



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 require 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



O₂ demand
exceeds supply

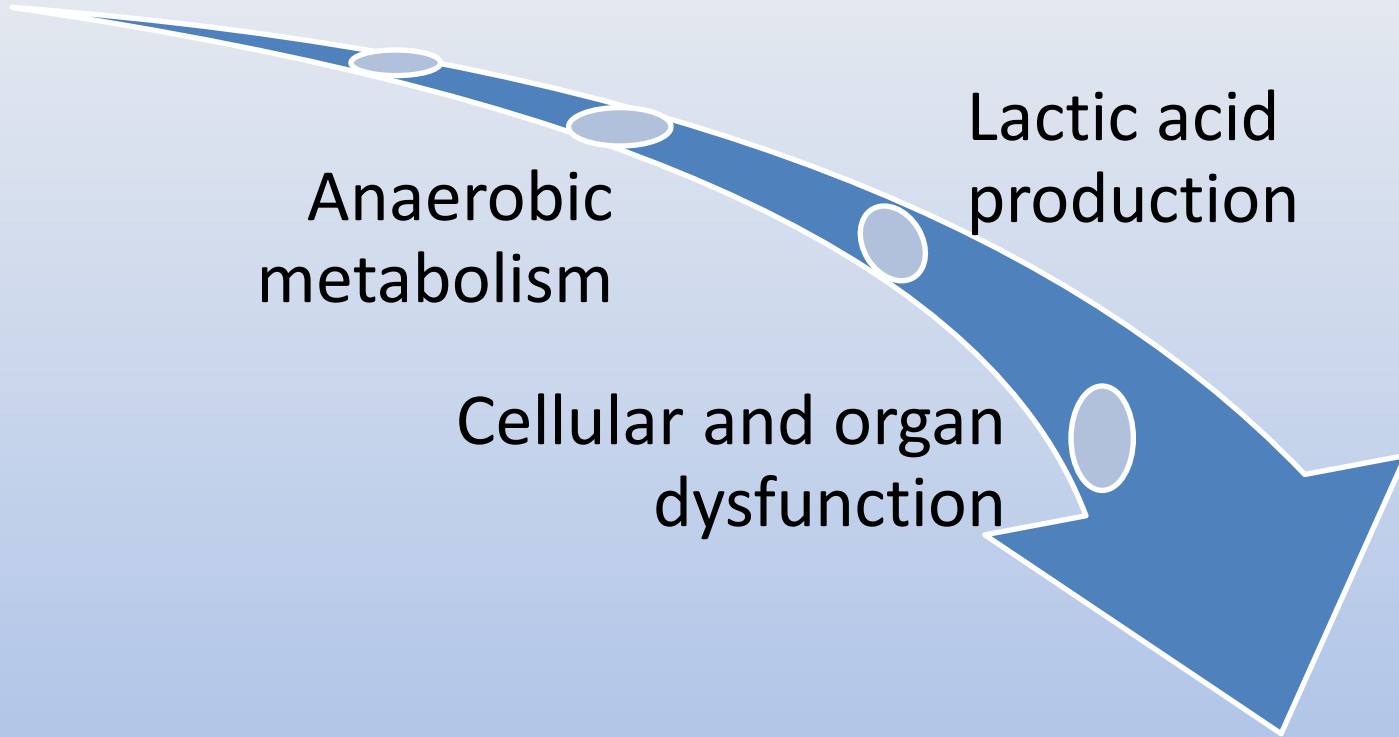
Hypoxia develops

Anaerobic
metabolism

Lactic acid
production

Cellular and organ
dysfunction

Cellular death



Respiratory System Anatomy

Upper airway

- Nose
- Mouth

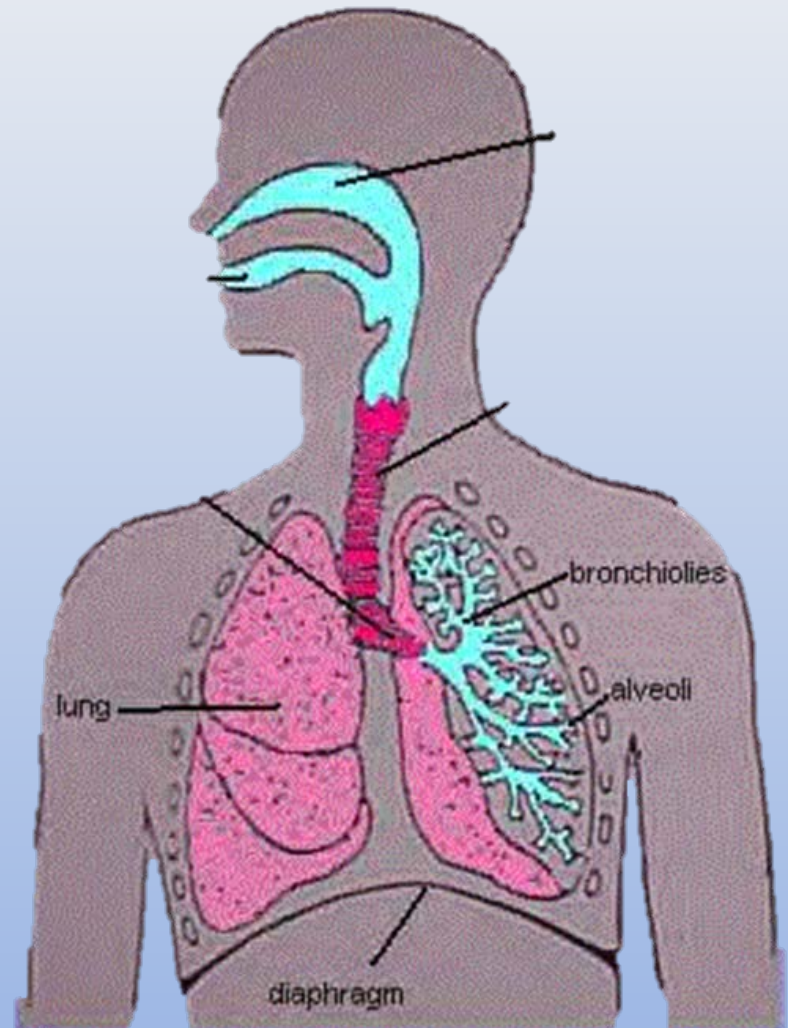
Lower airway

- Tracheobronchial tree
 - 23 divisions, begin at carina

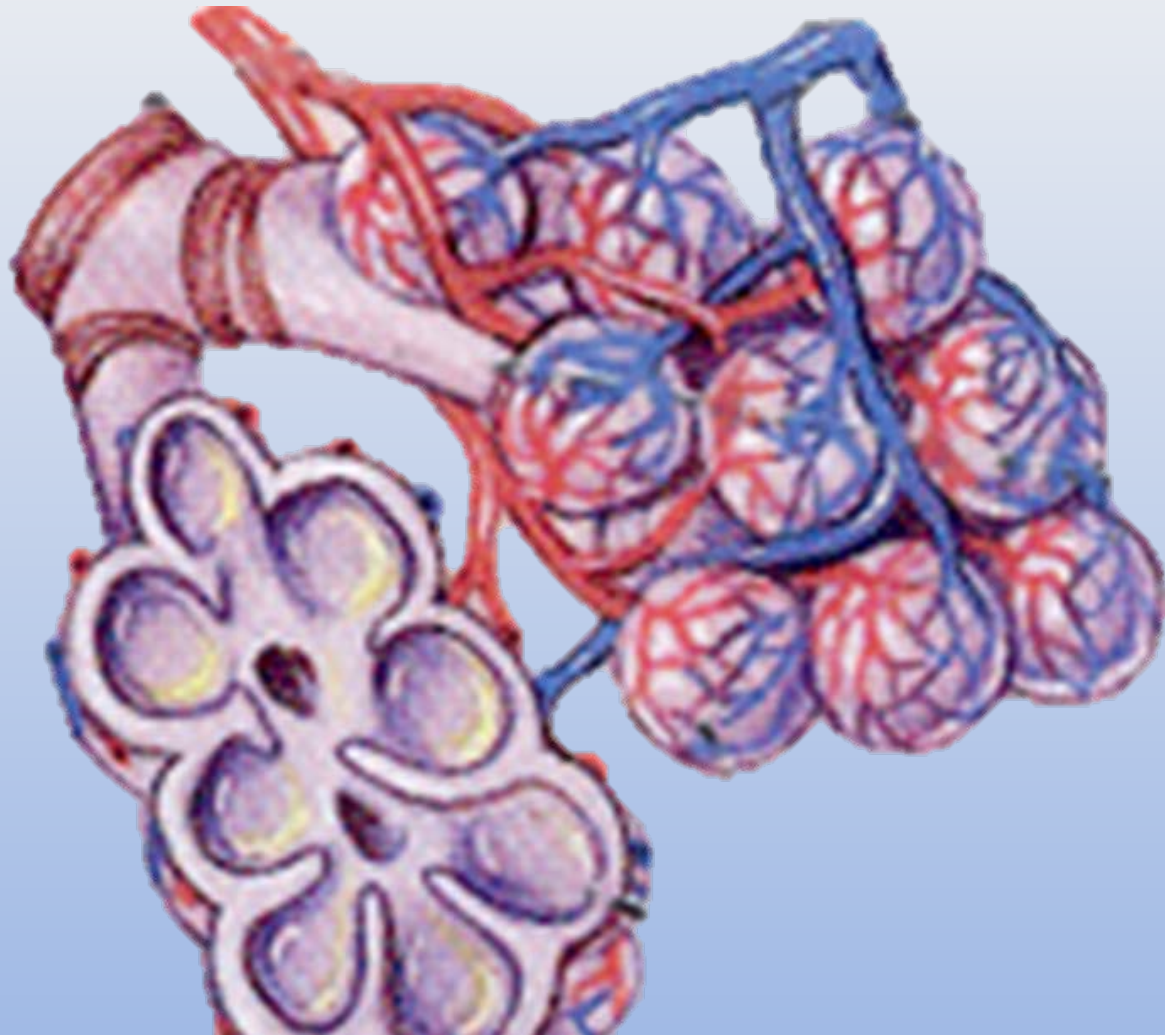
Alveoli

Chest wall & diaphragm

Neuro drive



Alveoli



Neuro Drive

Based on Chemoreceptors

Peripheral

Central

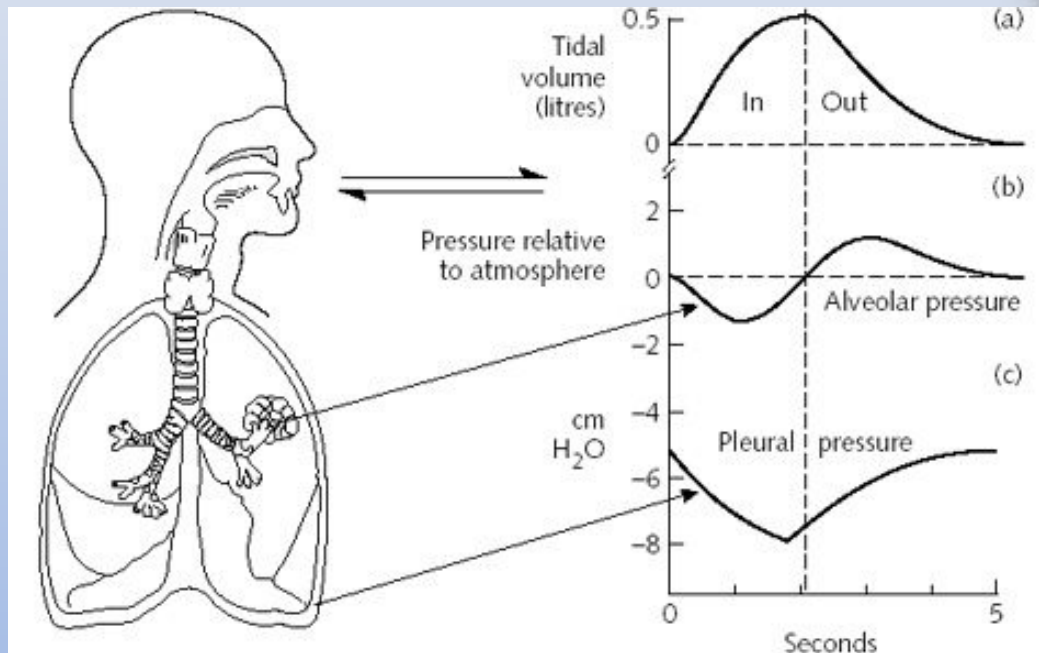
Carotid
artery

Aortic arch

Medulla

Secondary drive

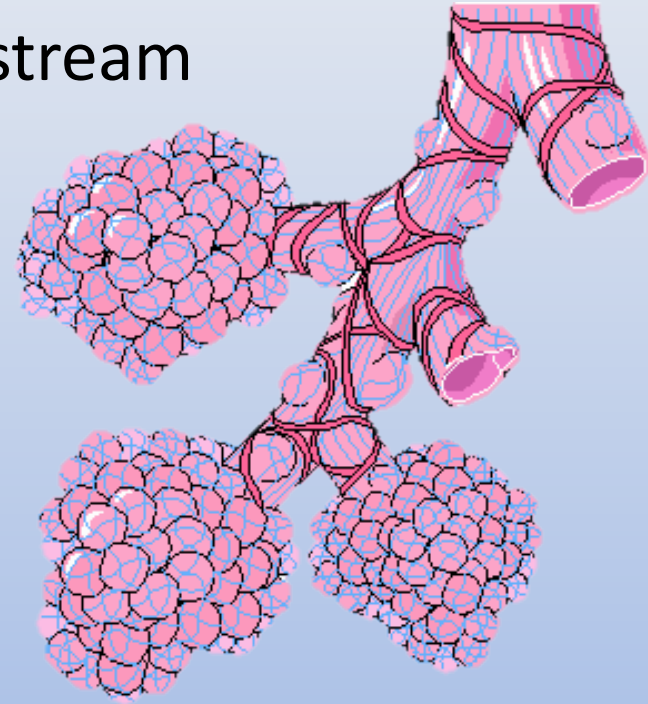
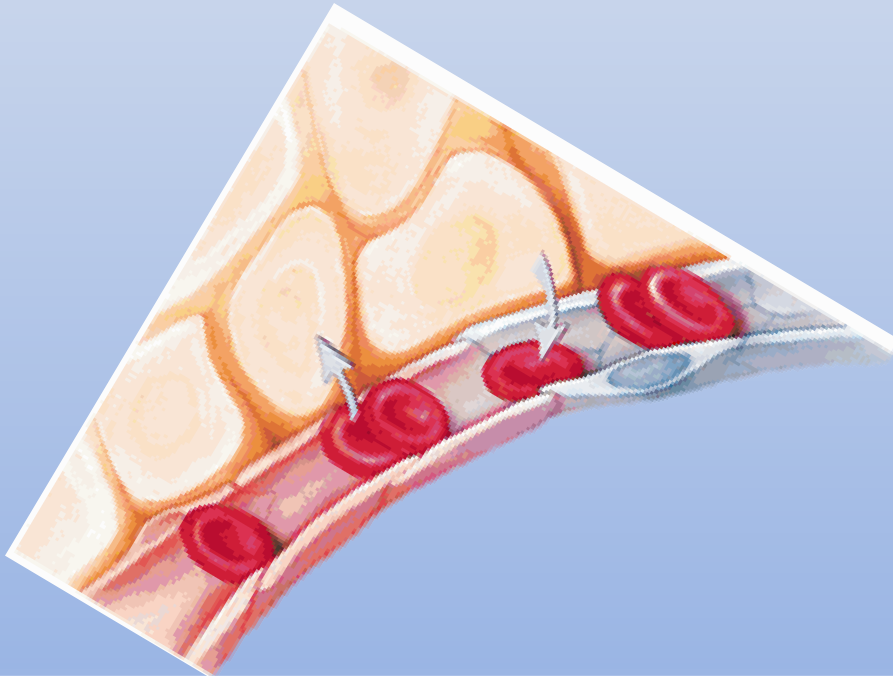
Ventilation



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%)

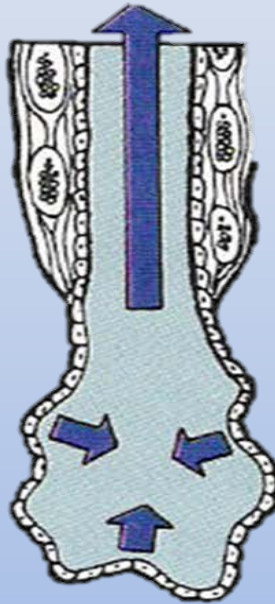
Water vapor
47mmHg (6.2%)



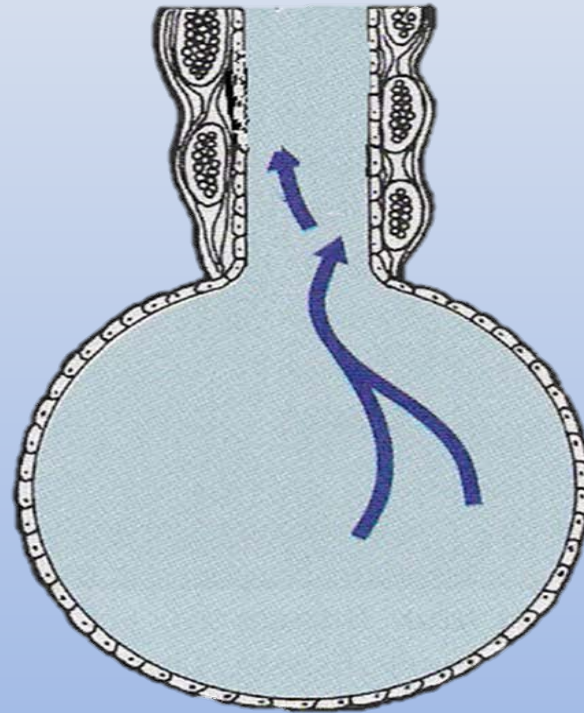
Nitrogen

Significance:

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

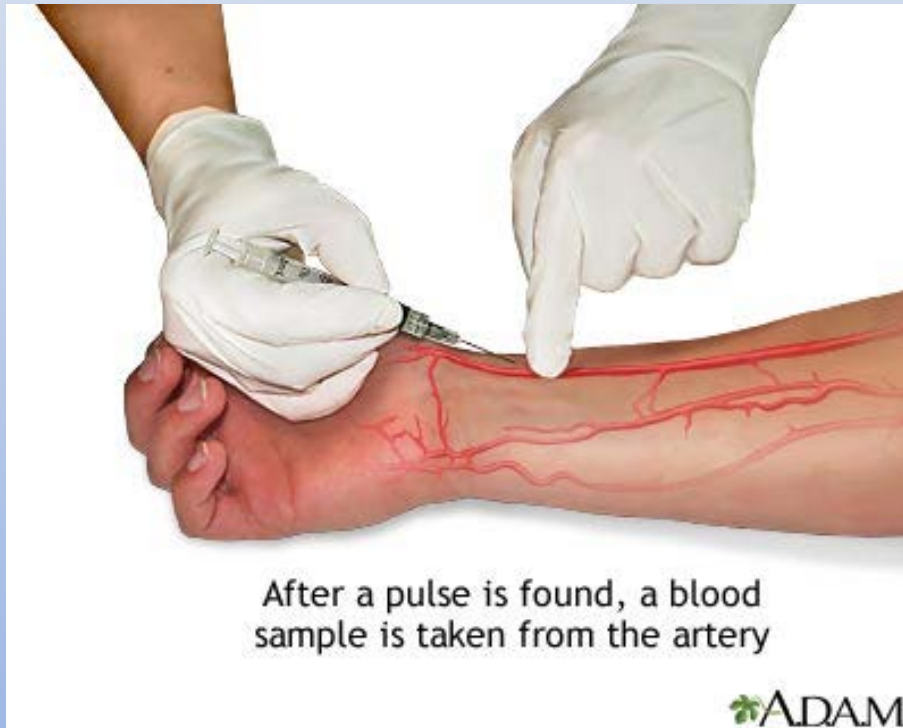


Without Nitrogen



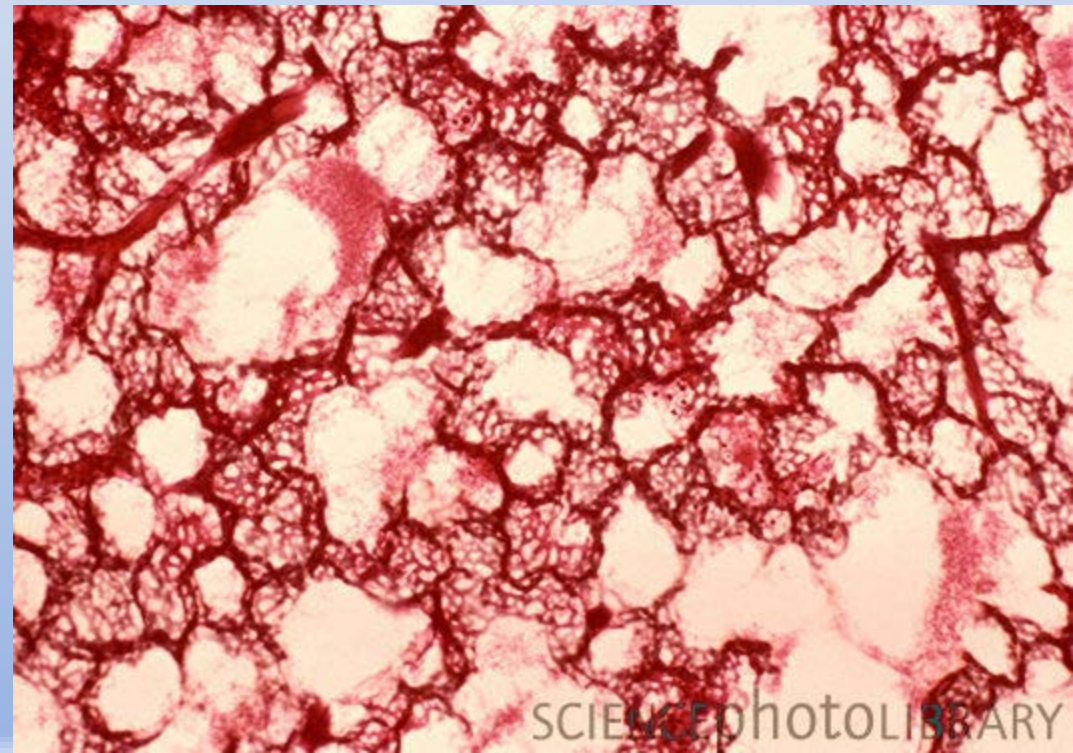
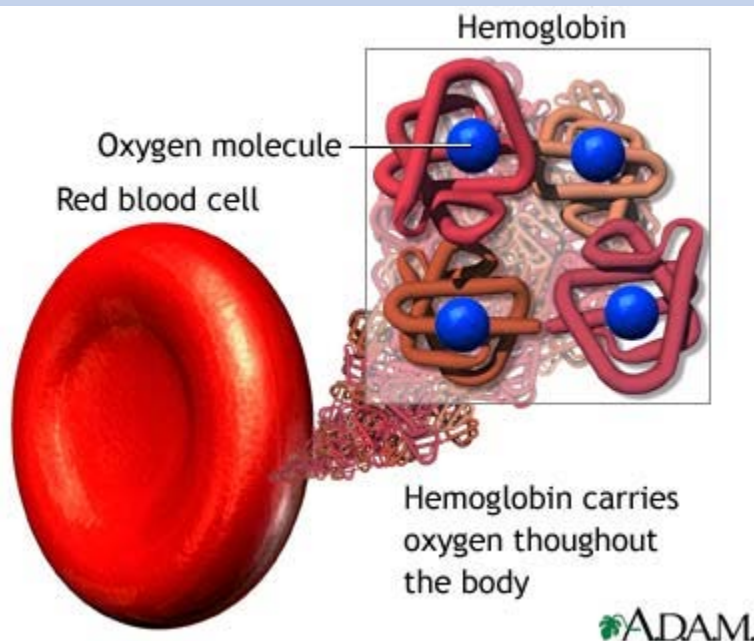
With Nitrogen

Objective 2: Correlate the ABG and Oxygen Administration



Oxygen Absorption

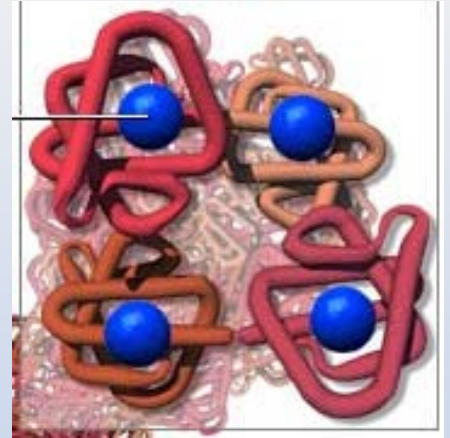
- When crosses alveolar membrane
 - Attaches to hemoglobin
 - Dissolves in plasma



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%

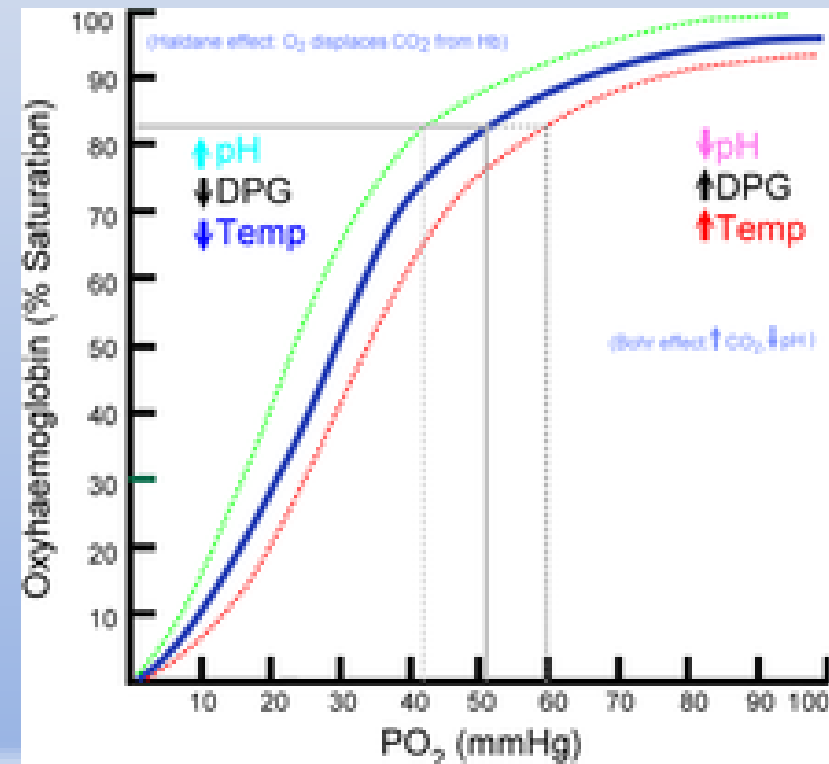
Dissolved Oxygen

Normally 2-5% of total oxygen in plasma

- 80-100mmHg

No maximum pressure

- Dalton's Law: $P_t = P_1 + P_2 + P_3 + \dots + P_n$
- 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

pH

7.35-7.45

PaCO₂

35-45mmHg (always higher than EtCO₂)

HCO₃⁻

22-26 mEq/L

PaO₂

80-100mmHg

Base excess

-2 – 2 mmol/L

Oxygen or Ventilation

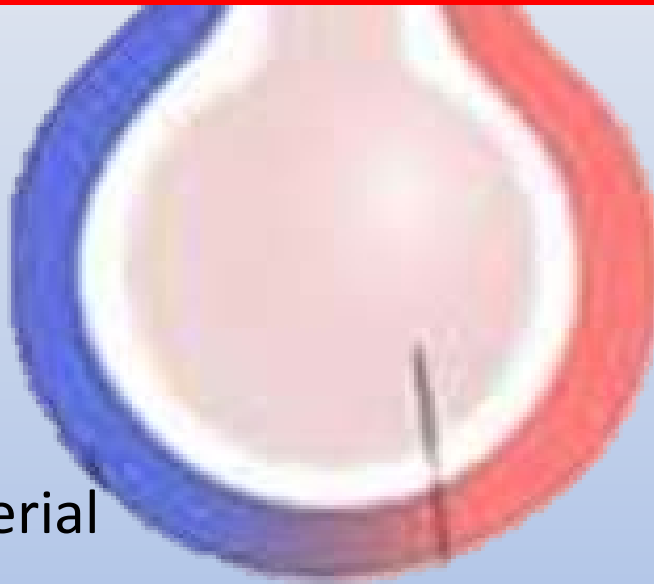
Determine Alveolar oxygen pressure (P_AO_2)

$$P_AO_2 = (FiO_2 \times 713) - (PaCO_2/0.8)$$

PCO_2 : 40

P_aO_2 : 45

P_AO_2 : 100



PCO_2 : 80

P_aO_2 : 45

P_AO_2 : 50

Alveolar-arterial

$$100 - 45 = 55$$

Wide A-a gradient

Alveolar-arterial

$$50 - 45 = 5$$

Normal A-a gradient

$$\text{Normal A-a gradient} = \text{Age} / 4 + 4$$



Measuring $\dot{V}O_2$

Total O_2 consumed by tissues a 1 minute

- Calculated
- Normal is $\sim 250\text{mL } O_2$

Influenced by

- Oxygen demand
- Oxygen availability
- Ability to carry oxygen
- Ability to carry and extract oxygen

Factors Increasing Oxygen Demand



Anxiety

Any increase in metabolism

Serious illness

Seizures

Surgery



Injury

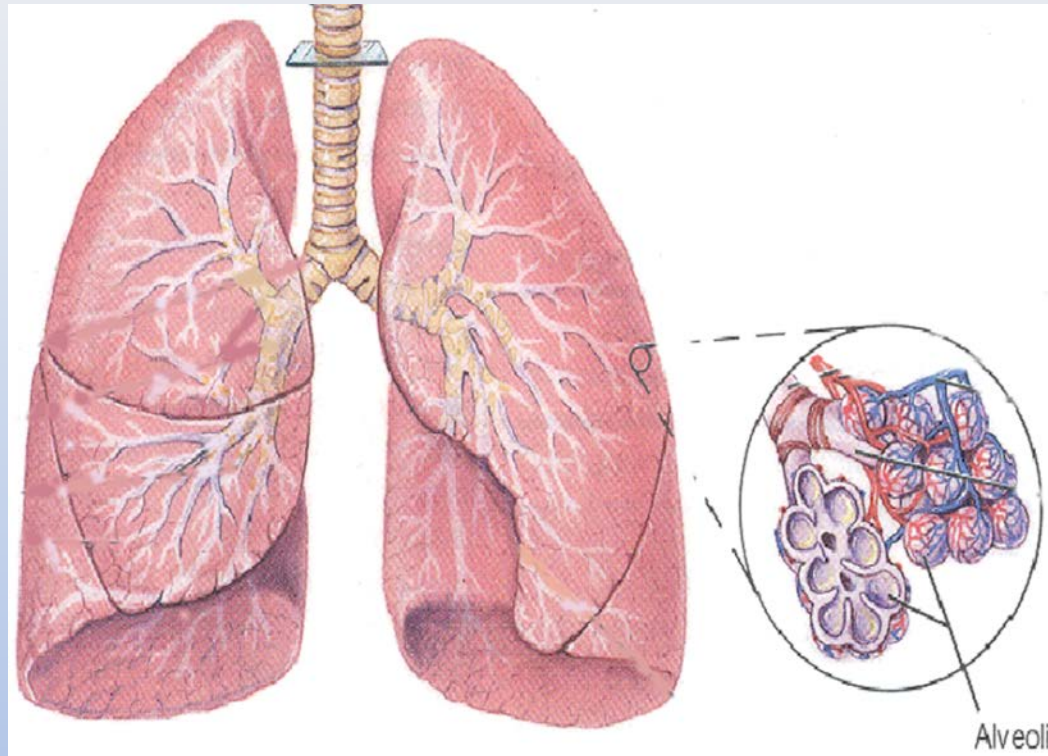


Pain



Sepsis

Oxygen Availability



Dependent on what's happening
back at the respiratory system

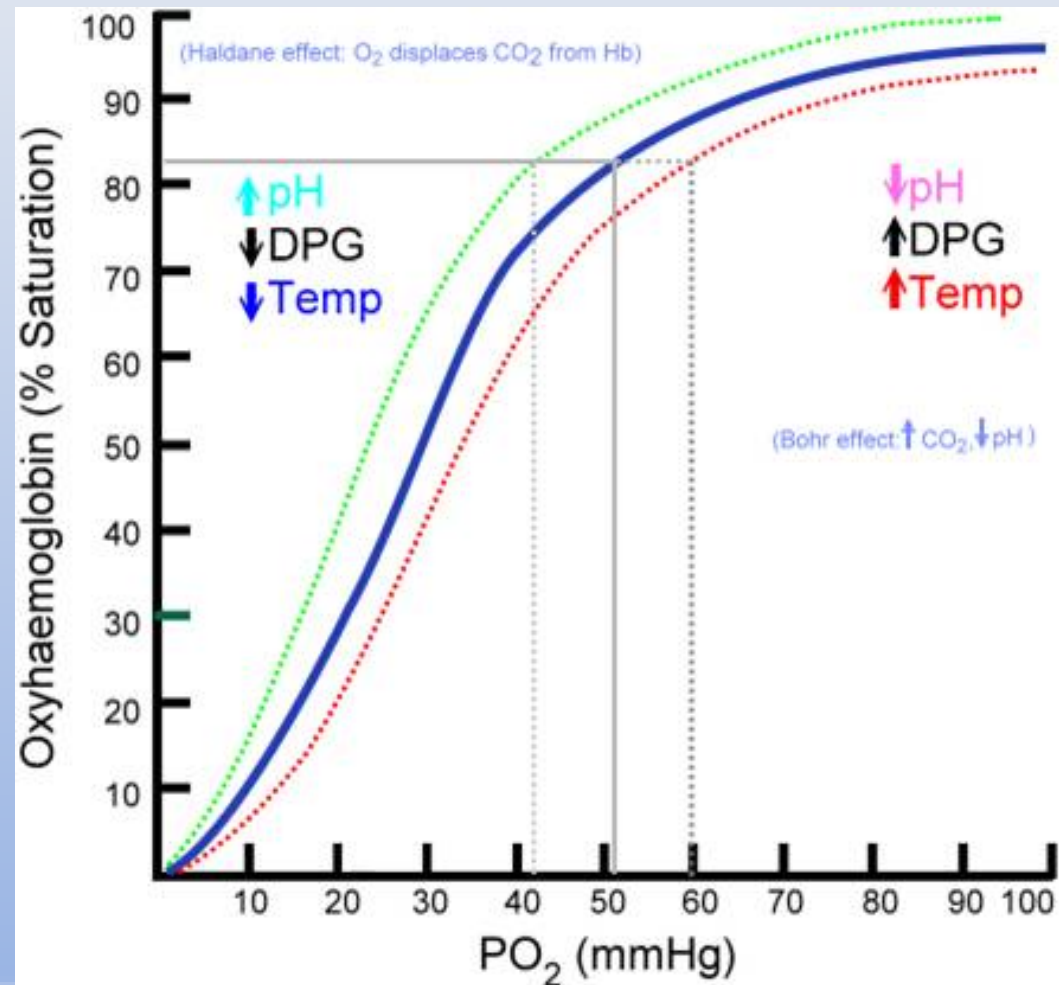
Oxygen Carrying & Transfer Capacity

Sickle-cell anemia

Anemia

Acidic environments

Alkalotic environments



Diagnosing Oxygen Debt

Clinical
oxygen debt:

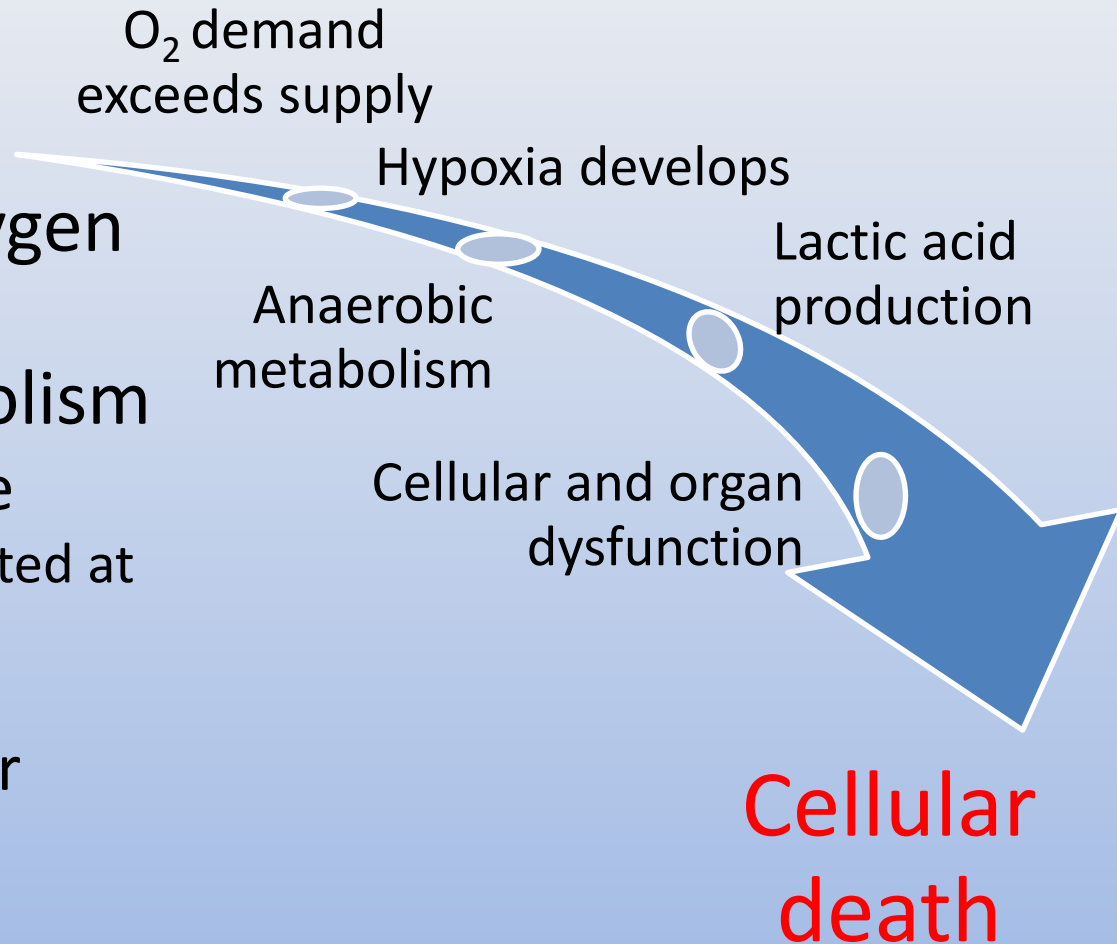
- Elevated lactate
- Ph < 7.35

$$S_v = S_a \times 0.75$$

If < then oxygen debt present

Cellular Changes From Hypoxia

- Normal cellular metabolism dependent on oxygen supply
- Anaerobic metabolism
 - Lactic acid release
 - Considered elevated at $>2.2\text{mm/L}$
 - Decreases pH
 - Decreased cellular function & ATP synthesis



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

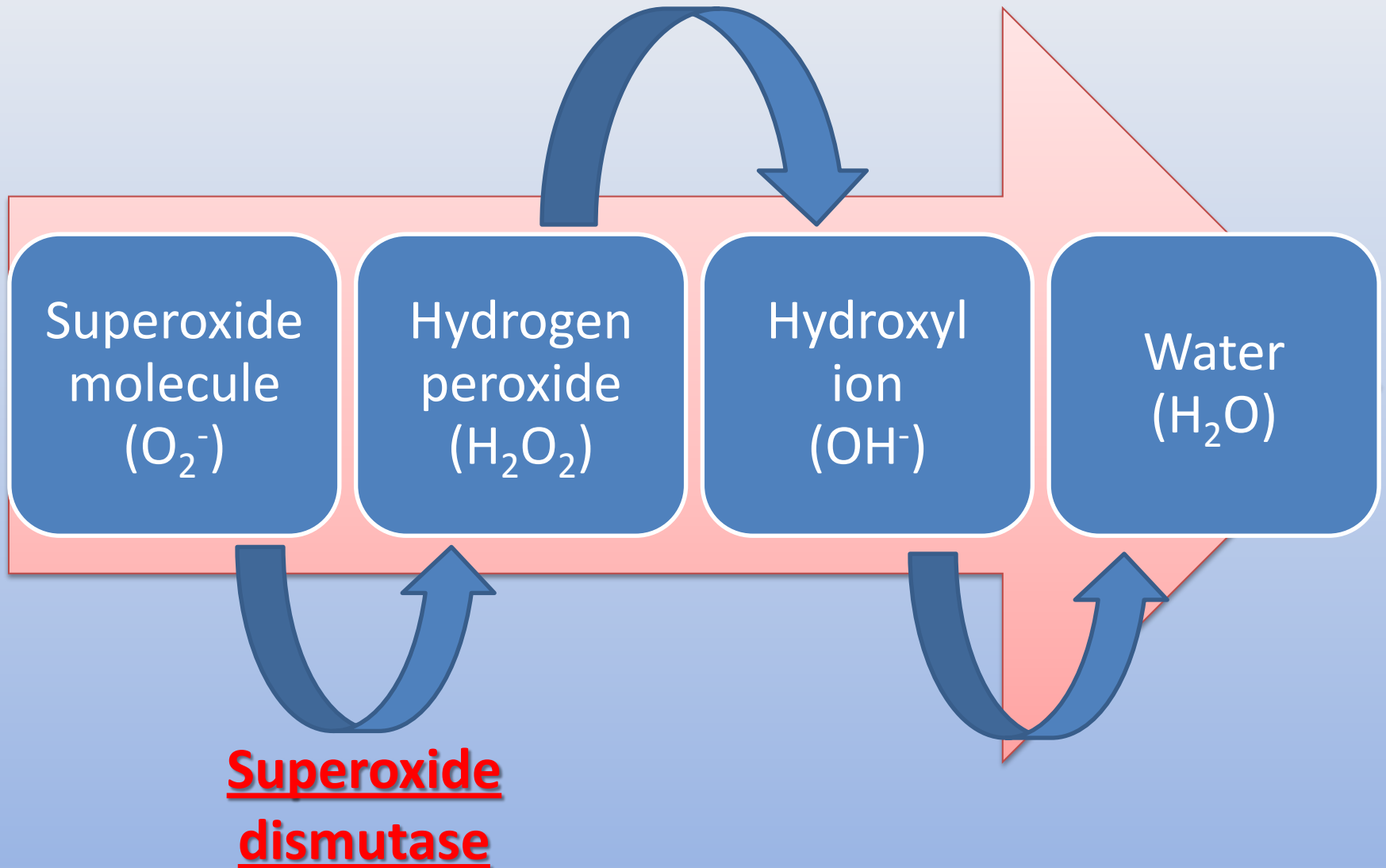
Prevents
hypoxia

Prevents
anaerobic
metabolism

Provided intact
circulation
system free of
occlusions



Cellular Metabolism in Mitochondria



The O_2^- Molecule

Damages cell membranes

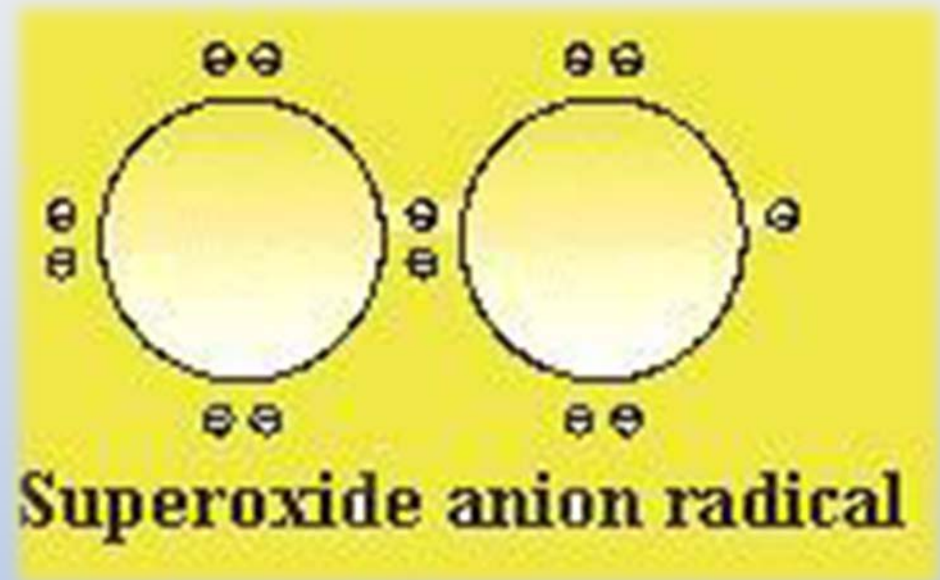
Considered "toxic"

Normally destroyed by enzymes within cell

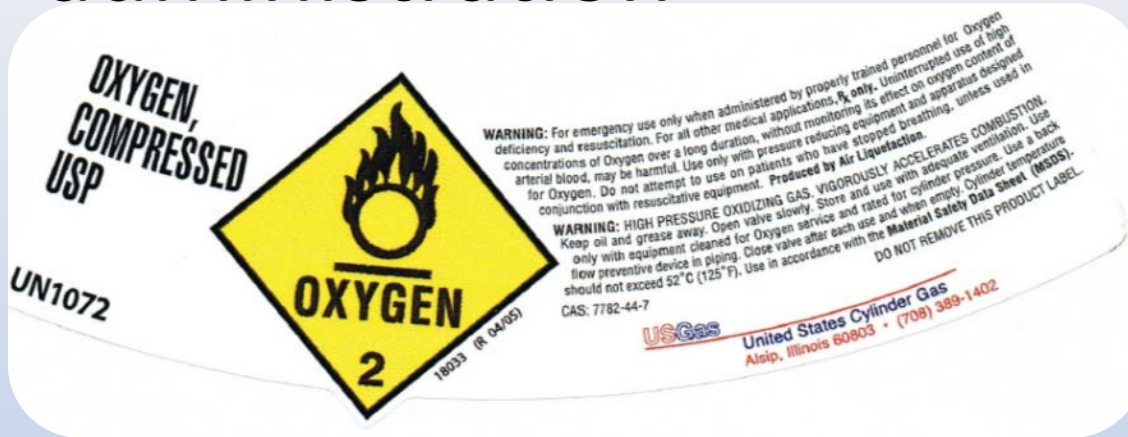
Superoxide dismutase

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

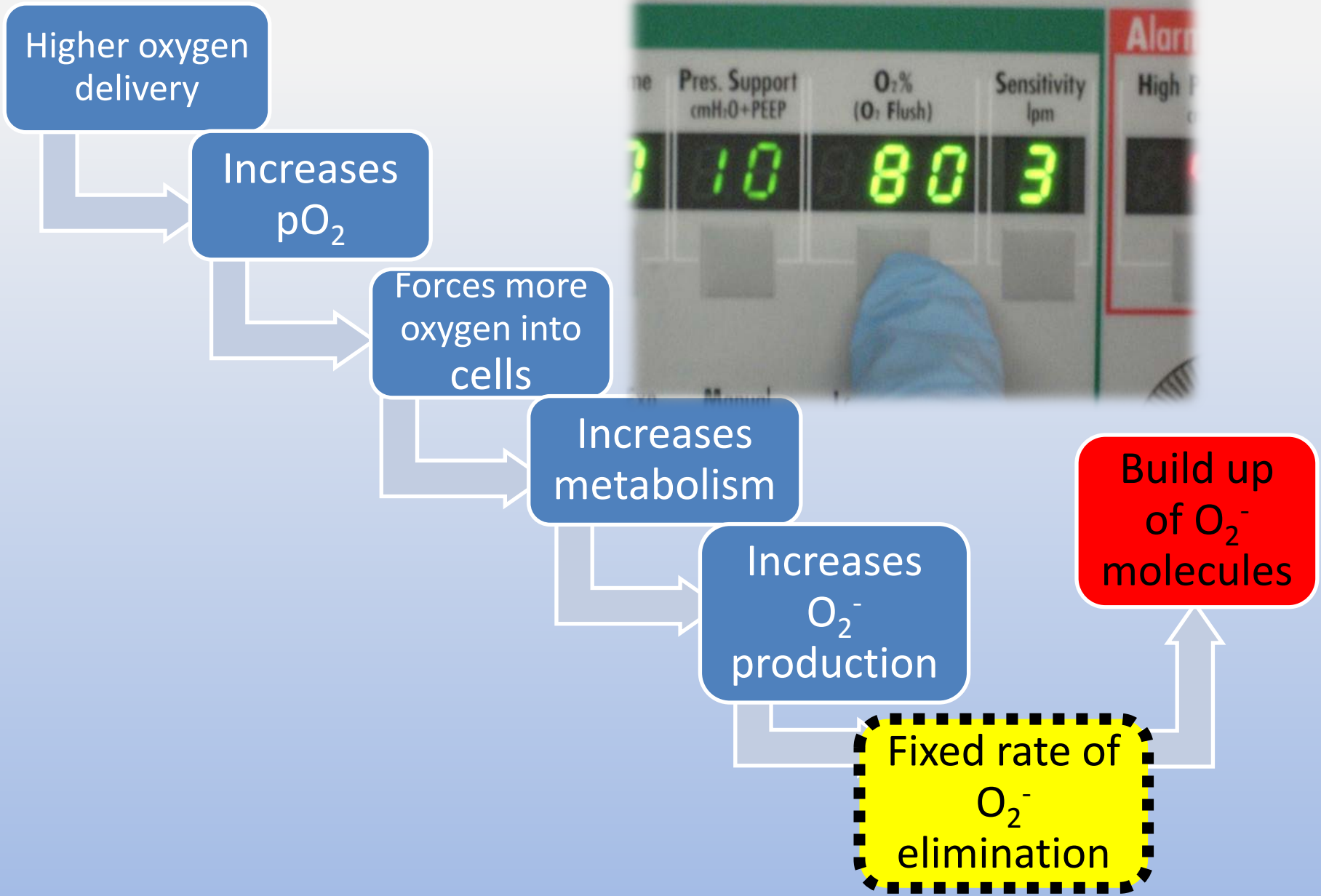
Can also cause epistaxis



Oxygen Toxicity

АААААА!





The Good News?

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



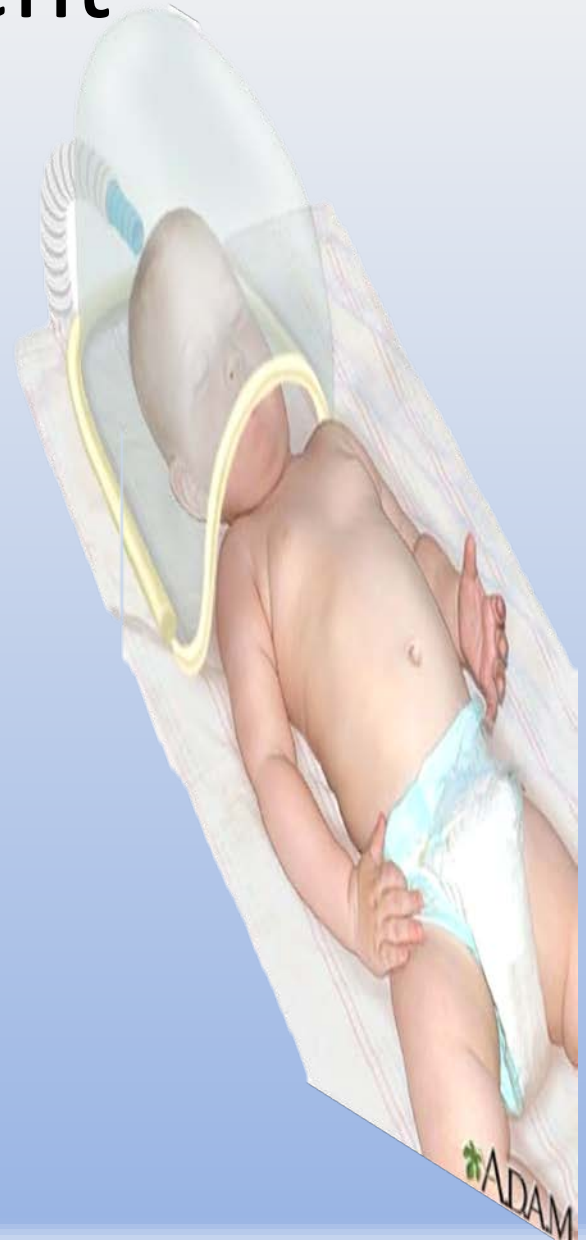
Oxygen Rich Environment

Healthy lungs $FiO_2 > 0.6$

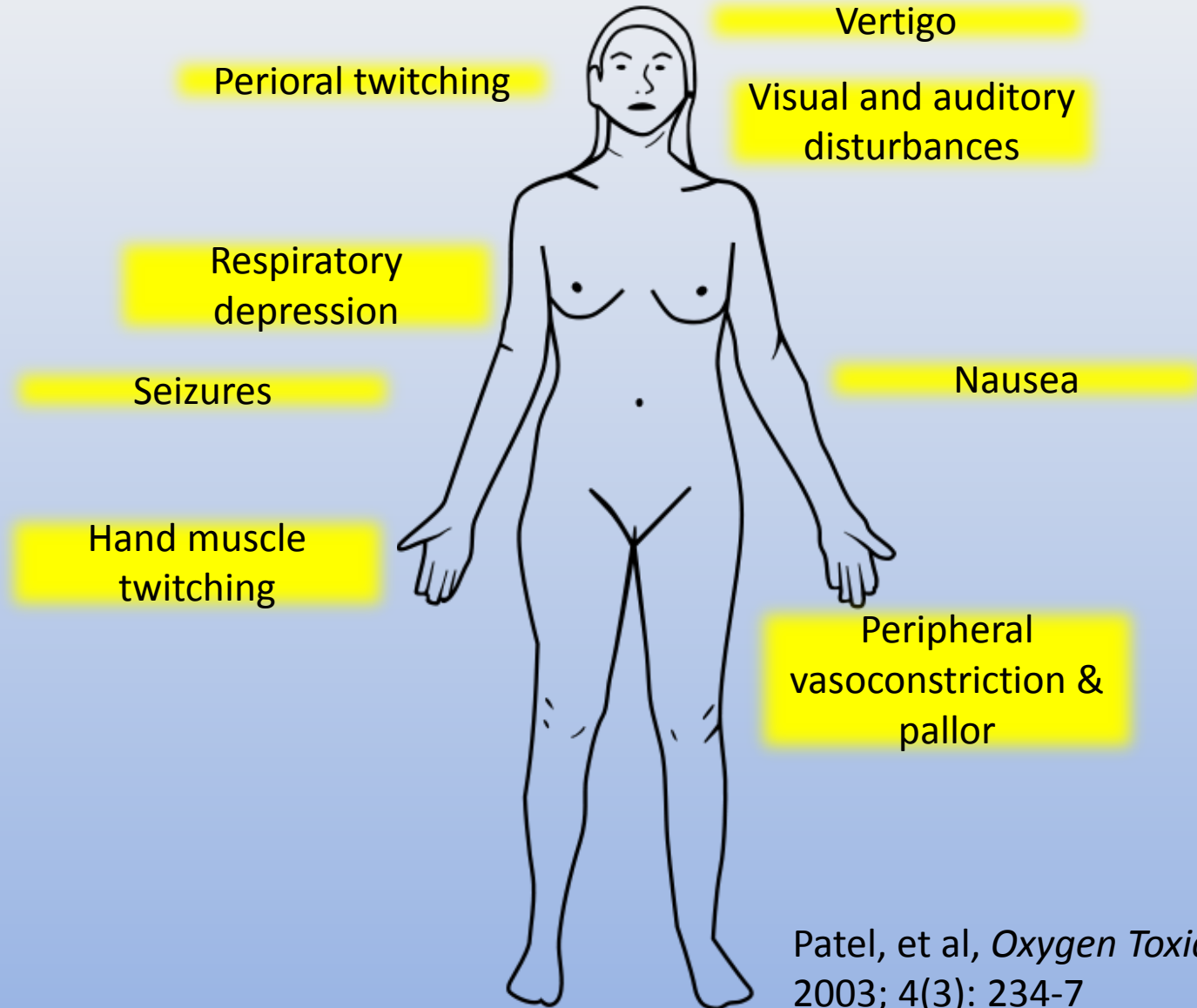
- Alveolar oxygen tensions of 350mmHg

Injured or diseased lungs $FiO_2 > 0.5$

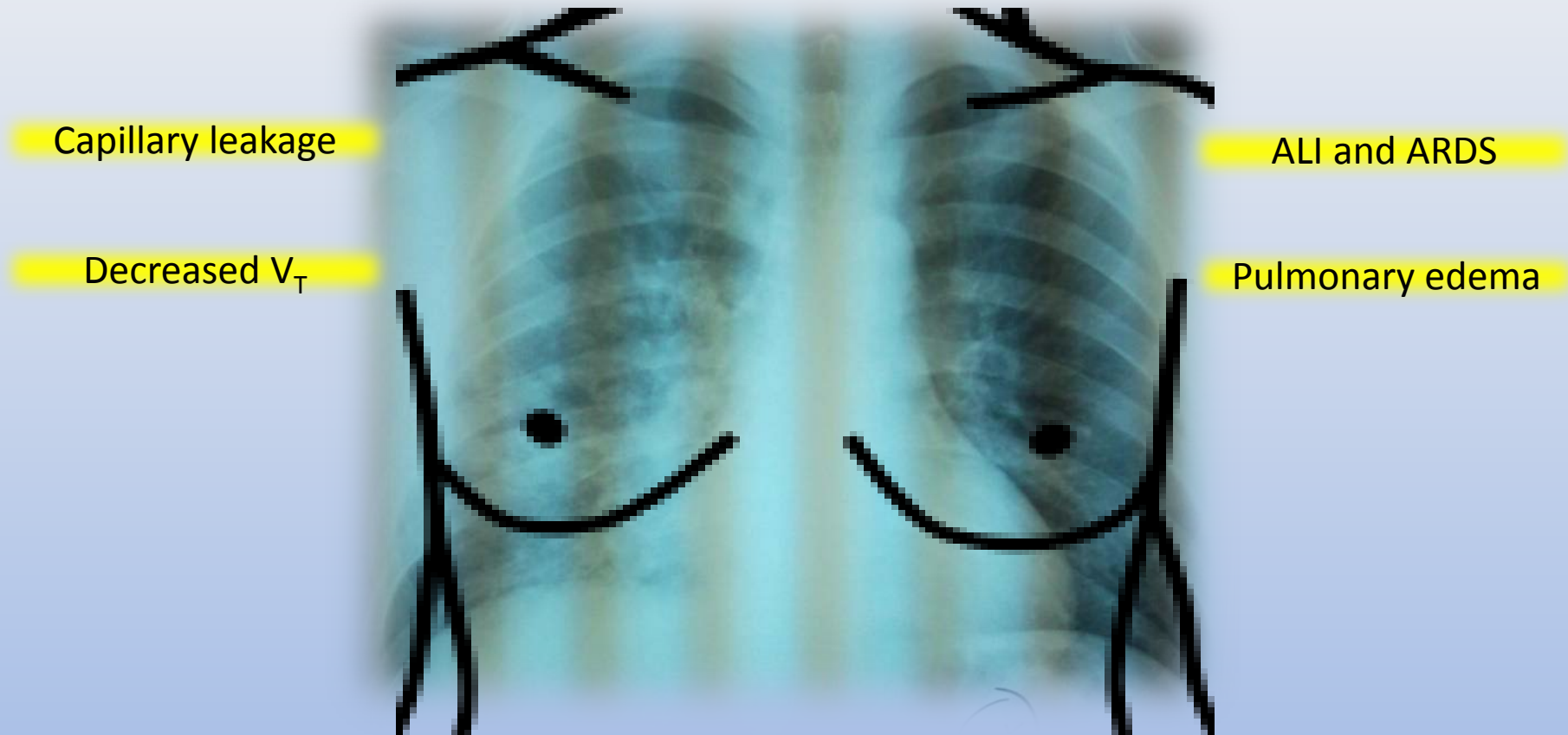
- Alveolar oxygen tensions of 250mmHg



Oxygen Toxicity CNS Symptoms

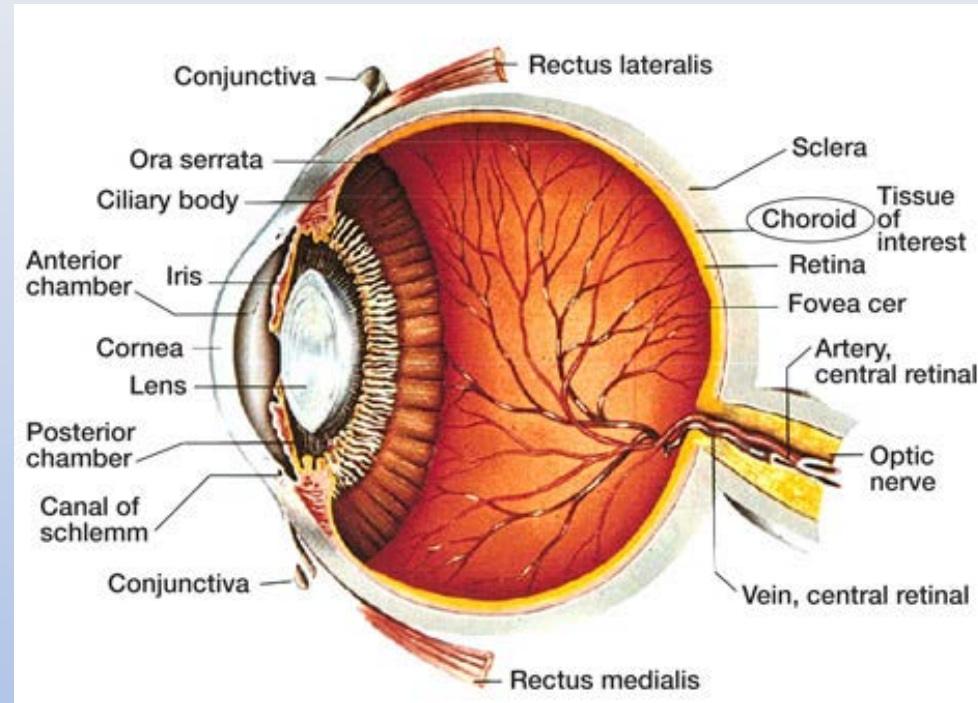


Oxygen Toxicity Pulmonary Symptoms



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

Muscular

- Twitching

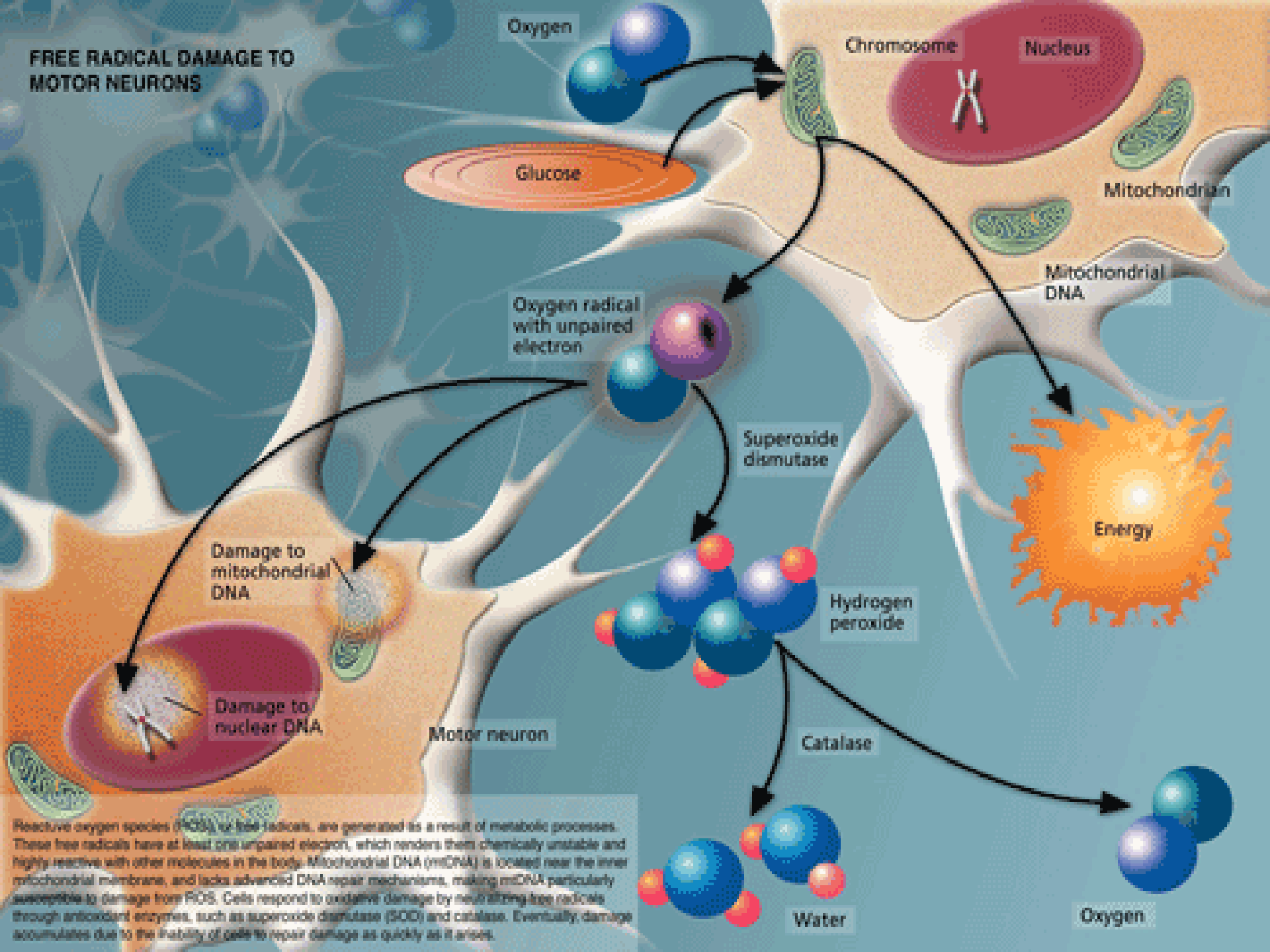
Central

- Seizures

Respiratory

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

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

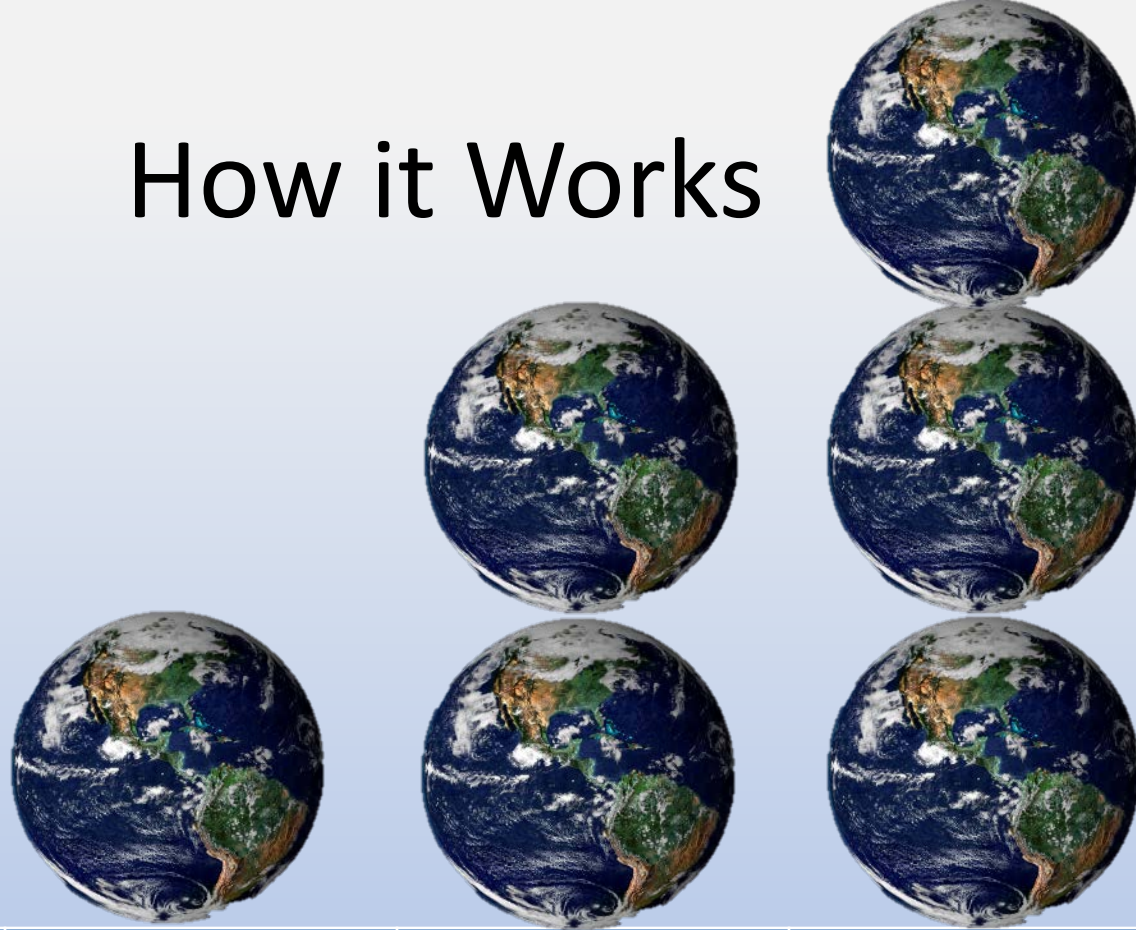
- Gas gangrene
- Refractory chronic osteomyelitis
- Infected burns

Trauma care

CO toxicity

Goal is to increase O_2 availability to cells

How it Works



	760mmHg	1520mmHg	2280mmHg
FiO ₂	Alveolar oxygen pressure		
0.21	101	202	303
0.4	304	608	912
0.6	456	912	1368
1.0	“510”	“1020”	“1530”

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 FiO_2 induces vasoconstriction, particular to temporal region of retina



Anticipated Lab Values

	Birth	Post 5 min	Post 24 hours
pH	≥7.20	7.2-7.34	7.35-7.4
PCO ₂	≤50mmHg	35-46mmHg	33-35mmHg
pO ₂	25-40mmHg	49-73mmHg	72-75mmHg
SaO ₂	>50%	>80%	>90%
HCO ₃ ⁻	≥15mmHg	16-19mmHg	>20mmHg

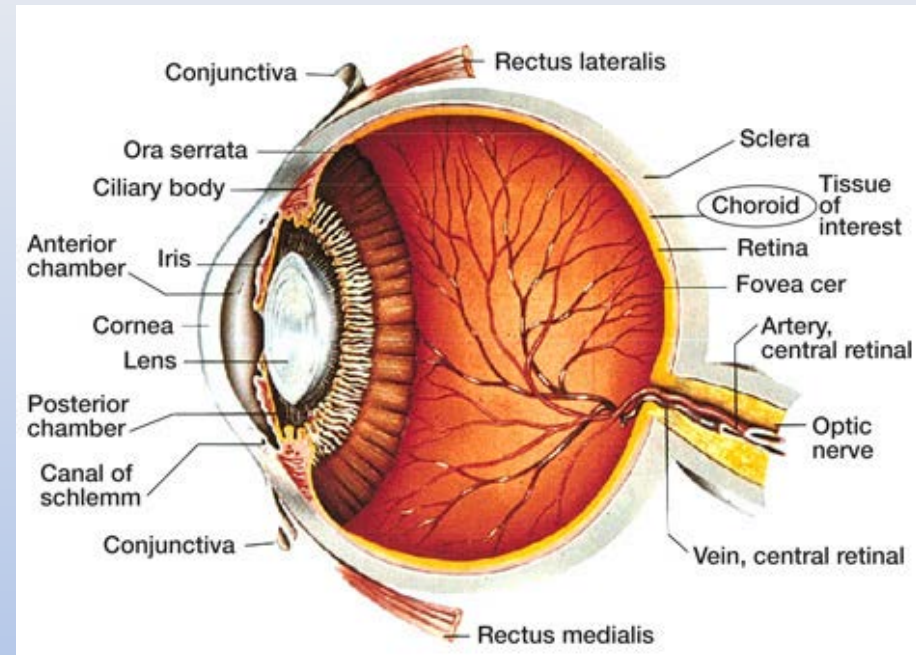
Neonatal Oxygen

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

Post-birth Age	SpO ₂
1 minute	60-65%
1 min	65-70%
3 min	70-75%
4 min	75-80%
5 min	80-85%
10 min	85-95%

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 blood flow to retina
- Becomes a risk when $\text{PaO}_2 > 80 \text{ mmHg}$

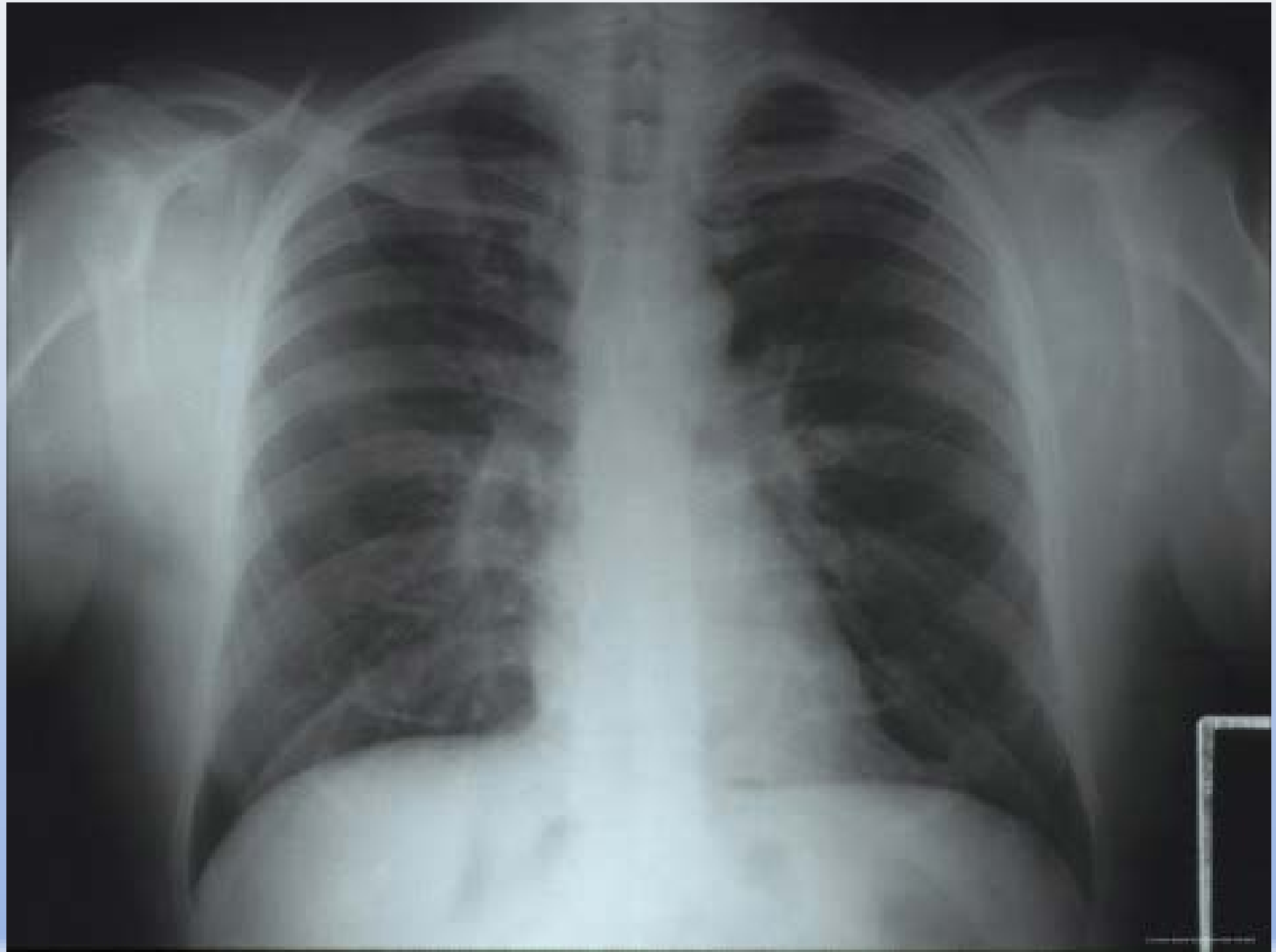




Neonatal Resuscitation Guidelines

(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 SpO₂ 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).

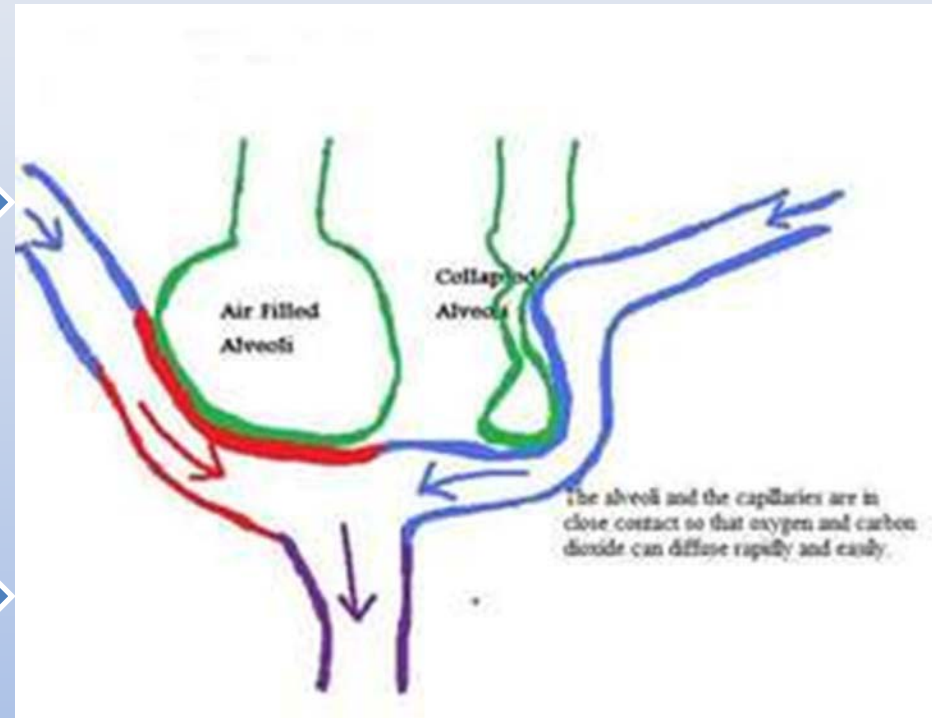
Absorbative Atelectasis



Nitrogen in the Alveoli

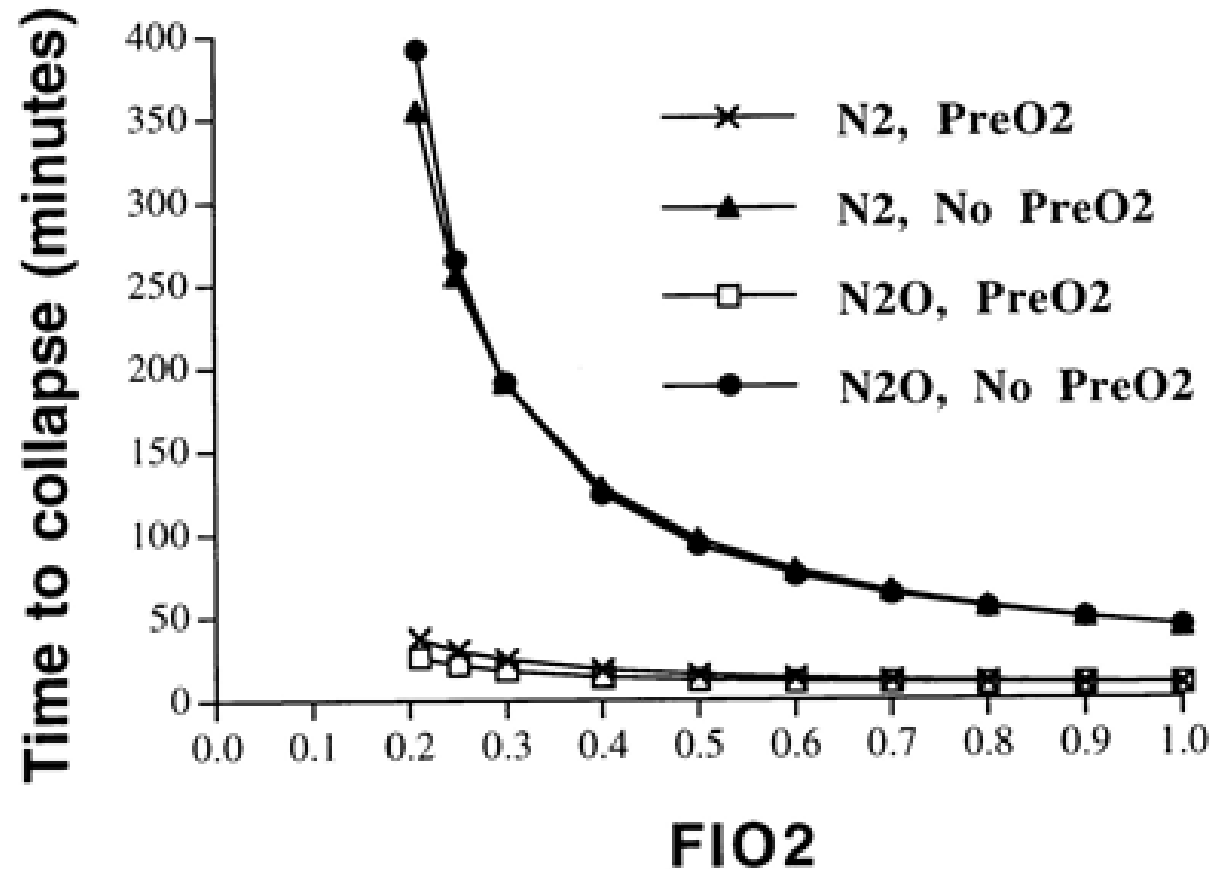
Recall that nitrogen is not well absorbed by body

When alveoli contract during periods of non activity nitrogen keeps the alveoli from collapsing.



Increasing FiO_2

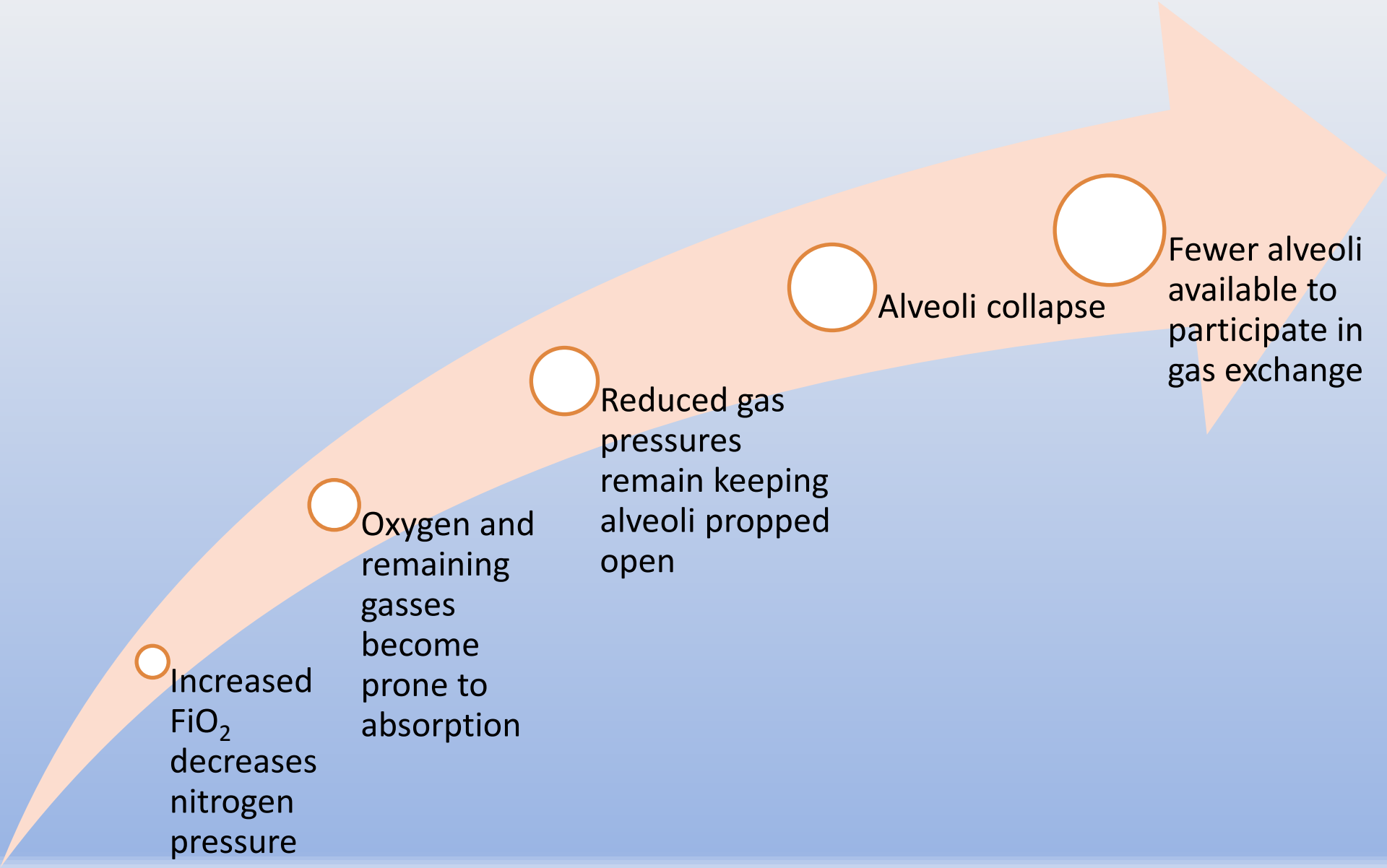
Time to collapse of unventilated lung compartment.



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

Journal of Applied Physiology

What's the Big Deal?



Increased FiO_2 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 V_t to maintain same ABG values on 100% O_2 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



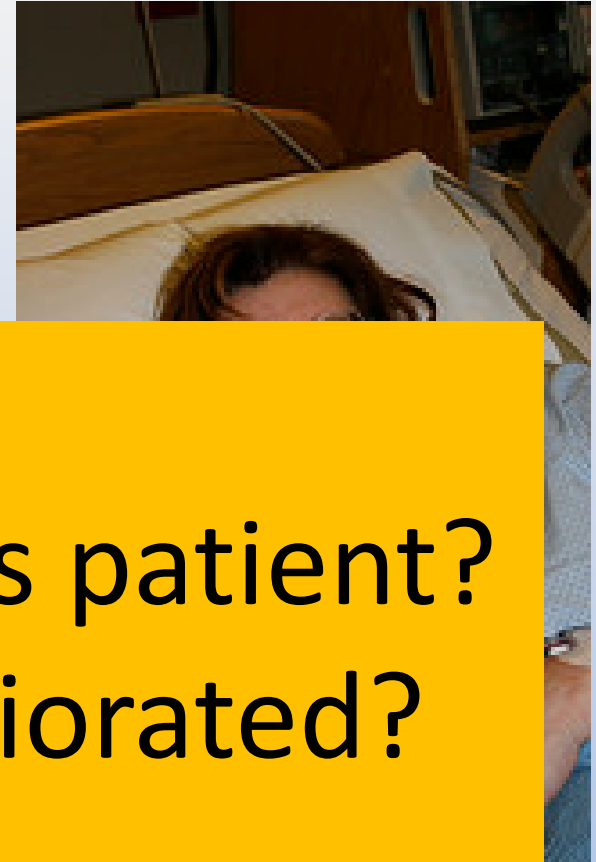
Initial ABG

pH	7.29
pO ₂	56
pCO ₂	64
HCO ₃ ⁻	20

Initial vitals

HR	104
RR	26
SpO ₂	79%
Temp	97.3°F

Case Study



ED Treatments

What happened to this patient?
Why have they deteriorated?

- Decreased respiratory effort
- Now on NRB

Repeat ABG

pH	7.24
pO ₂	80
pCO ₂	95
HCO ₃ ⁻	18

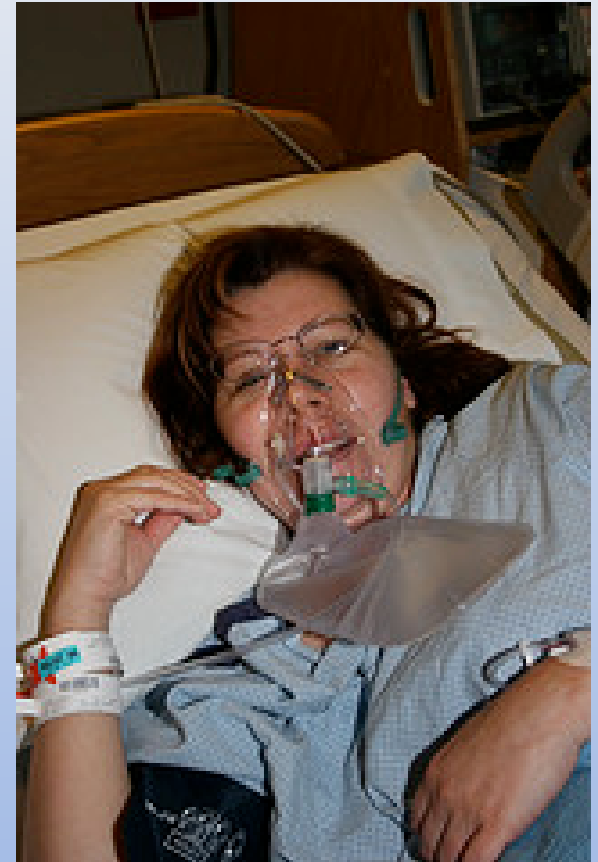
Carbon Dioxide Narcosis

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
 - Myasthenia gravis
 - Poliomyelitis
 - Head injury
 - Increased ICP



Chronic Respiratory Failure

Diagnosed by

- PaCO₂ >50mmHg
- PaO₂ <55mmHg

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

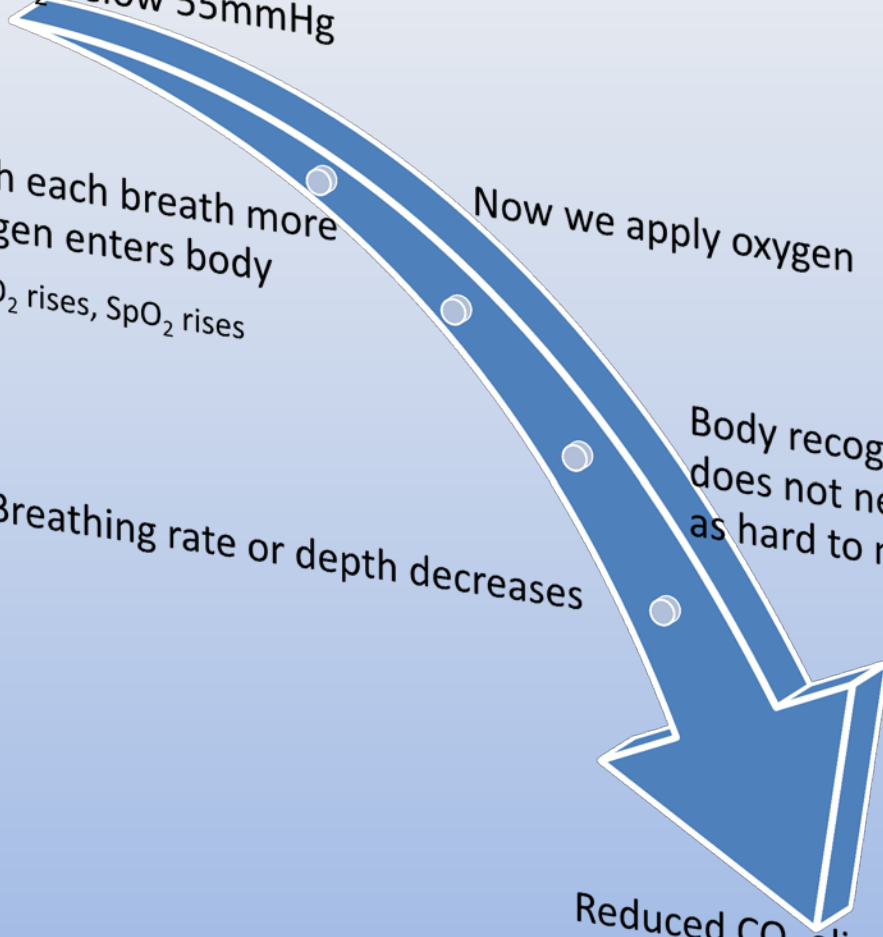
With each breath more oxygen enters body
• PaO₂ rises, SpO₂ rises

Now we apply oxygen

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

Breathing rate or depth decreases

Reduced CO₂ eliminated with each breath



Symptoms



Decreased respiratory rate

Decreased Vt

Measureable increases in CO₂ levels

- Sidestream CO₂
- PaCO₂

Mental status changes

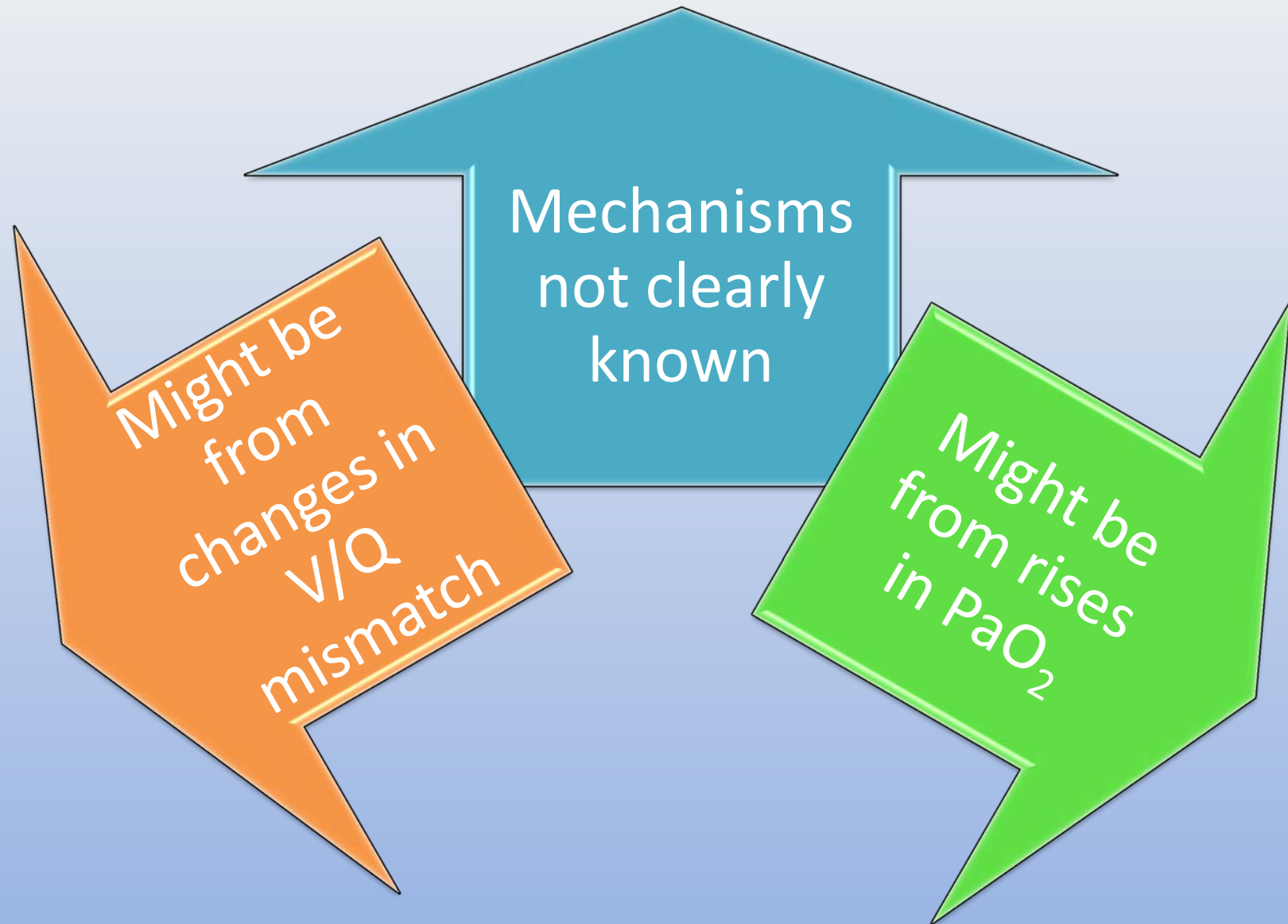
- Lethargy
- confusion
- Headache
- Somnolence

Sweating

Twitching

papillioedema

Caused by the “Hypoxic Drive?”



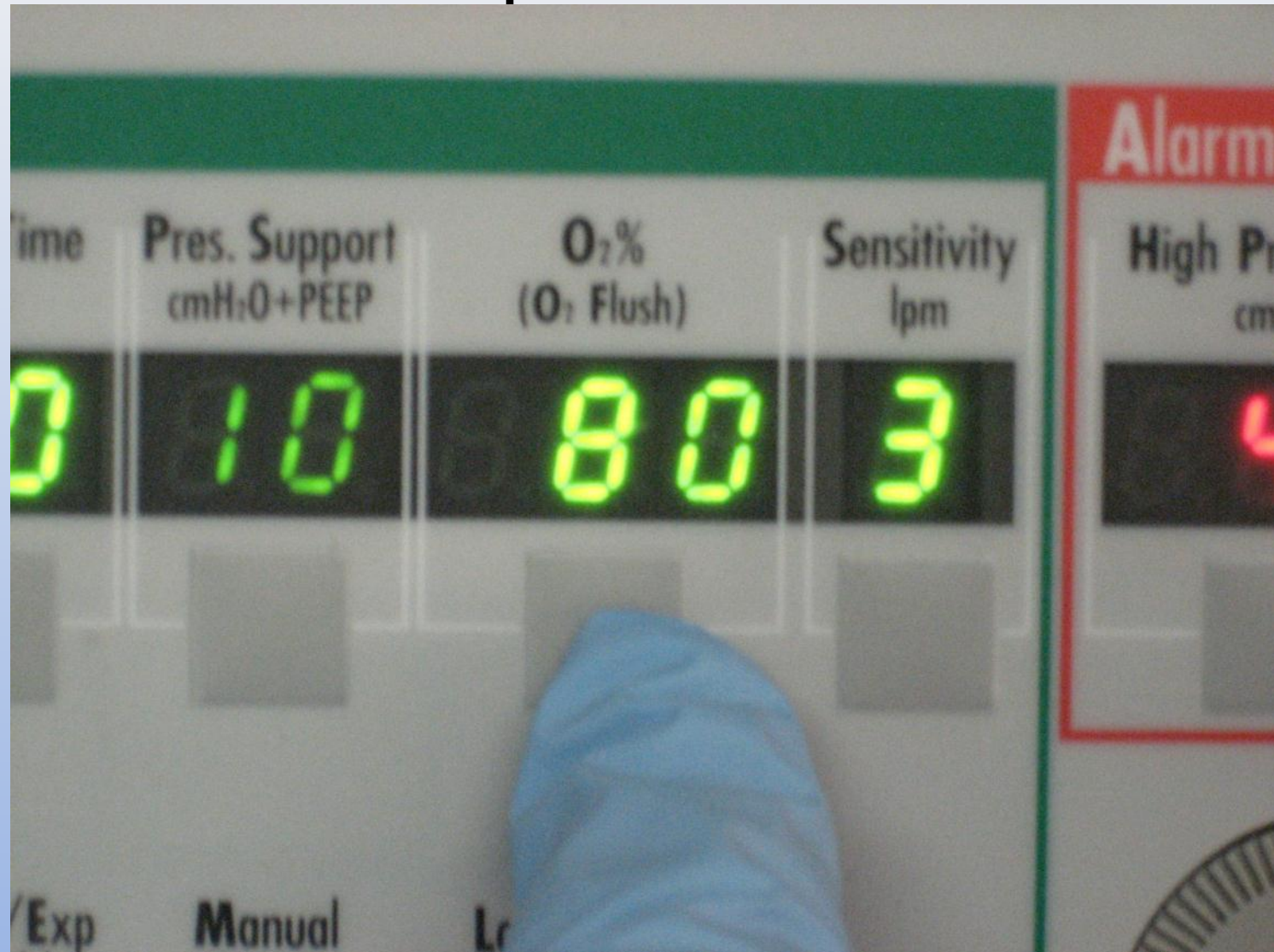
What do we KNOW?

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

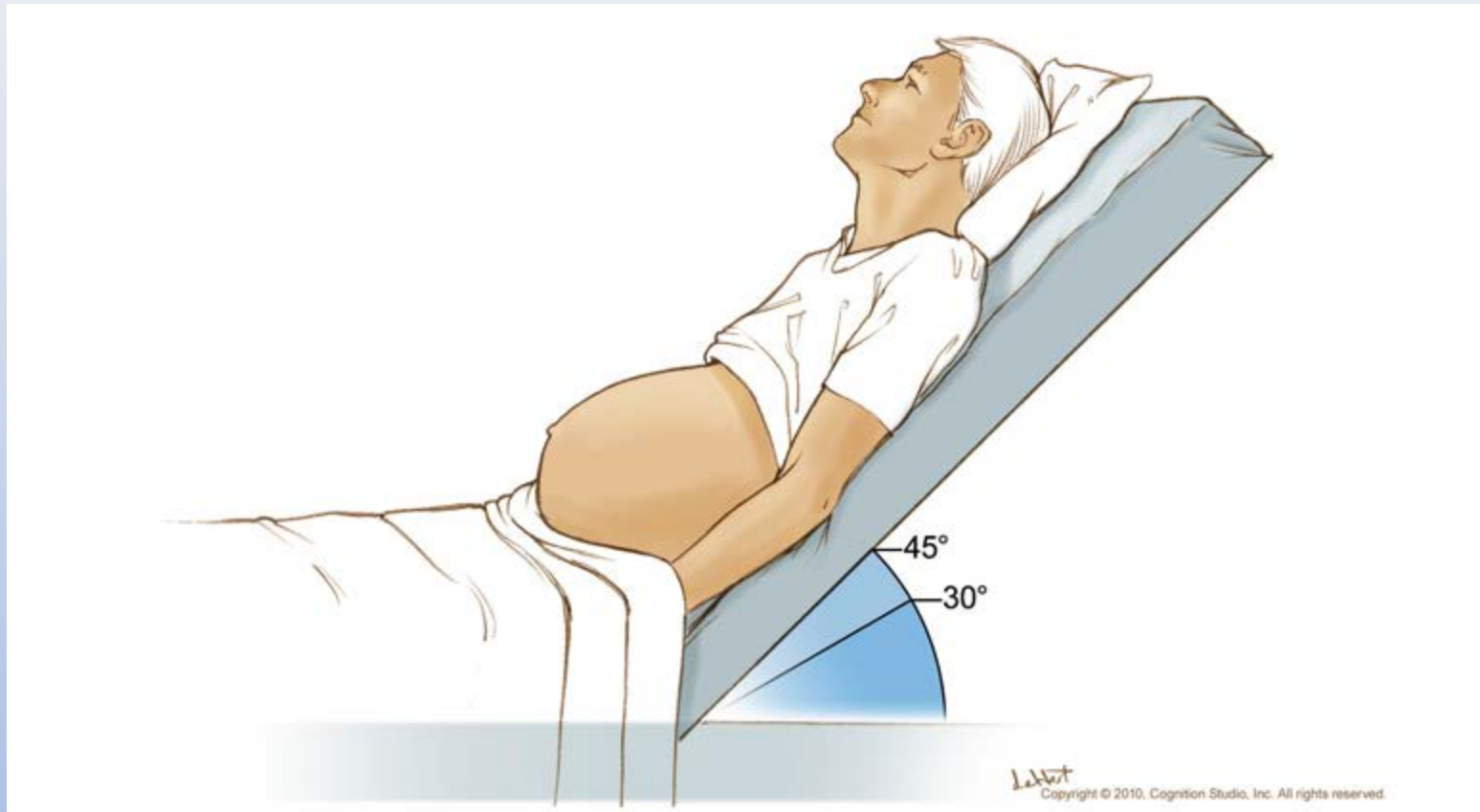
Any therapy over 4 LPM or $FiO_2 > .36$

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

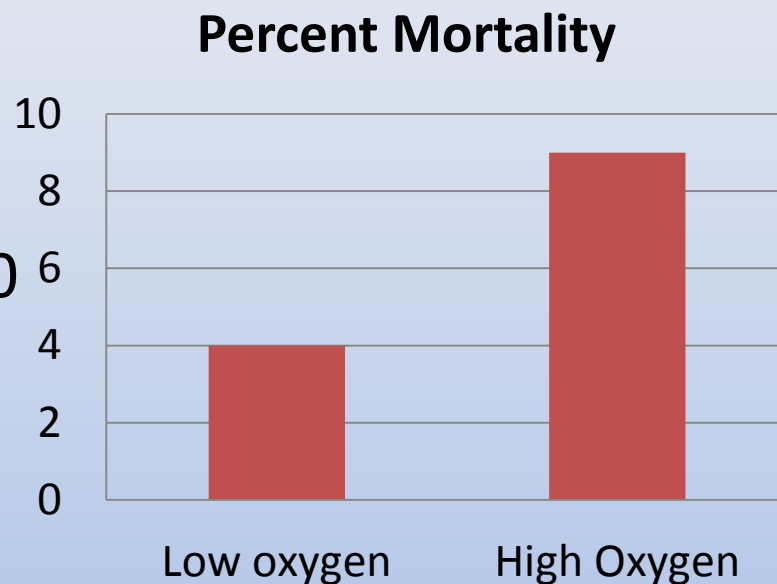
Oxygen Titration

- Never withhold oxygen, goal SpO₂ is 90-95%
 - 94-98% for patients <70yrs
 - 92-98% for patients >70yrs
- Remember SpO₂ of 90 Correlates to pO₂ 60mmHg
 - In patients with COPD aim for a PaO₂ of 50-55mmHg
- Oxygen's maximum benefit is in the 22-50% range



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 SpO₂ between 88-92%
- High oxygen group: NRB with oxygen at 10LPM



Austin M.A., et al., The Effect of High Flow Oxygen on Mortality in Chronic Obstructive Pulmonary Disease Patients in Prehospital Setting, BMJ 2010; 341

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%



What about STEMI?

2013 ACC/AHA Guidelines

- Limited data supports routine oxygen use
- It is appropriate for patients with $\text{SpO}_2 < 90\%$

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%

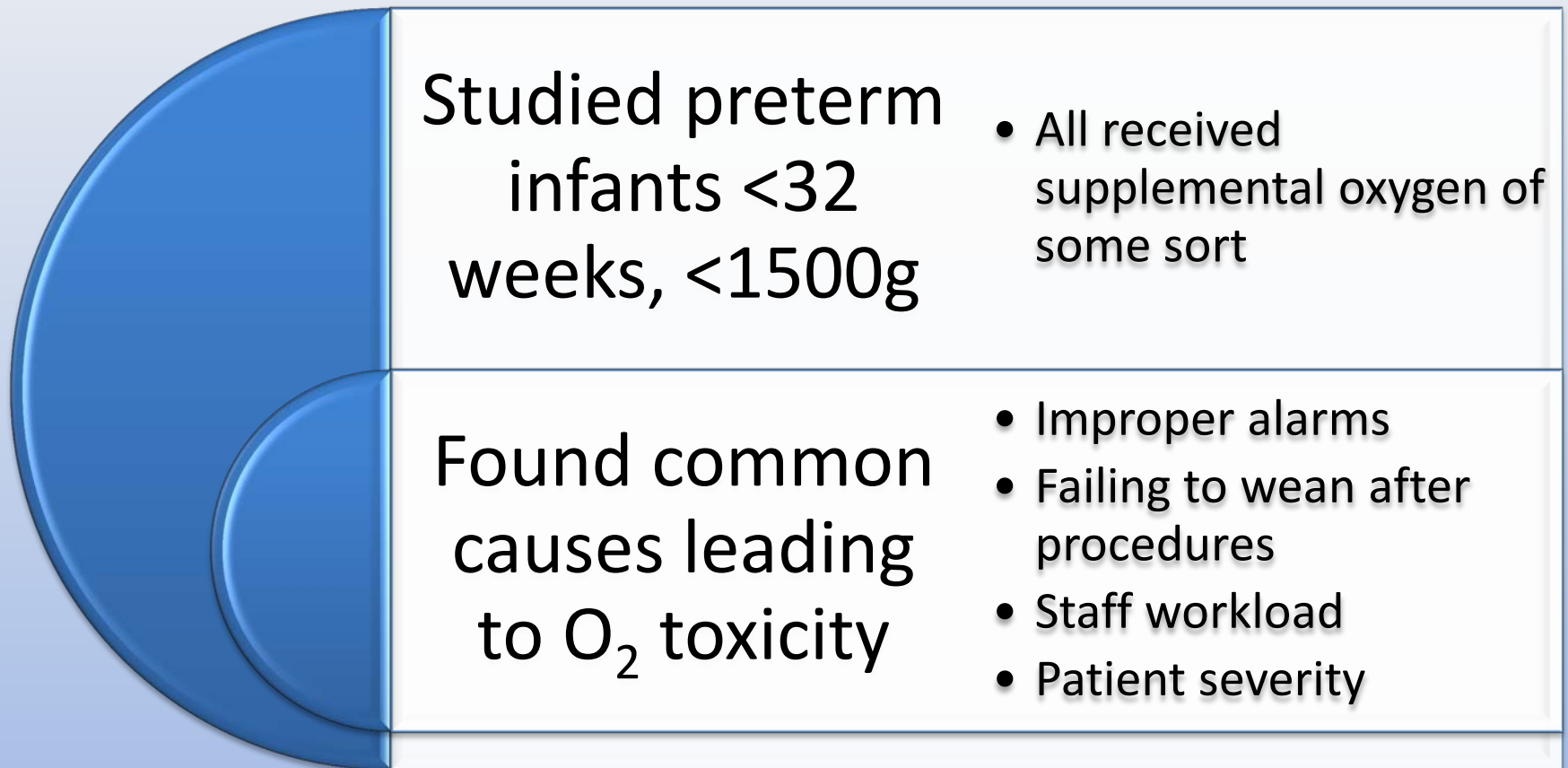
Oxygen in Pediatric Patients

- Utilize room air when possible
- Titrate up
- Use a pediatric flow meter
- Compare minute volume to oxygen LPM
- Keep PaO₂ below 160mmHg
 - Considered critical





SpO₂ in Preterm Infants



When Managing a Neonate

Utilize
 FiO_2 0.21-
0.30

- Lower mortality: 8% vs 13%
- Shorter period on supplemental oxygen
- Shorter ventilated period
- Similar time vs high FiO_2 to normal SpO_2

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

Bledsoe, et al, *Low-Fractional Oxygen Concentration Continuous Positive Airway Pressure is Effective in the Prehospital Setting*, Prehospital Emergency Care 2012; 16: 217-221



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 FiO_2 by 0.01 pO_2 will decrease PO_2 by 7mmHg



Ventilator Adjustments

$pO_2 < 60\text{mmHg}$

Increase FiO_2
to 0.6

Increase
peep

Increase FiO_2
above 0.6

$PaCO_2 > 45\text{mmHg}$
($EtCO_2 > 50\text{mmHg}$)

Increase V_r

Increase V_t

$SpO_2 > 95\%$

Reduce FiO_2
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_2 of 0.5, helps decrease the work of breathing, improves metabolism, decreases pain and anxiety, and
- Increasing the FiO_2 above 0.5-0.6 is necessary in some situations
- Utilize strategies to maximize the benefits of oxygen at the lowest oxygen setting possible

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See you at EMS
World Expo 2014