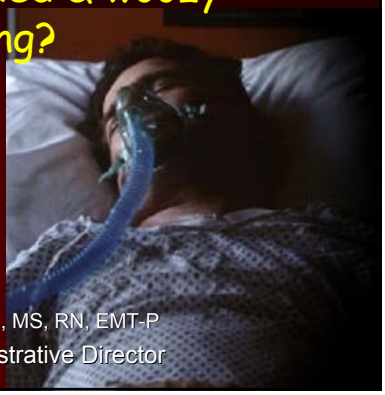
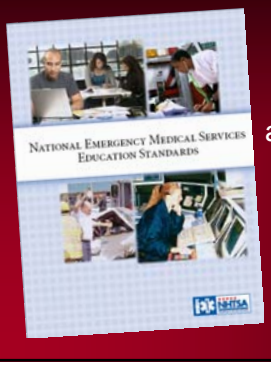
Weak, winded & woozy – what's wrong?



Connie J. Mattera, MS, RN, EMT-P
NWC EMSS Administrative Director

Upon completion, participants will do the following without critical error:



- Integrate assessment findings in pts who present w/ respiratory distress to form an accurate field impression.
- This includes developing a list of differential diagnoses using higher order thinking and critical reasoning.

Upon completion, participants will do the following without critical error:

- Compare and contrast pts who present w/ dyspnea, weakness, & possible AMS
- Weigh the indications and contraindications of possible interventions and sequence evidence-based EMS care

What audible sounds indicating airway or ventilatory impairment can be heard w/o a stethoscope when inspecting the airway?

- Snoring, gurgling
- Hoarseness
- Stridor, choking sounds
- Wheezes from larger bronchi
- Crackles heard through mouth
- Expiratory grunting

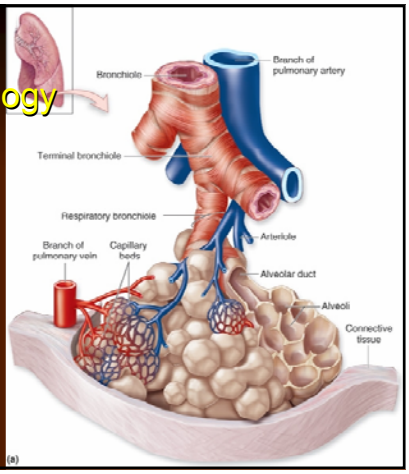
S&S airway impairment

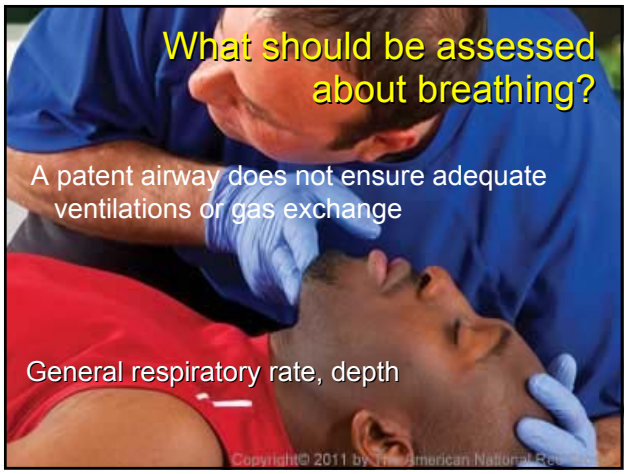
- Secretions/debris in airway
- Stridor, snoring, gurgling, grunting
- Restlessness, anxiety, dyspnea
- Apnea, agonal ventilations
- Use of accessory muscles; rocking chest motion
- Retractions, tracheal tugging
- Hypoxia, hypercarbia
- Unable to speak/make age-appropriate snds
- Faint/absent breath sounds

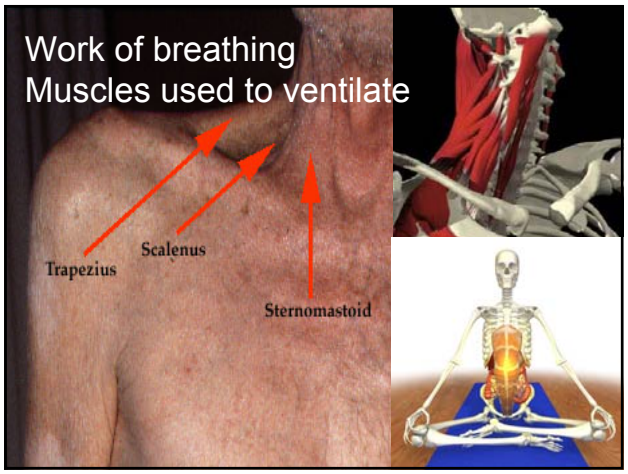
Pulmonary pathophysiology

All respiratory problems can be categorized as impacting

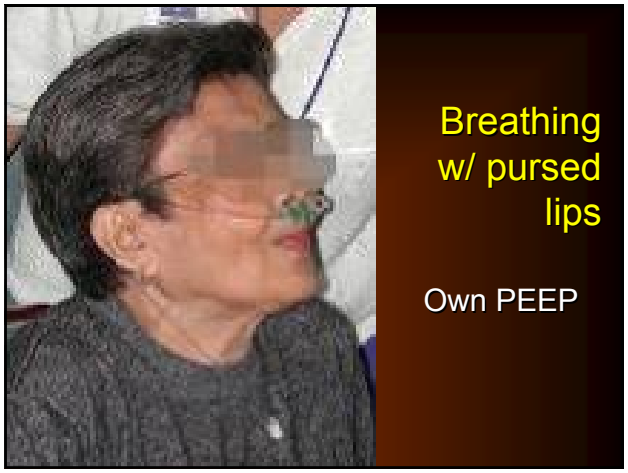
- Ventilation
- Diffusion
- Perfusion

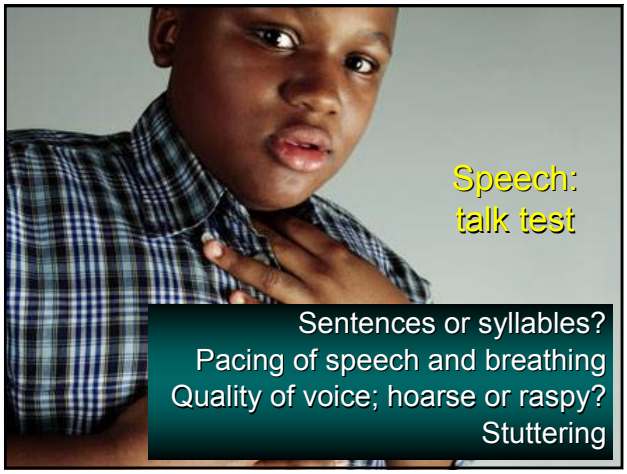


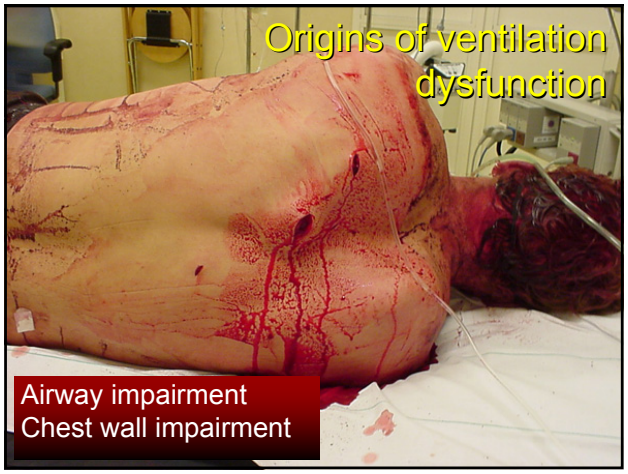

What should be assessed about breathing?
A patent airway does not ensure adequate ventilations or gas exchange
General respiratory rate, depth


Work of breathing
Muscles used to ventilate
Trapezius
Scalenus
Sternomastoid


Inspection cont.
Diaphragmatic, See Saw, Apical breathing?
Splinting?


Breathing w/ pursed lips
Own PEEP

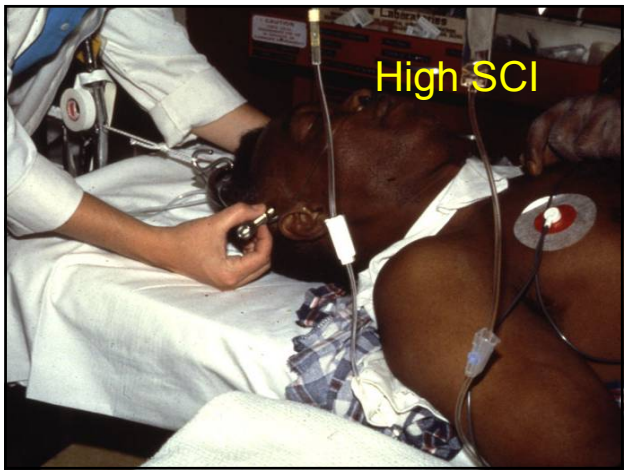

Speech: talk test
Sentences or syllables?
Pacing of speech and breathing
Quality of voice; hoarse or raspy?
Stuttering


Origins of ventilation dysfunction
Airway impairment
Chest wall impairment

Origins of ventilation dysfunction

Ventilatory depression or arrest
CNS: Trauma, stroke, medical neuro condition

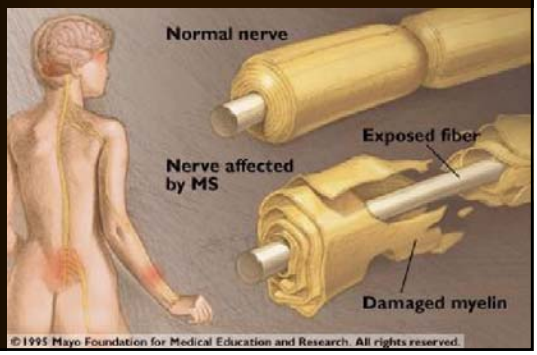
High SCI



Muscles:
- Myasthenia Gravis,
- muscular dystrophy

Ventilatory depression or arrest

Nerves: Polio, Guillain-Barrè syndrome, MS



Breathing considerations in obese pts

- Lungs 35% less compliant
- Weight of chest makes breathing difficult – ventilate at 8-10 mL/kg
- SpO_2 unreliable on finger – use central sensor
- Will desaturate if supine
- CO_2 retention probable
- CPAP useful

Factors that impair diffusion

- Thickening of alveolar walls
- Destruction or collapse of alveoli
- ↓ permeability
- Widened interspace
- ↓ in blood flow

Conditions w/ impaired diffusion

Atmospheric deficiency

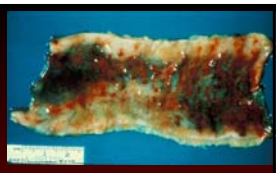
Alveolar pathology
- Asbestosis
- COPD
- Inhalation injuries
- Pneumonia

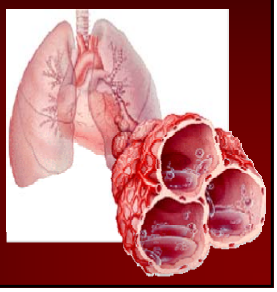
Interstitial pathology
- High pressures (HF)
- Pulmonary hypertension

Diffusion cont.

High permeability
- ALI; non-cardiogenic pulmonary edema
- Asbestosis
- Near-drowning
- Post-hypoxia
- Inhalation injury

Capillary bed pathology: atherosclerosis





Assess gas exchange/ evidence of hypoxia

What info is obtained by assessing skin color, temp & moisture?

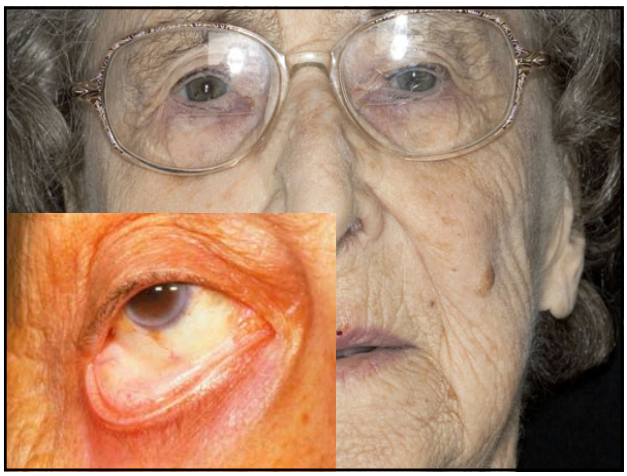
Adequacy of oxygenation
Adequacy of peripheral perfusion
Evidence of SNS stimulation/compensation

Cool, hot, pale, flushed, mottled, ashen, cyanotic, & diaphoretic skin must be explored for cause

What factors influence the detection of cyanosis?

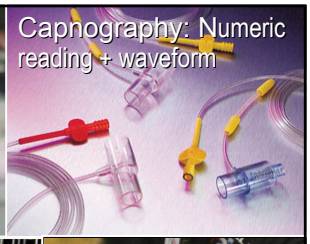
Rate of blood flow
Degree of desaturation (at least 5 Gm)
Type of light
Observer skill
Thickness and color of skin

Skin color unreliable: anemia & peripheral vasoconstriction



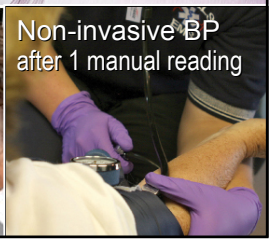
Start placing monitors prn

Pulse oximetry



Capnography: Numeric reading + waveform

ECG rhythm + 12 lead

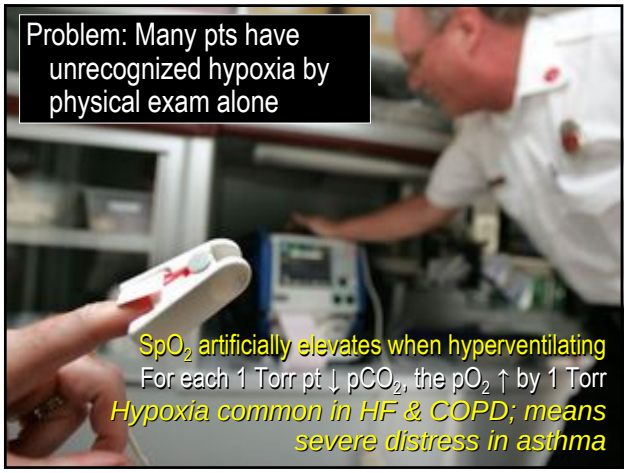


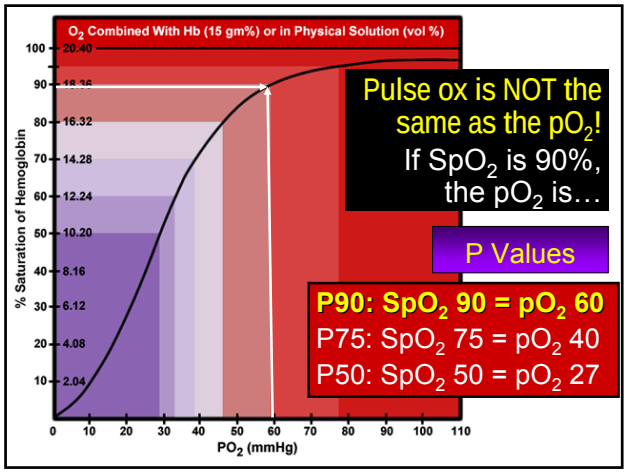
Non-invasive BP after 1 manual reading

What's the difference between capnography & pulse oximetry?

What does pulse ox measure?

A. Mean arterial pressure
B. Level of CO_2 in the blood
C. Amount of O_2 dissolved in plasma
D. % of hemoglobin bound with a gas





Pulse oximetry range guidelines

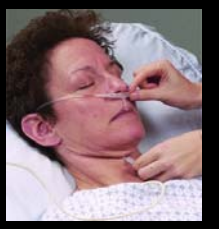
Ideal:	96%-100%
Mild-mod hypoxemia:	90%-95%
Severe hypoxia:	< 90%

Severely low SpO_2 (< 90%) predictor of poor outcomes

The affinity of hemoglobin for O_2 is altered by conditions in the tissue the blood is flowing through…

Which of these will influence the amount of O_2 delivered to cells?

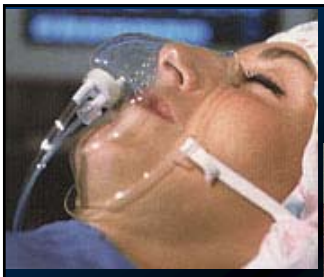
A. Acid-base status
B. Body temperature
C. The amount of hemoglobin
D. All of the above



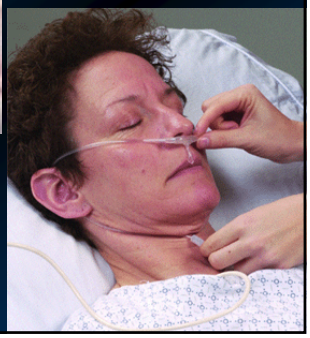
Capnography

Indicates adequacy of ventilations, perfusion, & dead space by detecting how much CO_2 is exhaled

Gives a numeric value & graphic waveform



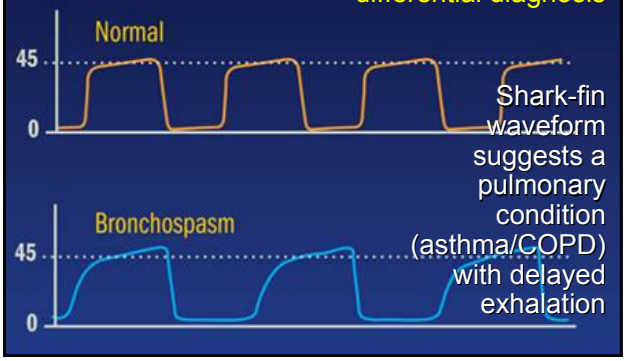
Capnography



Use on intubated and non-intubated pts with a NC attachment or mask

HF should have normal, squared off waveform

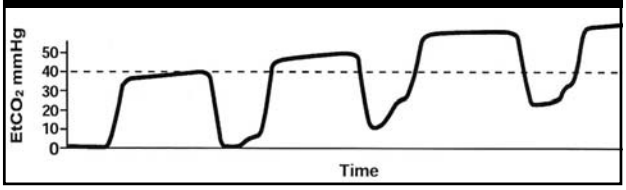
Waveform shape helps make a differential diagnosis



Shark-fin waveform suggests a pulmonary condition (asthma/COPD) with delayed exhalation

Asthma or COPD

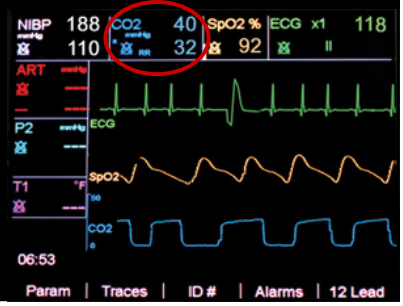
Capnography: Incomplete inhalation/ exhalation; CO_2 does not get completely washed out on inhalation

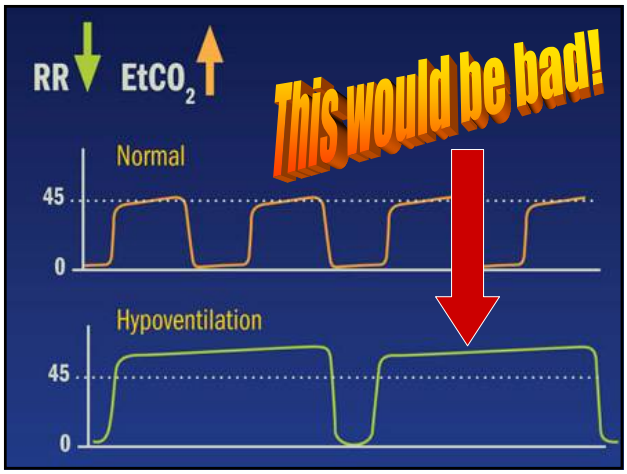


Capnography findings in HF

After CPAP started, $EtCO_2$ may briefly rise d/t improved ventilations, before it falls due to tachypnea

Severely ↑ $EtCO_2$ indicates ↑ pCO_2 levels and ventilatory failure



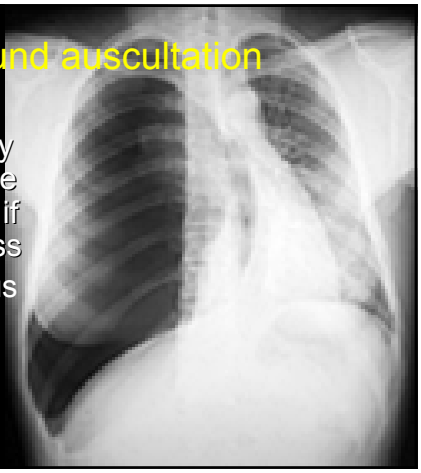


Causes of hypercarbia

Hypoventilation from any cause
- Airway obstruction
- Respiratory depression
- Ventilatory muscle impairment
- Pulmonary obstructive diseases

Treatment: Correct inciting cause

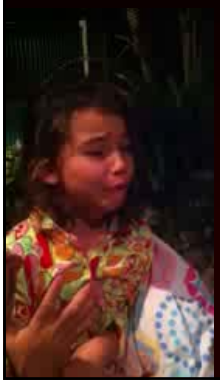
Breath sound auscultation



Listen immediately for evidence of air entry if pt in distress

Most ominous sound is silence

S&S compromised ventilations



Apnea
S&S hypoxia
Dyspnea; accessory muscle use
Upright, tripoding; orthopnea
Ventilatory efforts weak, shallow, labored, retracting
Adult RR < 10 or ≥ 24/min
$EtCO_2$ > 45; change in waveform

O_2 is a drug and must be given to specific pts based indications/contraindications and in correct doses by an appropriate route - being vigilant for adverse reactions

Which patients can be harmed by *hyper* oxia and need careful titration of oxygen?

Harmed by ***hyper***oxia

Uncomplicated Acute MI
Post-cardiac arrest
Acute exacerbations of COPD
Stroke
Neonatal resuscitation

Why?

Give O_2 to these pts only if evidence of hypoxia and titrate to dose that relieves hypoxemia without causing hyperoxia (SpO_2 94%)

Iscor, S. et al. (2011) Supplementary oxygen for nonhypoxemic patients: O(2) much of a good thing? Crit Care, 15(3), 305

Profound, prolonged hypoxia is also bad

So, who NEEDS O_2?

SpO_2 < 94% (Exception: COPD may be best managed w/ SpO_2 88-92%)

Actual or potential airway compromise (epiglottitis, airway burns)

DAI (to increase O_2 reserves)

Globally poor tissue oxygenation & perfusion (shock)

Who should get CPAP?

Pulmonary edema
COPD/asthma w/ severe distress
Flail chest w/o pneumothorax
Near drowning
Pneumonia (?)
Palliative care (?)
Diaphragmatic weakness
Post-extubation rescue

What do all these conditions have in common?

Severe dyspnea & refractory hypoxia
Poorly expanded lung fields
↑ WOB (↑ inspiratory muscle work)
↓ Minute ventilation
Inability to remove CO_2 from body
Hypercarbic ventilatory failure
Narcotic effect on brain → ↓ RR
Fatigue + ↓ RR = resp. arrest

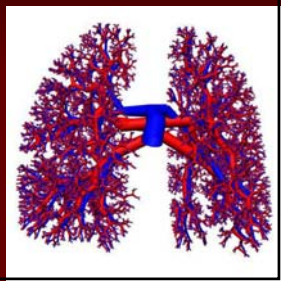
Which is NOT a possible complication of using CPAP?

A. Collapse of the alveoli
B. Decrease in blood pressure
C. Gastric distension and vomiting
D. Patient anxiety and claustrophobia

Pulmonary circulation (Q)

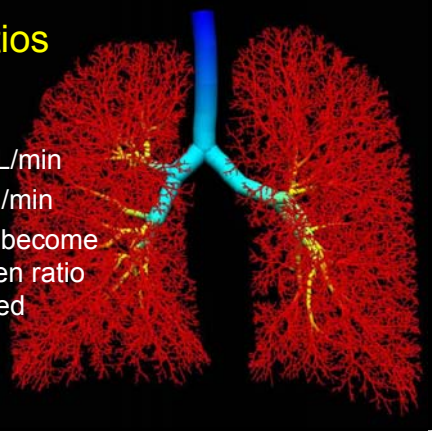
Depends on: Adequate blood volume, intact pulmonary vessels & efficient pump

Q = SV X HR
Q = 70 mL X 72 BPM
Q = 5,040 mL/min



V_A/Q ratios

Should be 1:1
V_A = 5,250 mL/min
Q = 5,040 mL/min
Patients may become hypoxic when ratio is imbalanced



Circulatory/Cardiac status

✓ **perfusion**: general pulse rate, quality, rhythmicity

Establish that underlying cause of respiratory difficulty is not cardiac in nature

✓general rate & quality of pulse: expect strong with HTN; weak if HF or dehydrated

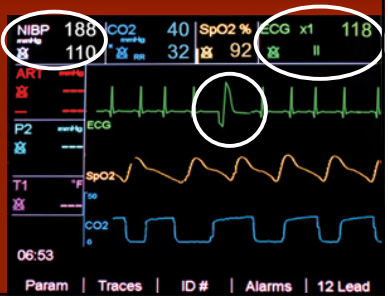
Tachycardia – hypoxia or early shock; ✓ pulse deficit

Palpitations or irregular pulse: Ask if hx of AF, PVCs common

BP (SBP ≥ 90; DBP ≥ 60)

Need MAP of 60 to fill CA

Often high in HF; if low suspect shock



Perfusion impairment

Inadequate blood volume

Inadequate hemoglobin: anemia, trauma

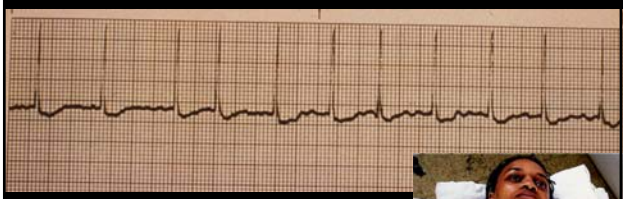
Impaired blood flow: pulmonary embolus

Capillary wall pathology: pulmonary contusion

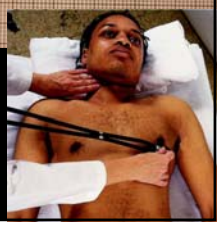
Monitor ECG

Bradycardia is bad!

Why is AF common in HF?



Why is it a concern?



12-lead IF:

Discomfort (nose to navel, shoulder, arm, back)

SOB/HF

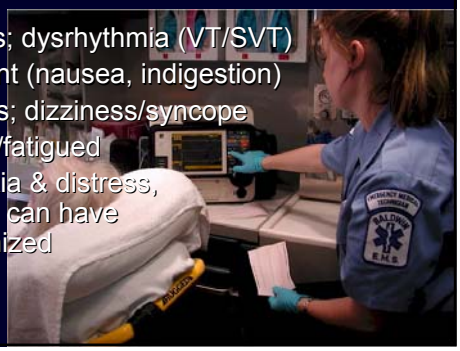
Palpitations; dysrhythmia (VT/SVT)

GI complaint (nausea, indigestion)

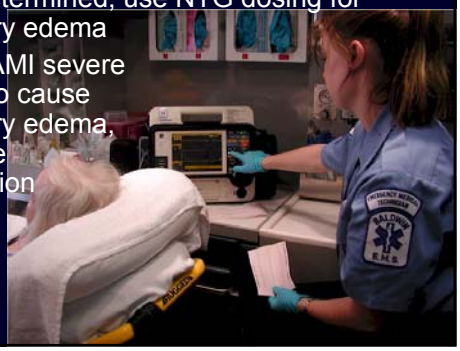
Diaphoresis; dizziness/syncope

Weak/tired/fatigued

With hypoxia & distress, many pts can have unrecognized ischemia

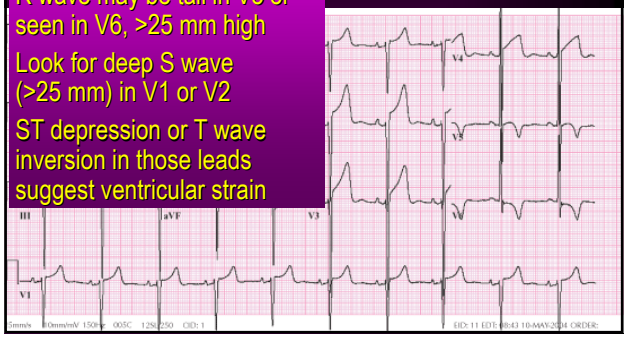


If HF & ACS suspected: 12-lead ASAP
If **acute** ischemia; give NTG per ACS
If age undetermined, use NTG dosing for pulmonary edema
Typically, AMI severe enough to cause pulmonary edema, will cause hypotension

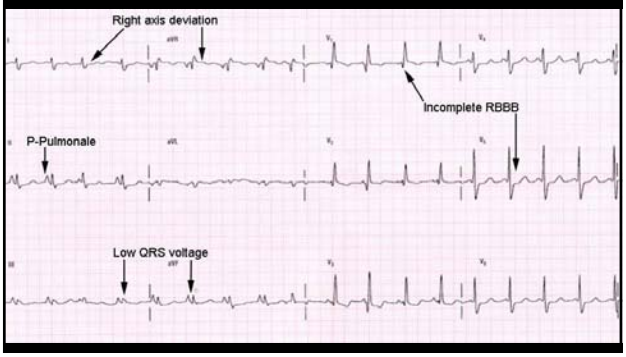


12 L implications

Ventricular hypertrophy may be evident in leads that overlie affected ventricle
R wave may be tall in V5 or seen in V6, >25 mm high
Look for deep S wave (>25 mm) in V1 or V2
ST depression or T wave inversion in those leads suggest ventricular strain



Tall, peaked P waves
"P pulmonale" pattern of COPD



Cerebral function may be affected by

Fatigue
Hypoxia; hypercarbia
Cardiac status

Assess mental status for:

Alertness, anxiety, apprehension, restlessness
AMS, confusion, disorientation, decreased LOC
Dizziness
Headache
Perioral tingling
Seizure, syncope, coma

Secondary assessment

SAMPLE history
Full set of VS
ROS
- Inspection
- Palpation
- Percussion
- Auscultation

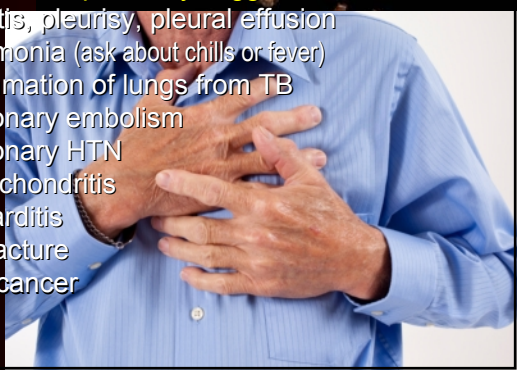
Dyspnea on exertion?
Dyspnea at rest?
OPQRST of S&S

Chief complaint

Ask pt to take a deep breath & ask about pain

Pleuritic chest pain may suggest

- Pleuritis, pleurisy, pleural effusion
- Pneumonia (ask about chills or fever)
- Inflammation of lungs from TB
- Pulmonary embolism
- Pulmonary HTN
- Costochondritis
- Pericarditis
- Rib fracture
- Lung cancer



Coughing?

Cough variant asthma is usually caused by airway irritation and/or constriction. Coughing may increase to retching causing bronchospasm & hypoxia.

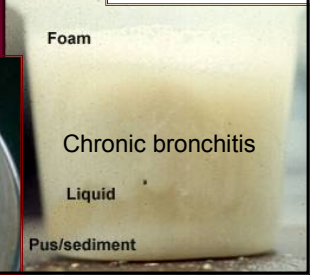
Cough differential

- Aspiration
- Smoke inhalation
- Secretions
- Irritation
- Hyperreactive airways
- Bronchospasm
- Productive or non-productive?

NOTE

Sputum quantity, color, consistency, odor

- Clear
- Yellow/Green
- Rusty/bloody



Frothy sputum due to pulmonary edema (pink-tinged means surfactant is washing out)

Quickly look for S&S LV failure (S3 heart sound)

Medications

Cardiac	Pulmonary
□ ACEIs: "prils"	□ Short/long-acting beta agonists
□ ARBs: "sartans"	□ Anticholinergics
□ Beta blockers: "lols"	□ Mast cell inhibitors
□ Ca Blockers	□ Leukotriene modifiers
□ Diuretics	□ Steroids
□ Vasodilators	□ Methylxanthines
□ Anticoagulants (AF)	□ Erectile dysf. drugs
□ Antiarrhythmics	□ Home oxygen
□ Digoxin	

Time and amount of last dose

Meds – ACE Inhibitors (ACEI)

Generic name ends in "pril"

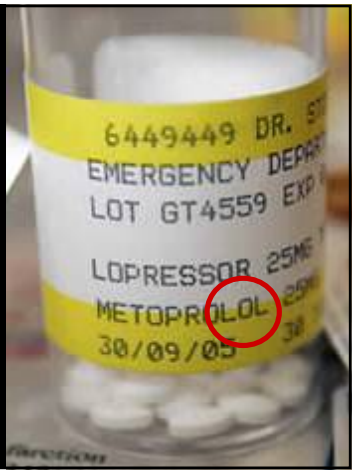
Blocks creation of angiotensin II:
Vasodilates pt, ↓ BP, prevents remodeling and ↓ the heart's workload

Benza*pril* / Lotensin	Moesipril / Univasc
Captopril / Capoten	Perindopril / Aceon
Enalapril / Vasotec	Quinapril / Accupril
Fosinopril / monopril	Ramipril / Altace
Lisinopril / Prinivil / Zestril	Trandolapril / Mavik

What can block the SNS?

Cardio-selective drugs

B-1 blockers: end in "lol"

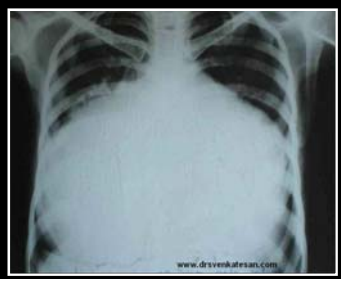


Beta 1 blockers

Acebutolol (Sectral)
Atenolol (Tenormin)
Betaxolol (Kerlone)
Bisoprolol (Zebeta)
Carvedilol (Coreg)
Labetalol
Metoprolol/Lopressor/Toprol
Nadolol (Corgard)
Pembutolol
Pindolol
Propranolol (Inderal)
Timolol (Blocadren)
Sotalol (Betapace)

…PMH

SOB, cough, dyspnea?
Asthma/COPD?
Other pulmonary conditions?
CVD: CAD, HTN, ACS, stroke
HF, dysrhythmias
Valve disease
Diabetes
Drug abuse
Chronic renal failure
Gastric surgery



Acute vs. Chronic in nature?

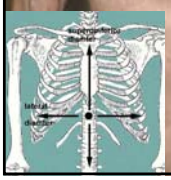
Classified by nature of onset
Acute: Rapid onset and short duration
Chronic: Slow onset, persists over time

Past medical history

Tobacco use
Report in pack years
# of packs smoked/day X
# of yrs they've smoked
Problems when pack years surpass 20

AP/Lateral diameter

Lateral diameter should be 2X AP diameter
Increased AP diameter (barrel chest) with diaphragmatic flattening associated w/ emphysema and lung hyperinflation
Rib flare?



Chest palpation

Palpation plays minor role in exam of normal chest; lungs covered by ribs & aren't palpable
Compress downward on sternum; inward on lateral chest wall (gently)
Point of maximum impulse (apical pulse)

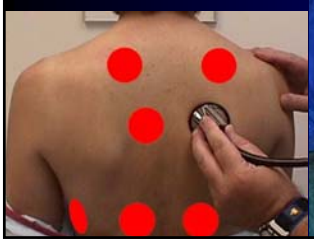
How can EMS miss findings or misdiagnose?

Poor equipment
Poor auscultation technique
Misinterpret cause of wheezes
Crackles isolated to 1 lobe
Diminished lung sounds not detected
Lack of adequate history

DIANA-hf-3-08

How to listen?

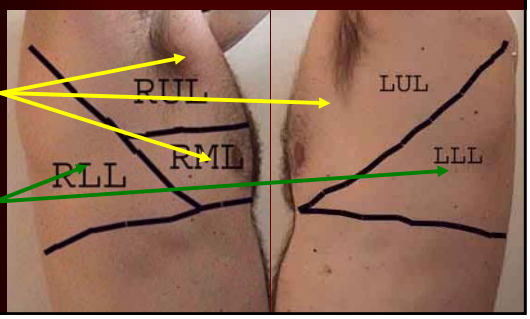
Sit pt up (if able)
Turn supine patient to assess back
Ask pt to breathe normally through open mouth
Stethoscope on skin – not over clothes



Technique:
Where should you listen?

All lobes, front and back

Front
Back

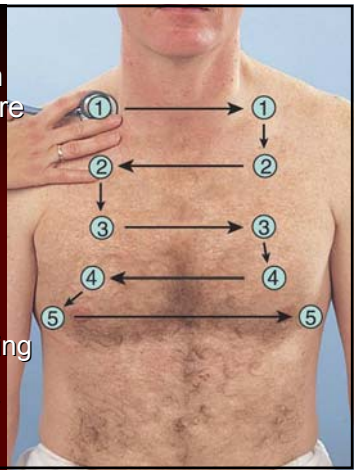


Where to start?

Auscultatory triangle in back
Medial/lower scapula; less muscle mass; easy to hear snds
Lower diaphragm attachment in back; fluid accumulates there first
Move up from posterior base compare side to side – one breath at each site

Anterior: Start at apex, move down to bases - compare side to side

TIPS
Have pt cough if sounds difficult to hear
Exhale forcefully to accentuate wheezing that is faint when breathing normally



What are adventitious sounds?

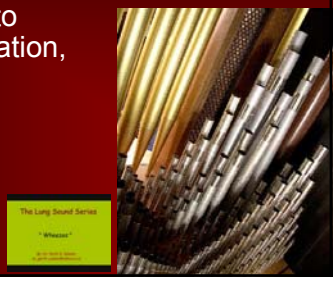
- Sounds that are super-imposed on normal breath sounds
- Crackles
- Wheezes
- Stridor
- Pleural friction rub

List 2 conditions that may present with crackles

- LV failure w/ *pulmonary edema*
- Poorly ventilated areas of *atelectasis*
- Localized over early or non-consolidating *pneumonia*
- Pulmonary fibrosis
- Tubercular lung cavities
- Lung abscess
- Terminally ill w/ depressed cough reflex

Wheezes

- Harmonic, musical sounds produced by turbulence when air passes through bronchi that fluctuate between closed & barely open
- Describe according to location, pitch, duration, timing, complexity



Wheezes

- Sibilant (asthma/emphysema)
- Sonorous (formerly called rhonchi)
- Louder/longer on expiration
- Severity does not correlate well w/ degree of airway obstruction – assess capnography
- Wheezes dissipate with bronchodilator therapy
- *No* wheezing if severe airway obstruction – assess ability to move air

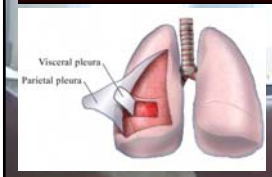
All that wheezes is not asthma Consider other causes

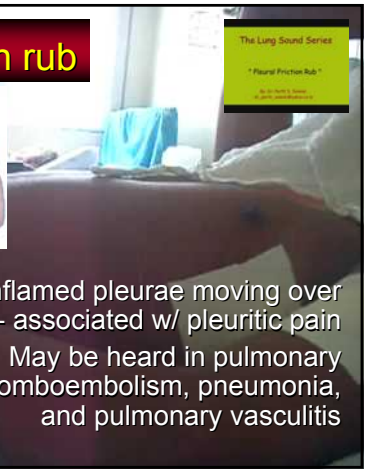
- **A**: Asthma
- **S**: Stasis: Pulmonary embolism
- **T**: Toxins/inhaled irritants
- **H**: Heart: HF; "cardiac asthma"
- **M**: Mechanical obstruction, FB, cancer
- **A**: Allergy/aspiration
- **TIC**: Trauma, infection, chronic (COPD)

Bilateral wheezes in cardiac conditions

- May be caused by external airway compression from interstitial water
- Fluid in alveoli irritating bronchioles causing bronchospasm
- Other mechanisms not well understood

Pleural friction rub





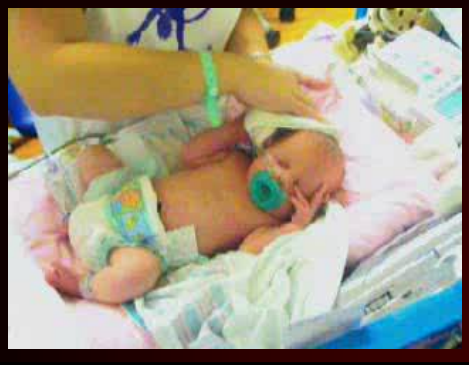
Produced by inflamed pleurae moving over one another - associated w/ pleuritic pain

May be heard in pulmonary thromboembolism, pneumonia, and pulmonary vasculitis

Differential

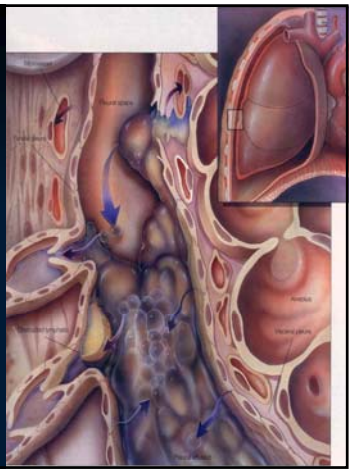
Pleural friction rub	Pericardial friction rub
Quality: Loud & grating, creaking or squeaking	Quality: Hard & grating, scratching or crunching
Location: Low axilla; anterior, lateral, or post. bases	Location: Lower L sternal border
Timing: Late inspiration/ early expiration; ceases if pt holds breath; persists during coughing	Timing: Occurs in relation to heartbeat; most noticeable during deep inspiration; continues if pt holds breath

Stridor

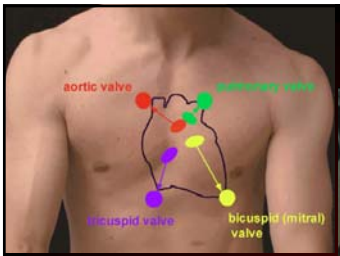


Decreased or absent sounds

- Fluid may move into pleural space, causing a pleural effusion
- Loss of surfactant → atelectasis

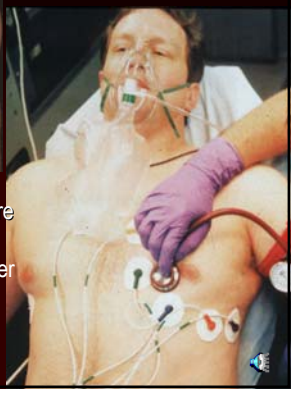


Heart sounds



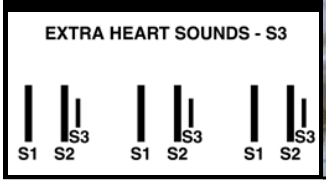
S1 (lub): Beginning of systole; closure of mitral and tricuspid valves. Most audible at apex & lower sternal border

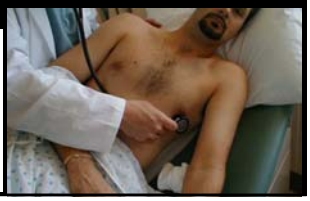
S2 (dub): Beginning of diastole; closure of aortic & pulmonic valves
Most audible at base



S3: Ventricular gallop

- 3rd sound occurs early in diastole immediately after "dub" with cadence like "Ken-tuck-y"
- Caused by early, rapid filling of a dilated LV
- Most audible at apex with pt on L side
- Abnormal > 30 years of age
- Present in 70% of pts > 40 yrs w/ EF < 30%



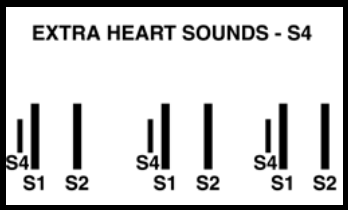


S4: Atrial diastolic gallop

Forceful ejection of blood into stiff ventricle

Most audible at apex

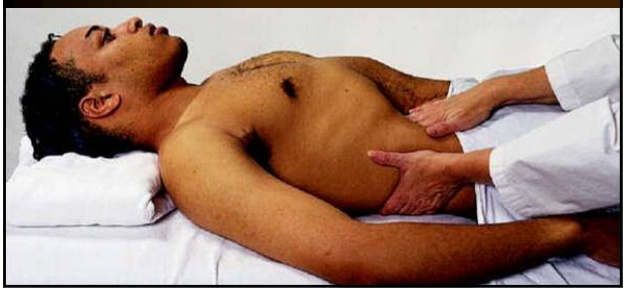
Occurs late in diastole and may be caused by pulmonic stenosis or any condition that affects left ventricular compliance, e.g., aortic stenosis, hypertension, MI, cardiomyopathy



Rapidly assess abdomen

✓ for ascites – chronic RV heart failure

✓Hepatojugular reflux – neck veins distend when liver palpated (sign of hepatomegaly)



Peripheral edema

New-onset HF

<25% of pts; rarely have peripheral edema

Causes:

- Ischemia or MI
- HTN crisis
- Arrhythmia – tachy, brady
- Pulmonary embolus

Acute decompensation of chronic Rt HF

~75% of pts (RV involved)

Often have recent wt gain (fluid) & dependent edema (sacral or feet/ankles)

911 called for a 68 y/o m w/ breathing problems

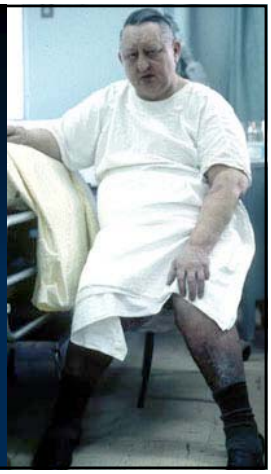
Pt confused but able to speak in short phrases

Exam: ↑ WOB w/ diffuse wheezes; no crackles

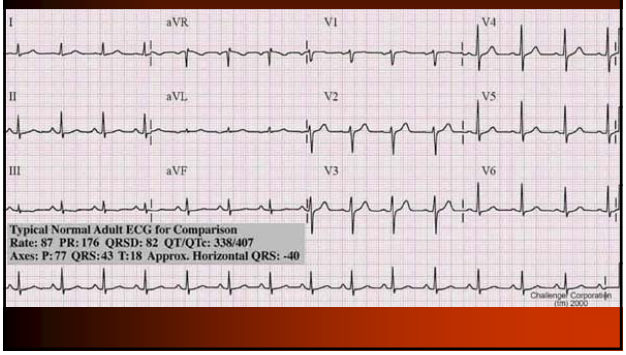
VS: BP 148/89; P 87; RR 32 & labored; SpO_2 84% on RA

Glucose: 145

12 L ECG as follows:



Look OK right now?



HPI: ↑ SOB for past 24 h w/ mild cough and confusion
Denies fever/chest pain
Meds: metformin, Benicar, Crestor, albuterol, ipratropium
Unsure if he took meds
PMH: CAD, HTN, high cholesterol, bypass surgery, HF, t2DM, chronic bronchitis

What's wrong?

Differential

Consider cause: Treat based on etiology

ALI
AMI
Anaphylaxis
Aspiration
Asthma
COPD
Heart failure
Panic disorders
Pleural effusion
Pulmonary edema
Pneumonia
Pneumothorax
Pulmonary embolism

Common causes of Lt HF

LV systolic dysfunction (60% of pts)
HTN (75% of pts)
CVD, CAD
Hx: MI
Faulty valves
Myocardial disease (myopathies, myocarditis)
Diabetes, renal failure

Path to pulmonary edema

When patient has an MI, uncontrolled HTN, valve disease, or dysrhythmia, LV becomes damaged and does not pump effectively

Compensatory mechanisms

↓ LV stroke volume sensed by receptors - trigger series of compensatory responses

Baro receptors in aortic arch, carotid sinus and kidneys

Osmoreceptors in brain

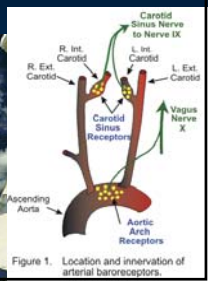


Figure 1. Location and innervation of arterial baroreceptors.

SNS tries to compensate…

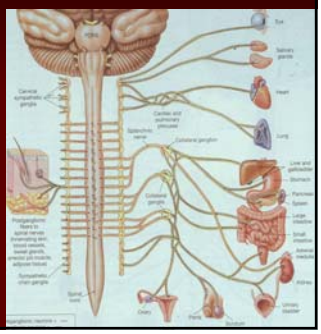
Norepi from nerve endings

Epi from adrenal glands

↑ heart performance & maintain MAP

Epi activates β receptors

Norepi activates α receptors



SNS Actions

	α	β
Heart		↑ rate, force, automaticity, conduction
Lungs	Constricts	Dilates
Vessels	Constricts	Dilates

Fight or flight response

Goal: Provide energy, O_2, & ability to react to stress

Pupils dilate

Arterioles constrict

Leg arteries dilate

↑ HR, contractility, BP

Bronchodilation, ↑ RR

ATP stimulated

Sweating

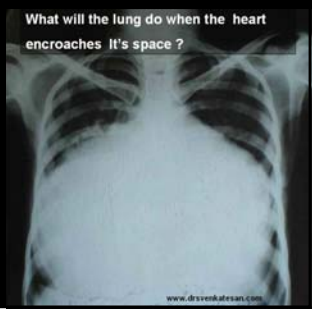
Nor-epi adds to afterload from atherosclerosis

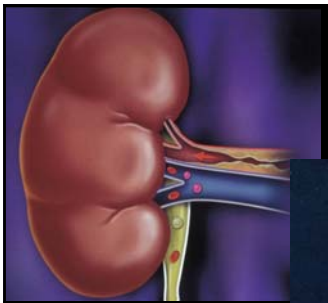
LV must work harder to overcome afterload & pump blood to systemic circulation

↑ workload = O_2 demand

⇒ enlarged heart

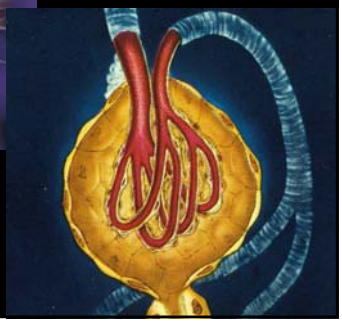
Heart becomes stiffer & more rigid, ↓ elasticity

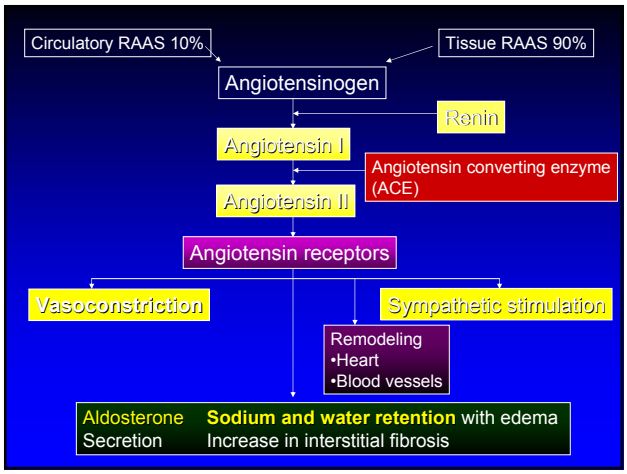




Kidneys sense drop in perfusion

Secrete renin…

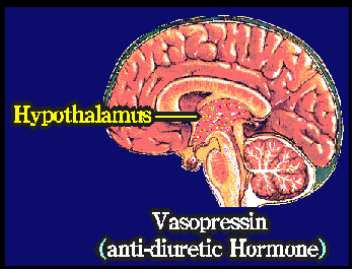




Secondary compensatory mechanisms

Endothelin from vascular lining results in vasoconstriction – helps perpetuate failure

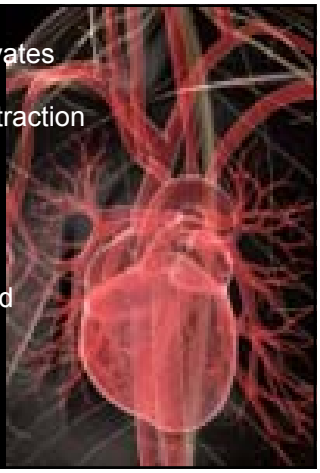
ADH acts on renal collecting ducts to hold water and vasoconstrict pt



Increased preload activates Starling's law to create greater contraction

RIGHT heart receives blood from body & pumps it into lungs

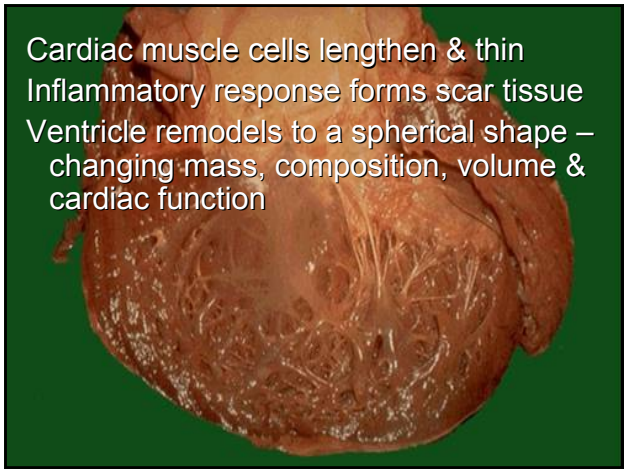
Over time, ventricles become overstretched & weak (chronically overinflated balloon)



Cardiac muscle cells lengthen & thin

Inflammatory response forms scar tissue

Ventricle remodels to a spherical shape – changing mass, composition, volume & cardiac function



Fluid dynamics in LV HF

Ventricular filling &/or emptying is impaired

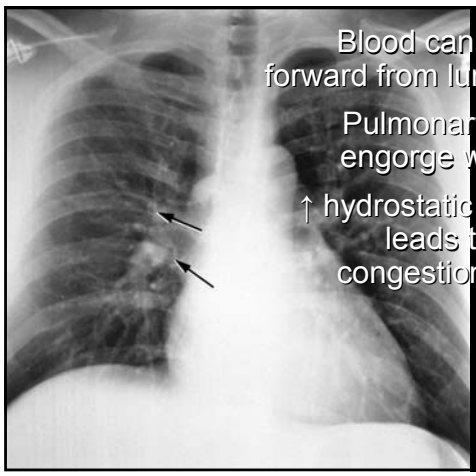
↑ blood remains in LV after systole

LV & LA overfill until they cannot hold more

Blood cannot move forward from lungs to LV

Pulmonary vessels engorge with blood

↑ hydrostatic pressure leads to venous congestion in lungs

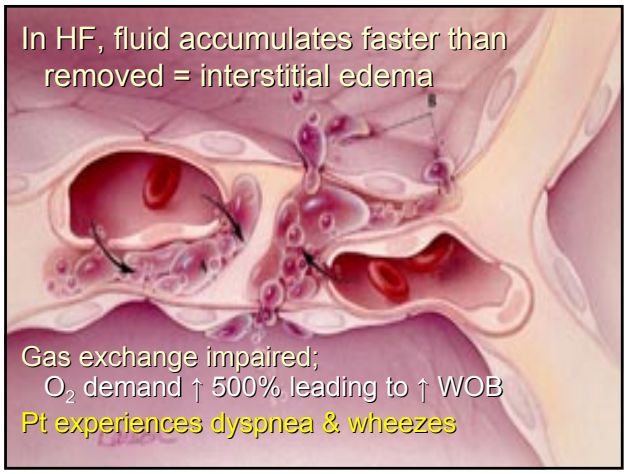


End result –
Pressure problem in lungs

Pressure in vessels > pressure in tissues → fluid leaks to interstitial spaces

Bronchovascular cuffs can hold ~500 mL

Lymph system can remove 10-20 mL/ hr in healthy lung; under stress, can remove more through ↑ flow



In HF, fluid accumulates faster than removed = interstitial edema

Gas exchange impaired;
O_2 demand ↑ 500% leading to ↑ WOB

Pt experiences dyspnea & wheezes

Then…

Alveolar walls damaged

Pressure in tissues > pressure in alveoli

Fluid floods into alveoli, washes out surfactant = atelectasis & alveolar pulmonary edema

Alveoli only open with considerable ventilatory effort

Gas exchange impaired, **crackles & hypoxia** develop

Basic problems…

Too much fluid (pressure) in lung vessels and tissues

Loss of surfactant, atelectasis & alveolar flooding

Impaired ventilations & gas exchange

↑ myocardial workload
↑ O_2 demand

How may they present?

Tachypnea w/ ↑ WOB

Accessory muscle use;
✓ for retractions

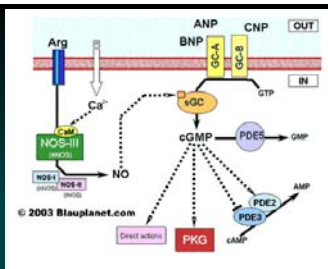
✓position (tripod?)
Orthopnea, PND, freq. nocturia: fluid returns to lungs & kidneys

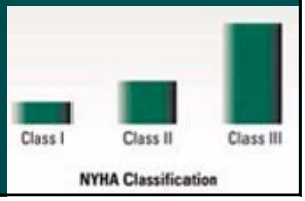
Prolonged expiration

Breathing w/ pursed lips – own PEEP

BNP

Released from damaged atria and ventricles, causes excessive neuroendocrine stimulation – vasodilates pt





Need more info?

Assess for hypoperfusion & cardiorespiratory compromise

Differentiate HF from COPD/ asthma by:

- History
- Meds
- Capnography
- S&S

COPD

Chronic Obstructive Pulmonary Disease?

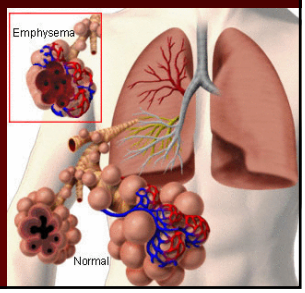
So, could it be his

Emphysema

Destruction of alveolar walls (distal to terminal bronchioles) & pulmonary capillaries

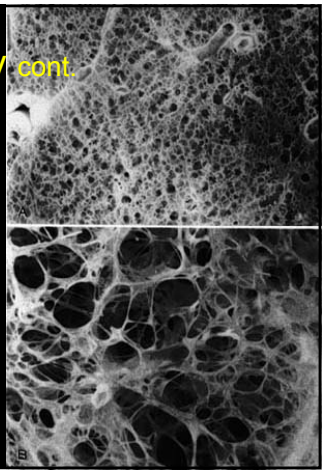
Fundamental problems:

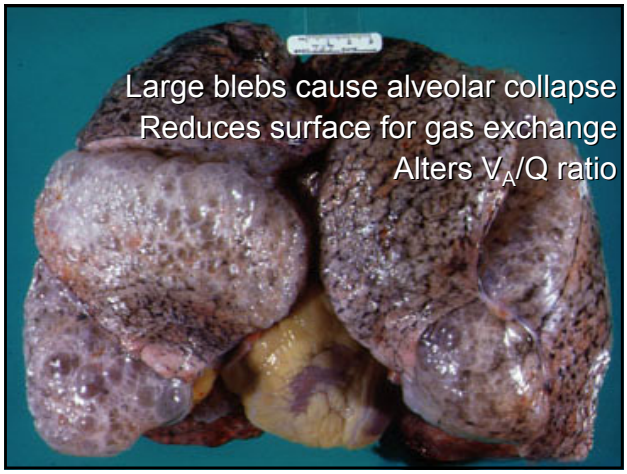
- Loss of elastic recoil causes bronchioles to collapse on expiration
- Decreased ability to oxygenate blood



Pathophysiology cont.

- Lungs become more compliant (stretchy) and distend
- Alveolar walls enlarge and decrease in #





Large blebs cause alveolar collapse
Reduces surface for gas exchange
Alters V_A/Q ratio

Pathophysiology cont.

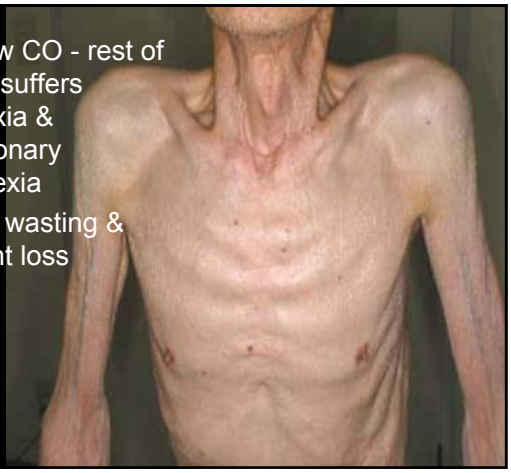
- Compensates w/ ↓CO & hyperventilation
- Limited blood flow thru relatively well oxygenated lung w/ normal ABGs & lung pressures

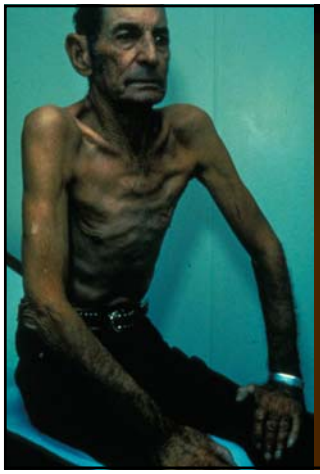
- As disease progresses, O_2 levels fall
- Stimulates RBC production (*polycythemia*)
- Better ABGs = **"Pink puffer"**

Pink Puffer - emphysema

- BP: Pulsus paradoxus
- P: Tachycardia
- RR: Tachypnea
  1-2 word dyspnea
- Increased WOB
- Pursed lip breathing
- Little/no cough

- With low CO - rest of body suffers hypoxia & pulmonary cachexia
- Muscle wasting & weight loss

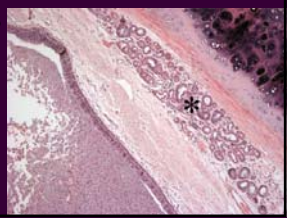




- Tripod position
- Chest:
- Barrel shape
- Hyperresonant; wheezing; HS distant
- Suprasternal retractions

Chronic bronchitis

- Mucous glands enlarge
- Airway walls infiltrated with inflammatory cells
- Bronchi thicken & become rigid (vasodilation, congestion, edema)
- Cilia don't clear bacteria & mucous
- Inflammation & secretions cause obstruction
- Relatively undamaged pulmonary capillary bed

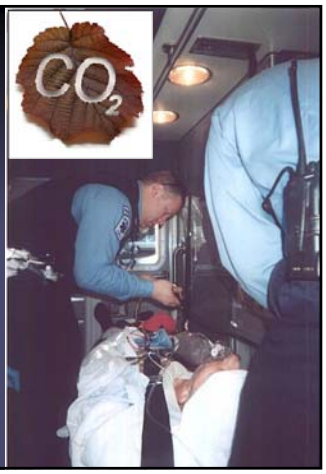


Pathophysiology

Body responds by
↓ventilation & ↑ CO

Rapid circulation in a poorly ventilated lung = hypoxemia, CO_2 retention

↑ pCO_2 causes AMS, headaches, and personality changes

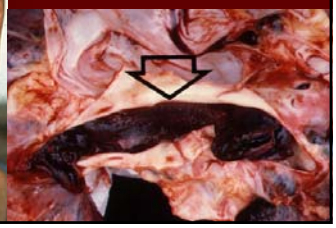


What form of COPD is more likely to develop Cor Pulmonale?

Emphysema or
Chronic Bronchitis

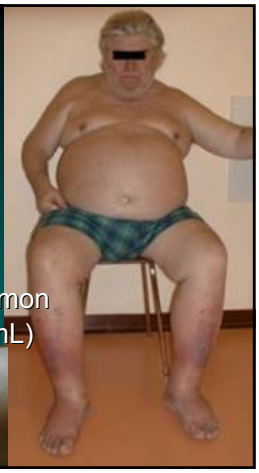
Causes of Cor Pulmonale

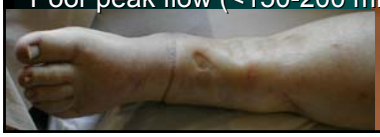
Increased RV strain due to pulm HTN
(Pulmonary embolism)
RV hypertrophy, venous congestion

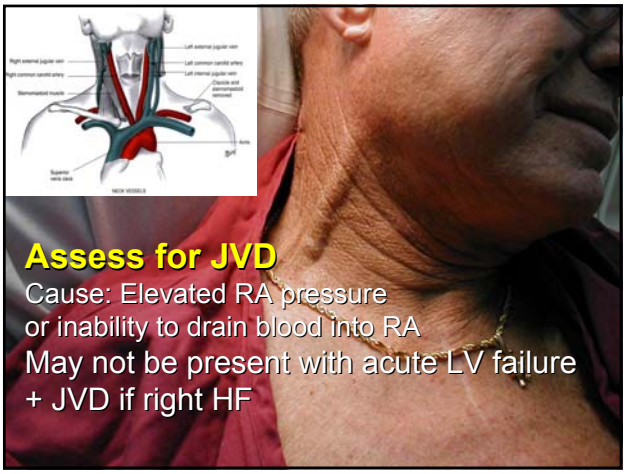


"Blue bloater"
Chronic bronchitis

Peripheral edema?
Cor Pulmonale, HF, renal failure, metastatic cancer
Cyanosis
May be obese
Accessory muscle use common
Poor peak flow (<150-200 mL)







Assess for JVD

Cause: Elevated RA pressure
or inability to drain blood into RA
May not be present with acute LV failure
+ JVD if right HF

COPD comparisons

Chronic bronchitis "Blue bloater"	Emphysema "Pink puffer"
Productive cough	Long hx of progressive dyspnea
Progression over time to intermittent dyspnea	Late onset non-productive cough
Frequent/recurrent pulmonary infections	Occasional mucopurulent relapses
Progressive cardiac/ respiratory failure w/ edema & weight gain	Eventual weight loss & respiratory failure

Acute hypoxic ventilatory failure

SpO_2 < 92 w/ FiO_2 > 60%
Restlessness, anxiety
Lightheaded
Disorientated, confused

Decision tool if patient is wheezing

PMH

Cardiac	Pulmonary
□ CVD: HTN; ACS; HF □ Stroke or TIA □ Dysrhythmias □ PVD □ Valve disease □ Diabetes; renal dx □ Drug abuse □ No Hx resp problem □ + cardiac risk factors	□ Asthma/COPD □ Pulmonary embolus risk factors □ Pneumothorax □ Pleural effusion □ TB, lung cancer □ Smoking; inhalation exposure

Decision tool: Adult wheezing

Clinical presentation

Cardiac	Pulmonary
□ Pain: non-pleuritic □ Cough: frothy □ DOE □ Orthopnea; PND □ Freq. nocturia	□ Pain: may be pleuritic □ Cough: mucoid, yellow, green □ Chills, fever, night sweats

Upright, (tripod?) position
Accessory muscle use; retractions
Breathing w/ pursed lips – own PEEP
Right heart failure?

Decision tool: Adult wheezing

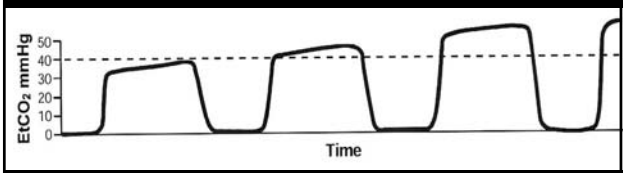
Vital signs

Cardiac	Pulmonary
Hyper/hypodynamic state Pulse deficits if fast HR or ectopics Weak pulse w/ hypotension	BP WNL unless very hypoxic / dehydrated

Tachypnea w/ ↑ WOB
↓SpO_2; evidence of hypoxia
↑$EtCO_2$; hypercarbia; shape of waveform
Pulsus paradoxus if air trapping/real trouble

Here's our patient

$PaCO_2$ > 50 mmHg at FiO_2 > 50%



Consider possible causes of S&S

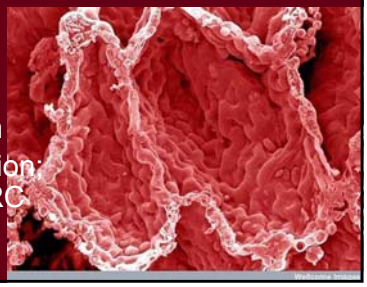
Stridor: F/B aspiration, croup, epiglottitis
Wheezing: Asthma/COPD, HF, ACS
Crackles: pneumonia (isolated), HF (diffuse)
Hypotension: Shock - rate, volume, vessel, pump or rhythm problem
Urticaria: Infection, anaphylaxis
Unconscious: AEIOU-TIPS; vasovagal syncope

Meld theory to practice: Main treatment goals

- ↓ cardiac workload
- ↓ fluid pressure in pulmonary vessels
- ↓ lung water
- ↓ WOB
- ↓ O_2 demands

Keep alveoli open

- ↑ O_2 & CO_2 diffusion; ↑ pulse ox; ↑ FRC
- ↑ cardiac output



If ventilatory effort is good, how should O_2 be applied *first* if both are available?

CPAP *IS* their oxygen delivery device

Why is the EMS goal to **avoid** intubating a patient in HF or with COPD/severe asthma?

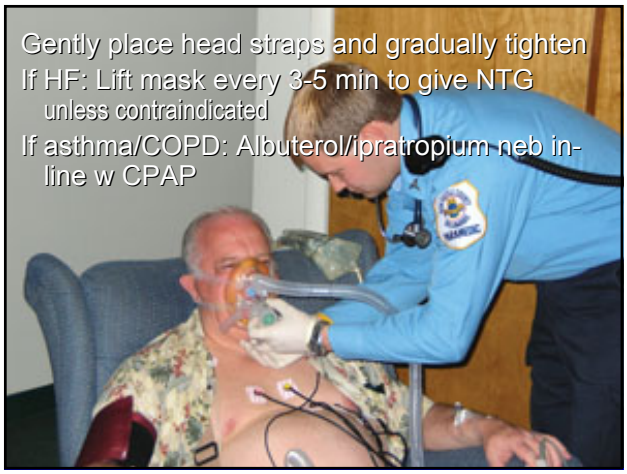
A. Intubated pts cannot be given PEEP
B. To avoid barotrauma, over sedation & infection
C. EMS personnel can rarely place the tube correctly
D. They are more likely to go into cardiac arrest after intubation

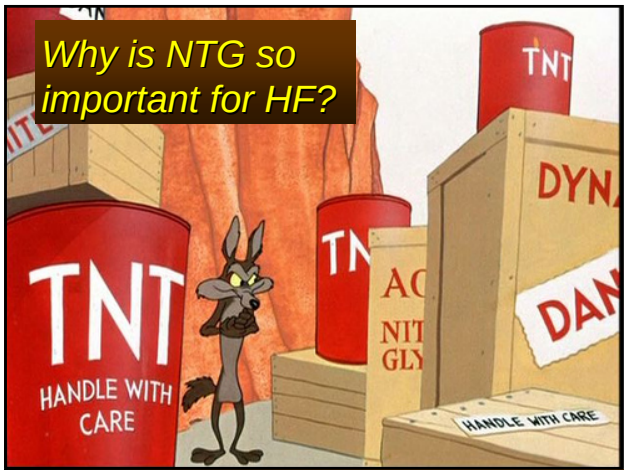
Vascular access needed?

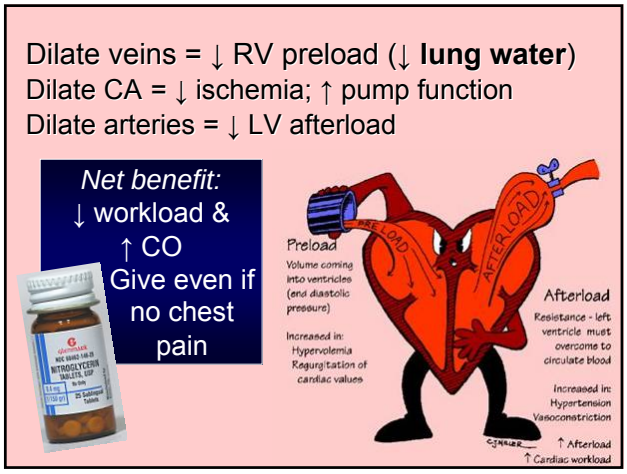
Must decide which meds

While prepping CPAP equipment, if suspect HF, quickly give ASA

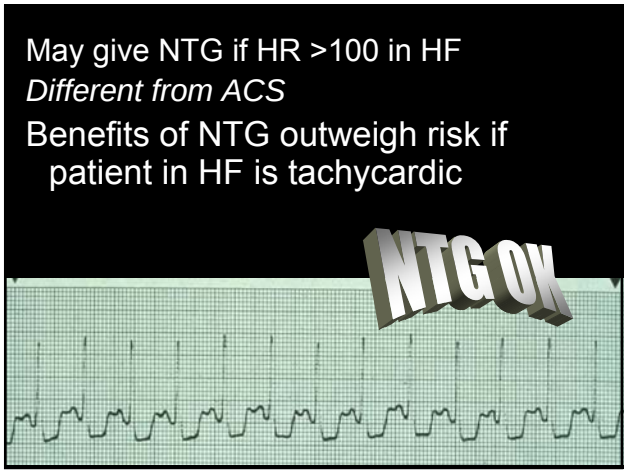
- **ASPIRIN 324 mg** (4 tabs 81 mg) PO unless contraindicated
- AMI cause of acute HF
- HF pts at ↑ risk for thromboembolic events
- AF promotes stasis & ↑ risk of thrombus formation
- May give small sip of water to swallow ASA prior to NTG

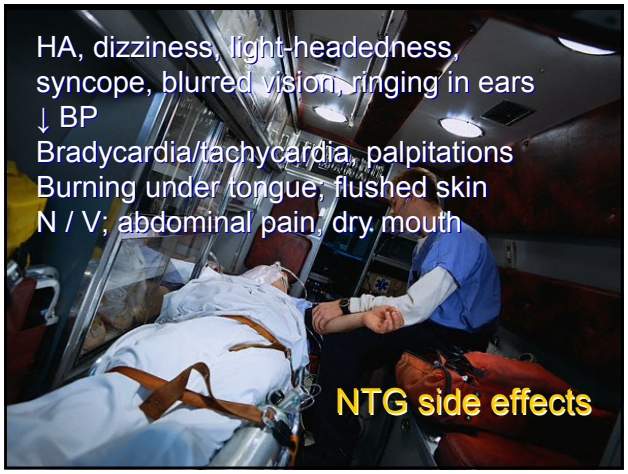

Gently place head straps and gradually tighten
If HF: Lift mask every 3-5 min to give NTG unless contraindicated
If asthma/COPD: Albuterol/ipratropium neb in-line w CPAP


Why is NTG so important for HF?
TNT
HANDLE WITH CARE
HANDLE WITH CARE

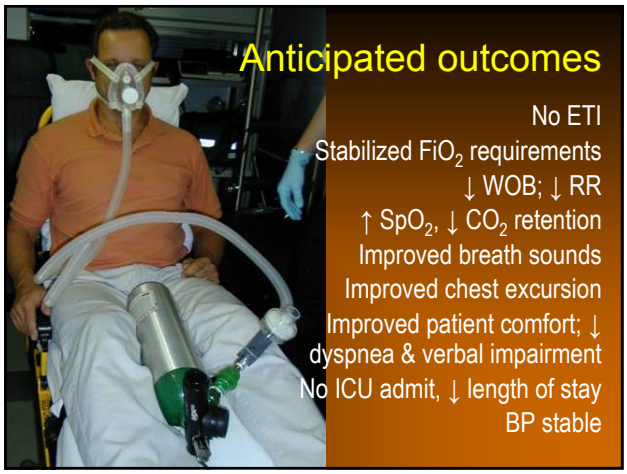

Dilate veins = ↓ RV preload (↓ lung water)
Dilate CA = ↓ ischemia; ↑ pump function
Dilate arteries = ↓ LV afterload
Net benefit:
↓ workload &
↑ CO
Give even if no chest pain
Preload
Volume coming into ventricles (end diastolic pressure)
Increased in: Hypervolemia Regurgitation of cardiac values
Afterload
Resistance - left ventricle must overcome to circulate blood
Increased in: Hypertension Vasoconstriction
↑ Afterload
↑ Cardiac workload


Adequate NTG critical
NTG 0.4 mg SL – give 1st dose right before CPAP applied
Onset 1-3 min; half life 5 min
If SBP ≥ 90: Repeat NTG q. 3-5 min
NO dose limit -
Need continuous action to ↓ pulmonary congestion & maintain other benefits
Lift CPAP mask to give more
Nitrostat

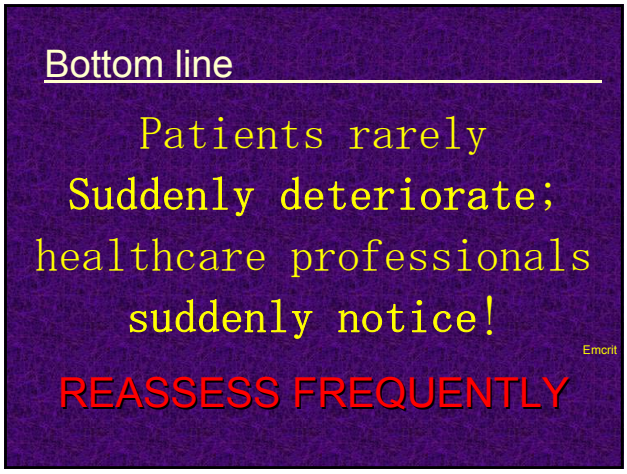

May give NTG if HR >100 in HF
Different from ACS
Benefits of NTG outweigh risk if patient in HF is tachycardic
NTG OK


HA, dizziness, light-headedness, syncope, blurred vision, ringing in ears
↓ BP
Bradycardia/tachycardia, palpitations
Burning under tongue, flushed skin
N / V; abdominal pain, dry mouth
NTG side effects


What about furosemide?
No longer indicated for acute pulmonary edema


Anticipated outcomes
No ETI
Stabilized FiO_2 requirements
↓ WOB; ↓ RR
↑ SpO_2, ↓ CO_2 retention
Improved breath sounds
Improved chest excursion
Improved patient comfort; ↓ dyspnea & verbal impairment
No ICU admit, ↓ length of stay
BP stable


Patient really needs to be in a cardiac cath lab!


Bottom line
Patients rarely
Suddenly deteriorate;
healthcare professionals
suddenly notice!
Emcrit
REASSESS FREQUENTLY


So we need an action plan…
Predetermine a course of action based on best practice models
Always put patients first
Work as a team
Head into action
Expect problems; solve them as they come
Always point to success (but learn from our failures)
Daily, give thanks for the privilege of serving those who need our care & expertise.