Weak, winded & woozy - what's wrong?

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

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

1. 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.

2. Compare and contrast pts who present w/ dyspnea, weakness, & possible AMS.

3. 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 sounds
- 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

Inspection cont.

Diaphragmatic, See Saw, Apical breathing? Splinting?

Speech: talk test

Sentences or syllables?
Pacing of speech and breathing
Quality of voice; hoarse or raspy?
Stuttering

Breathing w/pursed lips
Own PEEP

Origins of ventilation dysfunction

Airway impairment
Chest wall impairment
Weak, winded & woozy -
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Origins of ventilation dysfunction

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

Muscles:
Myasthenia Gravis, muscular dystrophy

Ventilatory depression or arrest
Nerves: Polio, Guillain-Barré syndrome, MS

Breathing considerations in obese pts
Lungs 35% less compliant
Weight of chest makes breathing difficult —
ventilate at 8-10 mL/kg
SpO₂ unreliable on finger —
use central sensor
Will desaturate if supine
CO₂ retention probable
CPAP useful

Factors that impair diffusion
Thickening of alveolar walls
Destruction or collapse of alveoli
↓ permeability
Widened interspace
↓ in blood flow
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**Atmospheric deficiency**
- Asbestosis
- COPD
- Inhalation injuries
- Pneumonia

**Alveolar pathology**
- Asbestosis
- COPD
- Inhalation injuries
- Pneumonia

**Interstitial pathology**
- High pressures (HF)
- Pulmonary hypertension

**Conditions w/ impaired diffusion**

**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**

- Capnography: Numeric reading + waveform
- Pulse oximetry
- Non-invasive BP after 1 manual reading
- ECG rhythm + 12 lead
What’s the difference between capnography & pulse oximetry?

What does pulse ox measure?
A. Mean arterial pressure  
B. Level of CO₂ in the blood  
C. Amount of O₂ dissolved in plasma  
D. % of hemoglobin bound with a gas

Problem: Many pts have unrecognized hypoxia by physical exam alone

SpO₂ artificially elevates when hyperventilating
For each 1 Torr pCO₂, the pO₂ 1 by 1 Torr
Hypoxia common in HF & COPD; means severe distress in asthma

Pulse ox is NOT the same as the pO₂!
If SpO₂ is 90%, the pO₂ is…

P90: SpO₂ 90 = pO₂ 60
P75: SpO₂ 75 = pO₂ 40
P50: SpO₂ 50 = pO₂ 27

Pulse oximetry range guidelines
Ideal: 96%-100%
Mild-mod hypoxemia: 90%-95%
Severe hypoxia: < 90%

Severely low SpO₂ (< 90%) predictor of poor outcomes

Use the right tool the right way!

If low, validate on another site - use a central sensor
The affinity of hemoglobin for O\textsubscript{2} is altered by conditions in the tissue the blood is flowing through...

Which of these will influence the amount of O\textsubscript{2} delivered to cells?
A. Acid-base status
B. Body temperature
C. The amount of hemoglobin
D. All of the above

The affinity of hemoglobin for O\textsubscript{2} is altered by conditions in the tissue the blood is flowing through...

Capnography
Indicates adequacy of ventilations, perfusion, & dead space by detecting how much CO\textsubscript{2} is exhaled
Gives a numeric value & graphic waveform

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

Asthma or COPD
Capnography: Incomplete inhalation/exhalation; CO\textsubscript{2} does not get completely washed out on inhalation

Capnography findings in HF
After CPAP started, ETCO\textsubscript{2} may briefly rise d/t improved ventilations, before it falls due to tachypnea
Severely ↑ ETCO\textsubscript{2} indicates ↑ pCO\textsubscript{2} levels and ventilatory failure
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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
- EtCO₂ > 45; change in waveform

**O₂ 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 hyperoxia and need careful titration of oxygen?**

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

**Why?**

- Harmed by **hyperoxia**
- Give O₂ to these pts only if evidence of hypoxia and titrate to dose that relieves hypoxemia without causing hyperoxia (SpO₂ 94%)
So, who NEEDS $O_2$?  
$SpO_2 < 94\%$ (Exception: COPD may be best managed w/ $SpO_2 88-92\%$)

Actual or potential airway compromise  
(spigletitis, 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
$\uparrow$ WOB ($\uparrow$ inspiratory muscle work)
$\downarrow$ Minute ventilation
Inability to remove $CO_2$ from body
Hypercarbic ventilatory failure
Narcotic effect on brain $\rightarrow$ $\downarrow$ RR
Fatigue + $\downarrow$ 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 ($Q$)

$Q = SV \times HR$

$Q = 70$ mL $\times 72$ BPM
$Q = 5,040$ mL/min

$VA/Q$ ratios

Should be 1:1
$VA = 5,250$ mL/min
$Q = 5,040$ mL/min
Patients may become hypoxic when ratio is imbalanced
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Circulatory/Cardiac status

✓ perfusion: general pulse rate, quality, rhythmicity
Establish that underlying cause of respiratory difficulty is not cardiac in nature

Perfusion impairment

Inadequate blood volume
Inadequate hemoglobin: anemia, trauma
Impaired blood flow: pulmonary embolus
Capillary wall pathology: pulmonary contusion

Monitor ECG

Bradydystonia is bad!

Why is AF common in HF?

Why is it a concern?

12-lead IF:

Discomfort (hose to navel, shoulder, arm, back)
SOB/HF
Palpitations; dysrhythmia (VT/STV)
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.

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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

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- 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?

Coughing?

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Cough differential

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

Sputum quantity, color, consistency, odor

NOTE

Clear
Yellow/Green
Rusty/bloody

Pneumonia
Chronic bronchitis

Frothy sputum due to pulmonary edema (pink-tinged means surfactant is washing out)
Quickly look for S&S LV failure (S3 heart sound)
Weak, winded & woozy -
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Allergies to food, meds, plants, pets, bites, stings, environmental triggers, exercise or others?

What are all those drugs they are taking?

Meds

EpiPen prescribed?
Taken anything to relieve symptoms?

Have they taken a NEW medication?

Meds – ACE Inhibitors (ACEI)
Generic name ends in “pril”
Blocks creation of angiotensin II:
Vasodilates pt, ↓ BP, prevents remodeling and ↓ the heart’s workload

Benzapril / Lotensin
Captopril / Capoten
Enalapril / Vasotec
Fosinopril / monopril
Lisinopril / Prinivil / Zestril

Moesipril / Univasc
Perindopril / Aceon
Quinapril / Accupril
Ramipril / Altace
Trandolapril / Mavik

Medications

Cardiac

- ACEIs: “prils”
- ARBs: “sartans”
- Beta blockers: “lols”
- Ca Blockers
- Diuretics
- Vasodilators
- Anticoagulants (AF)
- Antiarrhythmics
- Digoxin

Pulmonary

- Short/long-acting beta agonists
- Anticholinergics
- Mast cell inhibitors
- Leukotriene modifiers
- Steroids
- MethyIxanthines
- Erectile dysf. drugs
- Home oxygen

Time and amount of last dose

What can block the SNS?

Cardio-selective drugs
B-1 blockers: end in “lol”
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)
Must have significant disease before asymmetry of expansion can be identified on exam.

Point tenderness: Investigate painful areas
Deformity, instability

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

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

How to listen?

Technique: Where should you listen?

All lobes, front and back

Front

Back

RUL

LLL

RLL

RML

LUL

Where to start?

Auscultatory triangle in back
Medial/lower scapula; less muscle mass; easy to hear sounds
Lower diaphragm attachment in back; fluid accumulates there first
Move up from posterior bases; 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 with pulmonary edema
- Poorly ventilated areas of atelectasis
- Localized over early or non-consolidating pneumonia
- Pulmonary fibrosis
- Tubercular lung cavities
- Lung abscess
- Terminally ill with depressed cough reflex

Harmonic, musical sounds produced by turbulence when air passes through bronchi that fluctuate between closed & barely open

Wheeze

Describe according to location, pitch, duration, timing, complexity
- Sibilant (asthma/emphysema)
- Sonorous (formerly called rhonchi)
- Louder/longer on expiration
- Severity does not correlate well with degree of airway obstruction – assess capnography
- Wheezes dissipate with bronchodilator therapy
- No wheezes 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
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**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**

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

**Stridor**

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

**Decreased or absent sounds**

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%

**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

**EXTRA HEART SOUNDS - S3**

S1, S3
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;
SpO₂ 84% on RA
Glucose: 145
12 L ECG as follows:

Look OK right now?
Weak, winded & woozy -
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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

Heart Failure

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
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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

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
<table>
<thead>
<tr>
<th></th>
<th>α</th>
<th>β</th>
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<tbody>
<tr>
<td>Heart</td>
<td>Constricts</td>
<td>Dilates</td>
</tr>
<tr>
<td>Lungs</td>
<td>Constricts</td>
<td>Dilates</td>
</tr>
<tr>
<td>Vessels</td>
<td>Constricts</td>
<td>Dilates</td>
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</table>

↑ rate, force, automaticity, conduction

Fight or flight response
Goal: Provide energy, O₂, & 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 = O₂ demand
enlarged heart
Heart becomes stiffer & more rigid,
↓ elasticity

Kidneys sense drop in perfusion
Secrete renin...
Weak, winded & woozy -  
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**Circulatory RAAS 10%**
- **Angiotensinogen**
- **Angiotensin I**
- **Angiotensin II**
- **Renin**
- **Angiotensin converting enzyme (ACE)**
- **Angiotensin receptors**
- **Vasoconstriction**
- **Remodeling**
- **Increased preload activates Starling’s law**
  - to create greater contraction
- **Ventricular filling &/or emptying is impaired**
  - ↑ blood remains in LV after systole
  - LV & LA overfill until they cannot hold more
- **Cardiac muscle cells lengthen & thin**
  - Inflammatory response forms scar tissue
  - Ventricle remodels to a spherical shape – changing mass, composition, volume & cardiac function

**Tissue RAAS 90%**
- **Aldosterone**
- **Sodium and water retention with edema**
  - ↑ in interstitial fibrosis
- **Secondary compensatory mechanisms**
  - Endothelin from vascular lining results in vasoconstriction – helps perpetuate failure
  - **ADH** acts on renal collecting ducts to hold water and vasostrict pt
  - **Hypothalamus**
    - **Vasopressin** (anti-diuretic hormone)
  - **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)

**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
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: O₂ 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
  - ↑ O₂ 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
Assess for hypoperfusion & cardiorespiratory compromise
Differentiate HF from COPD/asthma by:
History
Meds
Capnography
S&S

Chronic Obstructive Pulmonary Disease?
So, could it be his Emphysema

C O P D
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
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
Compensates with ↓CO & hyperventilation
Limited blood flow thru relatively well oxygenated lung w/ normal ABGs & lung pressures.

As disease progresses, O₂ levels fall
Stimulates RBC production (polycythemia)
Better ABGs = “Pink puffer”

Pathophysiology cont.

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

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

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

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

Muscle wasting & weight loss
Weak, winded & woozy -
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Pathophysiology

Body responds by ↓ ventilation & ↑ CO
Rapid circulation in a poorly ventilated lung = hypoxemia, CO₂ retention
↑ pCO₂ 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

<table>
<thead>
<tr>
<th>Chronic bronchitis “Blue bloater”</th>
<th>Emphysema “Pink puffer”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Productive cough</td>
<td>Long hx of progressive dyspnea</td>
</tr>
<tr>
<td>Progression over time to intermittent dyspnea</td>
<td>Late onset non-productive cough</td>
</tr>
<tr>
<td>Frequent/recurrent pulmonary infections</td>
<td>Occasional mucopurulent relapses</td>
</tr>
<tr>
<td>Progressive cardiac/respiratory failure w/ edema &amp; weight gain</td>
<td>Eventual weight loss &amp; respiratory failure</td>
</tr>
</tbody>
</table>
**Acute hypoxic ventilatory failure**

- SpO$_2$ < 92 w/ FiO$_2$ > 60%
- Restlessness, anxiety
- Lightheaded
- Disoriented, confused

---

**Decision tool if patient is wheezing**

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

---

**Decision tool: Adult wheezing**

**Clinical presentation**

<table>
<thead>
<tr>
<th>Cardiac</th>
<th>Pulmonary</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Pain: non-pleuritic</td>
<td>□ Pain: may be pleuritic</td>
</tr>
<tr>
<td>□ Cough: frothy</td>
<td>□ Cough: mucoid, yellow, green</td>
</tr>
<tr>
<td>□ DOE</td>
<td>□ Chills, fever, night sweats</td>
</tr>
<tr>
<td>□ Orthopnea; PND</td>
<td></td>
</tr>
<tr>
<td>□ Freq. nocturia</td>
<td></td>
</tr>
</tbody>
</table>

**Upright, (tripod?) position**

- Accessory muscle use; retractions
- Breathing w/ pursed lips – own PEEP
- Right heart failure?

---

**Decision tool: Adult wheezing**

**Vital signs**

<table>
<thead>
<tr>
<th>Cardiac</th>
<th>Pulmonary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyper/hypodynamic state</td>
<td>BP WNL unless very hypoxic / dehydrated</td>
</tr>
<tr>
<td>Pulse deficits if fast HR or ectopics</td>
<td></td>
</tr>
<tr>
<td>Weak pulse w/ hypotension</td>
<td></td>
</tr>
</tbody>
</table>

**Upright, (tripod?) position**

- Accessory muscle use; retractions
- Breathing w/ pursed lips – own PEEP
- Right heart failure?

---

**Here’s our patient**

- PaCO$_2$ > 50 mmHg at FiO$_2$ > 50%

---

**Consider possible causes of S&S**

- **Stridor:** F/B aspiration, croup, epiglottitis
- **Wheeze:** 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
↓ O₂ demands
Keep alveoli open
↑ O₂ & CO₂ diffusion;
↑ pulse ox; ↑ FRC
↑ cardiac output

If ventilatory effort is good, how should O₂ be applied  \textit{first} if both are available?

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

\textbf{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?

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

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.

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

NTG side effects:

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.
Weak, winded & woozy -
Connie J. Mattera MS, RN, PM

**What about furosemide?**

No longer indicated for acute pulmonary edema

**Anticipated outcomes**

- No ETI
- Stabilized FiO₂ requirements
- ↓ WOB; ↓ RR
- ↑ Sp⁰₂; ↓ CO₂ retention
- Improved breath sounds
- Improved chest excursion
- Improved patient comfort; ↓ dyspnea & verbal impairment
- No ICU admit, ↓ length of stay
- BP stable

**Bottom line**

Patients rarely suddenly deteriorate; healthcare professionals suddenly notice!

**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.