INHALATION INJURY AND AIRWAY MANAGEMENT

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Outline

- Definition
- Anatomy and Pathophysiology
- Systemic Toxicities
- Diagnosis
- Management
- Prognosis
- Flash Burns (Smoking on Home Oxygen)

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Inhalation Injury

- **500k burns/ year, ~3.5k burn deaths/year, >60% of deaths are due to multi-organ failure**
  - **>30% have inhalation injury --> significantly contributes to an increased mortality**
    - **Increased fluid resuscitation requirements if cutaneous burns present**
- **Definition**: Nonspecific damage to the respiratory tract or lung tissue from heat, smoke, or chemical irritants
- **Classification based on location:**
  - Upper Airway
  - Lower Airway
  - Systemic Toxicity

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Table 16.1 Origin of Selected Toxic Compounds

Gases and Chemicals	Material	Source
Carbon monoxide	Polyvinyl chloride Cellulose	Upholstery, wire/pipe coating, wall, floor, furniture coverings Clothing, fabric Wood, paper, cotton
Cyanide	Wool, silk, cotton, paper, plastic, polymers Polyurethane Polyacrylonitrile Polyamide Melamine resins	Clothing, fabric, blankets, furniture Insulation, upholstery material Appliances, engineering, plastics Carpeting, clothing Household and kitchen goods
Hydrogen chloride	Polyvinyl chloride Polyester	Upholstery, wire/pipe coating, wall, floor, furniture coverings Clothing, fabric
Phosgene	Polyvinyl chloride	Upholstery, wire/pipe coating, wall, floor, furniture coverings
Ammonia	Wool, silk Polyurethane Polyamide Melamine resins	Clothing, fabric, blankets, furniture Insulation, upholstery material Carpeting, clothing Household and kitchen goods
Sulfur dioxide	Rubber	Tires
Hydrogen sulfide	Wool, silk	Clothing, fabric, blankets, furniture
Acrolein	Cellulose Polypropylene Acrylics	Wood, paper, cotton, jute Upholstery, carpeting Aircraft windows, textiles, wall coverings
Formaldehyde	Melamine resins	Household and kitchen goods
Isocyanates	Polyurethane	Insulation, upholstery material
Acrylonitriles	Polyurethane	Insulation, upholstery material

From Prien T, Traber DL. Toxic smoke compounds and inhalation injury: a review. *Burns* 1988;14:451–460.

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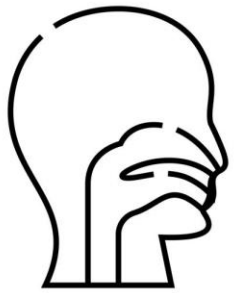
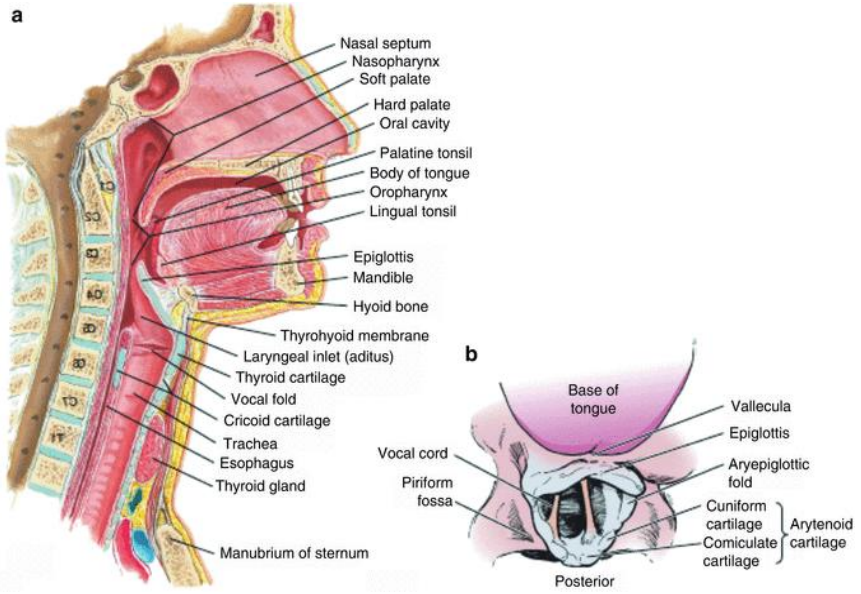
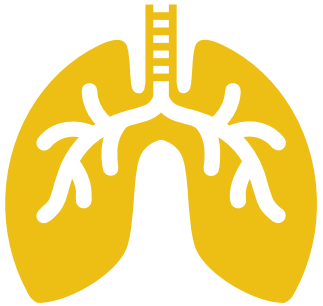


Upper Airway

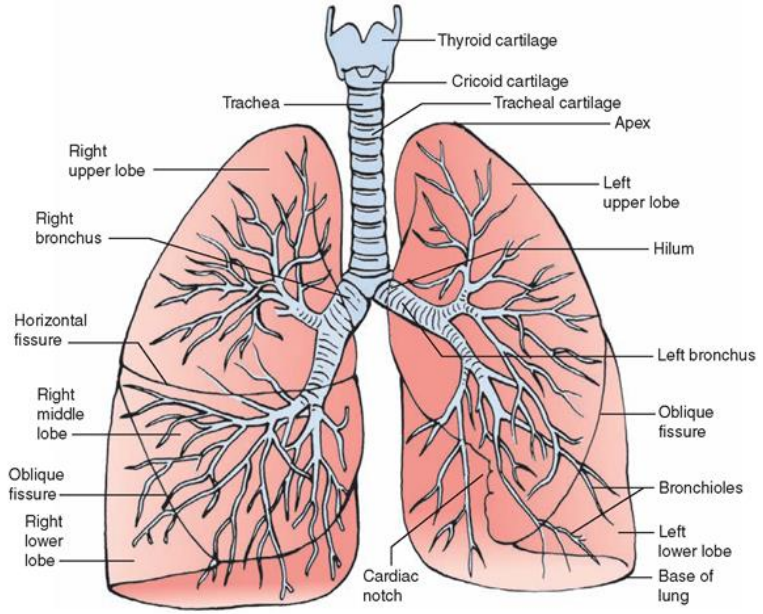
Anatomy





Lower Airway

Anatomy



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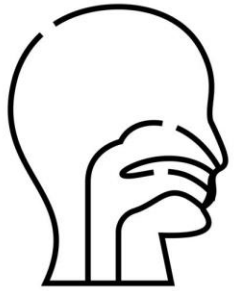
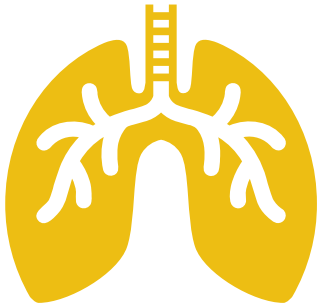


Upper Airway

Pathophysiology

- Thermal injury. Heat denatures proteins.
- Complement cascade activated, histamine released, ROS released, NO in endothelium = ↑permeability = edema
- Erythema, ulceration
- Upper airway protects lower airway via heat exchange.



Lower Airway

Pathophysiology

- Injury caused by chemicals in smoke
- Stimulates neuropeptides = induce bronchoconstriction, nitric oxide synthase (NOS) to generate ROS, and vasodilation
- ↑bronchial blood flow = delivers PMN and cytokines = inflammatory response
- disrupted bronchial epithelium= loss of proteins = exudate and cast formation --> alveolar collapse

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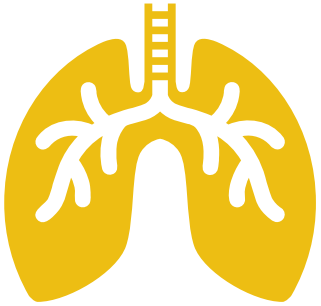

Parenchymal Injury

Pathophysiology?

-Delayed presentation

-Time difference from injury to decrease PaO2:FiO2 is correlated with severity of injury

-Atelectasis = increased transvascular fluid flux, decrease in surfactant, loss of hypoxic pulm vasoconstriction, increase permeability of small particles = impaired oxygenation

-Airway obstruction and atelectasis increases the risk for pneumonia

How To Diagnose Inhalation Injury

1. **History and Physical:**
   a. **Mechanism** (flame, smoke, steam)
   b. **Exposure** (duration)
   c. **Location** (enclosed space)
   d. **Disability**

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Physical Exam

Increased WOB

Change in Voice

Stridor

Issues Swallowing

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Case Study

59M presents to the ED trauma bay as a trauma delta. He was brought in by EMS after being found outside of a burning building.

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Labs and Adjuncts

Labs and Adjuncts

- **Trauma labs:** CBC, CMP, Mag, Phos, Lactate, UA
- COHb
- +/- Trauma Scans
- Diagnostic laryngoscopy

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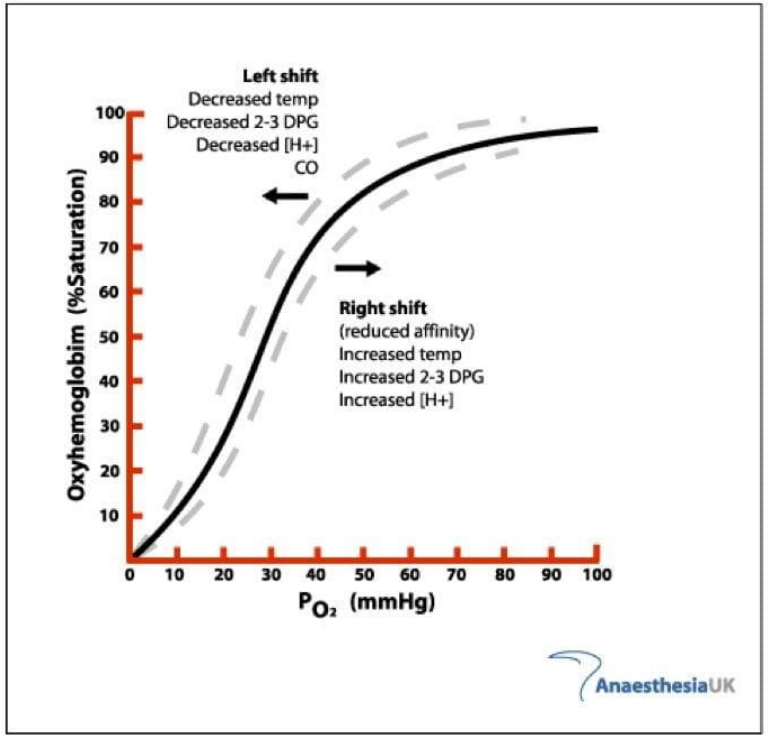
Carbon Monoxide Poisoning

- Odorless, colorless gas produced by combustion of many fuels
- Binds to hemoglobin = COHb (200-250x higher affinity than that of O2)
  - Competitive binding CO = decreased delivery of O2 to tissues = hypoxia



Carbon Monoxide Poisoning

- Sx manifest in organs with high O2 utilization
- **Dx**:
  - COHb levels
- **Tx:**
  - Cyanokit
  - 6h FiO2 100%

Table 16.2 Symptoms and Signs at Varying Concentrations of Carboxyhemoglobin (COHb)

COHb %	Symptoms
0–10	None
10–20	Tightness over forehead, slight headache, dilation of cutaneous blood vessels
20–30	Headache and throbbing in the temples
30–40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting, collapse
40–50	As above; greater possibility of collapse, syncope, increased pulse and respiratory rate
50–60	Syncope, increased pulse and respiratory rate, coma, intermittent convulsions, Cheyne-Stokes respirations
60–70	Coma, intermittent convulsions, depressed cardiac and respiratory function, possible death
70–80	Weak pulse, slow respirations, death within hours
80–90	Death in less than 1 h
90–100	Death within minutes

From Einhorn IN. Physiological and toxicological aspects of smoke produced during the combustion of polymeric materials. *Environ Health Perspect.* 1975;11:163–189; and Schulte JH. Effects of mild carbon monoxide intoxication. *Arch Environ Health* 1963;7:524–530.

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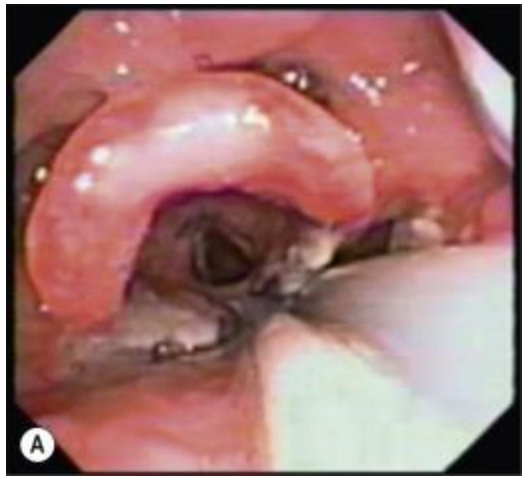
Diagnostic Laryngoscopy

- Upper airway thermal injury
- Performed in ED
- **Findings**:
  - Airway edema
  - Inflammation
  - Mucosal necrosis
  - Soot, charring
  - Tissue sloughing
  - Carbonaceous material



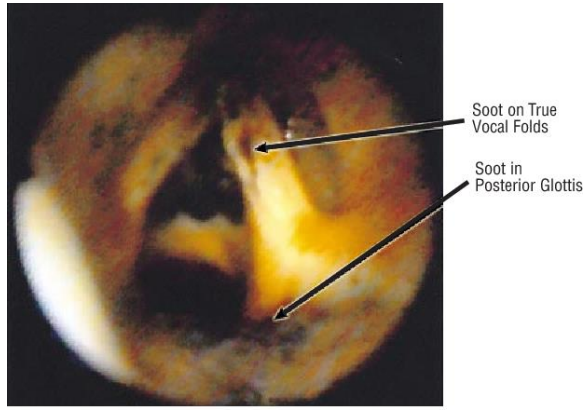


Figure 3. *Fiberoptic laryngoscopy revealed soot present in the larynx and*

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What About Bronchoscopy?

- Gold standard in confirming the diagnosis of inhalation injury
  - **Findings**:
    - Hyperemia, soot, edema
- Typically evaluates the lower airway
- Carries a procedural risk and sometimes can be contraindicated due to high oxygen requirements
- Not used as a diagnostic tool as the treatment would not change
- Considered when there is a therapeutic benefit or needed to help aid in the determination of prognosis

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To Intubate or To Not Intubate?

Moshrefi et al. 2019

- **Clear indicators for intubation:** resp distress, large FT TBSA, severe facial burns, oropharynx damage
- **Indirect physical exam findings that may indicate airway injury:** facial swelling, vocal changes, perioral burns, singed nasal hair, carbonaceous sputum, soot, hyperemic mucosa

Physical Exam Finding	% patients (out of 51 pts)
Facial Burn	Superficial (19%), PT (77 %), FT (4%)
Singed Nasal Hairs	63%
Nasal Soot	14%
Voice changes	10%
Throat pain	10%
SOB	10%
Wheezing	6%

(+) DL Findings	% patients
Edema	33%
Hyperemia	35%
Discoloration	8%
Soot	6%
Negative	20%

Avg 2.2 physical exam findings	+ DL = avg 2.3 physical exam findings
55% treated with supportive care	12% repeat DL – 1 pt intubated

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Intubation: Liberal vs Restrictive Criteria

Liberal Intubation Criteria (Local Criteria)	Restrictive Intubation Criteria (ABA 2011)
Facial Burn	FT Facial Burn
Stridor, Cough, Hoarse Voice	Stridor
Bronchospasm, Dyspnea, Chest Pain	Respiratory Distress
Hypoxia, Hypercarbia, Raised CO	Airway Edema on DL
Soot Around Mouth/Nose	Airway Trauma
LOC at Any Point	AMS
Enclosed Space	Hypoxia, Hypercarbia
Carbonaceous Sputum	Hemodynamic Instability

85 patients

58 pts = prolonged intubation

13 pts = not intubated

14 pts = extubated <48h

Criteria	Restrictive	Liberal
Sensitivity	0.84	0.98
Specificity	0.96	0.48
PPV	0.98	0.80
NPV	0.74	0.93

Predictive Ability for Need for Prolonged Intubation

Criteria	Restrictive	Liberal
Sensitivity	0.94	0.98
Specificity	0.86	0.05
PPV	0.94	0.70
NPV	0.86	0.50

Predictive Ability for Inhalation Injury

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The Intubation Question Continues!

Dyson et al. 2021

- Prevalence of confirmed inhalation injury = 11%
- 30% intubated by EMS, 41% non-burn center, 29% in burn center ED
  - 54% (EMS) had inhalation injury
  - 33% (non-burn center)
  - 58% (burn center ED)
- 30% of those intubated were extubated within 24h (78% with no inhalation injury)
  - 42% non-burn centers, 20% EMS, 23% ED burn center

TABLE 4. *Final multivariable model*†

	Adjusted odds of inhalation injury (95% confidence interval)	*P*-value
Per cent TBSA	1.05 (1.03–1.06)	<0.001
Flame	2.66 (1.14–6.20)	0.024
Enclosed space	2.98 (1.71–5.20)	<0.001
Face burns	2.24 (1.35–3.74)	0.002
Hoarse voice	3.40 (1.61–7.19)	0.001
Soot in mouth	4.34 (2.32–8.14)	0.020
Shortness of breath	2.27 (1.13–6.54)	0.025
Constant	0.01 (0.00–0.01)	<0.001

†C-statistic 0.87 (95% confidence interval 0.84–0.91), Hosmer–Lemeshow statistic χ^2 = 8.24, P = 0.410. TBSA, total body surface area.

Predicted risk of inhalation injury = exp(z)/1 + exp(z)

Where z = -4.760944 + 0.0462661 (per cent TBSA) + 0.9768775 (flame) + 1.092459 (enclosed space) + 0.8078726 (face burns) + 1.224117 (hoarse) + 1.468162 (soot) + 1.001516 (shortness of breath)

Example of use of predictor tool

1) A patient presents with 15% TBSA burned including facial burns that was caused by flame. Based on the formula above, the prediction of likelihood of inhalation injury equals 9%.

2) If this same patient was in an enclosed space when the burn occurred the likelihood of inhalation injury increases to 23%.

3) If the same patient is also presenting with a hoarse voice and soot in their mouth the likelihood of inhalation injury increases to 82%.

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Mechanical Ventilation

- If the patient needs intubation = larger ETT to allow for proper pulm hygiene +/- bronch
- **Consider lung protective strategies:**
  - ARDSnet protocol: TV 6-8mL/kg (IBW), airway pressures <35cm H2O
  - Optimize PEEP
  - Wean FiO2 as able after 6h 100% FiO2 is completed
- **Intubation comes with its own risks:**
  - Direct airway injury, bleeding, VAP, dental injury, stenosis (future), dysphonia, PTX, atelectasis, aspiration

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Benign Clinical Findings, (-) DL, w/o concerning lab findings

Airway Observation

"Healthy"
- CDU admit (no cutaneous burn)
- 12h airways obs
- NPO, IVF

Multiple Co-morbidities
- Stepdown
- ICU (critical care consult)

Clinical Signs of Lung Injury

100% FiO2 high flow oxygen via NRB (if not intubated) for 6h total

Cardiac monitoring, frequent neuro checks, circulatory support

Monitor labs
- LA
- COHb
- BMP
- ABG

Meds
- B-agonist inhalation
- NAC
- Nebulized heparin

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B-Agonist (albuterol)

- Decreased airflow resistance
- Improve airway compliance
- Decrease airway pressures via smooth muscle relaxation and inhibiting bronchospasm
- Increases PaO2:FiO2

NAC (mucomyst)

- Anti-oxidant and free radical scavenger
- Attenuates ROS damage
- Thins mucous secretions
- Aids in mobilization

Nebulized Heparin

- Anti-inflammatory properties
- Prevents formation of fibrin
- Inhibits cast formation

Administer albuterol with mucomyst Q4hrs

5000-10,000U Hep in 3mL Q4h alternate with albuterol

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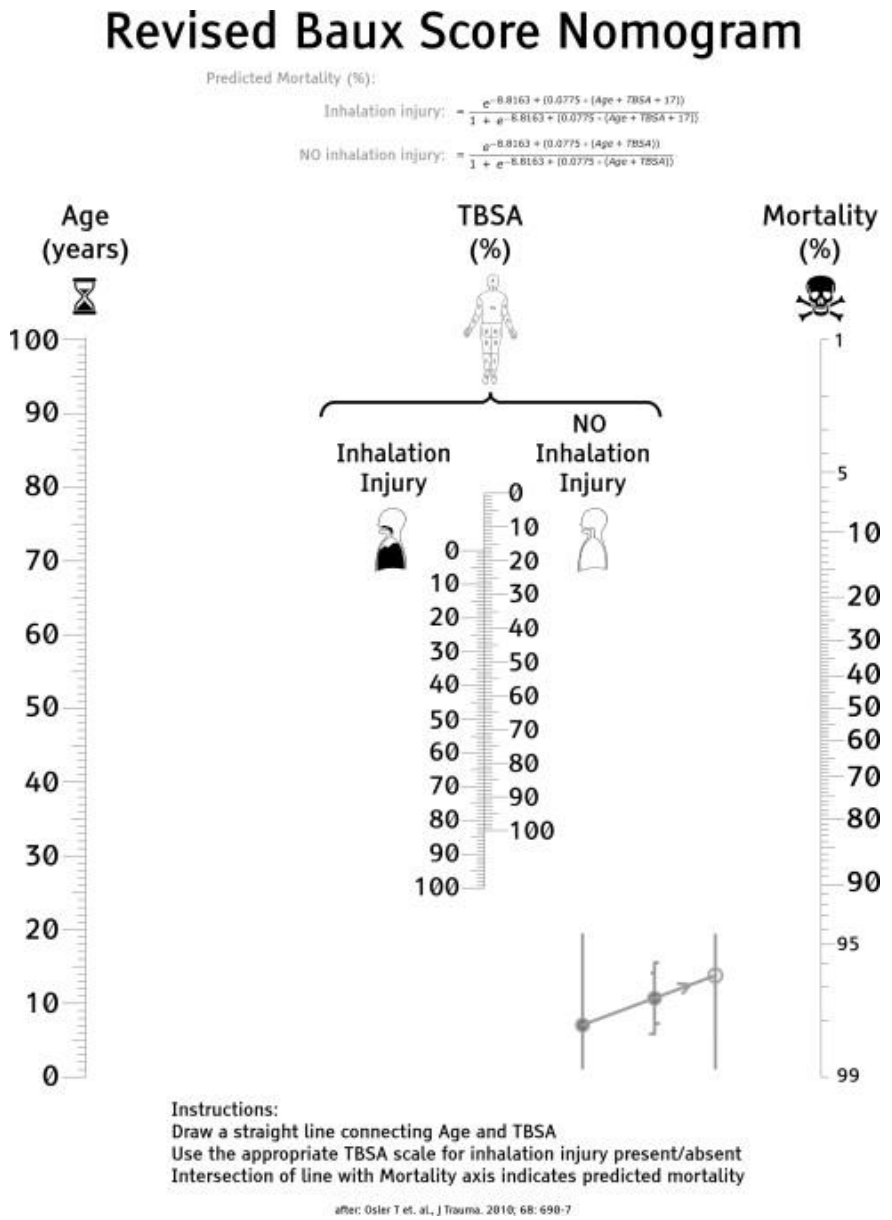
Revised Baux Score

- Score predicting mortality after a burn injury
- **Original Baux Score:** % Mortality = Age + TBSA
- **Revised Baux Score:** % Mortality = Age + TBSA + 17



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Hydrogen Cyanide

- Combustion of nitrogen and carbon containing substances
- Colorless gas, bitter almond odor
- Reversible inhibition of cytochrome c oxidase (thermal oxidase of resp chain) = suppresses cellular oxygenation = tissue anoxia
- **Dx**:
  - ECG changes (ST elevation) - suggestive
  - AG metabolic acidosis, non-correcting lactic acidosis
  - **Normal levels:** 0.02ug/mL (non-smokers) and 0.04ug/mL (smokers)
  - **Toxicity**: 0.1ug/mL ; **Death**: 1.0ug/mL

Table 16.3 Symptoms of Cyanide Toxicity

Symptoms in Low or Moderate Inhaled Cyanide Concentrations	Symptoms in Moderate or High Inhaled Cyanide Concentrations
Faintness	Prostration
Flushing	Hypotension
Anxiety	Tremors
Excitement	Cardiac arrhythmia
Perspiration	Convulsions
Vertigo	Stupor
Headache	Paralysis
Drowsiness	Coma
Tachypnea	Respiratory depression
Dyspnea	Respiratory arrest
Tachycardia	Cardiovascular collapse

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Hydrogen Cyanide

- **Tx**:
  - **Supplemental oxygenation**
    - Potentiate effects of antidotes, displaces cytochrome oxidases
  - **Methemoglobin generators:**
    - Competes with cytochrome oxidase for cyanide
    - Sodium Nitrite + Sodium Thiosulfate (Nithiodote)
  - **Direct binders to cyanide:**
    - Dicobalt edetate
    - Hydroxycobalamin (vitamin b12 precursor- Cyanokit)

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Flash Burns: Smoking on Oxygen

- 52% of patients with home oxygen continue to smoke despite known risks
- Typically have smaller TBSA (face/hands)
- Older, higher rate of inhalation injury, longer hospital stays
- **30-day mortality (COPD pt vs. COPD + burn): 0.49% vs 4.82%**
- ***Singer et al. 2020:***
  - Retrospective review, burn patients admitted smoking on home o2
  - **1-year mortality: 54.2%**
  - Increased age, increased mortality (amount of home o2 not statistically significant)
  - **Dispo**:
    - 44% home, 19% home health, 10% SNF, 6% LTACH, 21% died/hospice

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Flash Burns: Smoking on Oxygen

H&P, Physical Exam	Supportive Care	GOC Discussions
• Time of Incident • O2 requirements, hemodynamic status	• Supplemental O2 • Normal Saline Nasal Spray • +/- Opthalmic Baci	• Palliative Care Consult

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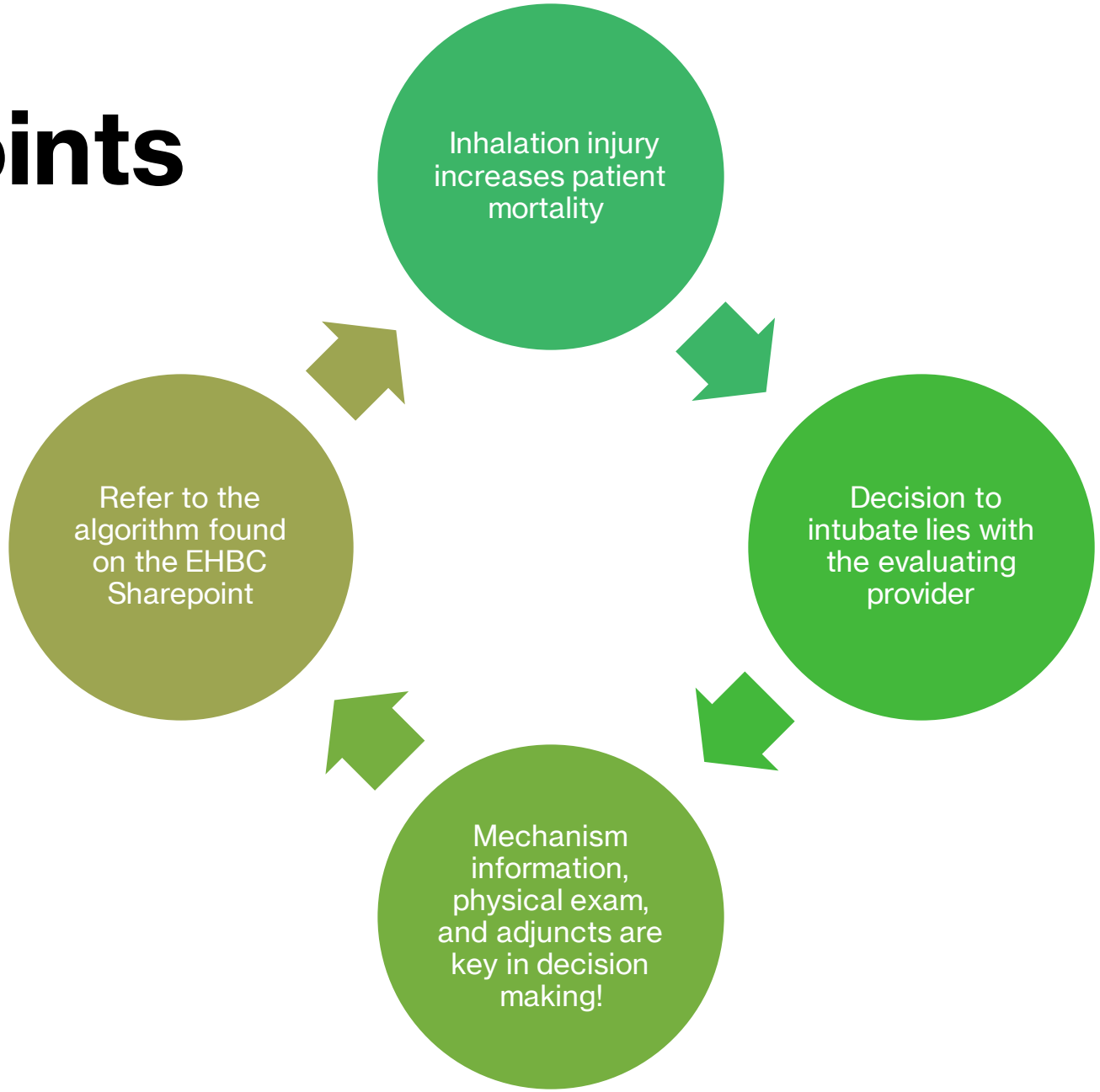
Key Points


Inhalation injury increases patient mortality
Decision to intubate lies with the evaluating provider
Mechanism information, physical exam, and adjuncts are key in decision making!
Refer to the algorithm found on the EHBC Sharepoint

References: Pictures

- Upper Airway Icon
- Upper Airway Anatomy
- Lower Airway Anatomy
- Inhalation Injury Diagram
- Singed Hair
- Facial Burn
- Carbonaceous Sputum and Soot
- Oxygen dissociation curve
- DL 1,
- DL 2
- DL 3
- Nithiodote
- Cyanokit
- Revised Baux Score

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