BURNING UP THE TRACT

Chandi Idicula, MS, RRT, CPFT
El Centro College
Dallas, TX
STARDUST NIGHTCLUB
1981 Dublin, Ireland
48 DEAD
MGM GRAND HOTEL
1980 Las Vegas, Nevada
84 DEAD
COCOANUT NIGHTCLUB
1942 Boston, Massachusetts
492 DEAD
U.S. FIRES

1,375,000
FIRES REPORTED

16,500
INJURIES

2800
DEATHS

32 min
1 DEATH

2012 NFPA
DEADLY ENVIRONMENT

Over 1000°C
AMBIENT TEMPERATURE

<2%
OXYGEN

CO, HCN, HCl
TOXIC GASES

<1 meter
VISIBILITY

1981 Dublin Investigation
Over 80% RESPIRATORY RELATED INJURIES
RESPIRATORY INJURIES

- Hypoxic/Anoxic
- Toxic Gas
- Airway
OBJECTIVES

Pathophysiology
Assessment
General Management
Ventilator Management
RESPIRATORY INJURIES

Hypoxic/Anoxic

Toxic Gas

Airway
HYPOXIC / ANOXIC

Low or No $O_2$ in tissue

Causes damage to brain, liver, and other organs within minutes
## HYPOXIC ENVIRONMENT

<table>
<thead>
<tr>
<th>When oxygen levels are at...</th>
<th>...a person experiences:</th>
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<tbody>
<tr>
<td>21%</td>
<td>Normal outside air</td>
</tr>
<tr>
<td>17%</td>
<td>Impaired judgment and coordination</td>
</tr>
<tr>
<td>12%</td>
<td>Headache, dizziness, nausea, fatigue</td>
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<tr>
<td>9%</td>
<td>Unconsciousness</td>
</tr>
<tr>
<td>6%</td>
<td>Respiratory arrest, cardiac arrest, death</td>
</tr>
<tr>
<td>&lt;2%</td>
<td>In Fire Environments</td>
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Carbon Monoxide
Decreases Hgb’s ability to pick up and deliver oxygen to the tissues

>240x more Hgb affinity than O₂

Left shift O₂-Hgb Dissociation curve
60-70% house fire deaths attributed to CO poisoning/anoxia
Hydrogen Cyanide

Disrupts tissue’s ability to utilize oxygen

Disrupts aerobic metabolism in tissues, forcing anaerobic metabolism

Lactic acid buildup leading to metabolic acidosis
RESPIRATORY INJURIES

Hypoxic/Anoxic

Toxic Gas

Airway
Low $O_2$ lead to “incomplete combustion”

Byproducts of “incomplete combustion” and burning of various materials, such as PVC, vinyl, plastics, household products, etc.
Different toxic gases can cause different effects from minor, itchy eyes and a sore throat to severe pulmonary edema and death.
TOXIC GAS INJURY

Highly Water-Soluble Gases
Aldehyde, Ammonia, Sulfur Dioxide
Mainly results in **upper airway damage**
Causes acid-base imbalances – results in edema

Poorly Water-Soluble Gases
Phosgene, Hydrogen Chloride, Oxides of Nitrogen
Mainly results in **lower airway damage**
Dissolves into cell membrane and causes delayed effects
RESPIRATORY INJURIES

Hypoxic/Anoxic

Toxic Gas

Airway
Upper Airway
- Nasopharynx
- Oropharynx
- Laryngopharynx

Lower Airway
- Trachea
- Bronchi
- Bronchioles
- Alveoli

AIRWAY
Glottis
AIRWAY CONCERNS

Upper Airway

Patency of airway
Heats & Humidifies
Filters

Lower Airway

Gas Exchange
Circulation
AIRWAY INJURY

Upper Airway
Heat-related injuries ("heat sink")
edema → obstruction

Lower Airway
Gas exchange
Circulation
Steam injury (thermal burns)
Superheated gases burn the respiratory tract. When the air is hot enough, one breath can kill.

Over 1000°C
TEMPERATURE
IN FIRE ENVIRONMENT
SEVERITY OF BURN INJURIES

First-Degree Burns
(minimal depth in skin)

Second-Degree Burns
(to deep thickness of skin)

Third-Degree Burns
(including tissue beneath skin)
SEVERITY OF BURN INJURIES

First-Degree Burns
Reddened skin, tenderness, no blisters
Healing time is about 6-10 days

Second-Degree Burns
Extending into dermis, blisters present
Healing in 7-21 days

Third-Degree Burns
Extending into underlying tissue, healing after 21 days
May need skin graft, Scars and granulation may present if skin graft was not performed
STAGES OF RESPIRATORY INJURY
RESPIRATORY INJURIES

Early Stage
(0-24 Hours)

Intermediate Stage
(2-5 Days)

Late Stage
(5+ Days)
Smoke inhalation injuries do not appear right away.

Pulmonary status goes from normal to very ill abruptly.
EARLY STAGE

Tracheobronchial tree (TBT):
- Inflamed
- Lots of secretion
- Bronchospasm
- Slowed mucosal ciliary transport

Non-cardiogenic pulmonary edema

Adult Respiratory Distress Syndrome (ARDS)
INTERMEDIATE STAGE

Upper airway thermal injury begins to improve

Tracheobronchial Tree (TBT)
- Mucus production continues
- Mucosal ciliary transport continue to decrease
- Mucosa becomes necrotic and sloughs
- Mucus plugging and atelectasis
INTERMEDIATE STAGE

Bacterial colonization
Gram + and Gram –

Inability to breathe deep and cough due to pain, narcotics, & airway resistance

Decrease lung and chest compliance
Infection from burns continues

Sepsis-induced multi-organ failure  
(common cause of death)

Pneumonia  
(a major problem)

Pulmonary embolism  
(from DVTs, within 2 weeks typically)

Restrictive lung disorder

Obstructive lung disorder
ASSESSMENT & DIAGNOSIS
ASSESSMENT & DIAGNOSIS

History
Found down or escaped?

Clinical Presentation

Diagnostic Procedures
Initial Chest X-Ray (routinely normal)
Fiber-Optic Bronchoscopy (gold-standard)
FIBER-OPTIC BRONCHOSCOPY (FOB)

Used to grade Injury
Initially may be negative
Intubation is typically required
FOB FINDINGS

Edema
Hyperemia
Soot
Mucosal Sloughing
Ulceration
Carbon Deposits
Sheridan R. A brief review of smoke inhalation. 1998
IMMEDIATE ASSESSMENT

Respiratory status
Cardiovascular status
The percentage of body burned (TBSA)
Depth of burns
INTUBATION

(Advanced Trauma Life Support – ATLS)

Absolute indications
- Burns > 40% TBSA
- Infant scald injuries > 20%
- Stridor

Relative indications
- Singed nasal hair
- Carbonaceous sputum
- Facial burns
- Burns to head and neck area
FLUID MANAGEMENT

Place an IV for fluids and meds

Use Ringer’s Lactate according to the Parkland formula (for first 24 hour)

\[
\text{Fluid required (RL)} = \text{TBSA (% burned) x } \frac{4 \text{mL}}{\text{kg}}
\]

Ex. 9L (RL) required = 30% burned x \( \frac{4 \text{mL}}{75 \text{kg}} \)
Monitor hemodynamic status
Urine output 30 - 50 ml / hr
CVP 2-6 mm Hg

Watch out for:
Overhydration
Pulmonary edema
Upper airway obstruction
TREATMENT FOR CO TOXICITY

\[ t_{1/2} \text{ (CO on room air)} = 240 \text{ min} \]
\[ t_{1/2} \text{ (CO on 100% oxygen)} = 40 \text{ min} \]

Administer 100% oxygen

Goal of therapy COHB <5%
HYPERBARIC OXYGEN THERAPY

Possibly for serious exposure
(\text{COHB} > 25\%)

Must be stable
Hemodynamic stability
No ongoing burn resuscitation
No transport needed for good general burn care
IS HBOT JUSTIFIED?

Reviewed 7 randomized controlled studies
Four – no benefit
Two – neurological improvement
One – unknown outcome

Our Conclusion:

The benefits do not outweigh the risks
Treatment is controversial

No simple rapid blood assay is available

“Cyanide Antidote Kit” – significant side effects
**CYANIDE TOXICITY TREATMENT**

**Hydroxocobalamin**
A hydroxylated active form of vitamin B12
(Drug developed due to chemical warfare concerns)

**Side effects**
Hypertension, bradycardia, changes in liver enzymes and electrolytes

**Aggressive supportive therapy**
to restore cardiovascular – enhances hepatic clearance
LOWER AIRWAY INJURY MANAGEMENT

- Supplemental humidified $O_2$
- Pulmonary Toilet
- Bronchodilators
- Aerosolized Heparin-Albuterol-Mucomist (HAM)
- Mechanical ventilation

Desai MH. J Burn Care Rehabilitation 1998
MECHANICAL VENTILATION

Goals:
- Prevention of ventilation-induced lung injury
- Aggressive pulmonary toilet
- Recruitment and stabilization of collapsed alveoli

Ventilation Strategy
There is no ideal respiratory support strategy
Patients with ALI & ARDS

Control: Tidal Volume 12ml / kg
Experimental: Tidal Volume 6ml / kg

Results:
Lower tidal volume → decreased mortality & increases the number of days without ventilation

Problems:
Burns > 30% TBSA excluded
Unclear how many inhalation injury patients

ARDS NETWORK STUDY
RG Brower MD, NEJM May 4, 2000 vol. 342 No. 18 P. 1301-09
Hypoxemia in early ARDS induced from:
- Systemic inflammation (sepsis)
- Alveolar edema → Intrapulmonary shunting

Hypoxemia in early smoke inhalation induced from:
- Small airway damage and obstruction → Intrapulmonary shunting
GENERAL VENTILATOR STRATEGY

- Prevent ventilator-induced lung injury
- Aggressive pulmonary toilet
- Recruit collapsed alveoli
HIGH FREQUENCY PERCUSSIVE VENTILATION

Decreased incidence of ventilator-associated pneumonia (VAP)

Decreased mortality

Acceptable $O_2$ saturation ($\geq 88-90\%$)

PEEP to support oxygenation

Low tidal volume (6-8 ml/kg)

Larger tidal volumes may be needed (12-15 ml/kg)

Plateau pressure 30-35 cm H$_2$O

Chest wall burn, plateau pressure may not be accurate

Permissive hypercapnia (pH $\geq 7.20$)
Pulsatile flowrate (PIP)

Oscillatory PEEP

Demand PEEP
VOLUMETRIC DIFFUSIVE RESPIRATION (VDR)

- Pneumatically powered
- Pressure limited
- Time cycled
- 200-900 breaths per minute
- Active inspiration
- Passive exhalation
- Stacks sub-tidal volume breaths to set PIP
Objective
Compare the 2 vent modalities in BICU Population: 62 burn patients >18 yrs requiring mech ventilation >24 hrs.

Setting
16 bed BICU at a military teaching hospital

Intervention
Randomly assigned 31 and 31
Results

Similar outcome (ventilator-free days in the 1\textsuperscript{st} 28 days) for burn patients with respiratory failure.

However, the low tidal volume strategy failed to achieve ventilation and oxygenation goals.

Kevin K Chung Critical Care Med 2010 Vol. 38. No 10
PRESIDENTIAL CITIZENS MEDAL

Dr. BIRD (2008)
The Inventor of all Birds, IPV, and VDR
NATIONAL MEDAL OF TECHNOLOGY AND INNOVATION

Dr. BIRD (2009)
The Inventor of all Birds, IPV, and VDR
QUESTIONS?

Chandi Idicula
iidicula@dcccdd.edu