee will understand current treatment approaches to CO.

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Smoke Inhalation

Its not just smoke

Heat
Soot (carbon particles +)
Cellular asphyxiants (CO, CN, H2S)
Irritants (acrolein, aldehydes, acids, particles)

Dynamic injury of airway and lung parenchyma evolving over hours

Smoke Inhalation

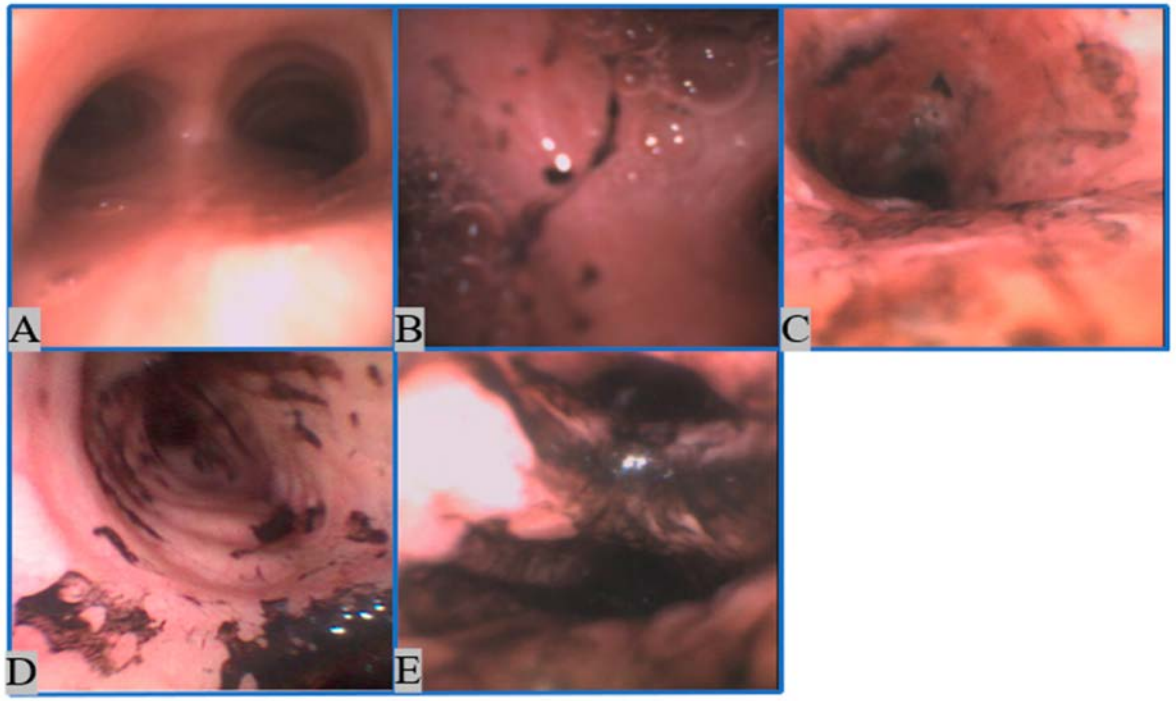
Upper Airway

Tracheobronchial Injury

Parenchymal Injury

Systemic Tox (CO, CN, H2S etc.)

Grading of inhalation injury using the abbreviated injury score (AIS). (**A**)—no injury; (**B**)—mild injury; (**C**)—moderate injury; (**D**)—severe injury; (**E**)—massive injury.



Li 2022 https://doi.org/10.3390/s22239430

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Abbreviated Injury Score Grading of inhalation injury by bronchoscopy

Grades

0 (no injury) – Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction

1 (mild injury) – Minor or patchy areas of erythema or carbonaceous deposits in the proximal or distal bronchi

2 (moderate injury) – Moderate degree of erythema, carbonaceous deposits, bronchorrhea, or bronchial obstruction

3 (severe injury) – Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, or obstruction

4 (massive injury) – Evidence of mucosal sloughing, necrosis, endoluminal obliteration

Smoke Inhalation

Leading cause of death from fires

Smoke contains numerous toxins from pyrolysis (burning)

Mixture of heated air, suspended particles, gases, fumes, aerosols, and vapors

Difficult to know composition of smoke – varies with fuels

Higher morbidity and mortality

Resp failure in 61% of burn victims with smoke inhalation versus 12% in burns only

History and Epidemiology

USA has one of highest fire death rates in the world

- 50-80% of deaths from smoke inhalation injuries

Injuries from various inhaled toxic xenobiotics and/or thermal burns

Injuries/death increase as more synthetic construction material used

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Fire Related Injuries

- Inhalation injury accounts 60-80% of fire-related deaths in the United States.
- Burn centers reduced the mortality from surface burns.
- The mortality from pulmonary injury has been increasing.
- Many have both smoke inhalation and thermal injury.
  - The co-presence of bronchopulmonary injury with cutaneous burns that exceed 30% of the total body surface area causes the mortality rate to increase more than 70%.
  - Other studies have shown that the incidence of inhalation injury increases with increasing burn size.

Components of Pyrolysis

Carbon soot particles not particularly toxic,

Delivery system for acid gases and other toxicants.

Cellular asphyxiants, including carbon monoxide (CO), nitrogen.

Mitochondrial cytochrome oxidase inhibition – Impairs ATP formation

Mechanisms:

displace oxygen from the air or

interfere with tissue oxygen delivery by blocking the action of hemoglobin or (e.g., CO, and/or cyanide)

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Fuel sources and products

Wool	CO, HCl, phosgene, chlorine, cyanide
Silk	Sulfur dioxide, HS, ammonia, cyanide
Nylon	Ammonia, cyanide
Wood, cotton, paper	CO, acrolein, acetaldehyde, methane, formic acid
PVC	CO, hydrogen chloride, phosgene
Rubber	Hydrogen sulfide, sulfur dioxide

Carbon Monoxide

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Carbon Monoxide Toxicology

Sources

Carbon containing material combustion

Confined or open exposure characteristics

Exposure

Present in minimal amounts 0.2-0.85% (heme oxygenase activity)

Up to ~ 10% COHb in heavy smokers

Automobile exhaust is about 1% (10,000 ppmv in states with exhaust testing)

- May be much higher is poorly functioning vehicles 30,000-40,000 ppmv

Dose

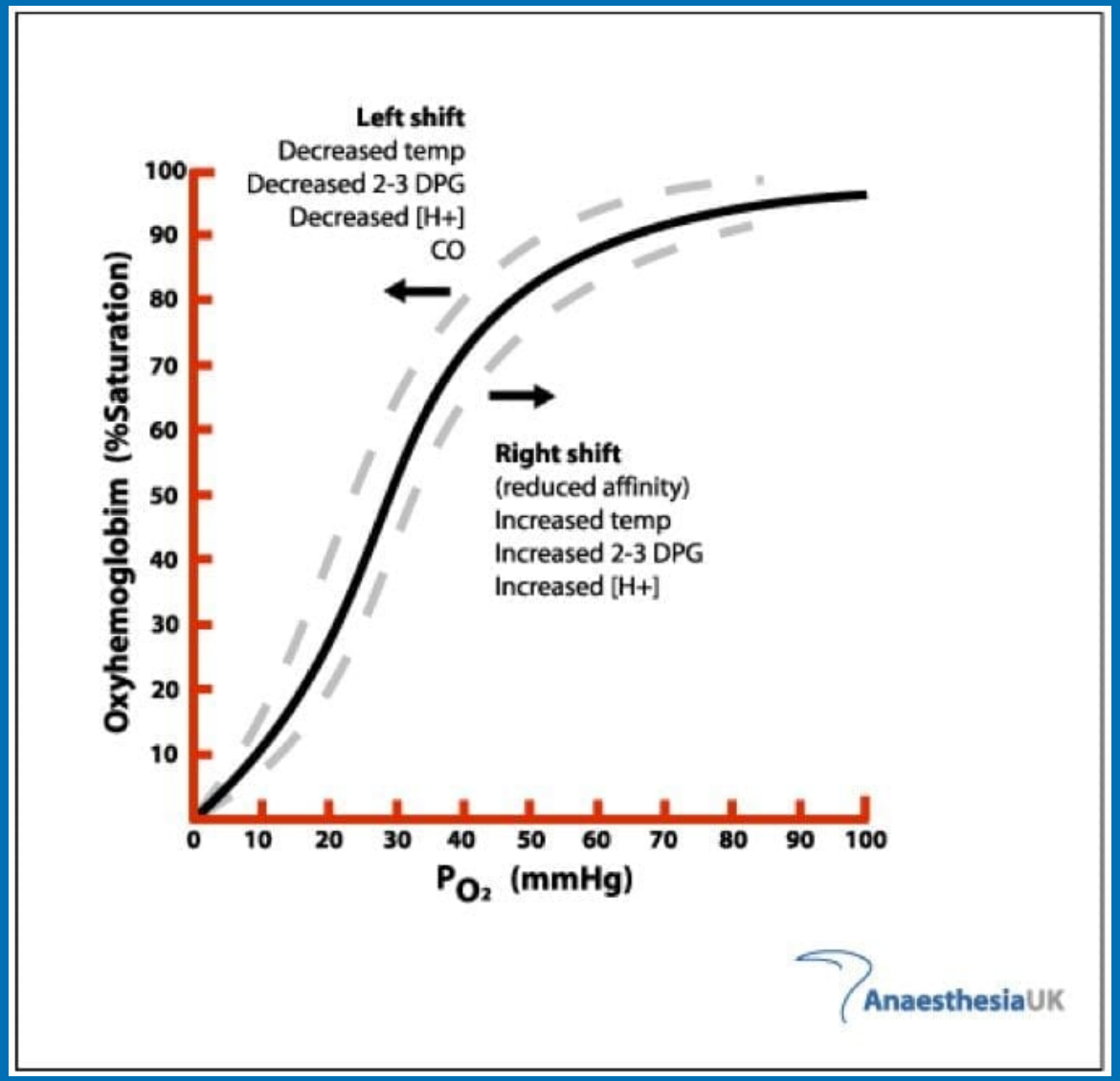
Function of concentration x time (plus: susceptibility & other exposures)

Soaking interval

Effects

Highly metabolic tissues

- Brain
- Heart



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CO Mechanism of Injury

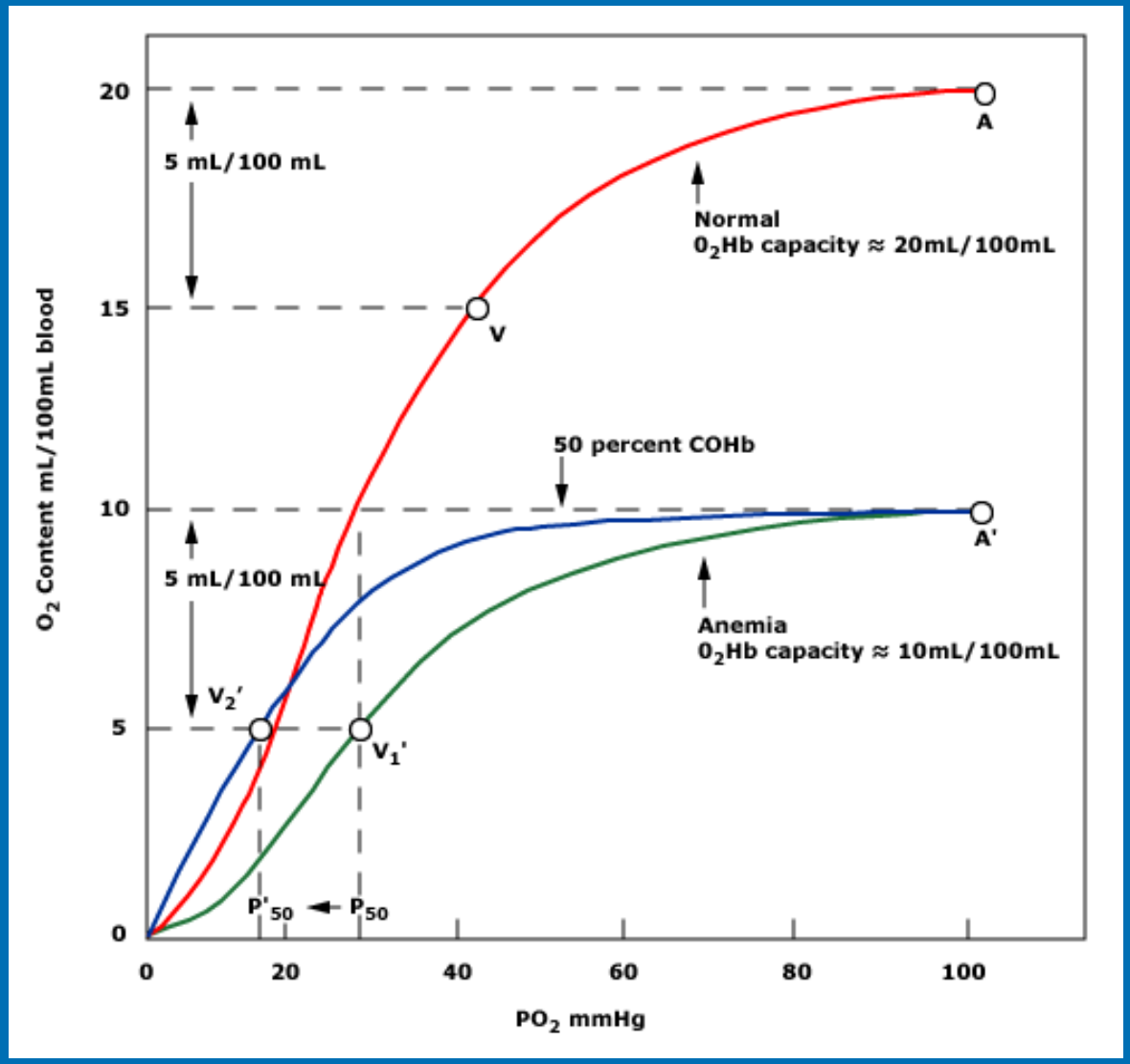
- **Impaired oxygen delivery**
  - CO binds to Fe moiety of heme, reduces off loading (left shift of oxyhemoglobin dissociation curve)
- **Impaired Oxygen utilization**
  - Cytochrome oxidase (Mitochondria Complex 4) inhibition
    - Blocks electron transfer, oxygen utilization and ATP formation
- **ROS**

•CO-induce production of ROS from different cellular sources: mitochondria, xanthine oxidase and NADPH oxidase.

•Stimulate oxidative stress with maximal impact on NADPH oxidase.

•CO-induced oxidative stress in neurons and astrocytes is a trigger for neuronal cell death.

CO Effect on O2 Content & Delivery



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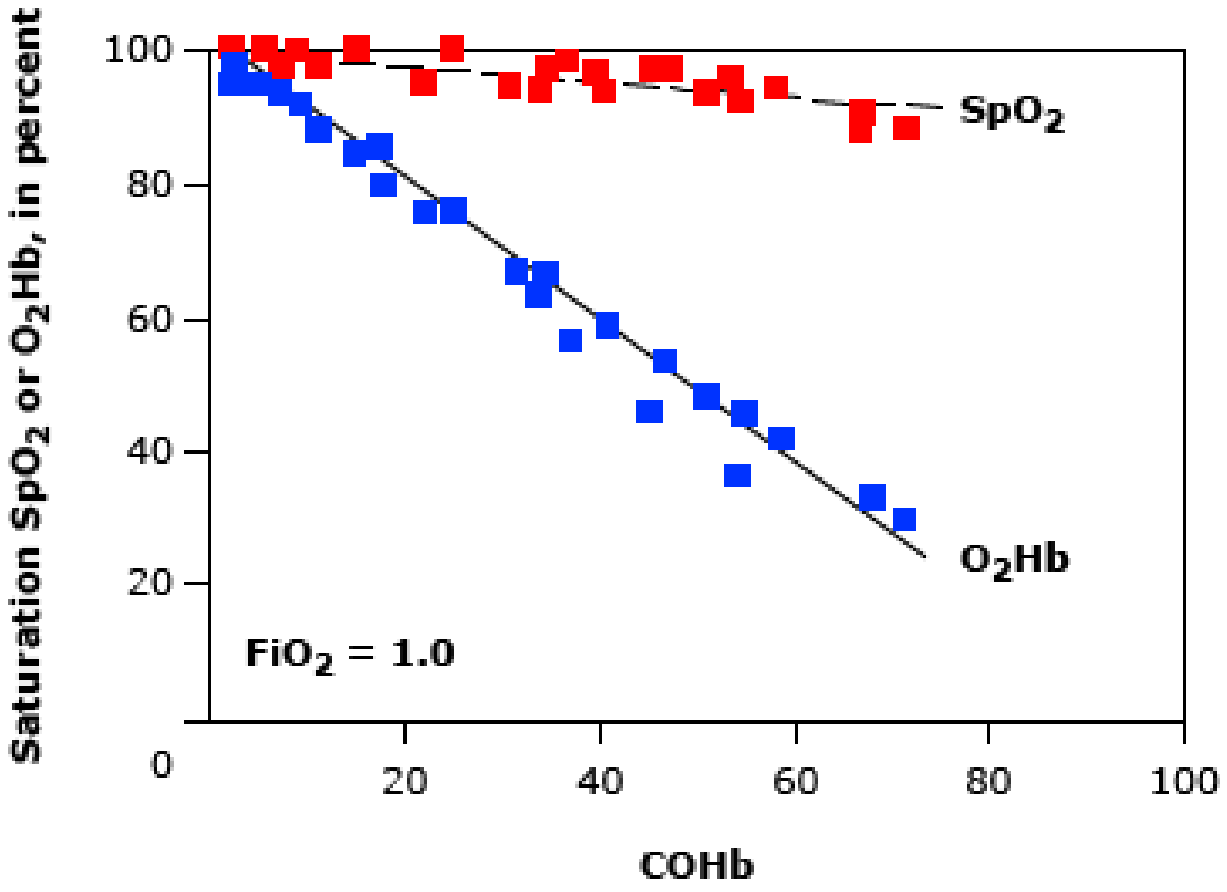
SpO2 and Co-oximetry vs carboxyhemoglobin (COHb)



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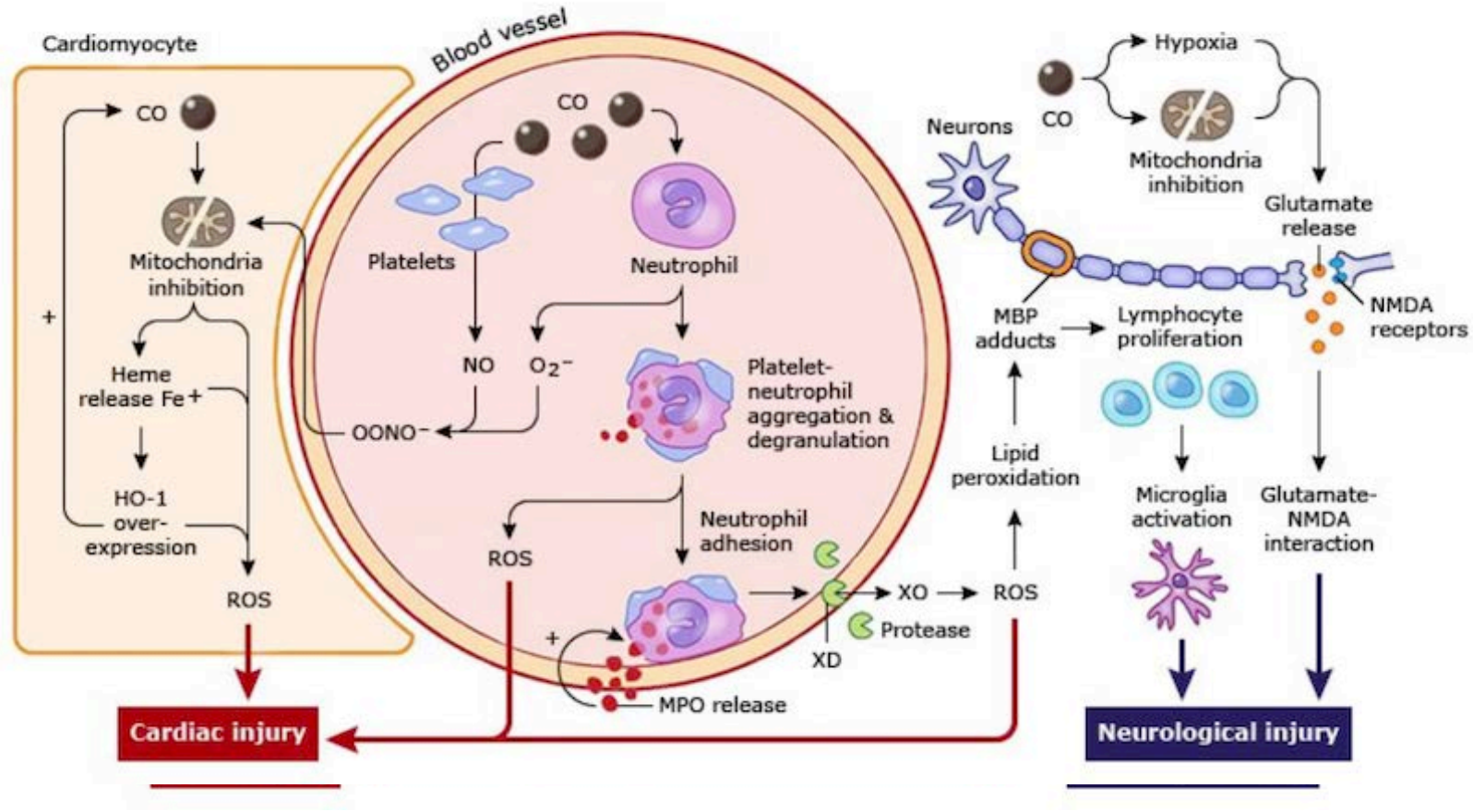
CO inflammatory response

Mechanism of Injury



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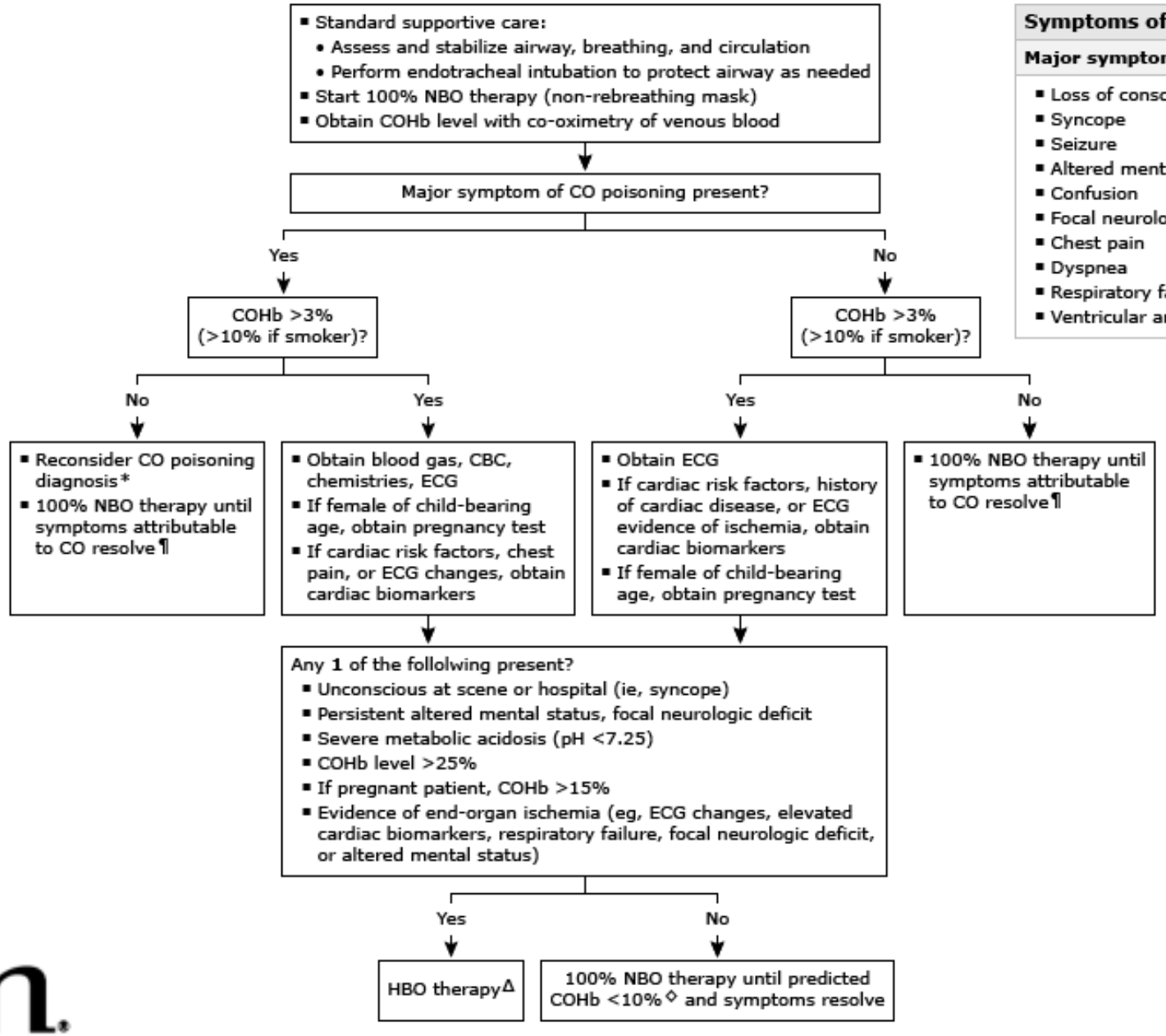

Symptoms of carbon monoxide poisoning

Major symptoms	Common symptoms
▪ Loss of consciousness ▪ Syncope ▪ Seizure ▪ Altered mental status ▪ Confusion ▪ Focal neurologic deficit ▪ Chest pain ▪ Dyspnea ▪ Respiratory failure ▪ Ventricular arrhythmia	▪ Headache ▪ Nausea ▪ Vomiting ▪ Dizziness ▪ Weakness ▪ Malaise ▪ Visual changes ▪ Difficulty concentrating

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What does all this mean?

Prevention is paramount

Smoke & CO detectors
No hydrocarbon powered machines use in an enclosed space
Exit plan for workplace and homes
Residential fire suppression systems

Patient Pearls

Smoke is a poly-tox exposure. (CO, CN, H2S, acrolein, aldehydes, acids, particles, etc.)
Cardio-pulmonary support

- pulmonary thermal or chemical injury

Likely significant metabolic acidosis from mitochondrial inhibition

- Lactate > 10 think CN
  - consider treating for CN with hydroxocobalamin

Treatment team (burn, crit care, pulmonologists, toxicologists, hyperbaricists)
If intentional, consider medication ingestions

Call your regional poison center for assistance – 1-800-222-1222 (nationwide)

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Thank you

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