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

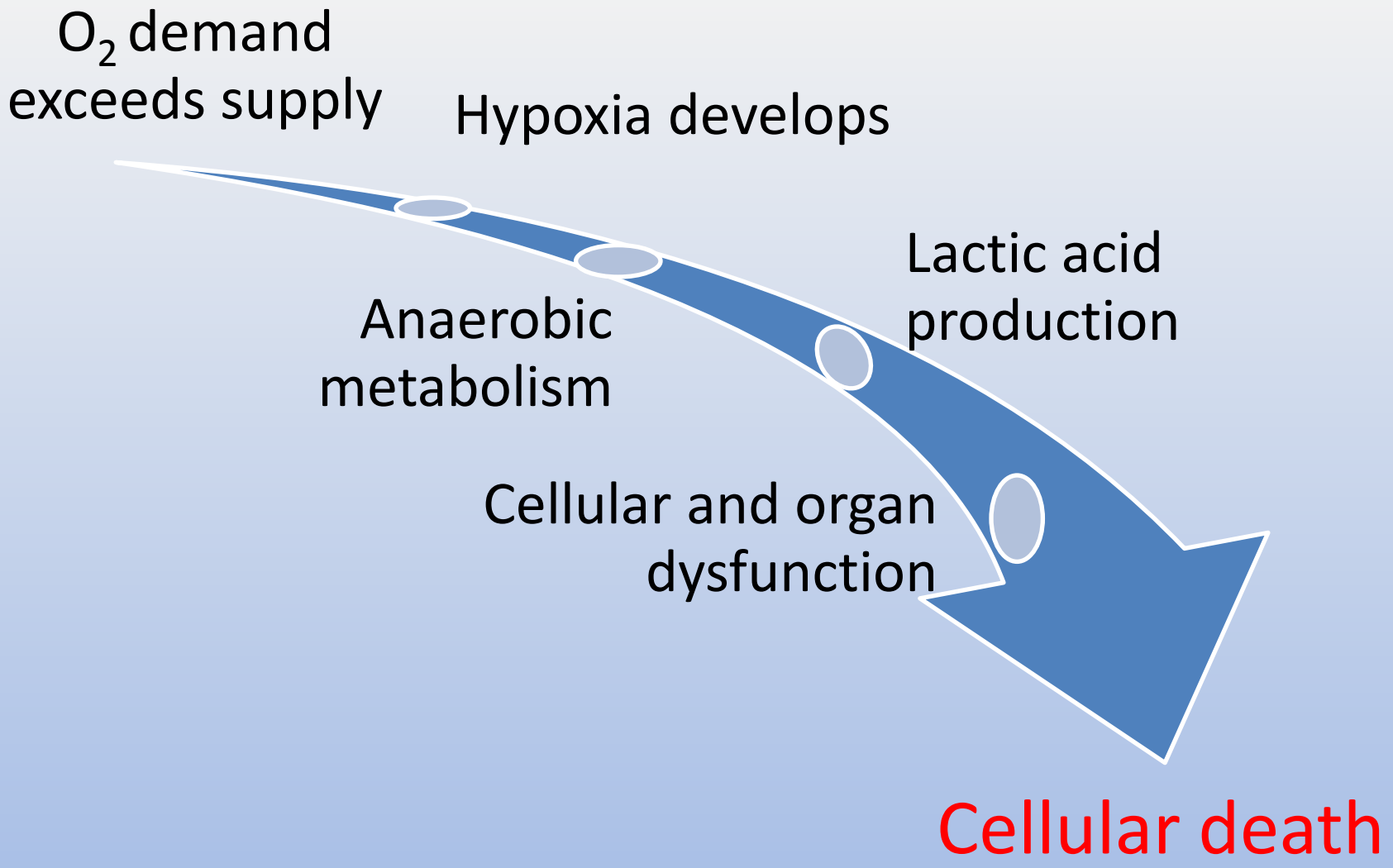
Objectives

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


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

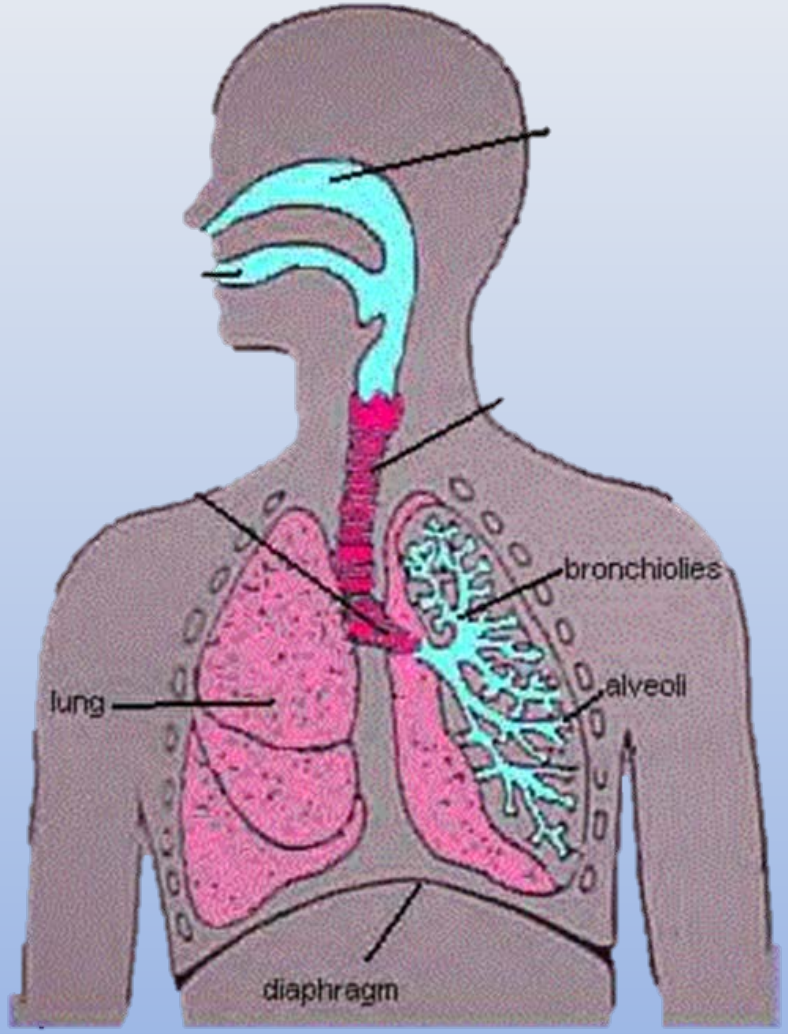
Lower airway

- Tracheobronchial tree
  - 23 divisions, begin at carina

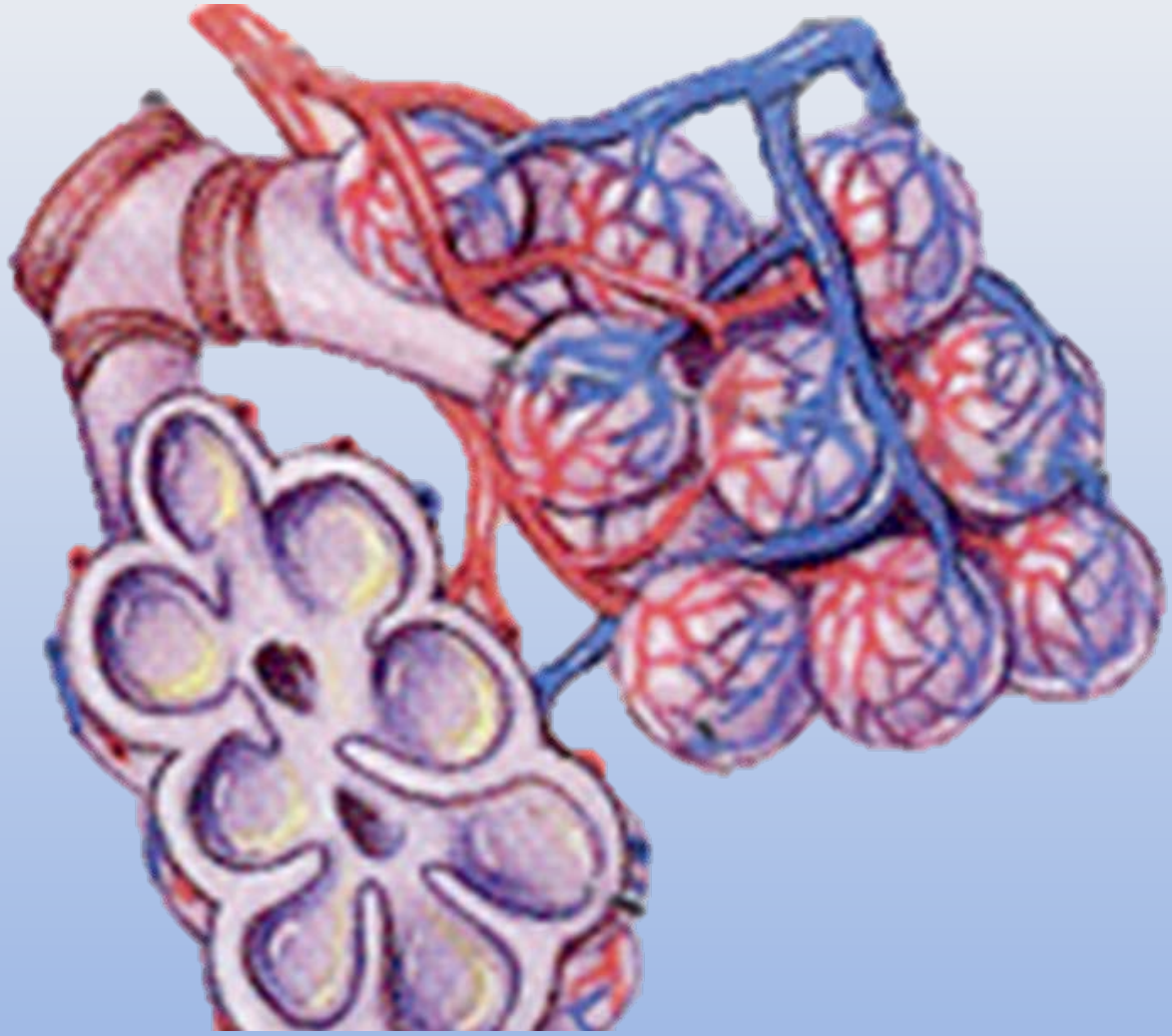
Alveoli

Chest wall & diaphragm

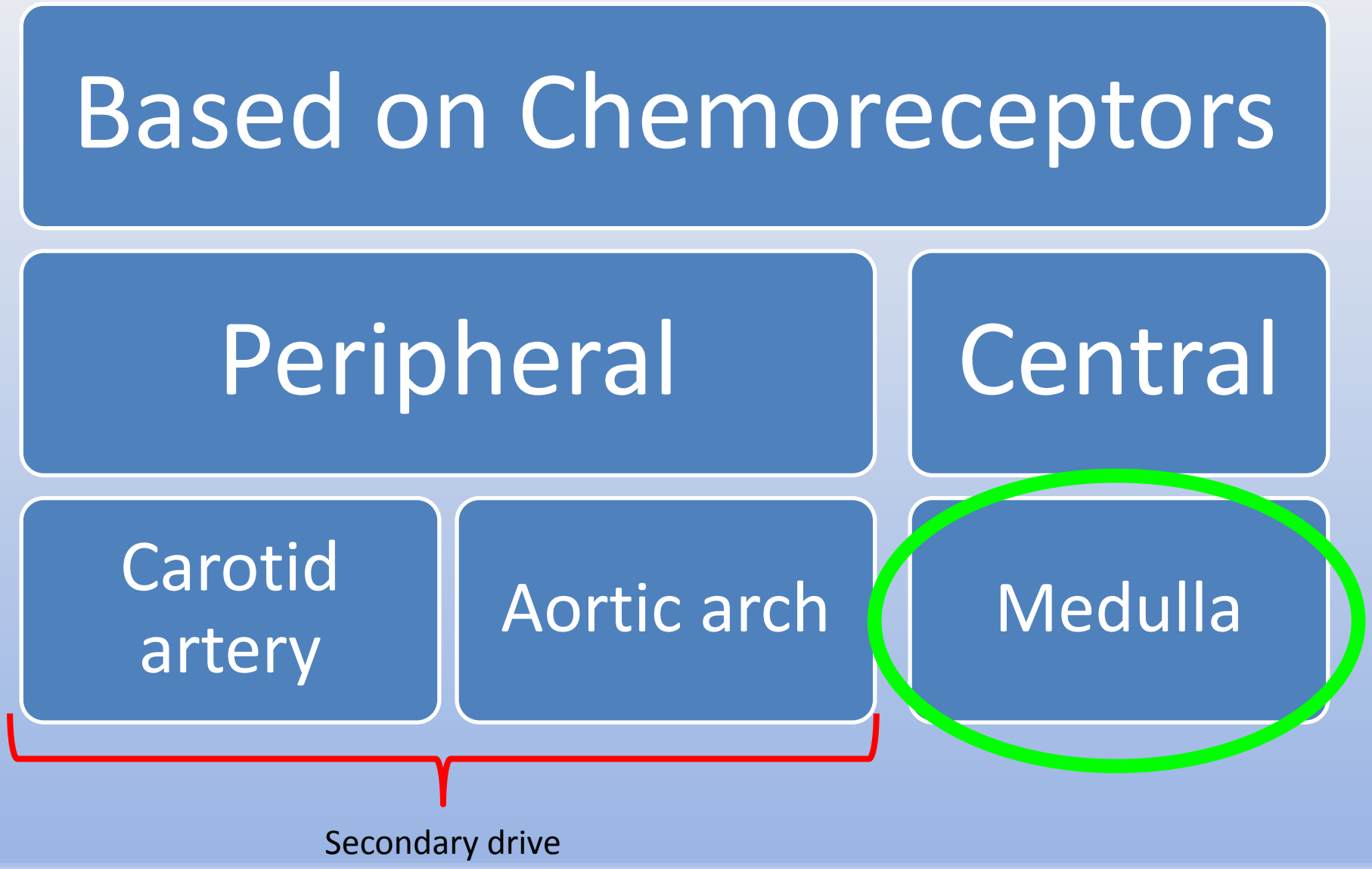
Neuro drive



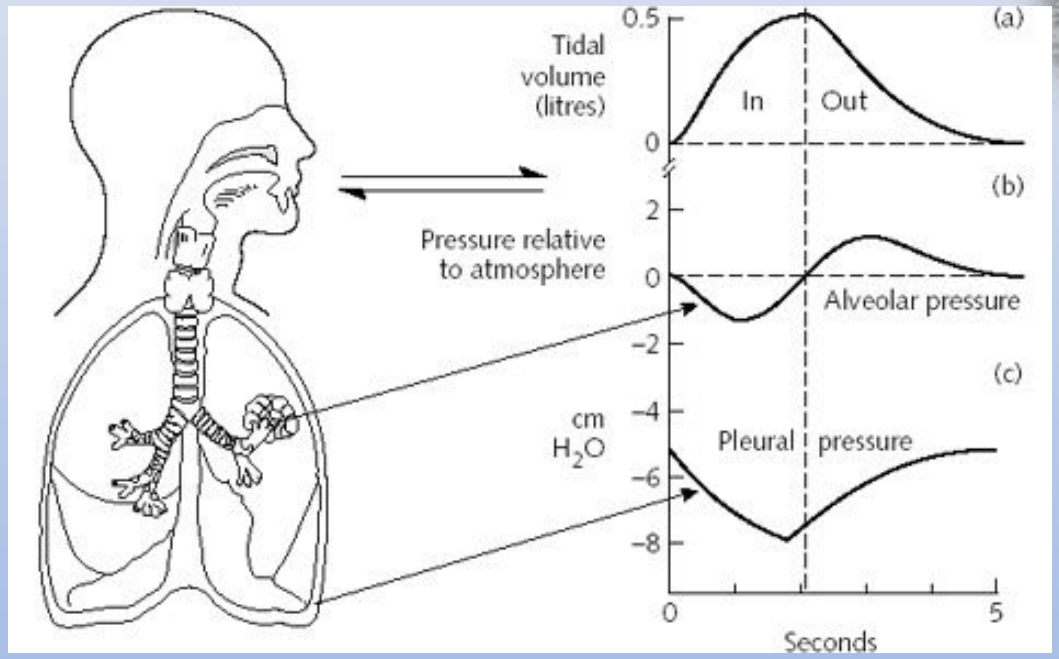
Alveoli



Neuro Drive


Based on Chemoreceptors
Peripheral
Central
Carotid artery
Aortic arch
Medulla
Secondary drive

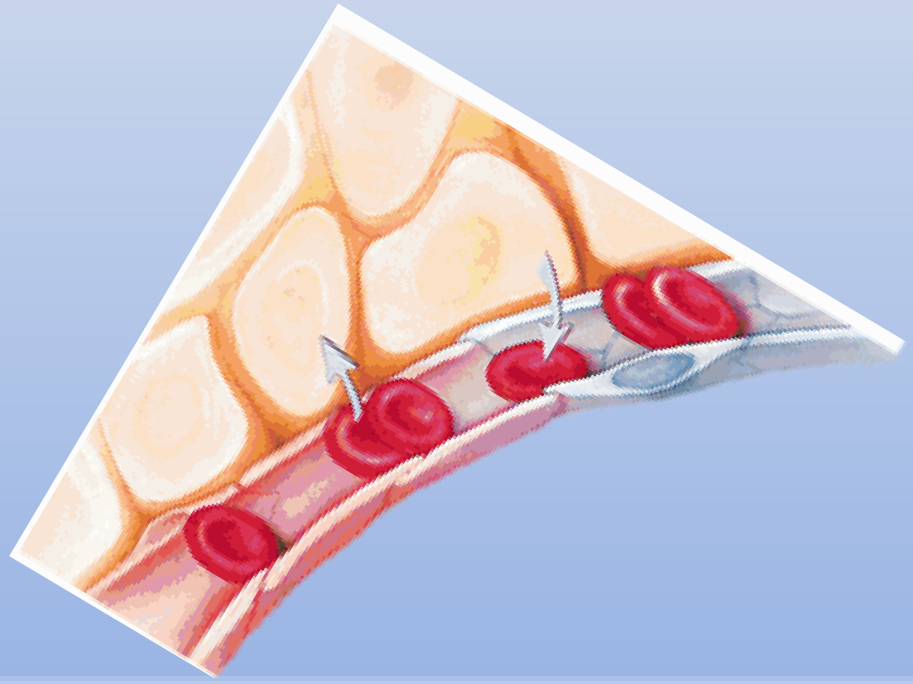
Ventilation

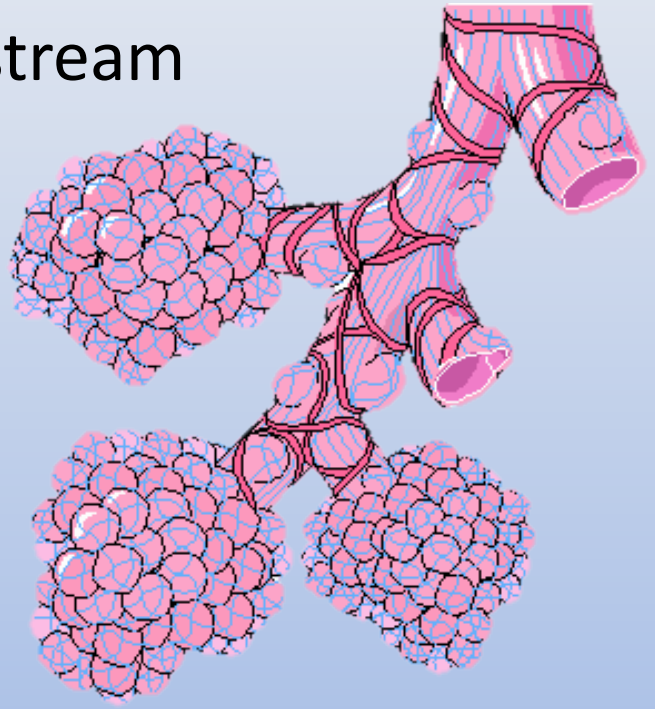


Ventilation is the exchange of gases into and out of the respiratory system

Respiration

- Alveolar respiration
  - Only gasses in alveoli and blood stream
  - Dead air space has no exchange
- Cellular respiration





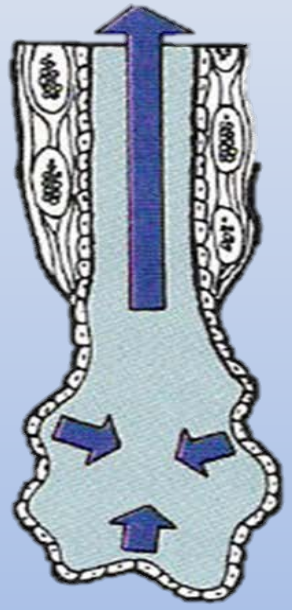
Normal Air Components

- 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%)

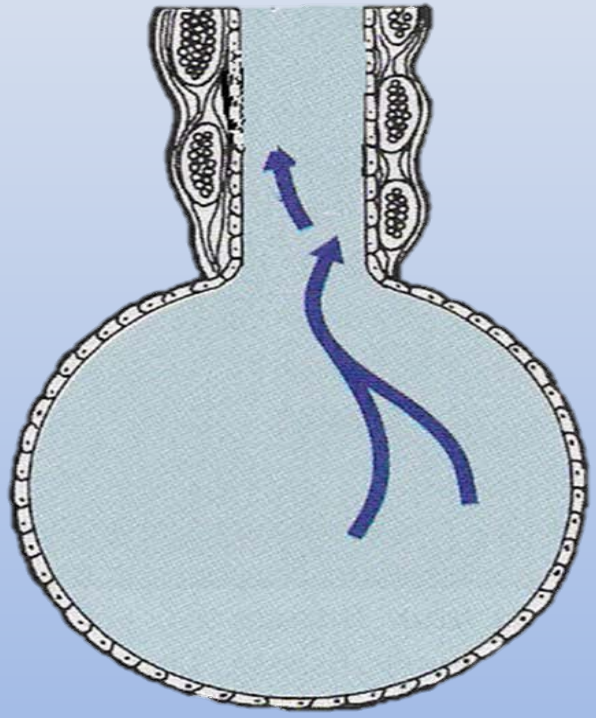
Nitrogen

Significance:

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



Without Nitrogen

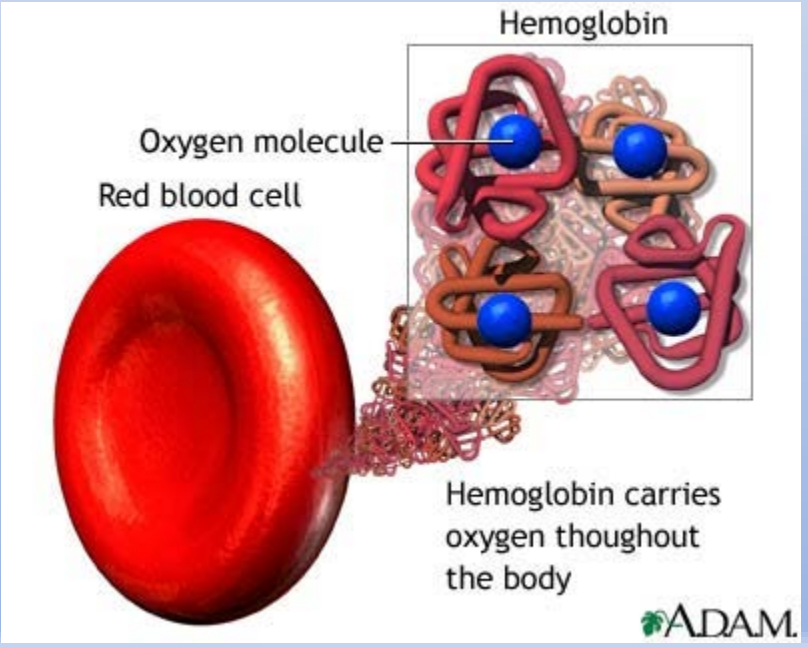


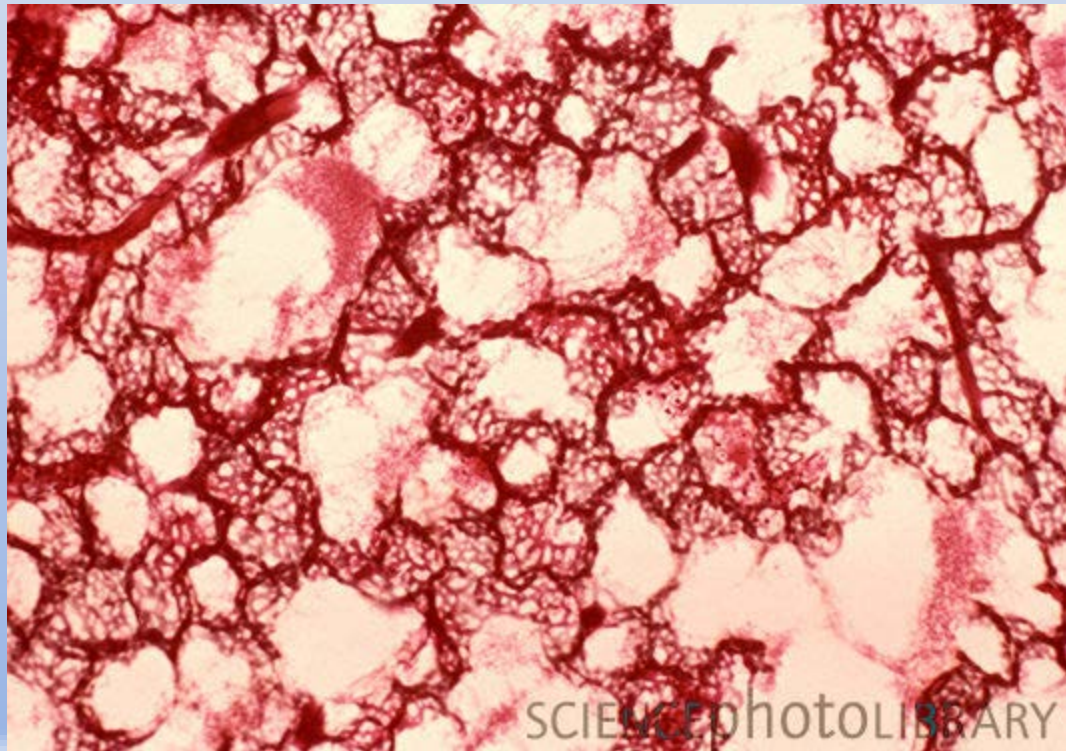
With Nitrogen

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 SaO_2
- SpO_2 similar but same as SaO_2
- Can't distinguish CO from O_2
- Normally 95% of O_2 attached to hemoglobin

Highest reading SaO_2 or SpO_2 can be 100%

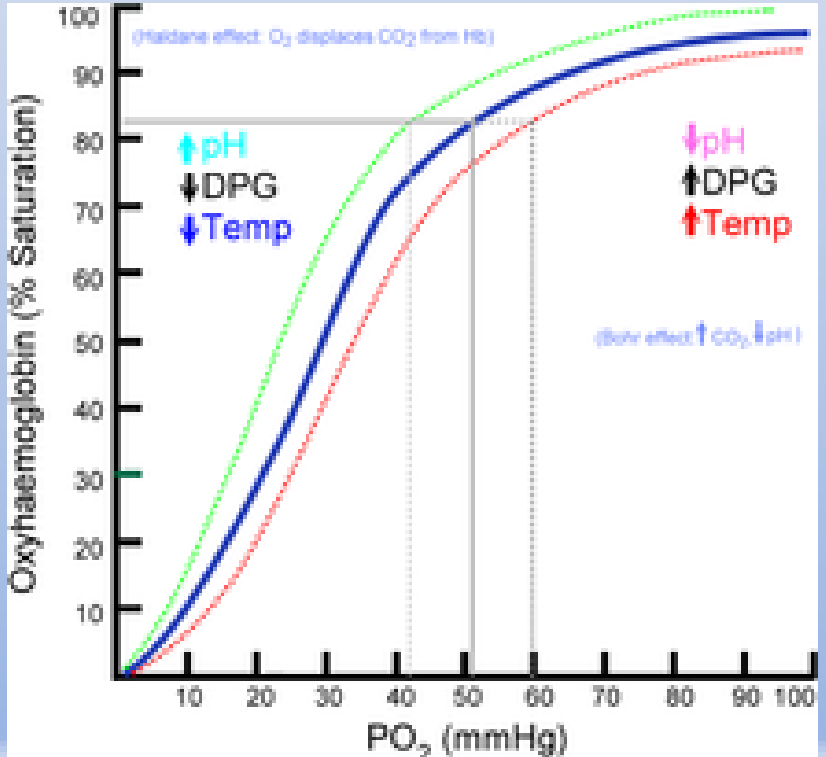
Dissolved Oxygen

Normally 2-5% of total oxygen in plasma

- 80-100mmHg

No maximum pressure

- Dalton's Law: Pt=P1+P2+P3+…Pn
- 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
$PaCO_2$	35-45mmHg (always higher than $EtCO_2$)
HCO_3^-	22-26 mEq/L
PaO_2	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

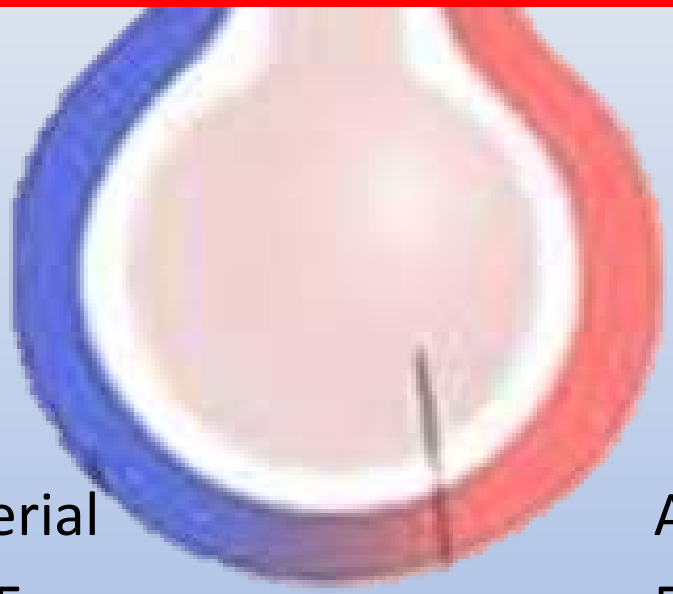
P_aO_2: 45

P_AO_2: 100

Alveolar-arterial

100 – 45 = 55

Wide A-a gradient



PCO_2: 80

P_aO_2: 45

P_AO_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

Any increase in metabolism

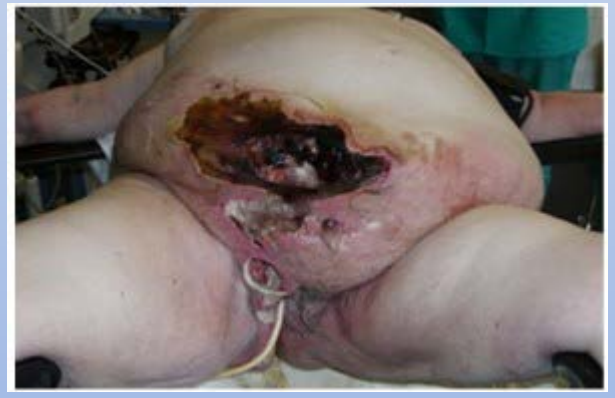
Serious illness

Seizures

Surgery

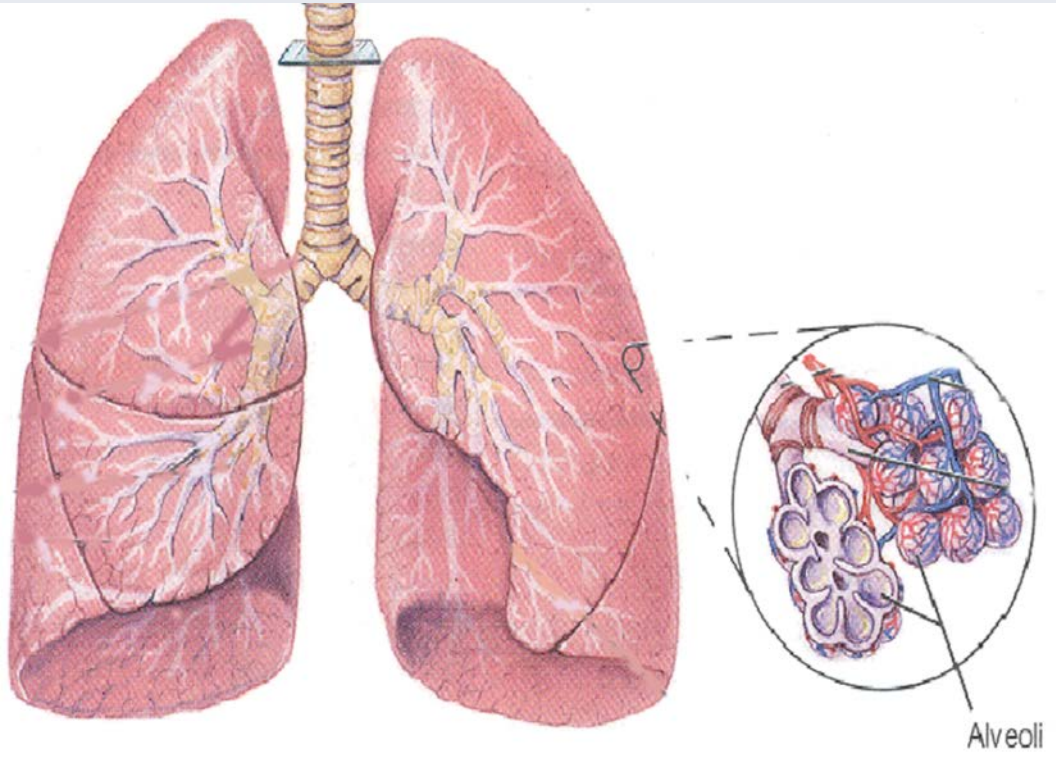
Injury

Pain



Sepsis

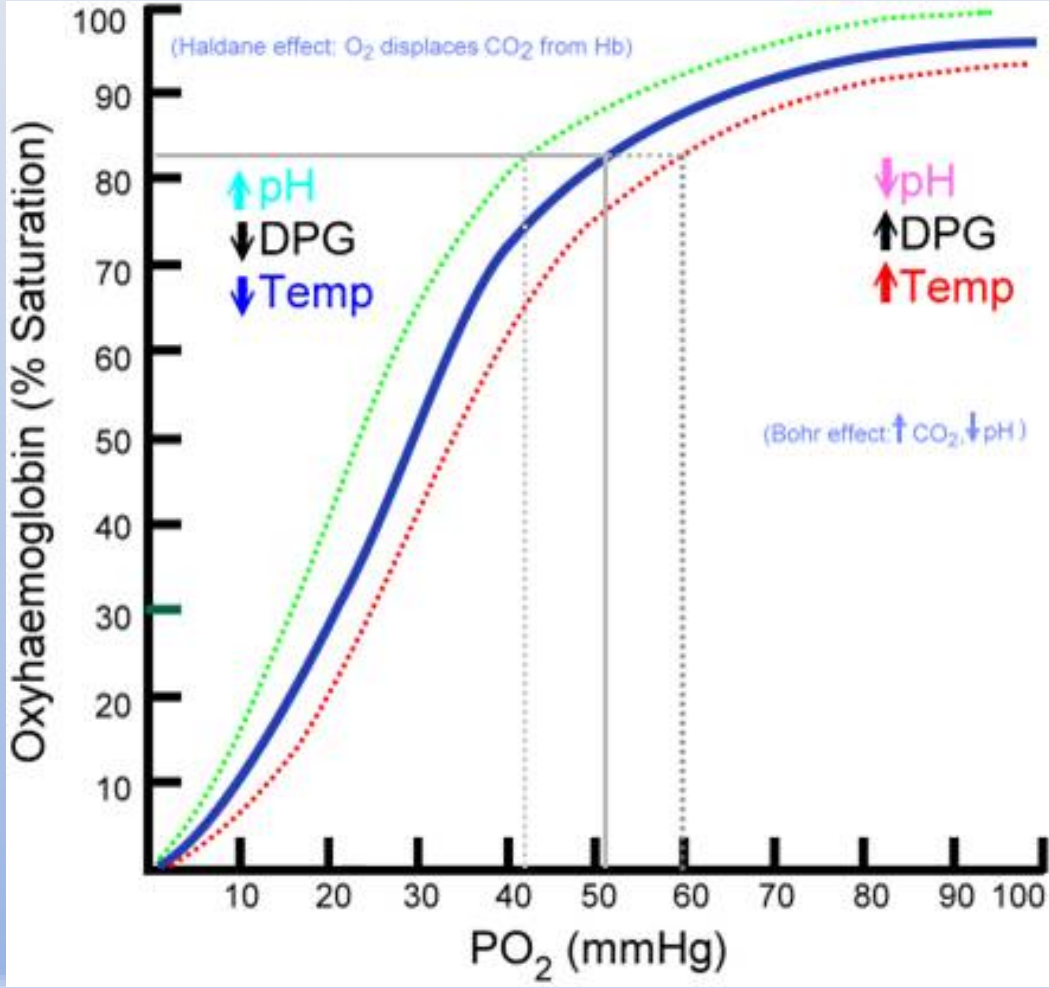
Oxygen Availability



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:

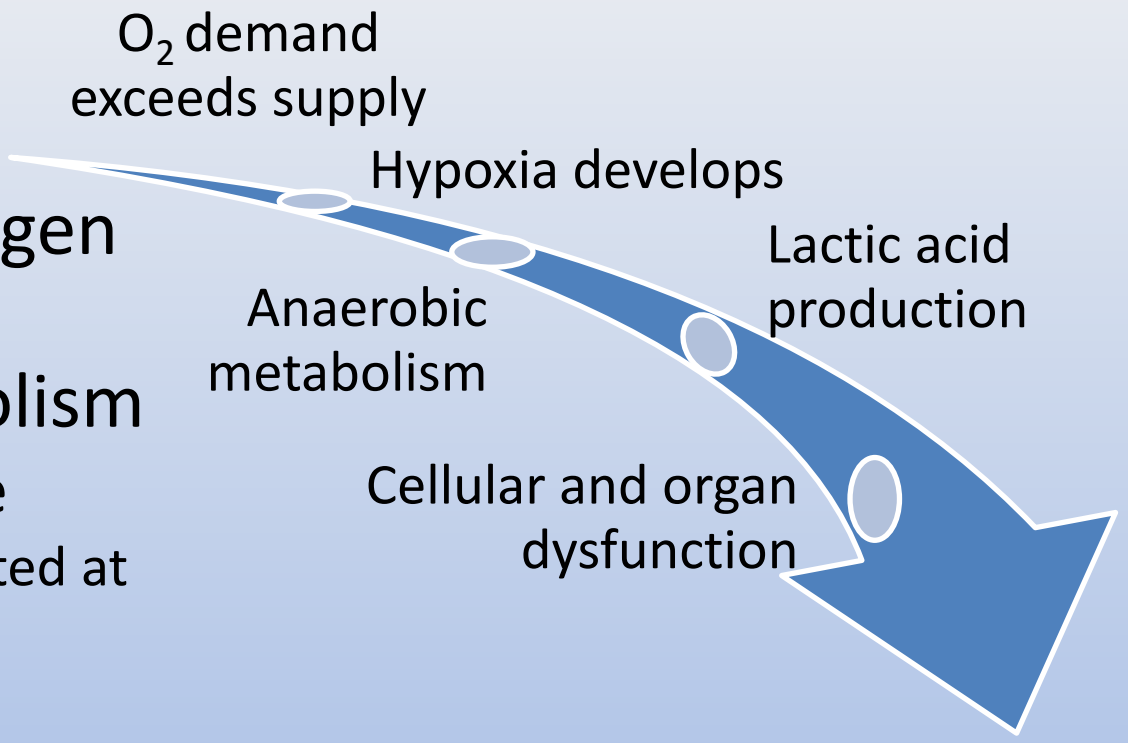
- Elevated lactate
- Ph <7.35

$$S_v = S_a \times 0.75$$

If < then oxygen debt present

Cellular Changes From Hypoxia

- Normal cellular metabolism dependent on oxygen supply
- Anaerobic metabolism
  - Lactic acid release
    - Considered elevated at >2.2mm/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 SpO_2
    - Defined as >94%
  - Use minimal amount of oxygen to maintain normal SpO_2

O'Connor, Robert E., et al, Acute Coronary Syndromes: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, *Circulation 2010*; 122; S787-817

HOLD ON…

Oxygen is good

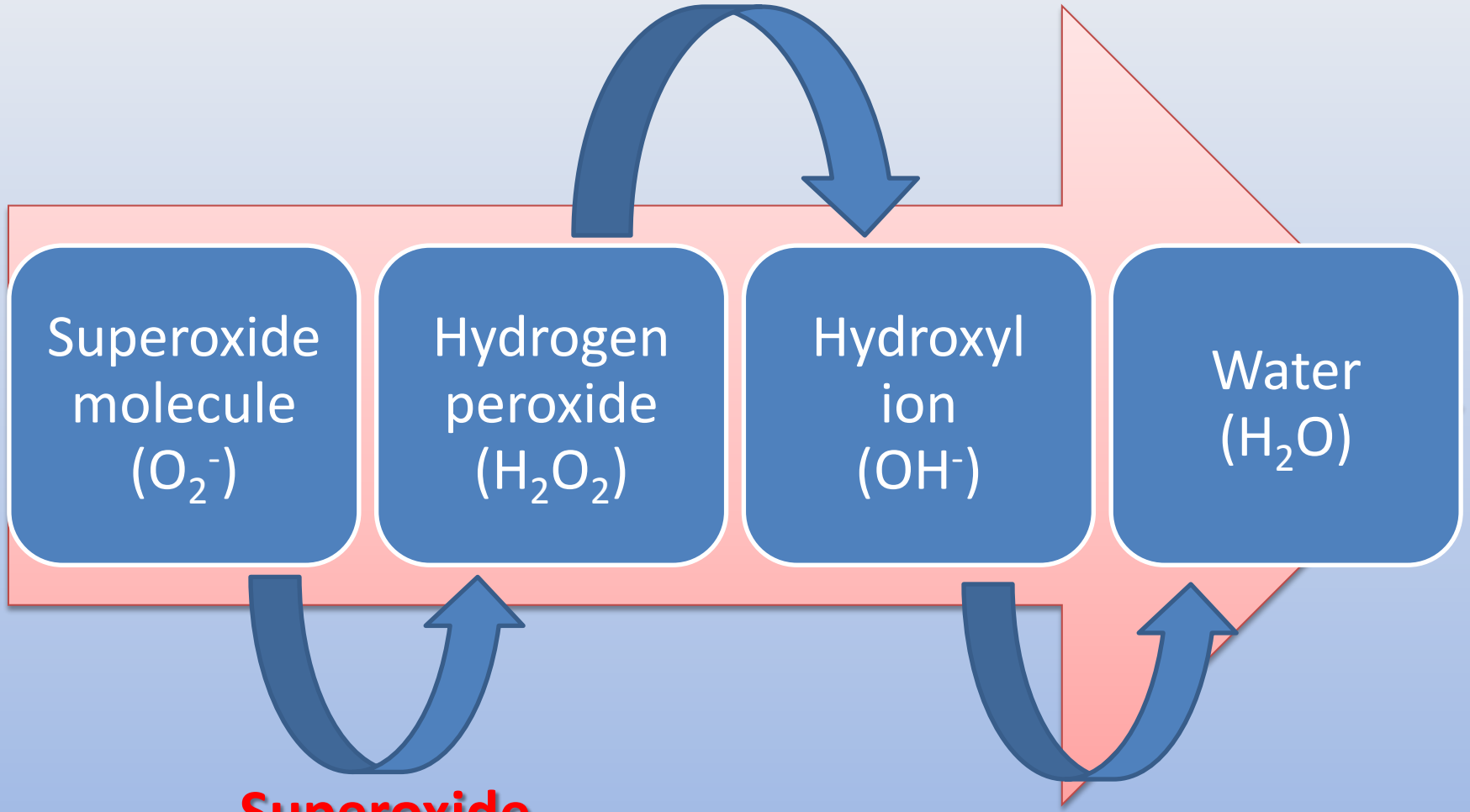
Needed to promote normal cell function

Prevents hypoxia

Prevents anaerobic metabolism

Provided intact circulation system free of occlusions

Cellular Metabolism in Mitochondria



The O_2^- Molecule

Damages cell membranes

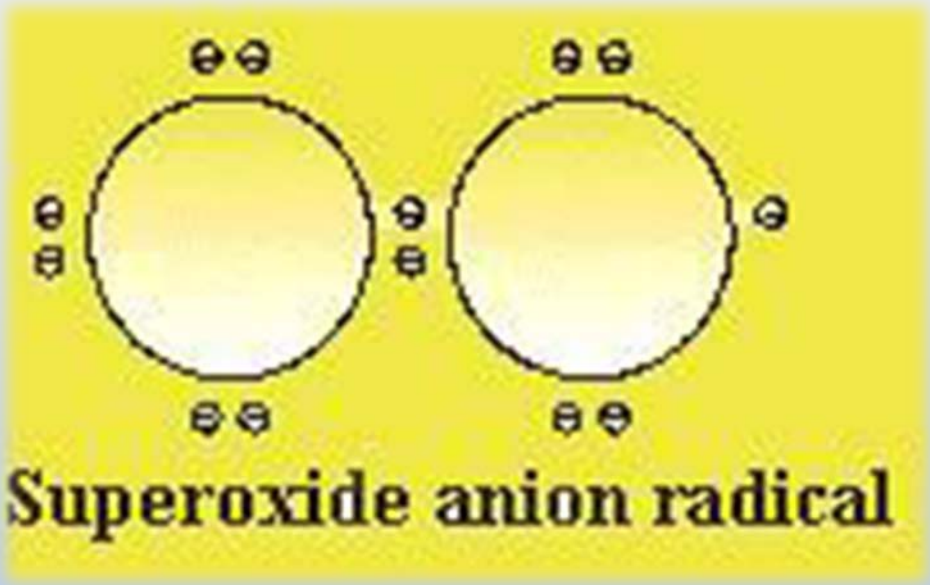
Considered "toxic"

Normally destroyed by enzymes within cell

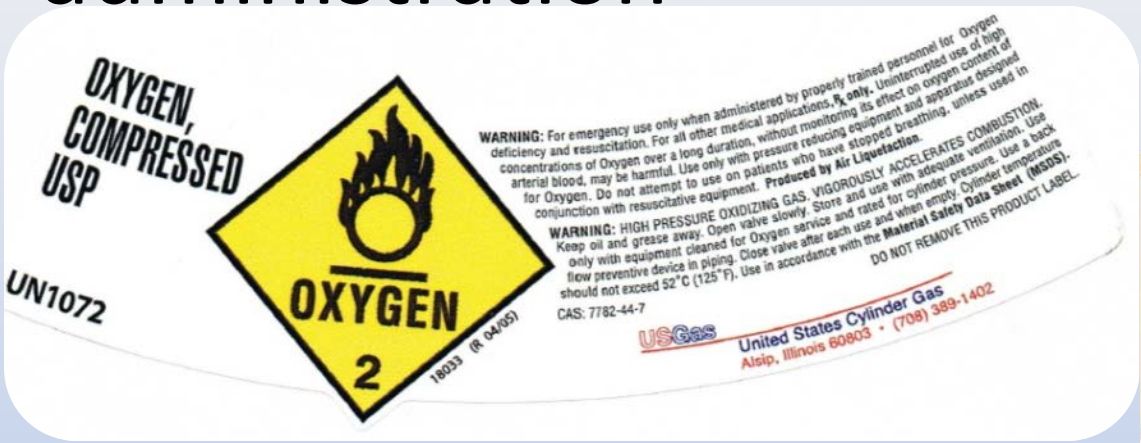
Superoxide dismutase

Enzymes produced at fixed rate

Enzyme production rate does not increase with metabolism

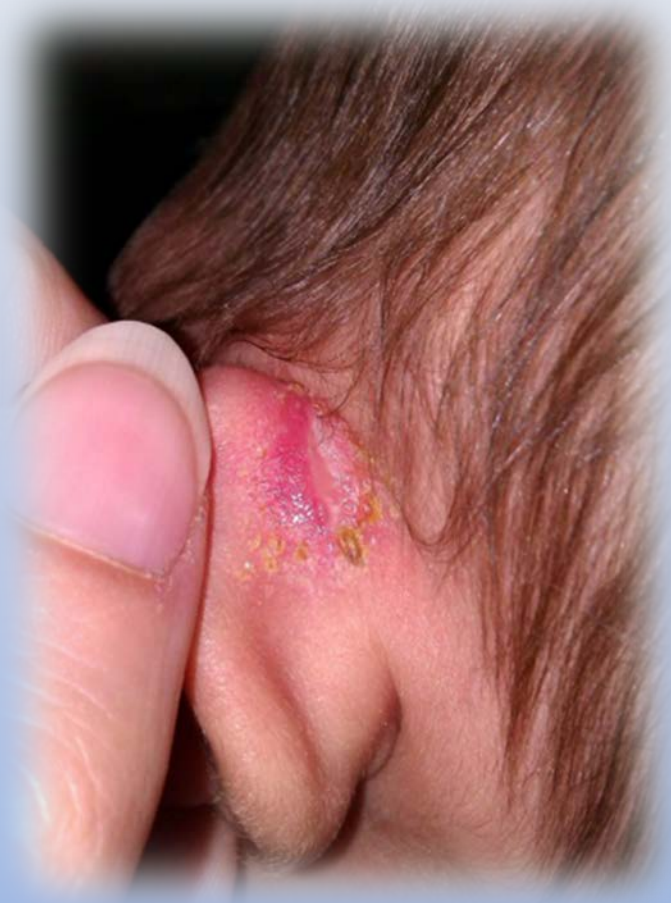


Objective 4: Complications of oxygen administration



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

Higher oxygen delivery

Increases pO_2

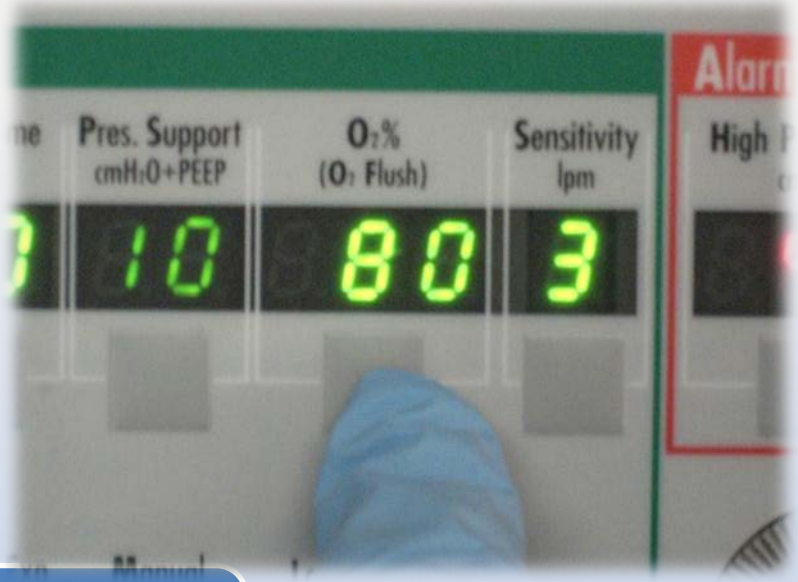
Forces more oxygen into cells

Increases metabolism

Increases O_2^- production

Fixed rate of O_2^- elimination

Build up of O_2^- molecules



The Good News?

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

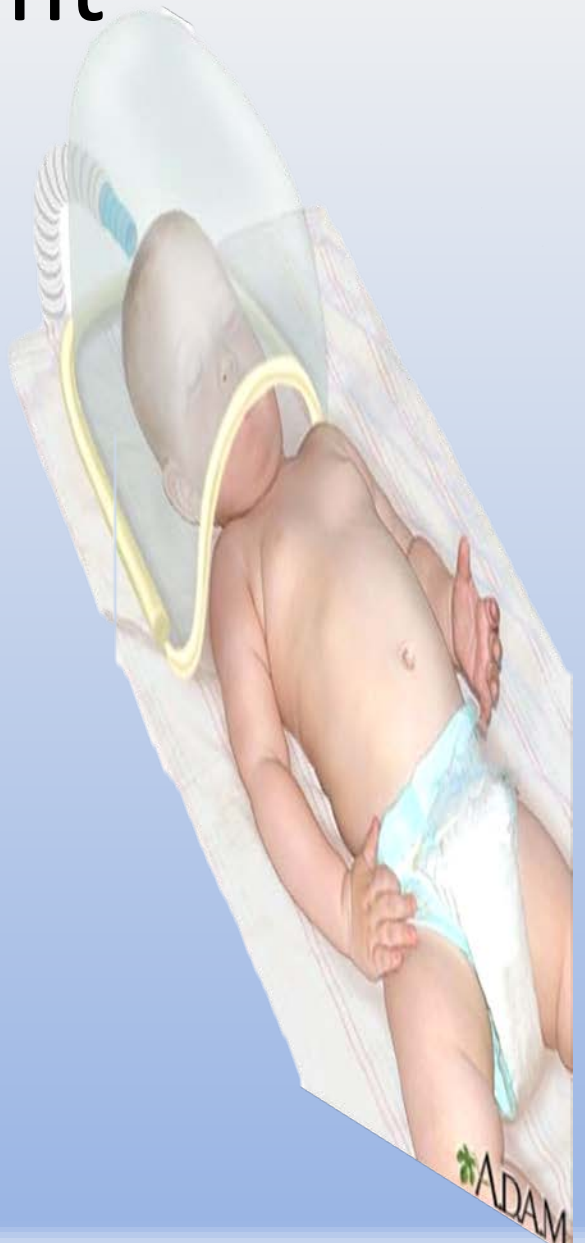
Oxygen Rich Environment

Healthy lungs FiO_2 >0.6

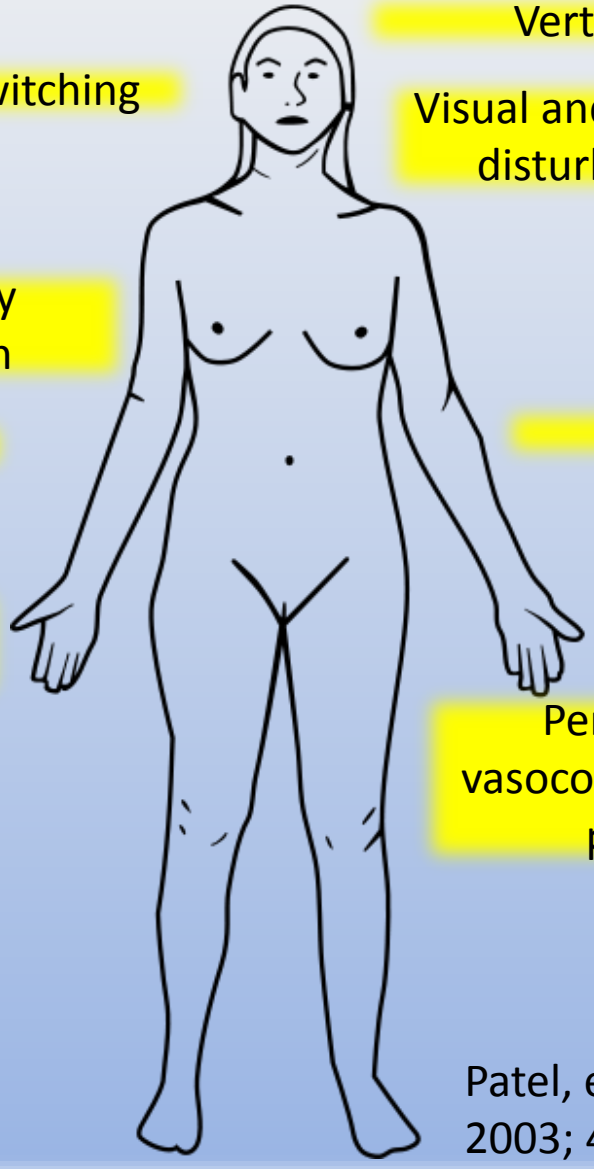
- Alveolar oxygen tensions of 350mmHg

Injured or diseased lungs FiO_2 >0.5

- Alveolar oxygen tensions of 250mmHg



Oxygen Toxicity CNS Symptoms



Vertigo

Perioral twitching

Visual and auditory disturbances

Respiratory depression

Seizures

Nausea

Hand muscle twitching

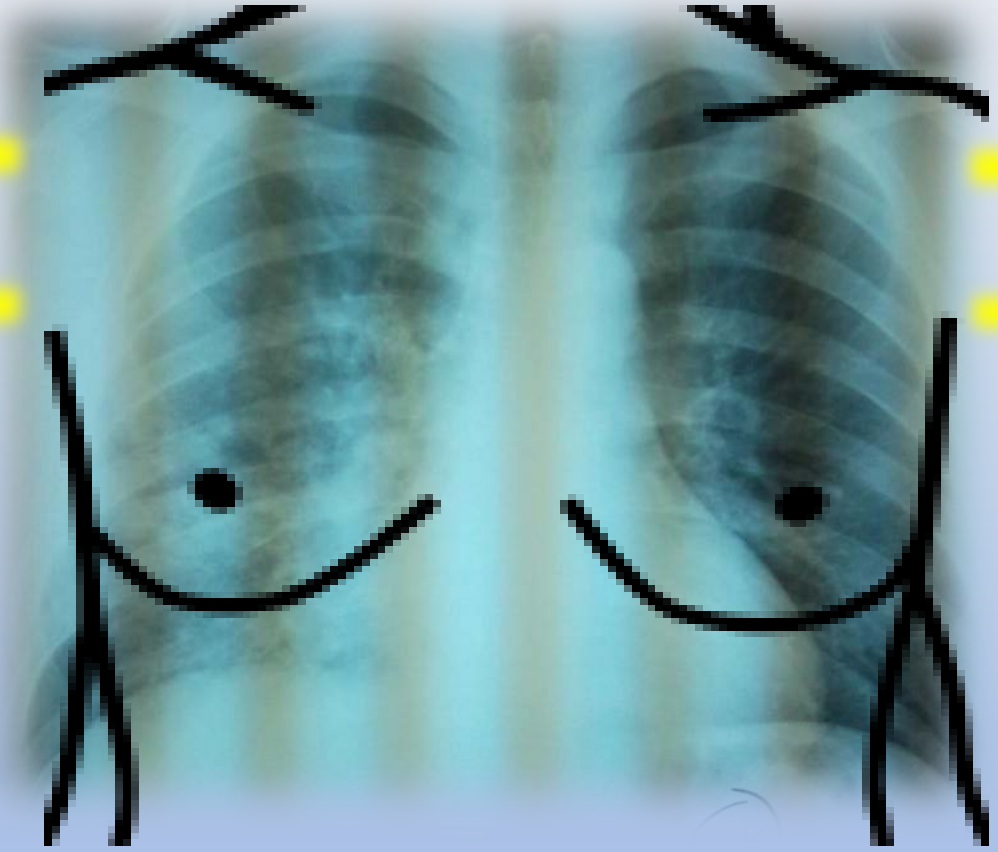
Peripheral vasoconstriction & pallor

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

Oxygen Toxicity Pulmonary Symptoms

Capillary leakage

Decreased V_T



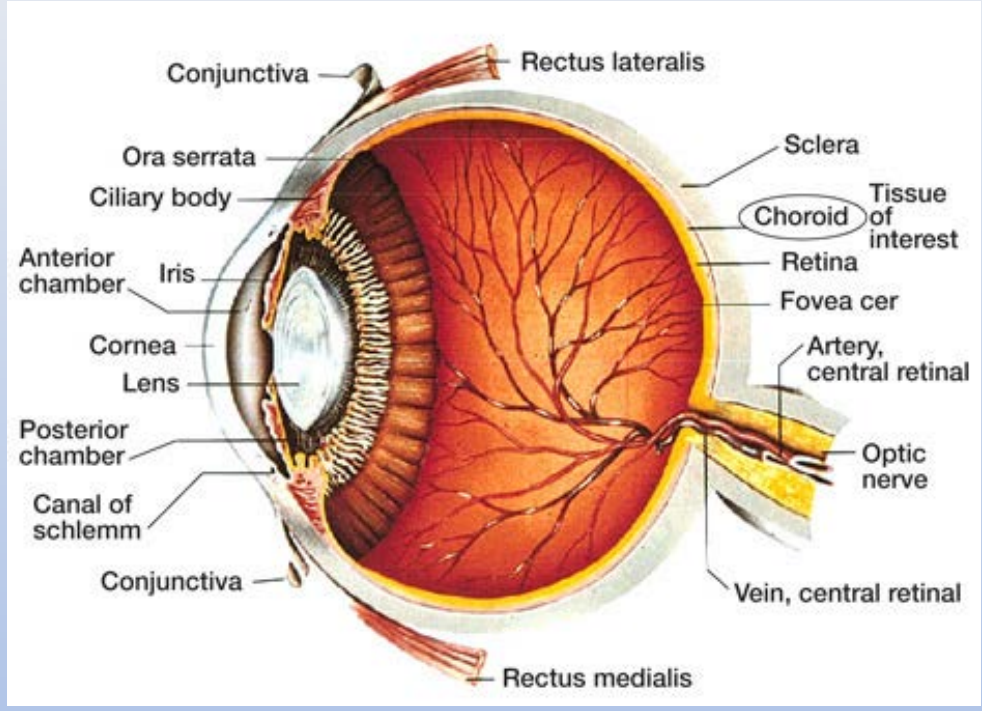
ALI and ARDS

Pulmonary edema

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

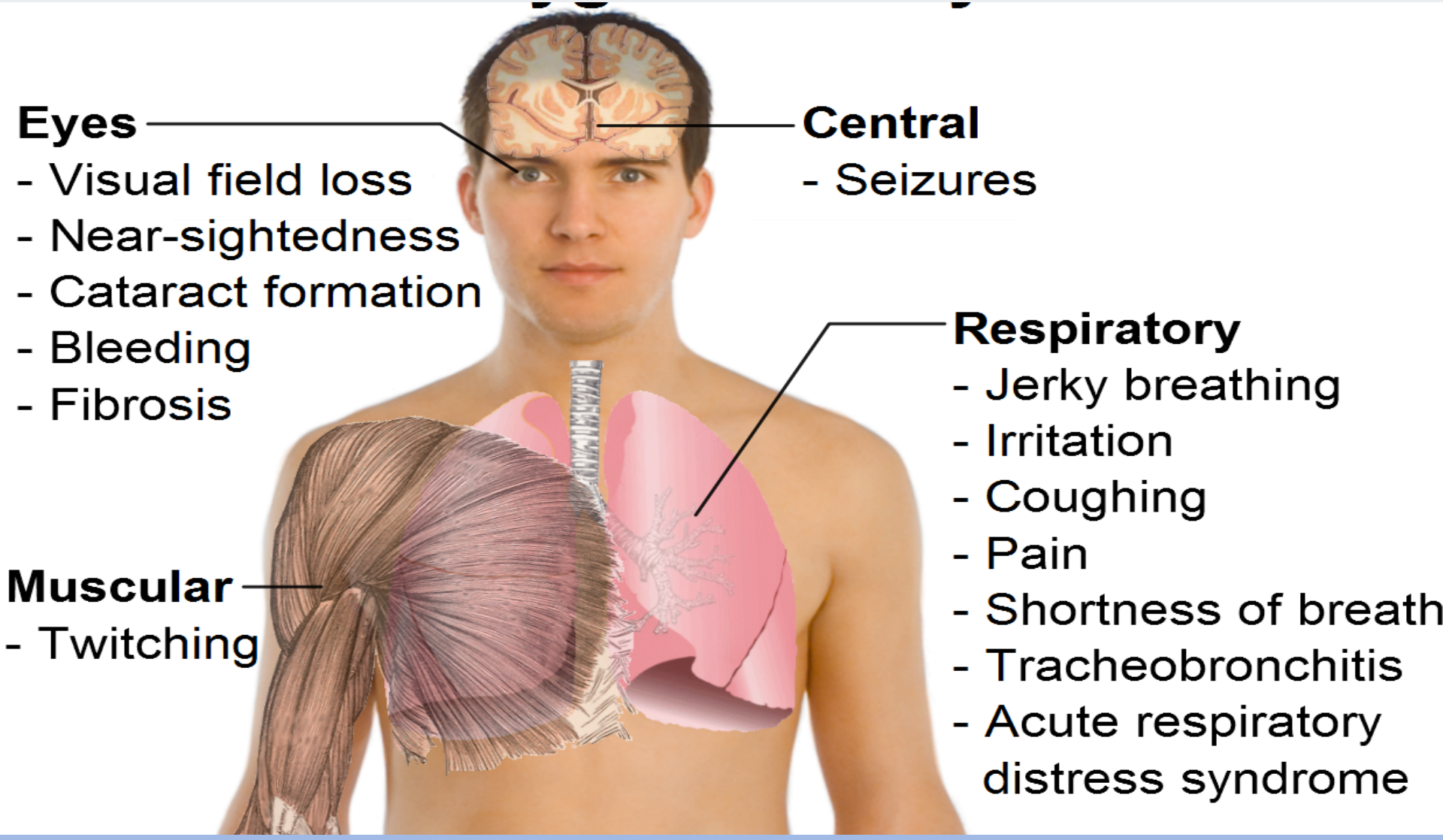
Oxygen Toxicity Symptoms

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



Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

Summary of Symptoms



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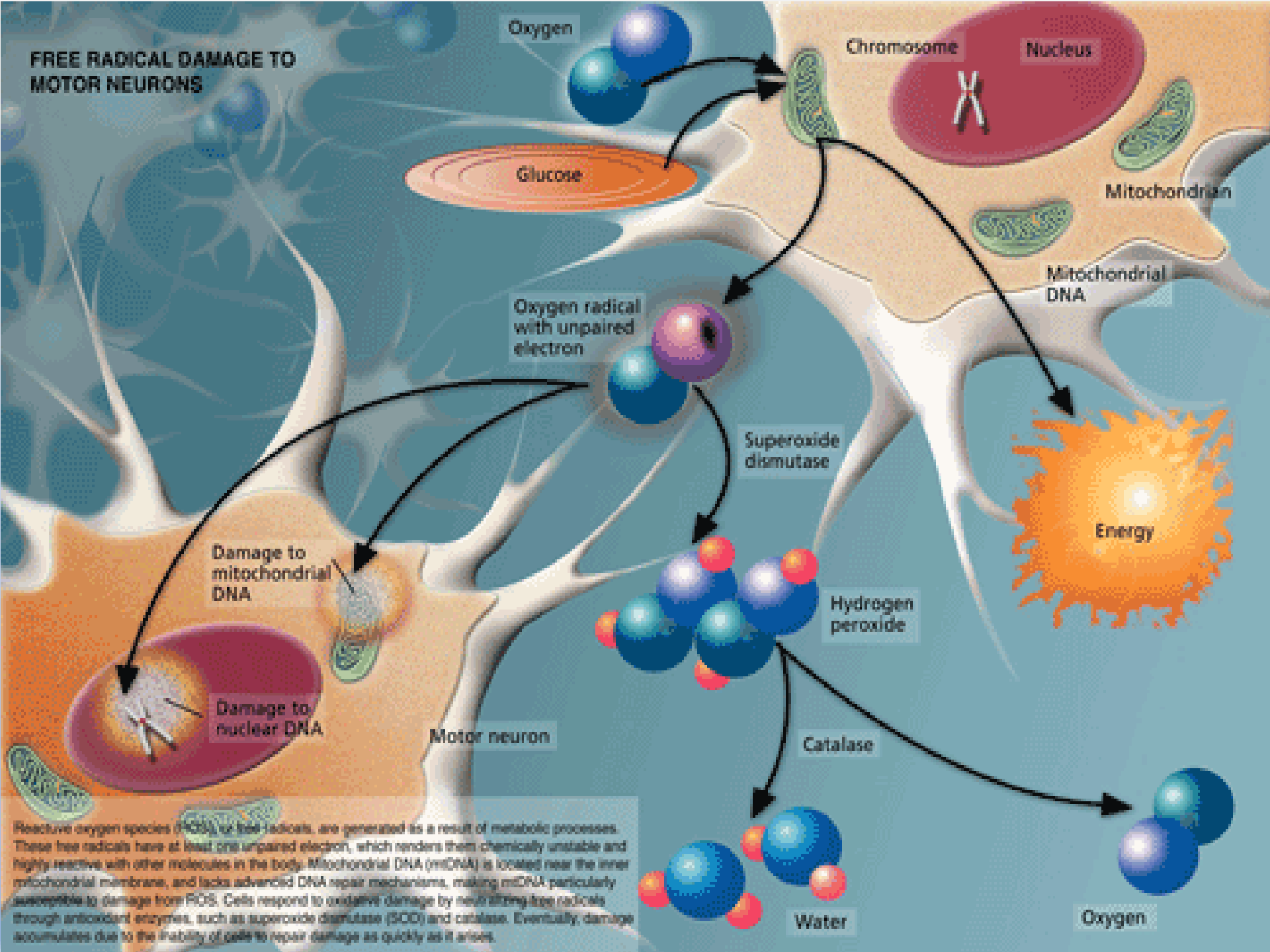

FREE RADICAL DAMAGE TO MOTOR NEURONS
Oxygen
Chromosome
Nucleus
Glucose
Mitochondrion
Mitochondrial DNA
Oxygen radical with unpaired electron
Superoxide dismutase
Energy
Damage to mitochondrial DNA
Hydrogen peroxide
Damage to nuclear DNA
Motor neuron
Catalase
Water
Oxygen
Reactive oxygen species (ROS), 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

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

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 Resuscitation; American Fam Physician 2011 April 15

Neonatal Oxygen

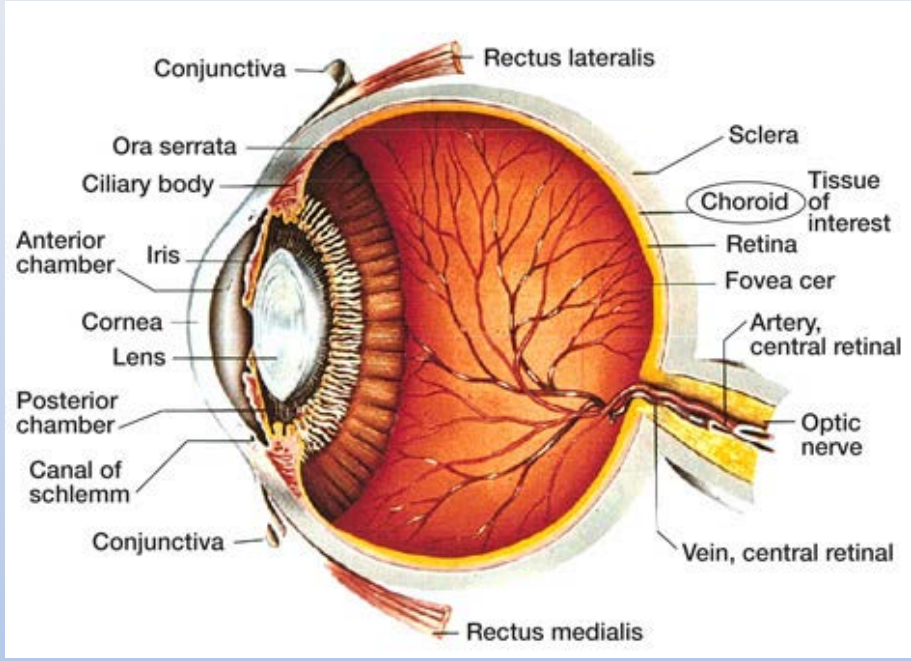
- Traditionally common, particularly during resuscitation
- Causes pO_2 to rapidly well exceed normal levels

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 Resuscitation; American Fam Physician 2011 April 15

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 PaO_2 >80 mmHg

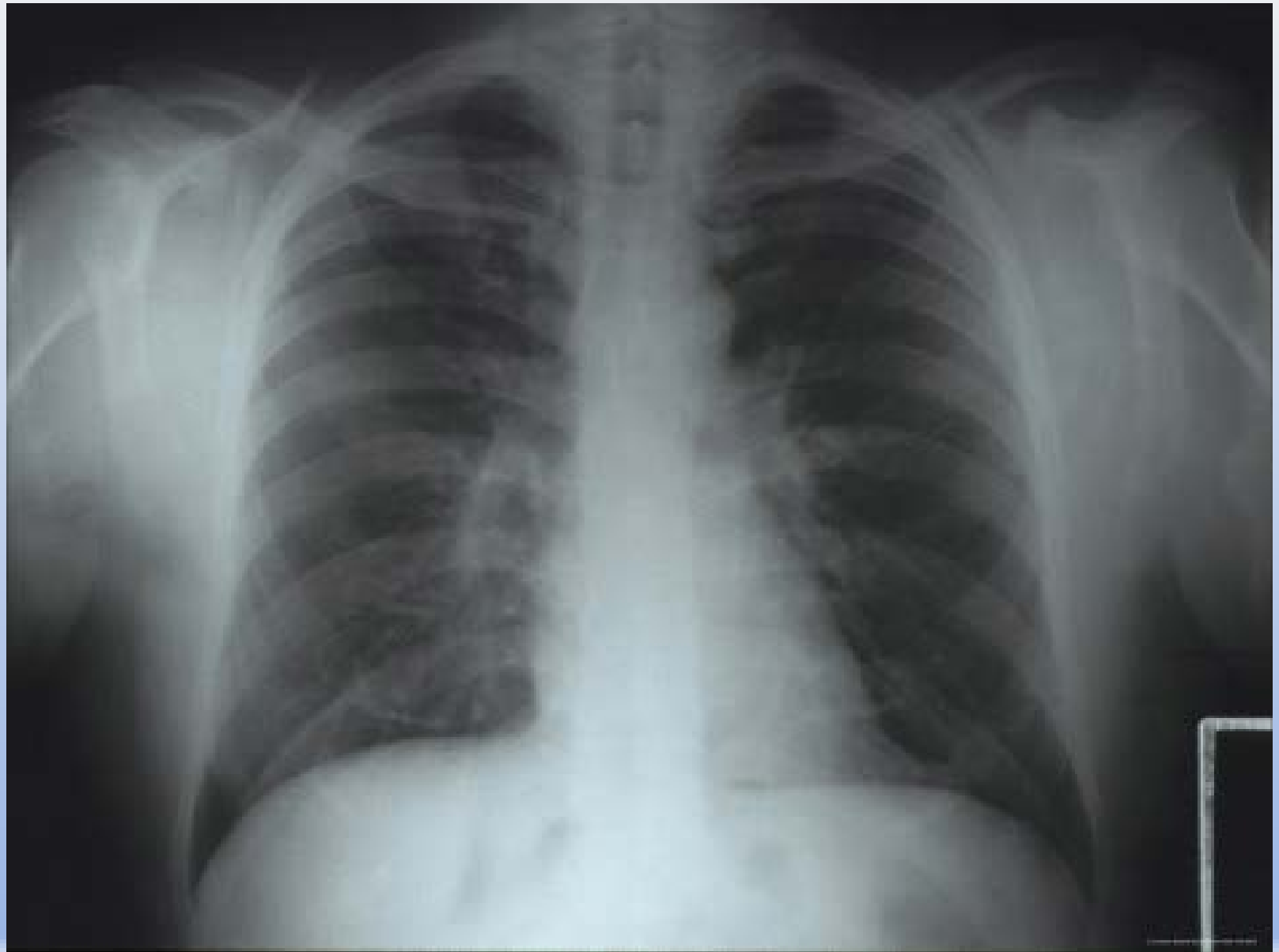


Neonatal Resuscitation Guidelines

(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 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

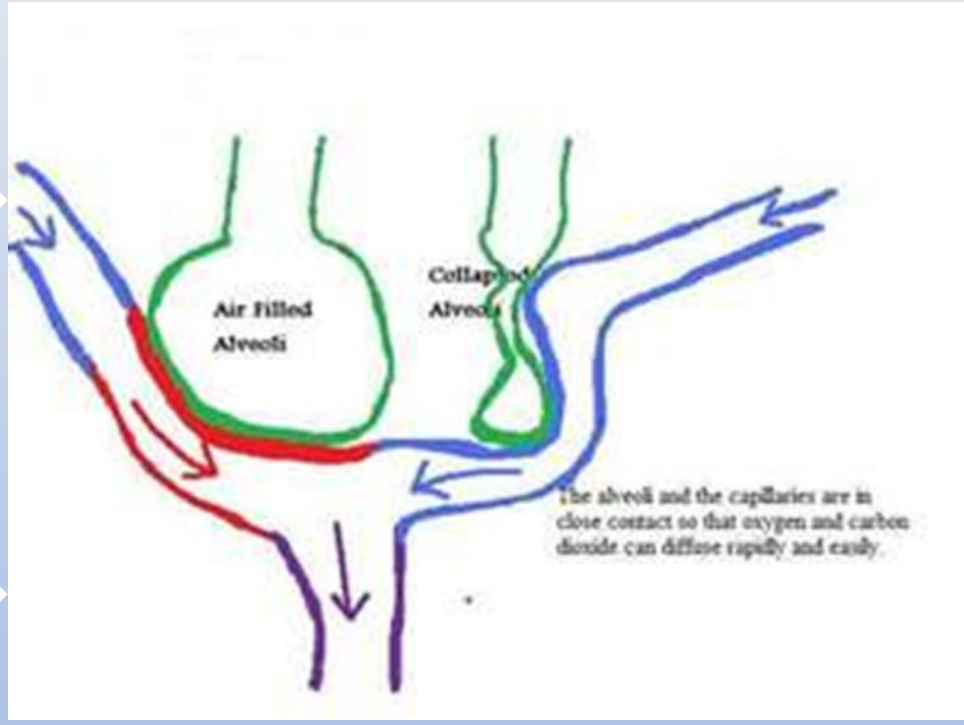
Absorbative Atlectasis



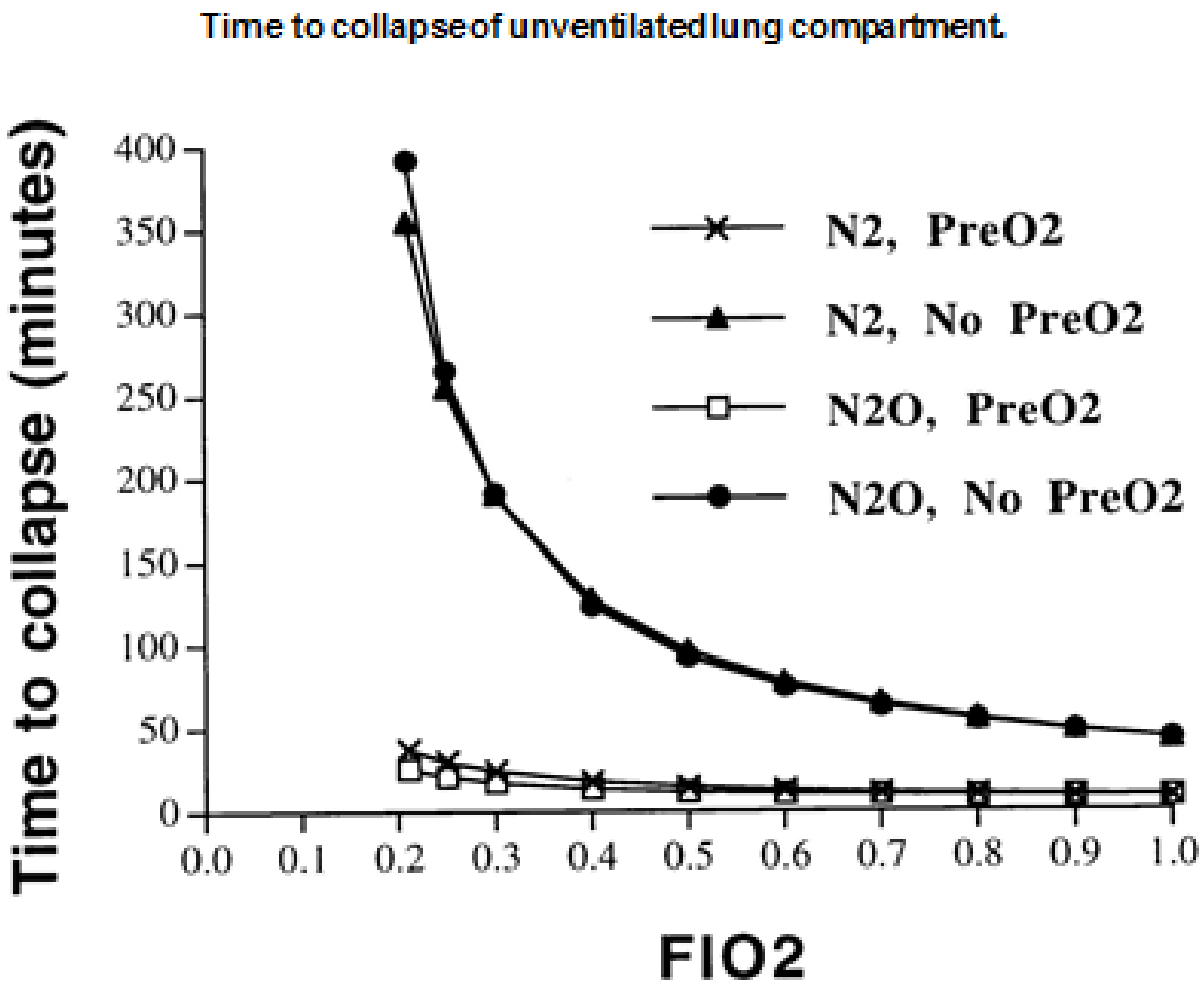
Nitrogen in the Alveoli

Recall that nitrogen is not well absorbed by body

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



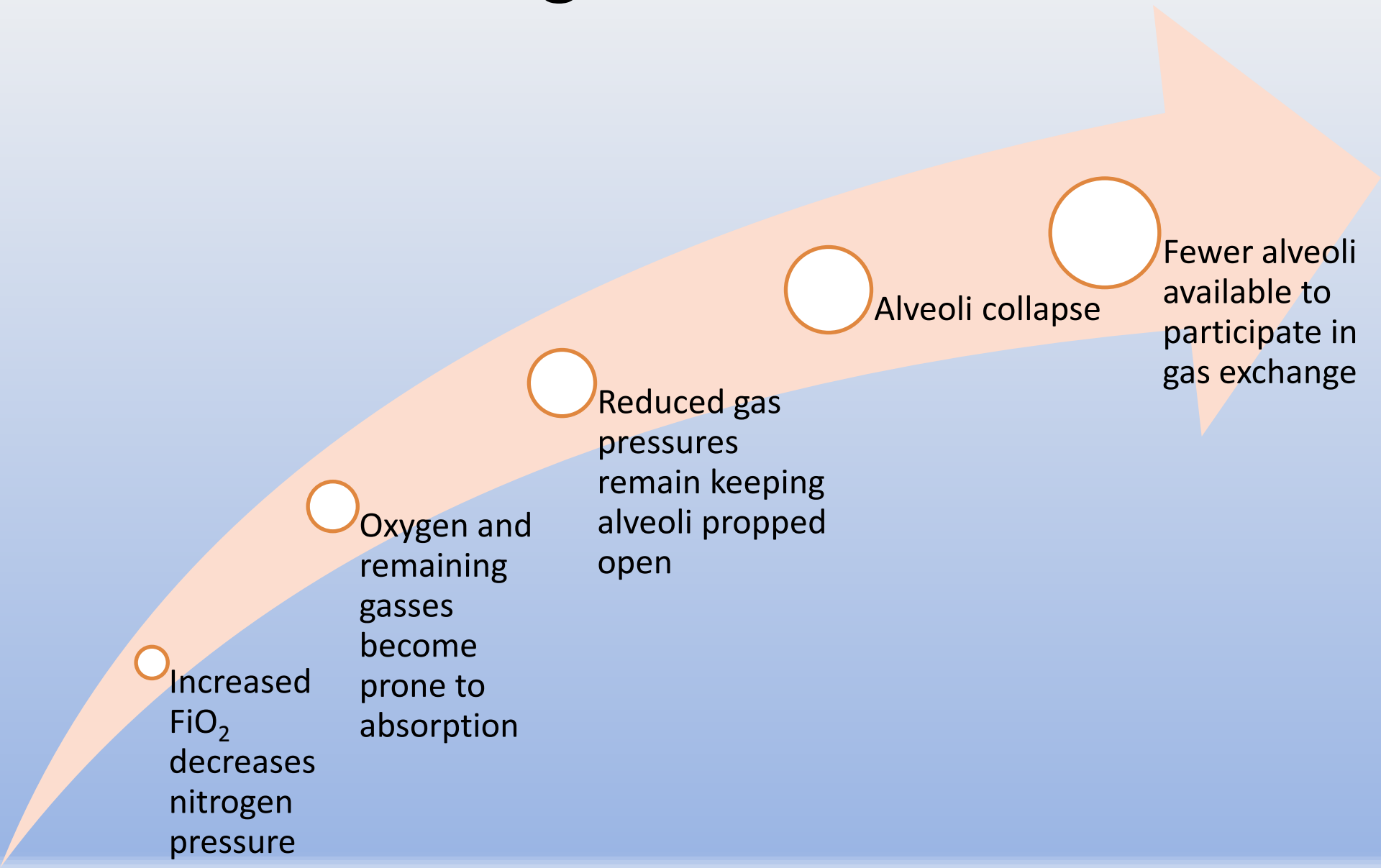
Increasing FiO_2



Joyce C J , Williams A B J Appl Physiol 1998;85:1116-1125

Journal of Applied Physiology

What's the Big Deal?



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% O_2 vs lower concentraitons

Case Study

- 49 year old female
- PMH
  - CHF
  - COPD with home O_2
- Presented to ED via EMS with respiratory distress and 2-word dyspnea

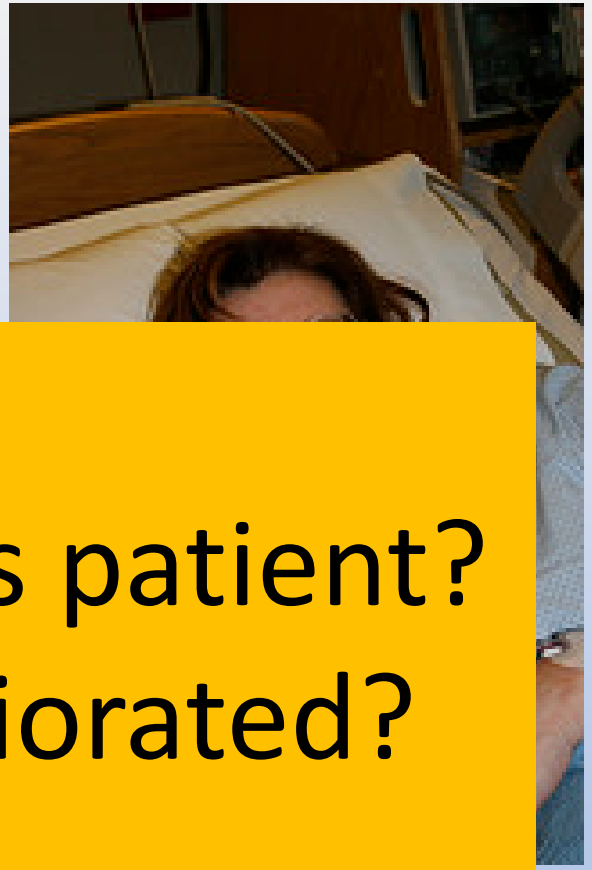
Initial ABG

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

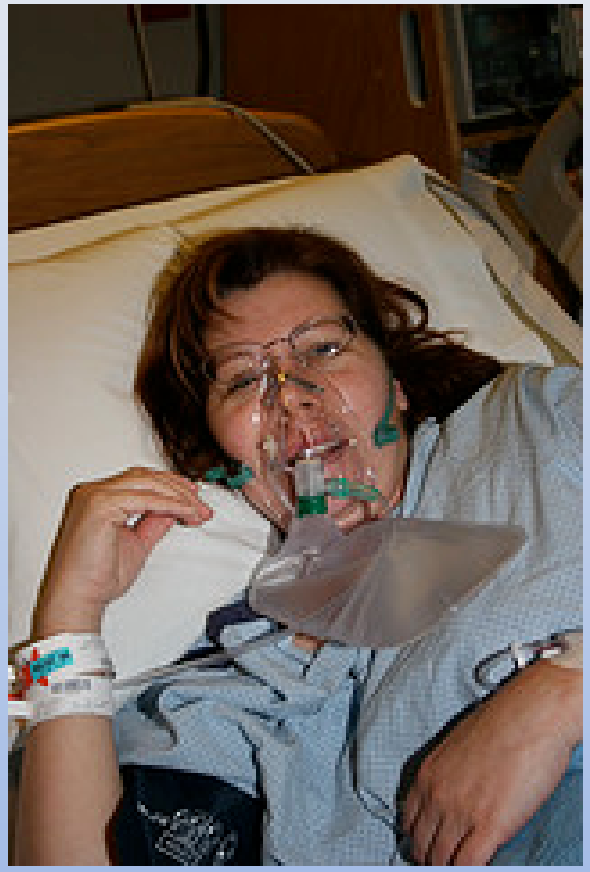
Carbon Dioxide Narcosis

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
    - Mysthenia gravis
    - Poliomyelitis
    - Head injury
    - Increased ICP

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

Chronic Respiratory Failure

Diagnosed by

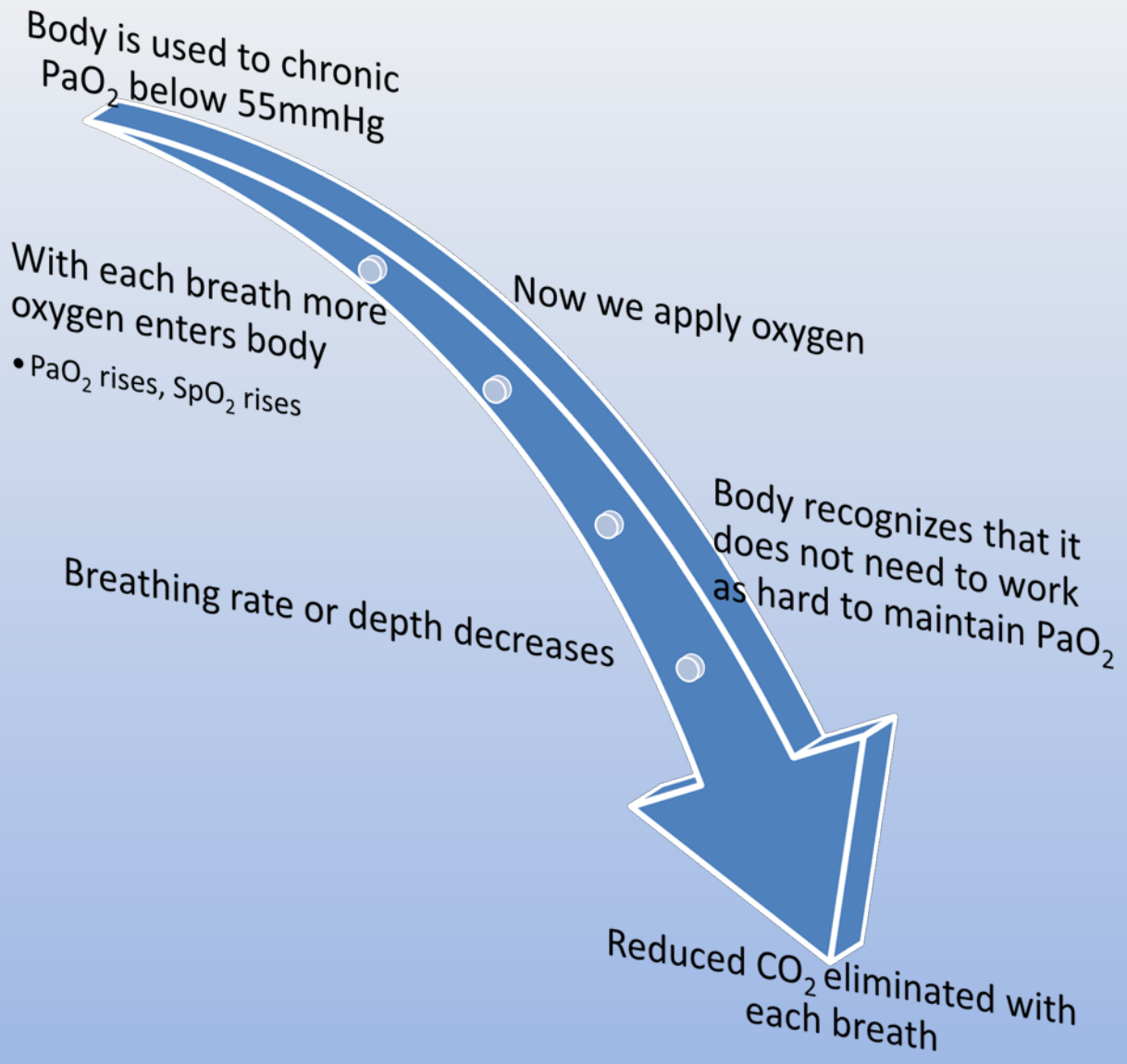
- $PaCO_2$ >50mmHg
- PaO_2 <55mmHg

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

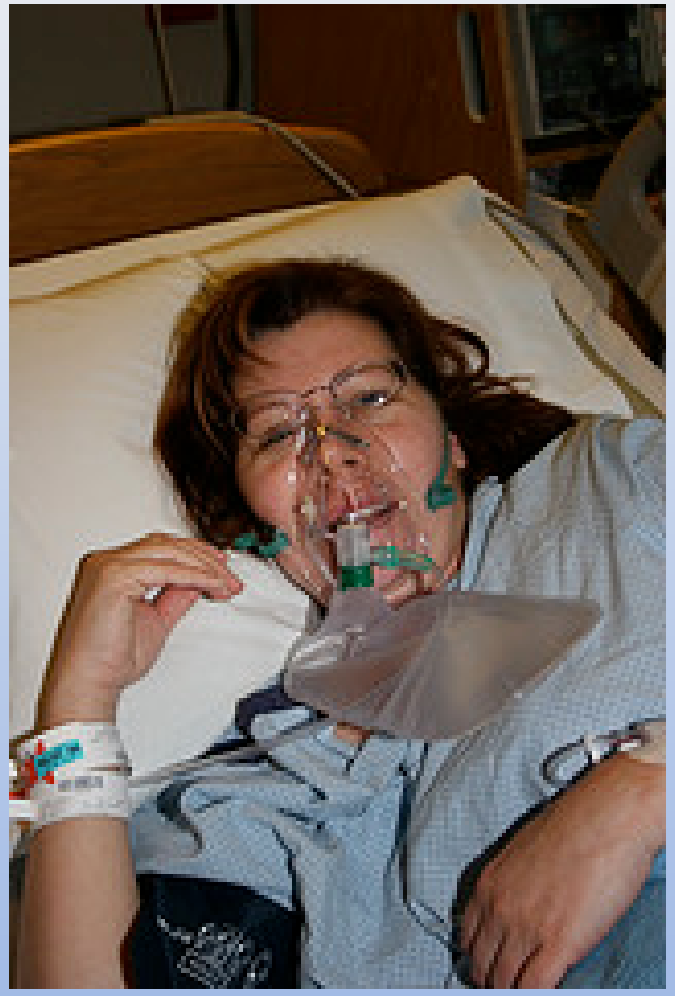
Common Causes of CRF

- Chronic Bronchitis
- Emphysema
- Bronchiectasis
- Cystic Fibrosis
- Pneumoconiosis
- Tuberculosis
- Fungal disease
- kyphoscoliosis

Physiology


Body is used to chronic PaO_2 below 55mmHg
With each breath more oxygen enters body
• PaO_2 rises, SpO_2 rises
Now we apply oxygen
Body recognizes that it does not need to work as hard to maintain PaO_2
Breathing rate or depth decreases
Reduced CO_2 eliminated with each breath

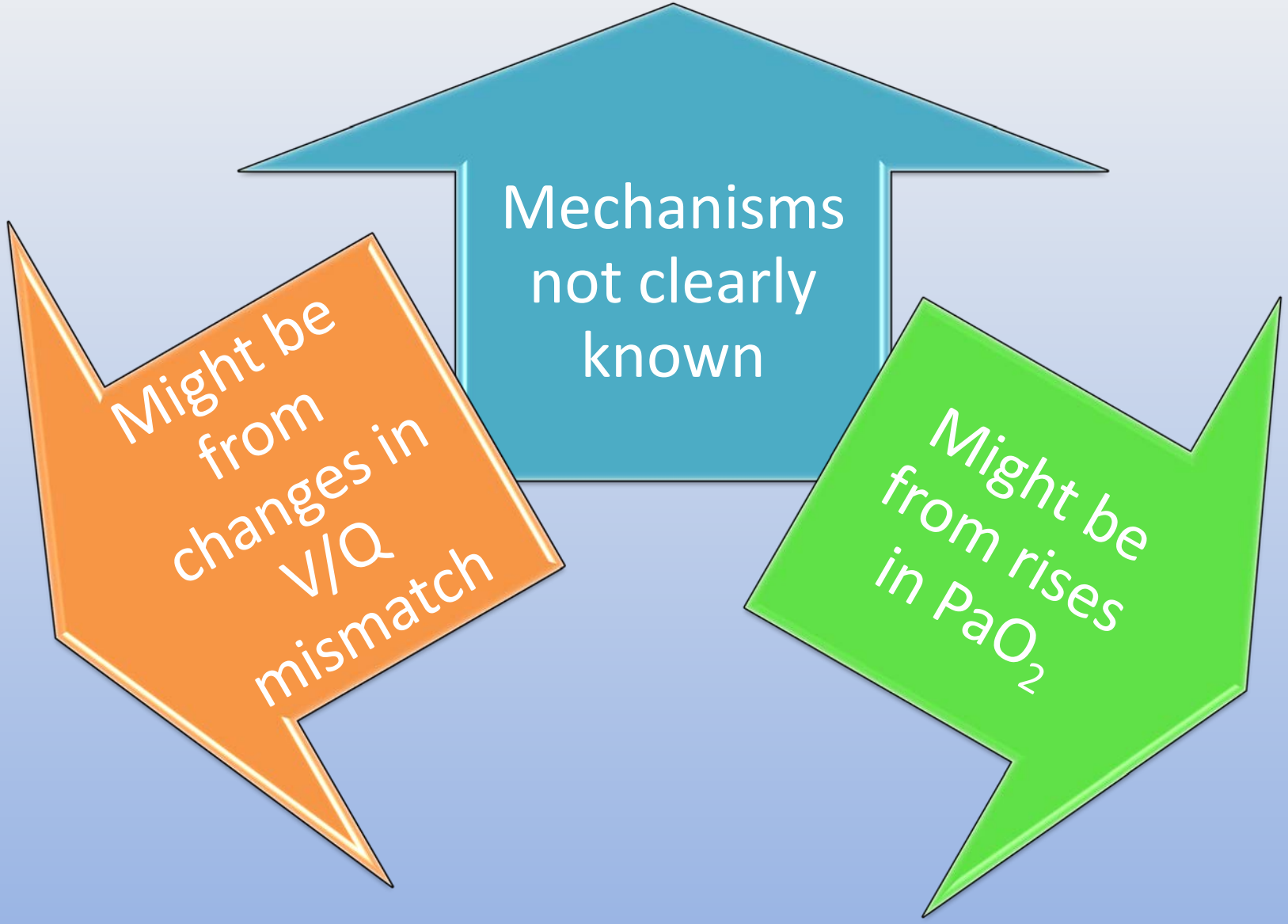
Symptoms



- Decreased respiratory rate
- Decreased Vt
- Measureable increases in CO_2 levels
  - Sidestream CO_2
  - $PaCO_2$
- Mental status changes
  - Lethargy
  - confusion
  - Headache
  - Somnolence
- Sweating
- Twitching
- papillioedema

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

Caused by the “Hypoxic Drive?”



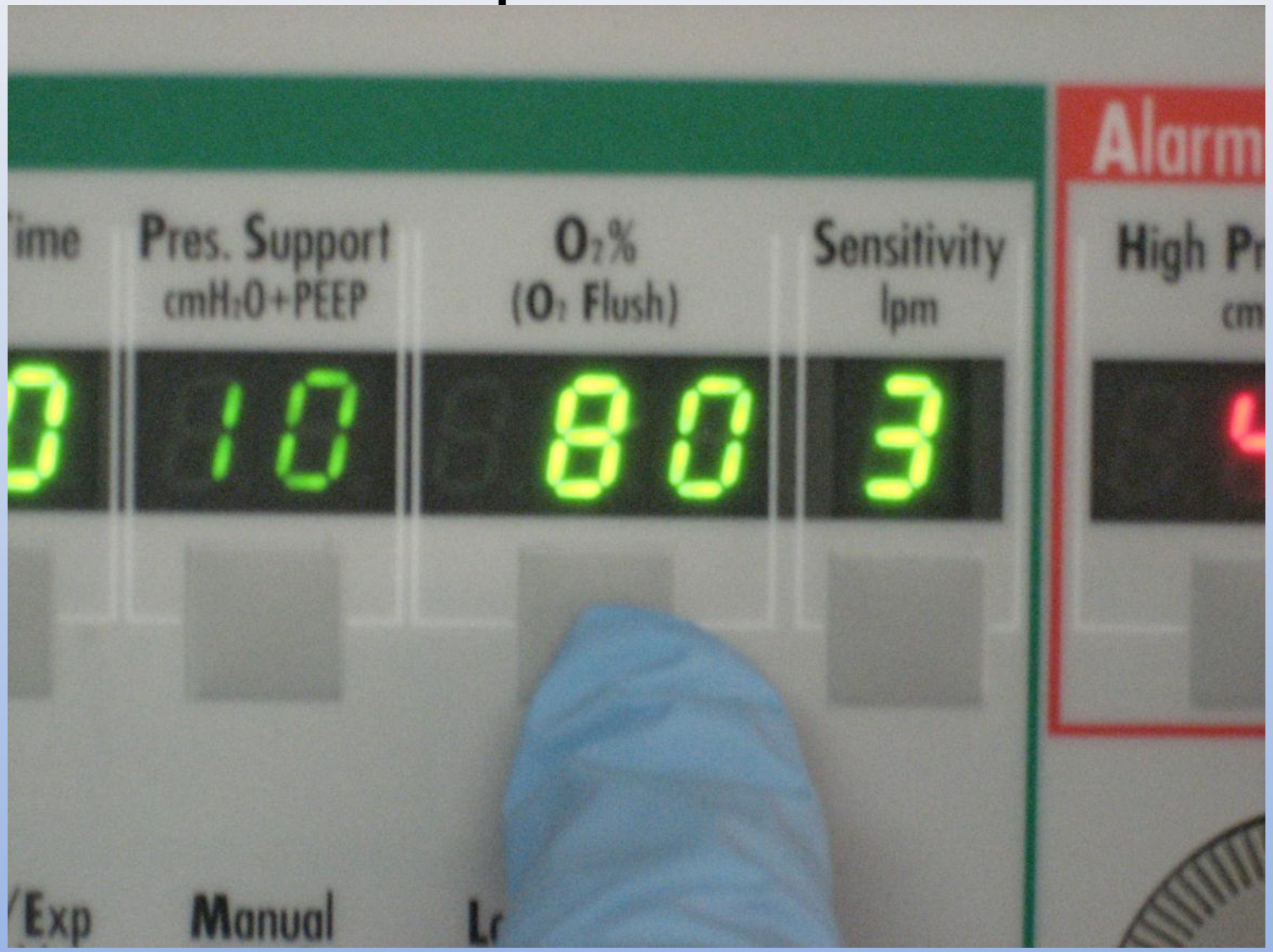
What do we KNOW?

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

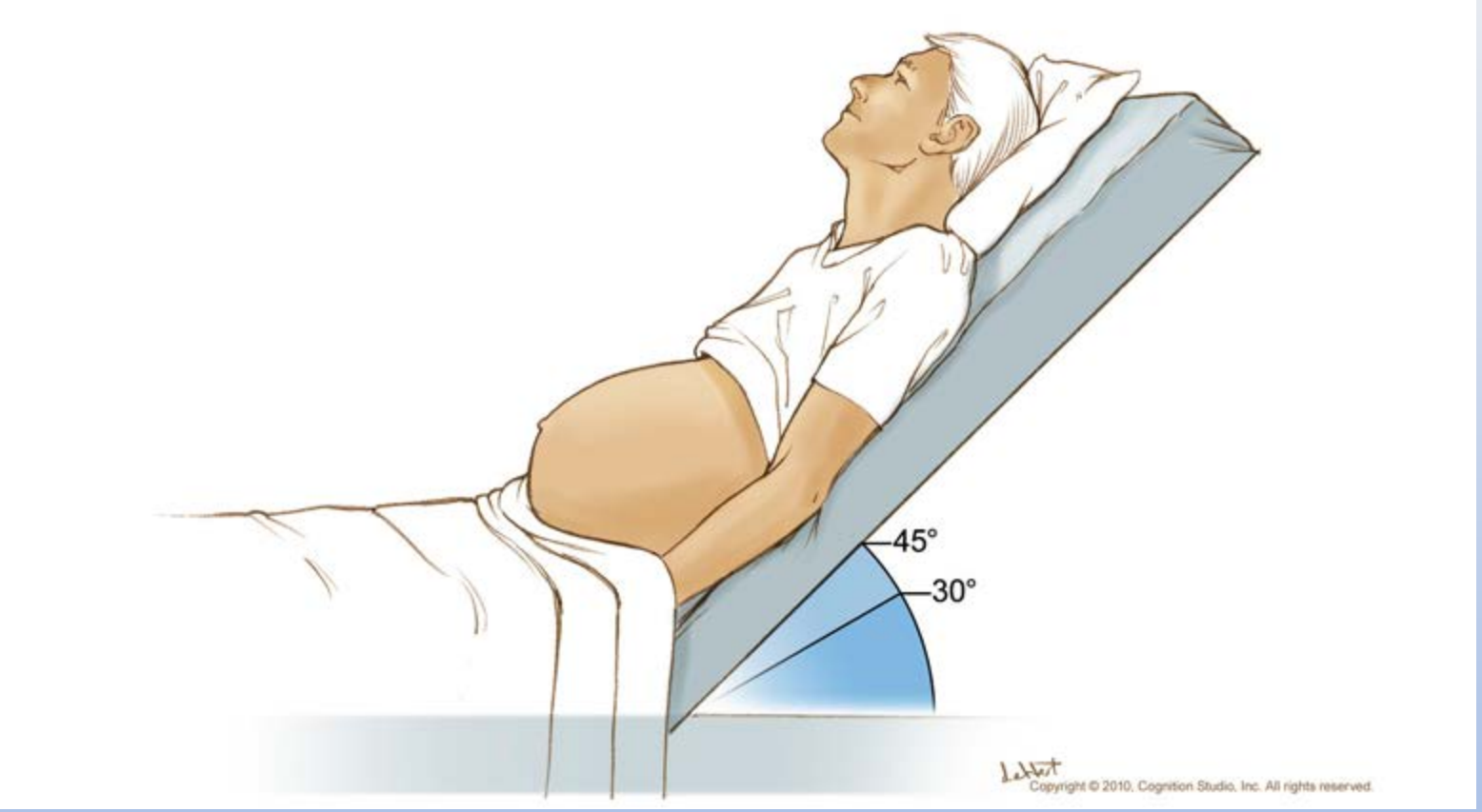
Humidify Oxygen

Any therapy over 4 LPM or FiO_2 > .36

Standard in hospitals, not routine in all CCT

- Cost?
- Duration of transport?
- Inconvient?

Optimize Patient Position



Maximizes lung expansion
Decreases work of breathing
Prevents aspiration

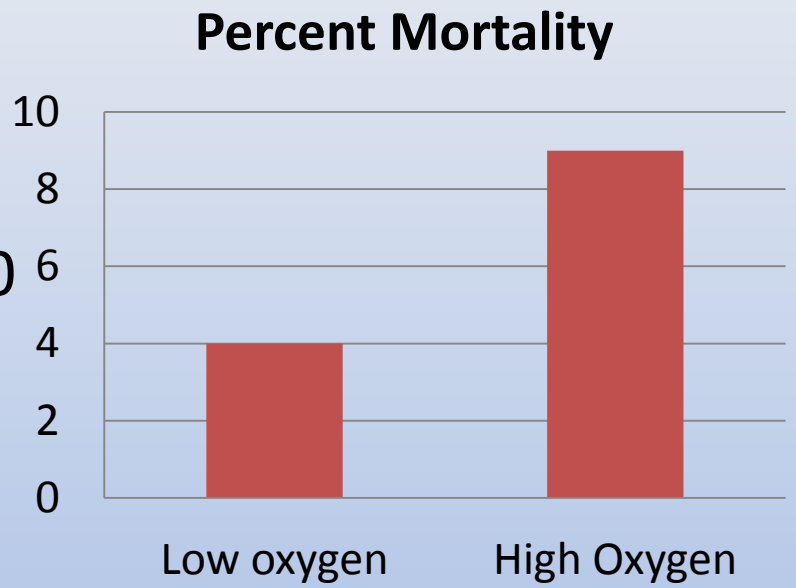
Oxygen Titration

- 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



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

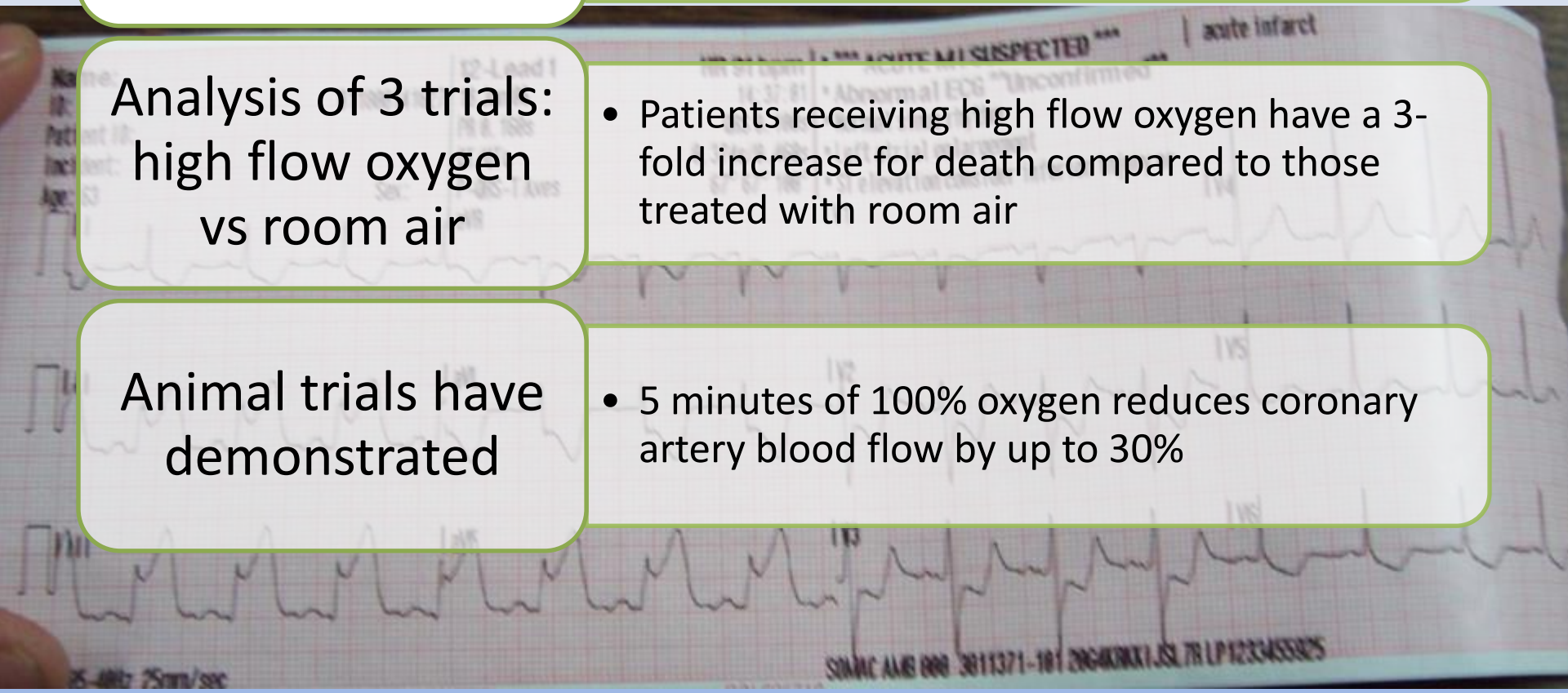
- 1425 Units = predicted loss of 10% vital capacity

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7

When to use 100%

What about STEMI?

2013 ACC/AHA Guidelines	• Limited data supports routine oxygen use • It is appropriate for patients with 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%



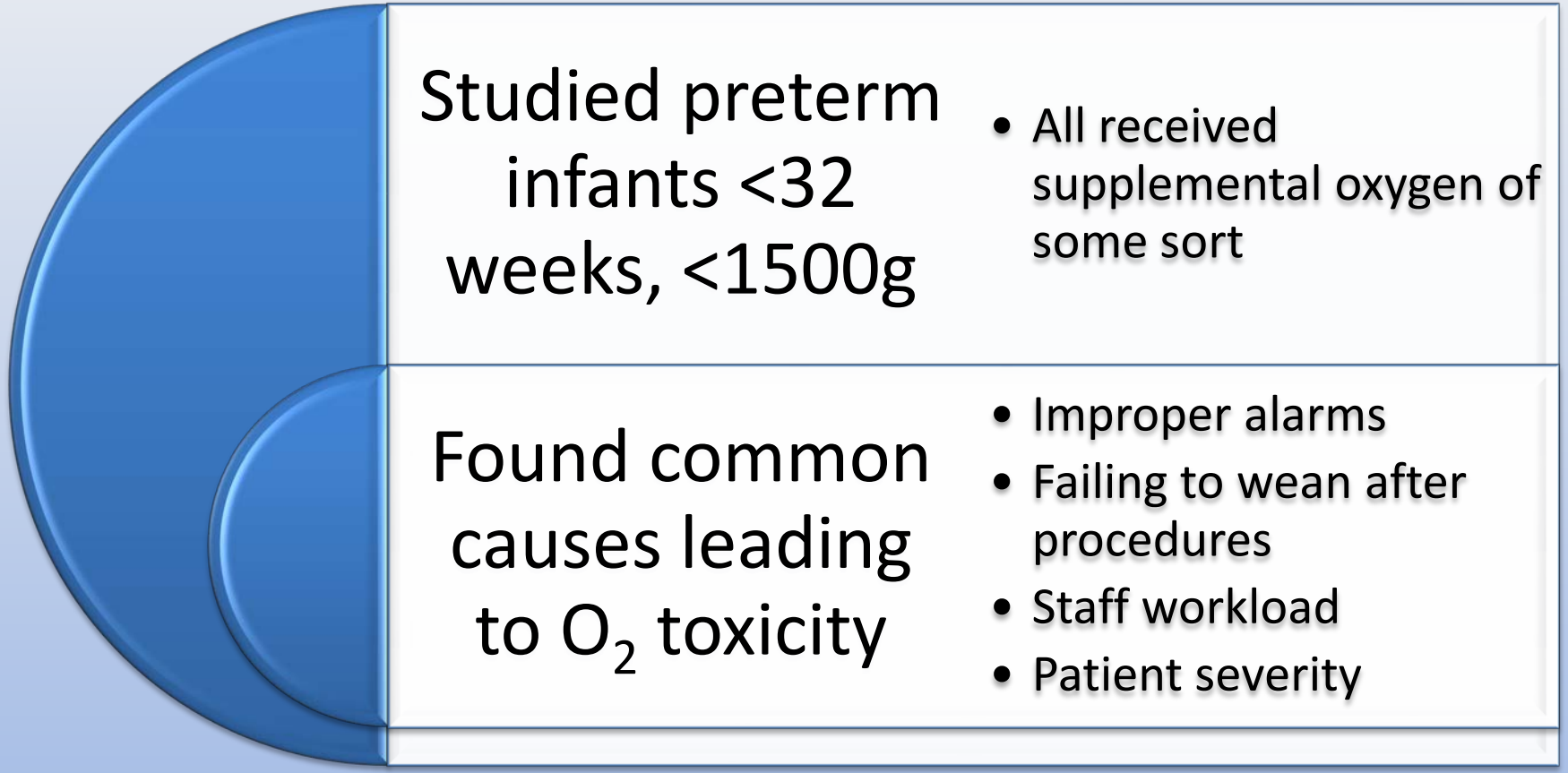
Journal Am Coll Cardiol. 2013; 61(4): e78-e140

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

Patel, et al, *Oxygen Toxicity*, JIACM 2003; 4(3): 234-7ox

SpO_2 in Preterm Infants



Lau, et al, *Maintaining Optimal Oxygen Saturations in Premature Infants*, The Permanente Journal, Winter 2011, Volume 15 (1)

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

Lau, et al, *Maintaining Optimal Oxygen Saturations in Premature Infants*, The Permanente Journal, Winter 2011, Volume 15 (1)

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 SpO_2 and respiratory rate

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

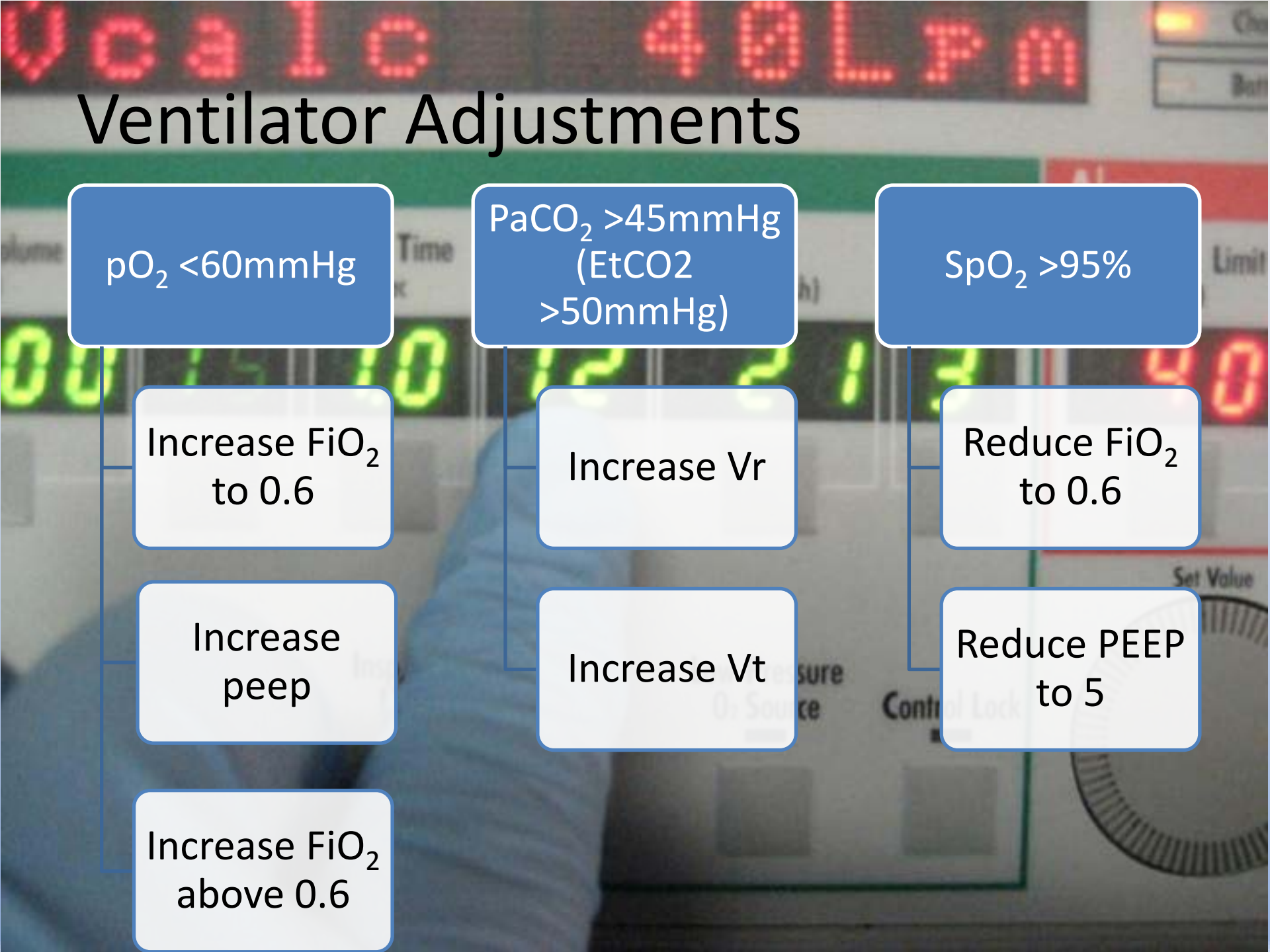
Point of Care Testing

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 <60mmHg
  - Increase FiO_2 to 0.6
  - Increase peep
  - Increase FiO_2 above 0.6
- $PaCO_2$ >45mmHg (EtCO2 >50mmHg)
  - Increase Vr
  - Increase Vt
- 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 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

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See you at EMS World Expo 2014