Transition to the New National EMS Education Standards: EMT-B to EMT

Module Two

Objectives:

Upon completion, each participant will do the following to a degree of accuracy that meets the Ntl EMS Education Standards:

- Discuss the requirements for adequate perfusion
- Discuss the composition of ambient air and how this relates to FiO_2
- Comprehend how the Boyle law influences ventilation
- Differentiate the muscles of inhalation and expiration as well as the accessory muscles

Objectives cont.

- Understand how compliance and resistance affect ventilation
- Differentiate general causes of upper and lower airway dysfunction
- Apply the concepts of minute and alveolar ventilation to EMS
- Summarize the role of chemoreceptors, and how changes in pH effect patient presentation

Objectives cont.

- Discuss hypoxic drive in COPD patients
- Compare and contrast the roles of the lung receptors
- Differentiate between the respiratory centers in the brain stem and understand how they influence patient presentation
- Comprehend how a V/Q mismatch effects patient presentation and treatment
- Discuss how O_2 and CO_2 are transported in the blood

Objectives cont.

- Compare and contrast the treatment for ventilatory disturbances and perfusion disturbances
- Differentiate respiratory distress from ventilatory failure and discuss appropriate treatments
- Discuss the positive and negative aspects of positive pressure ventilation

Objectives cont.

- Support the need to ventilate spontaneously breathing pts suffering from ventilatory disturbances
- Understand the need for and action of CPAP
- Discuss shock as it relates to ventilation and perfusion

Respiratory pathophysiology

The New EMS Standards incorporate pathophysiology concepts and have increased the depth and breadth of existing material

So how will this new pathophysiology stuff change what I do?

Old
- EMT-B used to react to a situation

New
- EMT will be better able to anticipate the situation

This will result in
- better pt. outcomes
- a more confident and competent EMT as a member of the EMS team

Adequate perfusion

Based on:
- Airway
- Ventilation
- Oxygenation
- Circulation

Provides oxygenated blood

Removes waste products

Composition of ambient air and FiO_2

Ambient air contains:

21%	Oxygen
79%	Nitrogen
.9%	Argon
.03%	Carbon Dioxide

FiO_2 is the fraction of inspired air expressed as a fraction or decimal rather than percentage

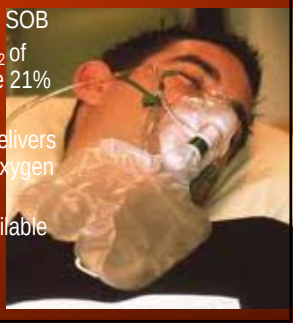
What does that have to do with EMS?

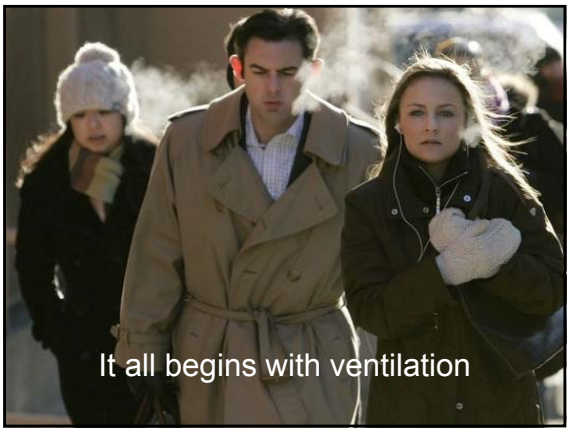
EMT responds for a pt. with SOB

Atmospheric air has an FiO_2 of 0.21 which would indicate 21% oxygen

NRB mask is applied and delivers an FiO_2 of 0.95, or 95% oxygen

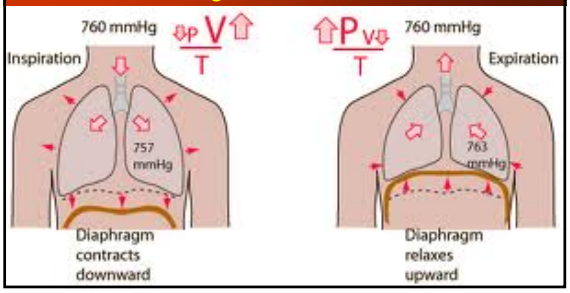
The pts. SOB is relieved by increasing amount of available oxygen





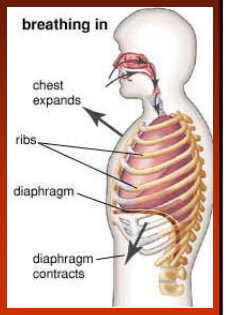
The Boyle law

By increasing the size of a closed container pressure decreases which creates a vacuum allowing air to enter



Inhalation

- Diaphragm contracts
- External intercostal muscles move slightly downward
- Ribs lift upward and outward
- Pleural linings force lung to expand
- Elastin creates an opposite pull, which creates negative pressure



Accessory muscles of inhalation

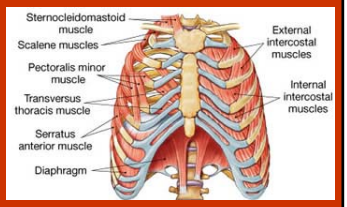
During compromise accessory muscles are employed to assist in inhalation

These include

sternocleidomastoid
- lifts sternum upward

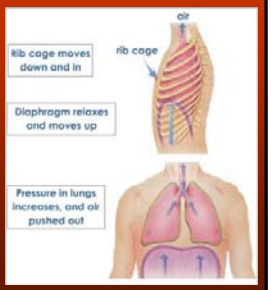
Scalene
- elevates ribs 1 & 2

pectoralis minor
- elevates ribs 3-5



Exhalation

- Diaphragm relaxes and moves upward
- Intercostals relax
- Ribs fall inward
- Chest cavity decreases in size
- Pressure in chest cavity increases
- Air is forced from lungs



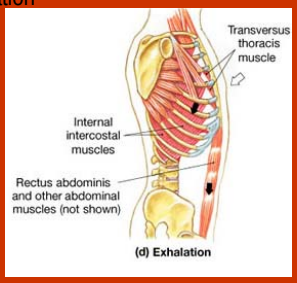
Accessory muscles of exhalation

Used during forced exhalation

Abdomen contracts
- diaphragm forced upward

Internal intercostals
- contract
- chest wall pulled inward

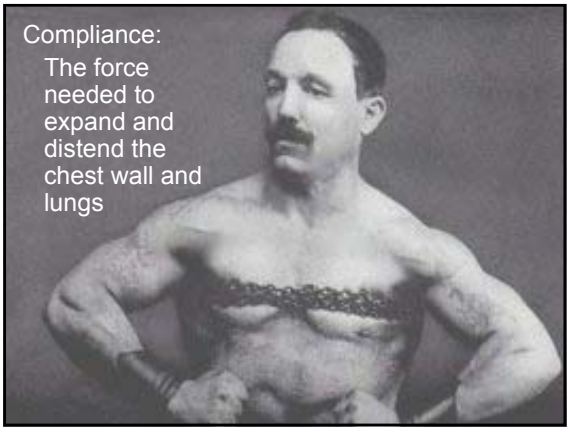
Intrathoracic pressure
- increases
- higher pressure forces air out



Mechanics of ventilation

Compliance:

- The force needed to expand and distend the chest wall and lungs



Airway resistance

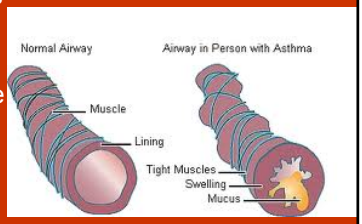
Patency of the airway

- Patency can be compromised by obstructions in upper airway or lower airway
- Upper airway obstruction occurs above the trachea and is typically caused by a foreign body or the tongue

Figure

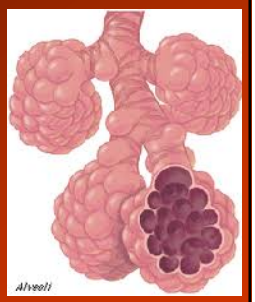
Lower airway obstruction occurs below the cricoid cartilage may be caused by:

- Foreign objects
- Bronchiole constriction
- Smooth muscle spasm of bronchioles



Alveolar ventilation

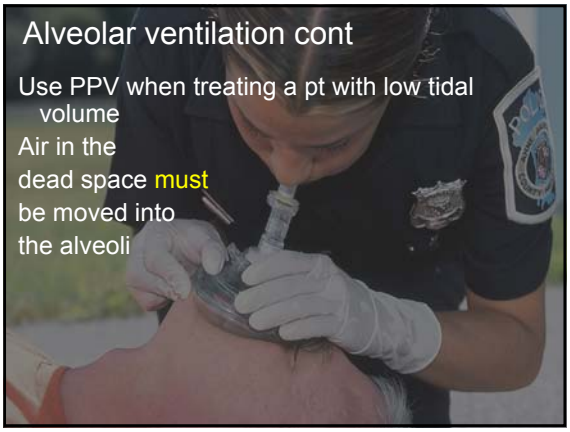
- Amount of air moved in and out of alveoli in 1 minute
- 150 mL of dead space where no gas exchange occurs
- Alveolar ventilation = (tidal volume minus dead space) x respiratory rate



Alveolar ventilation cont

- Decreased tidal volume or respiratory rate profoundly affect alveolar ventilation
- Despite a compensatory increase in respirations a low tidal volume will affect gas exchange in the alveoli
- Dead air space is first area fill

Alveolar ventilation cont

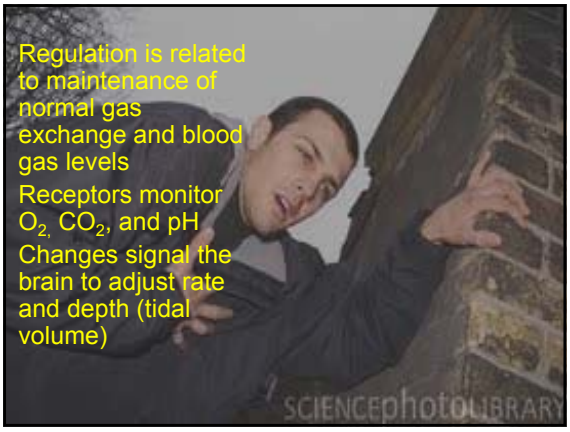


- Use PPV when treating a pt with low tidal volume
- Air in the dead space must be moved into the alveoli

Alveolar ventilation cont

- Placing a pt with low tidal volume on a NRB will do nothing but increase the level of O_2 in dead space

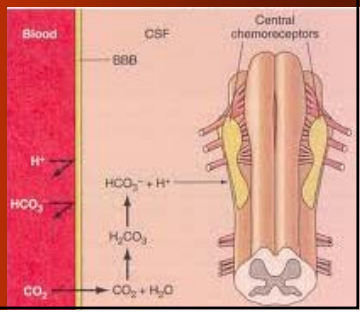
Concepts for the regulation of ventilation



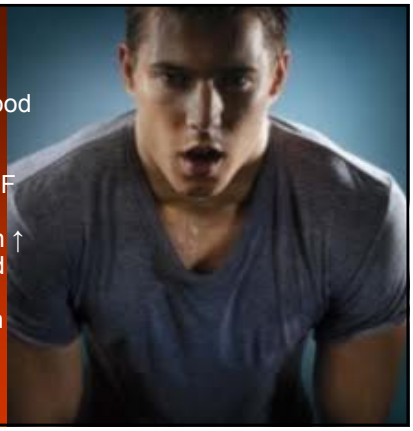
- Regulation is related to maintenance of normal gas exchange and blood gas levels
- Receptors monitor O_2, CO_2, and pH
- Changes signal the brain to adjust rate and depth (tidal volume)

Central chemoreceptors

- Most sensitive to changes in pH of cerebrospinal fluid, and CO_2 in arterial blood
- Located near respiratory center of the medulla

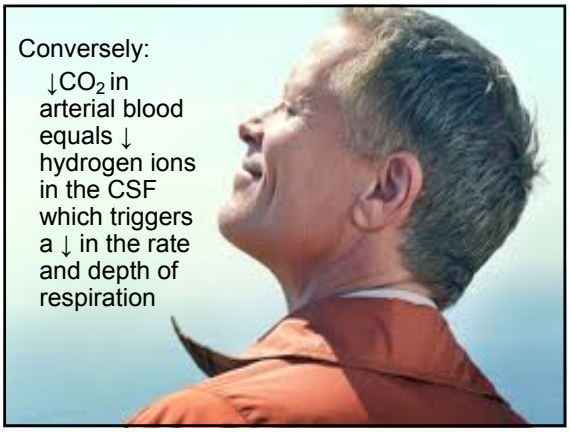


- ↑ CO_2 in arterial blood equals ↑ hydrogen ions in CSF which triggers an ↑ in rate and depth of respiration



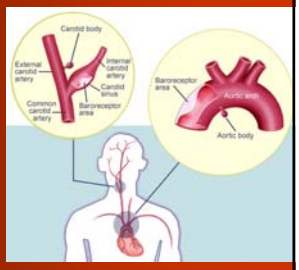
Conversely:

- ↓CO_2 in arterial blood equals ↓ hydrogen ions in the CSF which triggers a ↓ in the rate and depth of respiration



Peripheral chemoreceptors

- Located in the aortic arch and carotid artery bodies
- Strongest stimulus is arterial oxygen level
- Significant decrease in arterial oxygen content needed to trigger peripheral chemoreceptors

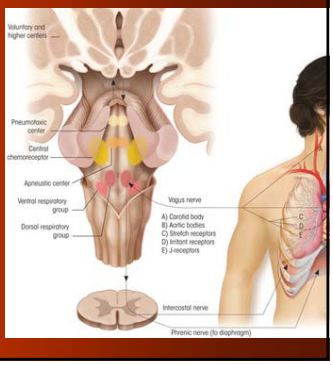


Lung Receptors

- Irritant
  - found in the airways
- Stretch
  - smooth muscle in airway
- J
  - capillaries surrounding the alveoli

Respiratory centers

- Four centers in the brainstem:
- Dorsal respiratory group (DRG)
- Ventral respiratory group (VRG)
- Apneustic Center
- Pneumotaxic Center

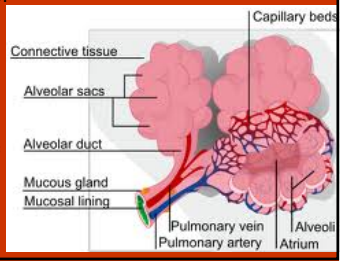


Which brings us to the V/Q ratio

The ventilation / perfusion (V/Q) ratio represents amount of ventilation in alveoli and amount of perfusion through alveolar capillaries

Bottom line

Amount of O_2 entering blood and amount of CO_2 offloading from blood



Ventilatory disturbances

Less oxygenated blood is available in alveoli, compared to amount of blood in the capillaries

Less oxygen saturates blood

Less oxygen delivered to cells

Result-cellular hypoxia

Gas exchange in the lungs

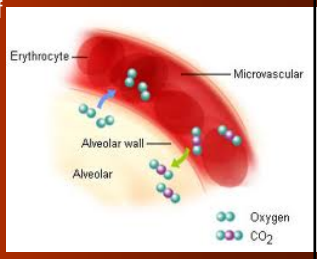
Gas diffuses from an area of high to an area of low concentration

Alveoli-O_2 rich

Venous capillary blood-CO_2 rich

O_2 diffuses to pulmonary capillaries for distribution

CO_2 diffuses to alveoli to be exhaled



O_2 and CO_2 transport in the blood

97-98.5% of O_2 is carried by hemoglobin
1.5-3% carried by plasma

70% of CO_2 is in the form of bicarbonate

23% of CO_2 is carried by hemoglobin

7% of CO_2 is dissolved in plasma

Gas exchange in the cells

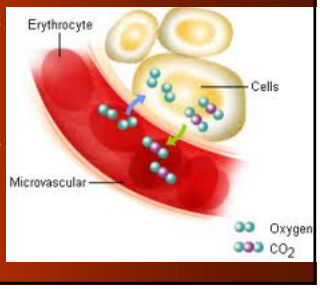
Diffusion from capillaries to body cells

Arterial capillary blood-O_2 rich

Cells-CO_2 rich

O_2 diffuses to cells

CO_2 diffuses to capillaries



Assessment

2% of ED visits from respiratory distress

Many causes / same treatment

Immediate recognition of inadequacy

Treatment for ventilation perfusion disturbances

Ventilatory disturbance treated by increasing amount of oxygenated blood in alveoli

Perfusion disturbance treated by increasing blood flow through pulmonary capillaries

Consider asthma

Ventilation disturbance

Constricted bronchioles reduce airflow resulting in less oxygenated air available at alveoli

Blood pressure is not affected and pulmonary capillaries are normal

Cellular hypoxia results from less oxygen available for amount of capillary blood

Consider severe bleeding

Compensatory mechanisms provide more O_2 to alveoli

Perfusion disturbance results in less blood for pulmonary capillary diffusion

Until bleeding is stopped cells become more hypoxic

SO…..

Assess:

Rate-respiratory & heart

Accessory muscle use

Mental status

Lung sounds

Retractions

Positioning

Speech

Skin

Differentiating respiratory distress from respiratory failure

Respiratory distress

- compensatory mechanisms sustain normal function

Respiratory Failure

- compensatory mechanisms fail

Recognize respiratory distress

Signs and symptoms

- tripoding
- accessory muscle use
- increased respiratory rate

Treatment for distress

Prevent distress from becoming failure

Support compensatory efforts with supplemental O_2 if tidal volume and rate permit

Take time to focus on lung sounds during secondary assessment

Respiratory failure

Signs and symptoms

AMS

cyanosis

low O_2 sats

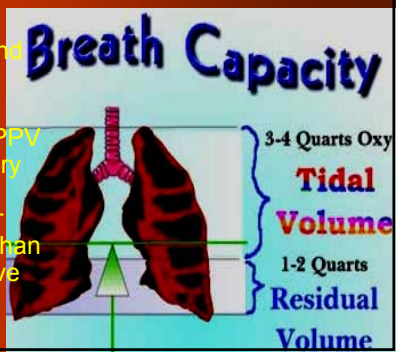
slow or irregular rate

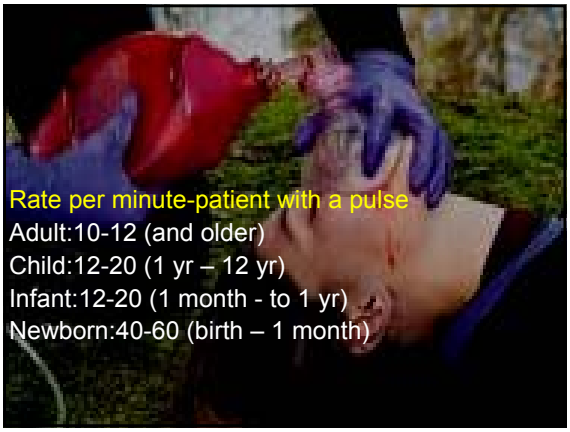
symptoms persist despite supplemental O_2

PPV for failure

Requires immediate recognition and intervention

Increase tidal volume with PPV delivered every 3rd- 5th breath with a greater tidal volume than pt. can achieve on their own





Rate per minute-patient with a pulse

Adult:10-12 (and older)

Child:12-20 (1 yr – 12 yr)

Infant:12-20 (1 month - to 1 yr)

Newborn:40-60 (birth – 1 month)

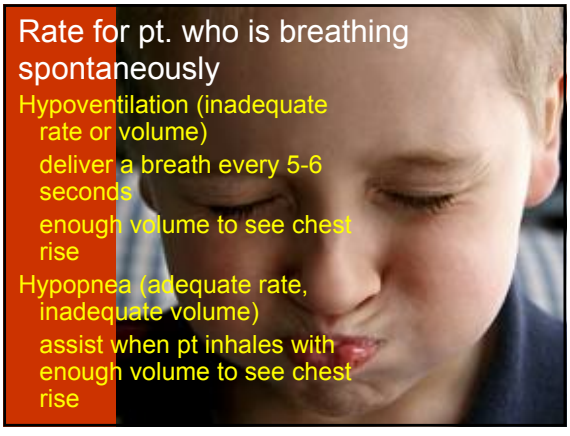
Rate for pt. who is breathing spontaneously

Hypoventilation (inadequate rate or volume)

- deliver a breath every 5-6 seconds
- enough volume to see chest rise

Hypopnea (adequate rate, inadequate volume)

- assist when pt inhales with enough volume to see chest rise



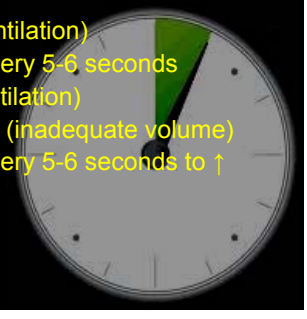
Rate for pt. who is breathing spontaneously

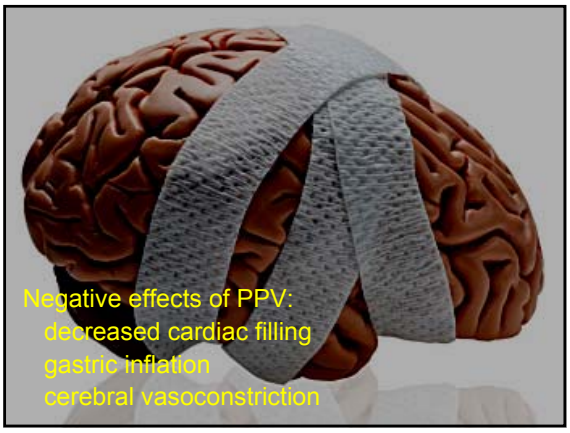
Bradypnea (slow ventilation)

- deliver a breath every 5-6 seconds

Tachypnea (fast ventilation)

- leads to hypopnea (inadequate volume)
- deliver a breath every 5-6 seconds to ↑ volume and ↓ rate

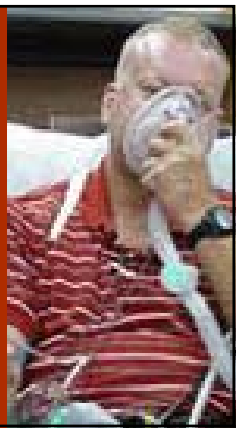




Negative effects of PPV:
- decreased cardiac filling
- gastric inflation
- cerebral vasoconstriction

CPAP

Used during acute pulmonary edema to:
- Keep alveoli from collapsing
- Prevent fluid from crossing alveolar membrane

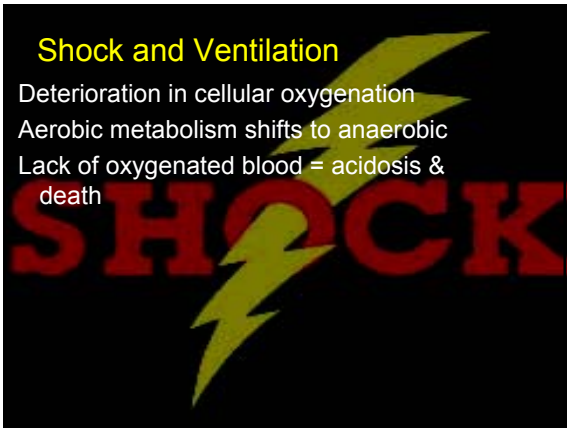


Shock and Ventilation

Deterioration in cellular oxygenation

Aerobic metabolism shifts to anaerobic

Lack of oxygenated blood = acidosis & death



Anaerobic metabolism

Acidosis = failure of normal metabolic activities

Cells die

Enzymes released & kill neighboring cells

Widespread cell death cascades

Leads to cycle of hypoperfusion, hypoxia, acidosis and eventual death

Anaerobic cont

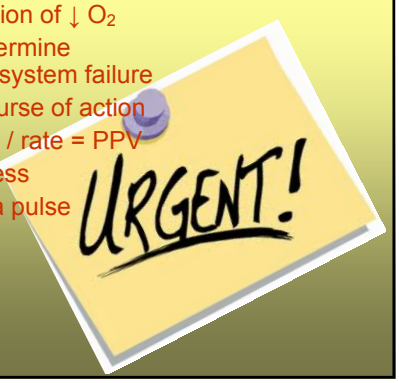
Compensatory mechanisms:
- Tachypnea
- Tachycardia
- Release of epi & nor epi
- Release of renin-angiotensin-aldosterone hormones

Early recognition of ↓ O_2

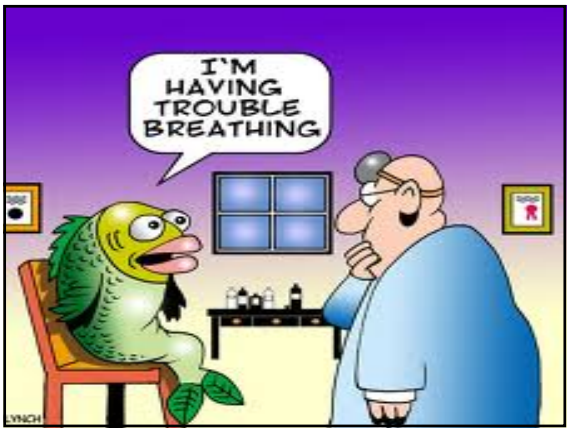
Use hx to determine physiologic system failure

Determine course of action

↓ tidal volume / rate = PPV
- 8-10 pulseless
- 10-12 with a pulse



- Be aggressive when approaching ventilatory issues
- Be diligent in assessing tidal volume
- Use PPV whenever tidal volume or rate is inadequate
- Just because a patient is breathing does not mean they are perfusing
- Don't wait for distress to become failure



Although it seems distant…..we'll make it in time !