Are We Killing Our Patient?
The Hidden Dangers Of Oxygen Administration

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Why Give Oxygen?

- Treat hypoxemia
  - Particularly from decreased alveolar oxygen tension

- Decrease the work of breathing
  - Fewer breaths get same minute oxygen
  - Smaller breaths are required less energy

- Decrease myocardial work
  - Blood needs to circulate fewer times to provide same or more volume of oxygen
Objectives

1. Review oxygen absorption and consumption physiology
2. Correlate oxygen administration to the Arterial Blood Gas
3. Explain oxygen induced complications
4. Apply oxygen administration strategies to prevent complications
Objective 1: Oxygen Absorption and Consumption

Oxygen delivery and absorption are both essential

- Cellular
- Tissues
- Organs
O$_2$ demand exceeds supply → Hypoxia develops → Anaerobic metabolism → Lactic acid production → Cellular and organ dysfunction → Cellular death
Respiratory System Anatomy

Upper airway
- Nose
- Mouth

Lower airway
- Tracheobronchial tree
  - 23 divisions, begin at carina

Alveoli

Chest wall & diaphragm

Neuro drive
Alveoli
Neuro Drive

Based on Chemoreceptors

Peripheral
- Carotid artery

Central
- Aortic arch
  - Medulla

Secondary drive
Ventilation

Ventilation is the exchange of gases into and out of the respiratory system.
Respiration

• Alveolar respiration
  – Only gasses in alveoli and blood stream
  – Dead air space has no exchange

• Cellular respiration
Normal Air Components

**Ambient gas (760 mmHg)**
- Oxygen 159mmHg (20.9%)
- Nitrogen 600mmHg (79.0%)
- Others 1mmHg (0.1%)

**Alveolar gas (760 mmHg)**
- Oxygen 101mmHg (13.3%)
- Nitrogen 572mmHg (75.2%)
- Carbon Dioxide 40mmHg (5.3%)
- Water vapor 47mmHg (6.2%)
Nitrogen

Significance:

- Not absorbed by body easily
- Creates pressure inside alveoli promoting inflation

Without Nitrogen

With Nitrogen
Objective 2: Correlate the ABG and Oxygen Administration

After a pulse is found, a blood sample is taken from the artery.
Oxygen Absorption

- When crosses alveolar membrane
  - Attaches to hemoglobin
  - Dissolves in plasma
Oxygen Saturation

~98% oxygen absorbed onto hemoglobin

- Each 1g Hgb carries ~1.34mL of oxygen
- Actually measured as an SaO₂
- SpO₂ similar but same as SaO₂
- Can’t distinguish CO from O₂
- Normally 95% of O₂ attached to hemoglobin

Highest reading SaO₂ or SpO₂ can be 100%
Dissolved Oxygen

Normally 2-5% of total oxygen in plasma

- 80-100mmHg

No maximum pressure

- Dalton’s Law: $P_t = P_1 + P_2 + P_3 + \ldots + P_n$
- Diffusion occurs for each gas based on its own pressure gradient
- Fick’s law describes that the rate of gas exchange is proportional to the tissue thickness and the difference in gas pressures on both sides
## Arterial Blood Gas

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>PaCO$_2$</td>
<td>35-45 mmHg</td>
</tr>
<tr>
<td>HCO$_3^-$</td>
<td>22-26 mEq/L</td>
</tr>
<tr>
<td>PaO$_2$</td>
<td>80-100 mmHg</td>
</tr>
<tr>
<td>Base excess</td>
<td>-2 – 2 mmol/L</td>
</tr>
</tbody>
</table>
Oxygen or Ventilation

Determine Alveolar oxygen pressure ($P_A O_2$)

$$P_A O_2 = (FiO_2 \times 713) - (PaCO_2 / 0.8)$$

**Case 1:**
- $PCO_2$: 40
- $PaO_2$: 45
- $PAO_2$: 100

Alveolar-arterial

$$100 - 45 = 55$$

Wide A-a gradient

**Case 2:**
- $PCO_2$: 80
- $PaO_2$: 45
- $PAO_2$: 50

Alveolar-arterial

$$50 - 45 = 5$$

Normal A-a gradient

Normal A-a gradient = Age / 4 + 4
Measuring VO$_2$

Total O$_2$ consumed by tissues a 1 minute

- Calculated
- Normal is ~250mL O$_2$

Influenced by

- Oxygen demand
- Oxygen availability
- Ability to carry oxygen
- Ability to carry and extract oxygen
Factors Increasing Oxygen Demand

- Anxiety
- Serious illness
- Seizures
- Surgery
- Injury
- Sepsis

Any increase in metabolism
Oxygen Availability

Dependent on what’s happening back at the respiratory system
Oxygen Carrying & Transfer Capacity

- Sickle-cell anemia
- Anemia
- Acidic environments
- Alkalotic environments
Diagnosing Oxygen Debt

Clinical oxygen debt:
- Elevated lactate
- Ph < 7.35

\[ S_v = S_a \times 0.75 \]

If < then oxygen debt present
Cellular Changes From Hypoxia

• Normal cellular metabolism dependent on oxygen supply

• Anaerobic metabolism
  – Lactic acid release
    • Considered elevated at >2.2mm/L
    • Decreases pH
  – Decreased cellular function & ATP synthesis

O₂ demand exceeds supply
Hypoxia develops
Lactic acid production
Anaerobic metabolism
Cellular and organ dysfunction
Cellular death
Cellular Oxygen Metabolism

Cells function better in oxygen rich environments

- More oxygen means higher function (to a point)
- AHA 2010 Circulation recommendations
  - Titrate oxygen to normalize SpO₂
    - Defined as >94%
  - Use minimal amount of oxygen to maintain normal SpO₂

HOLD ON...

Oxygen is good

Needed to promote normal cell function

Prevents hypoxia

Prevents anaerobic metabolism

Provided intact circulation system free of occlusions
Superoxide molecule ($O_2^-$)
Hydrogen peroxide ($H_2O_2$)
Hydroxyl ion ($OH^-$)
Water ($H_2O$)

Superoxide dismutase
The $\text{O}_2^-$ Molecule

- Damages cell membranes
- Considered "toxic"
- Normally destroyed by enzymes within cell

- Superoxide dismutase
- Enzymes produced at fixed rate
- Enzyme production rate does not increase with metabolism
Objective 4: Complications of oxygen administration

Oxygen is a drug
Skin Irritation

- Produced by plastic systems
  - N/C
  - NRB

Common areas
- Behind ears
- Bridge of nose
Mucous Membrane Drying

- Supplemental oxygen often has no moisture content
- Upper airway warms, humidifies, filters air
- With supplemental oxygen, more moisture pulled from membranes
  - More of a discomfort
- Can also cause epistaxis
Oxygen Toxicity
Higher oxygen delivery

- Increases pO$_2$
- Forces more oxygen into cells
- Increases metabolism
- Increases O$_2^-$ production
- Build up of O$_2^-$ molecules
- Fixed rate of O$_2^-$ elimination
The Good News?

Typically takes 24 hours in an oxygen rich environment to build up enough $O_2^-$ to develop evidence of cellular damage.
Oxygen Rich Environment

Healthy lungs $\text{FiO}_2 > 0.6$
- Alveolar oxygen tensions of 350mmHg

Injured or diseased lungs $\text{FiO}_2 > 0.5$
- Alveolar oxygen tensions of 250mmHg
Oxygen Toxicity CNS Symptoms

- Perioral twitching
- Hand muscle twitching
- Respiratory depression
- Seizures
- Peripheral vasoconstriction & pallor
- Vertigo
- Visual and auditory disturbances
- Nausea

Oxygen Toxicity Pulmonary Symptoms

- Capillary leakage
- Decreased $V_T$
- Pulmonary edema
- ALI and ARDS

Oxygen Toxicity Symptoms

- Ocular effects
  - Decreased field of vision
  - Progressive myopia
  - Retrolental fibroplasia
    - A primary cause of childhood blindness
- More common when in oxygen tent

Summary of Symptoms

Eyes
- Visual field loss
- Near-sightedness
- Cataract formation
- Bleeding
- Fibrosis

Central
- Seizures

Respiratory
- Jerky breathing
- Irritation
- Coughing
- Pain
- Shortness of breath
- Tracheobronchitis
- Acute respiratory distress syndrome

Muscular
- Twitching

http://0.tqn.com/d/chemistry/1/0/h/X/1/Symptoms_of_oxygen_poisoning.png
FREE RADICAL DAMAGE TO MOTOR NEURONS

Reactive oxygen species (ROS), or free radicals, are generated as a result of metabolic processes. These free radicals have at least one unpaired electron, which renders them chemically unstable and highly reactive with other molecules in the body. Mitochondrial DNA (mtDNA) is located near the inner mitochondrial membrane, and lacks advanced DNA repair mechanisms, making mtDNA particularly susceptible to damage from ROS. Cells respond to oxidative damage by neutralizing free radicals through antioxidant enzymes, such as superoxide dismutase (SOD) and catalase. Eventually, damage accumulates due to the inability of cells to repair damage as quickly as it arises.
High Risk Patients

- Hyperbaric therapy
- Neonatal patients
- Ventilated patients
Hyperbaric Medicine

Diving emergencies

Wound management
- Gas gangrene
- Refractory chronic osteomyelitis
- Infected burns

Trauma care

CO toxicity

Goal is to increase O₂ availability to cells
### How it Works

<table>
<thead>
<tr>
<th>FiO₂</th>
<th>760mmHg</th>
<th>1520mmHg</th>
<th>2280mmHg</th>
</tr>
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<tbody>
<tr>
<td>0.21</td>
<td>101</td>
<td>202</td>
<td>303</td>
</tr>
<tr>
<td>0.4</td>
<td>304</td>
<td>608</td>
<td>912</td>
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<tr>
<td>0.6</td>
<td>456</td>
<td>912</td>
<td>1368</td>
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<tr>
<td>1.0</td>
<td>“510”</td>
<td>“1020”</td>
<td>“1530”</td>
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</table>
Ventilated Patients

Already compromised

Increased alveolar oxygen pressure

Increased sensitivity for

- ARDS
- Hypoxemia
- Lung disease

When damage starts is not known!
Neonatal Patients

Those <30 weeks or <1500 G at birth

Fetal hemoglobin has > affinity than adults

Why a problem?

• Normal retinal vascularization occurs shortly after birth
• high FiO₂ induces vasoconstriction, particular to temporal region of retina

Anticipated Lab Values

<table>
<thead>
<tr>
<th></th>
<th>Birth</th>
<th>Post 5 min</th>
<th>Post 24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>≥7.20</td>
<td>7.2-7.34</td>
<td>7.35-7.4</td>
</tr>
<tr>
<td>PCO₂</td>
<td>≤50mmHg</td>
<td>35-46mmHg</td>
<td>33-35mmHg</td>
</tr>
<tr>
<td>pO₂</td>
<td>25-40mmHg</td>
<td>49-73mmHg</td>
<td>72-75mmHg</td>
</tr>
<tr>
<td>SaO₂</td>
<td>&gt;50%</td>
<td>&gt;80%</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>≥15mmHg</td>
<td>16-19mmHg</td>
<td>&gt;20mmHg</td>
</tr>
</tbody>
</table>

Neonatal Resuscitation; American Fam Physician 2011 April 15
Neonatal Oxygen

- Traditionally common, particularly during resuscitation
- Causes $pO_2$ to rapidly well exceed normal levels

<table>
<thead>
<tr>
<th>Post-birth Age</th>
<th>$SpO_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 minute</td>
<td>60-65%</td>
</tr>
<tr>
<td>1 min</td>
<td>65-70%</td>
</tr>
<tr>
<td>3 min</td>
<td>70-75%</td>
</tr>
<tr>
<td>4 min</td>
<td>75-80%</td>
</tr>
<tr>
<td>5 min</td>
<td>80-85%</td>
</tr>
<tr>
<td>10 min</td>
<td>85-95%</td>
</tr>
</tbody>
</table>
Neonatal Eye Damage

• Most significant complication is vascular constriction
  – Normal part of shift to extra-uterine life
  – Exacerbates this constriction however
• Can cause loss of blow flow to retina
• Becomes a risk when $\text{PaO}_2 > 80 \text{ mmHg}$
Neonatal Resuscitation Guidelines

(Class IIb, LOE B). These targets may be achieved by initiating resuscitation with air or a blended oxygen and titrating the oxygen concentration to achieve an $\text{SpO}_2$ in the target range as described above using pulse oximetry (Class IIb, LOE C). If blended oxygen is not available, resuscitation should be initiated with air (Class IIb, LOE B). If the baby is bradycardic (HR $<$60 per minute) after 90 seconds of resuscitation with a lower concentration of oxygen, oxygen concentration should be increased to 100% until recovery of a normal heart rate (Class IIb, LOE B).

Kattwinkel, John et al, Neonatal Resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care; Circulation 2010; 122; S909-S919
Absorbative Atlectasis
Nitrogen in the Alveoli

Recall that nitrogen is not well absorbed by the body.

When alveoli contract during periods of non-activity, nitrogen keeps the alveoli from collapsing.
Increasing FiO₂

Time to collapse of unventilated lung compartment.

- N₂, PreO₂
- N₂, No PreO₂
- N₂O, PreO₂
- N₂O, No PreO₂

Time to collapse (minutes)

FIO2

Joyce C J, Williams A B J Appl Physiol 1999;86:1116-1125

Journal of Applied Physiology

©1999 by American Physiological Society
What’s the Big Deal?

Increased FiO₂ decreases nitrogen pressure.

Oxygen and remaining gasses become prone to absorption.

Reduced gas pressures remain keeping alveoli propped open.

Alveoli collapse.

Fewer alveoli available to participate in gas exchange.
Clinical Significance

Difficult to visualize

Leads to decreased tidal volume

Clues

• Awake patient on oxygen experiences increased SOB, may complain of not enough air with each breath

• Vented patients need increased Vt to maintain same ABG values on 100% O₂ vs lower concentrations
Case Study

- 49 year old female
- PMH
  - CHF
  - COPD with home O₂
- Presented to ED via EMS with respiratory distress and 2-word dyspnea

**Initial vitals**
- HR 104
- RR 26
- SpO₂ 79%
- Temp 97.3°F

**Initial ABG**
- pH 7.29
- pO₂ 56
- pCO₂ 64
- HCO₃⁻ 20
Case Study

ED Treatments

What happened to this patient? Why have they deteriorated?

- Decreased respiratory effort
- Now on NRB

Repeat ABG

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>pH</td>
<td>7.24</td>
</tr>
<tr>
<td>pO₂</td>
<td>80</td>
</tr>
<tr>
<td>pCO₂</td>
<td>95</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>18</td>
</tr>
</tbody>
</table>
Carbon Dioxide Narcosis
aka *Oxygen induced Hypercapnia*

Central chemoreceptors found in medulla

Central chemoreceptors sensitive to drops in pH

- when patients have a chronically low pH due to chronically high CO$_2$ the central receptors become desensitized
- Primary respirations triggered by peripheral chemoreceptors
Whose at Risk?

- PMH
  - status asthmaticus
  - COPD
  - weakness in respiratory muscles
    - Mysthenia gravis
    - Poliomyelitis
    - Head injury
    - Increased ICP

Chronic Respiratory Failure

**Diagnosed by**
- $\text{PaCO}_2 > 50 \text{ mmHg}$
- $\text{PaO}_2 < 55 \text{ mmHg}$

**Patients do still need oxygen, particularly when oxygen is below baseline**

**Common Causes of CRF**
- Chronic Bronchitis
- Emphysema
- Bronchiectasis
- Cystic Fibrosis
- Pneumoconiosis
- Tuberculosis
- Fungal disease
- Kyphoscoliosis
Physiology

Body is used to chronic PaO₂ below 55mmHg

With each breath more oxygen enters body
  - PaO₂ rises, SpO₂ rises

Breathing rate or depth decreases

Now we apply oxygen

Body recognizes that it does not need to work as hard to maintain PaO₂

Reduced CO₂ eliminated with each breath
Symptoms

- Decreased respiratory rate
- Decreased Vt
- Measureable increases in CO₂ levels
  - Sidestream CO₂
  - PaCO₂
- Mental status changes
  - Lethargy
  - Confusion
  - Headache
  - Somnolence
- Sweating
- Twitching
- papilloedema

Caused by the “Hypoxic Drive?”

- Mechanisms not clearly known
- Might be from changes in V/Q mismatch
- Might be from rises in PaO₂
What do we KNOW?

Hypercapnia via ventilation changes takes hours to days to develop

Appears in patients with advanced COPD who is otherwise asymptomatic and has oxygen applied when they are

- Relaxed
- Unstimulated
- In no distress
Objective 5: Oxygen administration and side-effect prevention
Goals of Oxygen Administration

- Stabilize arterial oxygen
- Establish eupnea
- Decrease anxiety
- Eliminate shortness of breath

Accomplish these with the least amount of oxygen necessary
Pad Oxygen Devices

- Inspect any skin & oxygen devices at the start of transports
- Behind ears
- Keep ties loose

- If irritation is present consider changing device type
Humidify Oxygen

Any therapy over 4 LPM or FiO$_2$ > .36

Standard in hospitals, not routine in all CCT

• Cost?
• Duration of transport?
• Inconvenient?
Optimize Patient Position

Maximizes lung expansion
Decreases work of breathing
Prevents aspiration
Oxygen Titration

• Never withhold oxygen, goal SpO$_2$ is 90-95%
  – 94-98% for patients <70yrs
  – 92-98% for patients >70yrs

• Remember SpO$_2$ of 90 Correlates to pO$_2$
  60mmHg
  – In patients with COPD aim for a PaO$_2$ of 50-55mmHg

• Oxygen’s maximum benefit is in the 22-50% range

Patel, et al, Oxygen Toxicity, JIACM
2003; 4(3): 234-7
The Effect of high flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting

- Included patients
  - >35yrs
  - Shortness of breath
  - Known history of COPD or >10 pack-year smoking history
- Low oxygen group: oxygen titrated to maintain SpO$_2$ between 88-92%
- High oxygen group: NRB with oxygen at 10LPM

Predict Toxicity and Atelectasis

• 1 minute of FiO$_2$ at 1.0 = a Unit of Pulmonary Toxicity Dosage

• 1425 Units = predicted loss of 10% vital capacity

When to use 100%

- Cardiac arrest
- Major resuscitations
What about STEMI?

2013 ACC/AHA Guidelines

- Limited data supports routine oxygen use
- It is appropriate for patients with SpO₂ <90%

Analysis of 3 trials: high flow oxygen vs room air

- Patients receiving high flow oxygen have a 3-fold increase for death compared to those treated with room air

Animal trials have demonstrated

- 5 minutes of 100% oxygen reduces coronary artery blood flow by up to 30%

Oxygen in Pediatric Patients

- Utilize room air when possible
- Titrate up
- Use a pediatric flow meter
- Compare minute volume to oxygen LPM
- Keep PaO$_2$ below 160mmHg
  - Considered critical

SpO$_2$ in Preterm Infants

Studied preterm infants <32 weeks, <1500g

- All received supplemental oxygen of some sort

Found common causes leading to O$_2$ toxicity

- Improper alarms
- Failing to wean after procedures
- Staff workload
- Patient severity

When Managing a Neonate

Utilize FiO₂ 0.21-0.30

- Lower mortality: 8% vs 13%
- Shorter period on supplemental oxygen
- Shorter ventilated period
- Similar time vs high FiO₂ to normal SpO₂

NiPPV

Two forms
- CPAP
- BiPAP

Can support ventilation rate and depth

Does not mean need to apply oxygen
- 340 patient study
- CPAP with <30% oxygen improved SpO₂ and respiratory rate

Point of Care Testing

Arterial blood gasses can change in 15-20min

Current gasses allow for accurate adjustments to oxygen and ventilator settings

For every decrease of $\text{FiO}_2$ by 0.01 $\text{pO}_2$ will decrease $\text{PO}_2$ by 7mmHg
**Ventilator Adjustments**

- **pO₂ < 60 mmHg**
  - Increase FiO₂ to 0.6
  - Increase peep
  - Increase FiO₂ above 0.6

- **PaCO₂ > 45 mmHg** (EtCO₂ > 50 mmHg)
  - Increase Vr
  - Increase Vt

- **SpO₂ > 95%**
  - Reduce FiO₂ to 0.6
  - Reduce PEEP to 5
Summary

• Oxygen is an essential element needed by every cell in our body
• Supplemental oxygen up to an FiO\textsubscript{2} of 0.5, helps decrease the work of breathing, improves metabolism, decreases pain and anxiety, and
• Increasing the FiO2 above 0.5-0.6 is necessary in some situations
• Utilize strategies to maximize the benefits of oxygen at the lowest oxygen setting possible
Contact Information

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See you at EMS
World Expo 2014