Northwest Community EMS System
Continuing Education: January 2012

RESPIRATORY ASSESSMENT
Independent Study Materials

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COGNITIVE OBJECTIVES

Upon completion of the class, independent study materials and post-test question bank, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Integrate complex knowledge of pulmonary anatomy, physiology, & pathophysiology to sequence the steps of an organized physical exam using four maneuvers of assessment (inspection, palpation, percussion, and auscultation) and appropriate technique for patients of all ages. (National EMS Education Standards)
2. 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. (National EMS Education Standards)
3. Describe the signs and symptoms of compromised ventilations/inadequate gas exchange.
4. Recognize the three immediate life-threatening thoracic injuries that must be detected and resuscitated during the “B” portion of the primary assessment.
5. Explain the difference between pulse oximetry and capnography monitoring and the type of information that can be obtained from each of them.
6. Compare and contrast those patients who need supplemental oxygen and those that would be harmed by hyperoxia, giving an explanation of the risks associated with each.
7. Select the correct oxygen delivery device and liter flow to support ventilations and oxygenation in a patient with ventilatory distress, impaired gas exchange or ineffective breathing patterns including those patients who benefit from CPAP.
8. Explain the components to obtain when assessing a patient history using SAMPLE and OPQRST.
9. Compare and contrast the past medical history, risk factors, medications generally prescribed, and clinical presentations for patients with a cardiovascular vs. respiratory condition.
10. Describe the techniques of a thought pulmonary assessment.
11. Explain the physiologic etiology of normal, abnormal, and adventitious breath sounds.
12. Recognize normal and abnormal breath sounds including the adventitious sounds of crackles, wheezes, pleural friction rub and stridor.

PSYCHOMOTOR OBJECTIVE

1. Compare and contrast the intensity of breath sounds obtained when a person breathes through their nose as opposed to breathing through the mouth.

AFFECTIVE OBJECTIVE

1. Defend the need to complete thorough histories and physical exams on patients as indicated or permitted to reach an appropriate EMS impression that directs initial medical care.

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I. **Action strategy overview**

A. Regardless of the patient's problems, a **systematic approach** should be followed in assessing all patients. What varies somewhat are the priorities and time frames within which certain procedures must be done.

B. The prioritized steps in this presentation are sequenced in the order of their importance for purposes of clarity; however, some are frequently accomplished simultaneously. The importance of obtaining a thorough history and analysis of mechanism of injury/illness is paramount. Obtaining patient and event histories may account for 90% of the prehospital impression/diagnosis. If this step is abbreviated or omitted, major illnesses or injuries may be missed or pre-existing medical diseases that may compromise the patient's outcome or responses may be overlooked.

C. While some parts of a PE are universal, the specialized portions of an exam are tailored to each patient's situation. Always consider and rule-out the worst possible scenario based on the patient's age, sex, and presenting complaint. "The eye does not see and the hand does not feel what the mind does not think of". Consider that all patients have a life-threatening event in process until it is ruled out. Build mental checklists of possible diagnoses and rule each in or out in your own mind as you proceed.

II. **Scene size up: PENMAN:** Simple acronym that may be used for the initial size-up of an incident. It reminds the first arriving incident commander of the primary scene priority: safety. It can also be used during the event the parameters of the incident evolve (Miller, 2011).

A. **(P)** Personal and personnel safety

B. **(E)** Environmental hazards. An environmental hazard is anything that can reach out and hurt you; therefore, "E" also addresses safety.

C. **(N)** Number of victims

D. **(M)** Mechanism of injury or nature of illness.

E. **(A)** Additional resources from within the responder's own agency.

F. **(N)** Need for outside agencies

III. **Primary assessment** (Assessment should take less than 2 minutes if no impairments are found)

A. **Purpose:** Detect and resuscitate all clinically evident, immediate life threats. All physiological factors assessed during the initial assessment are of such a critical nature that major deviations from normal require immediate intervention. The initial assessment may be completed at a glance if the patient is alert, communicative and has minor distress, but each of the components must be included in the assessment.

B. **Appreciate the value of time**: Examine, reexamine. Catastrophic conditions may present with maximal symptoms immediately. Others take time to reveal their true nature. It is not always easy to determine at what stage the patient is in during the initial assessment. Use the passage of time as an ally and reevaluate patients frequently.

Form a general impression

C. **While walking up to the person, observe/inspect/do the following**:

1. Age, gender, developmental influences
2. How ill or injured does the patient appear?
3. **Level of consciousness**

a. ✓ for agitation, confusion, weakness

b. Changes in mental status suggest a host of problems that signal a need for immediate evaluation of the patient's airway, oxygenation, ventilation, perfusion, glucose and neurologic status.

c. Describe the patient's initial responses to stimuli. If patient appears to be sleeping or comatose, how easily can they be aroused?

(1) **A**: **A**lert
(2) **V**: Responds to **v**erbal stimulus
(3) **P**: Responds to **p**ainful stimulus; if no responses to a peripheral stimulus, check CNS site (pinch ear lobe)
(4) **U**: **U**nresponsive to any stimulus

d. **Causes of altered mental status**

(1) **A**: Alcohol and ingested drugs and toxins
(2) **E**: Endocrine/exocrine, particularly liver; electrolytes
(3) **I**: Insulin; hyper or hypoglycemia
(4) **O**: Oxygen, opiates, OD
(5) **U**: Uremia, renal causes including hypertensive problems
(6) **T**: Trauma, temperature changes
(7) **I**: Infections, both neurologic and systemic
(8) **P**: Psychiatric, poisons
(9) **S**: Space occupying lesions, stroke, subarachnoid bleed, shock, seizures

e. Quickly determine if an awake patient is oriented to person, place, time, and situation. Observe their ability to follow simple commands; quality and rapidity of their responses.

f. **General appearance, affect, behavior and cognition**: Restlessness is one of the earliest signs of hypoxemia and suggests the possibility of serious underlying problems.

4. **Observe the patient's position – Is there a position of comfort?**

a. **Sitting upright** - High Fowler's position will increase lung volumes and vital capacity, allow fluid to descend to the lung bases, and hopefully reduce work of breathing and open more lung surface area for diffusion in the upper lobes and decrease venous return to the heart.

b. **Tripod position** will help expand chest; affirms presence of muscle tone

c. **Orthopnea** (inability to lie flat): Indicates progression of dyspnea

D. **Rapidly assess pt for clinically evident immediate life-threats; resuscitate as found**

1. **A = Airway access/maintenance -** Impairment is a frequent cause of death

a. Obstruction may be acute, insidious, progressive or recurrent. Maintain a high index of suspicion.

b. **Anatomical airway distinctions**

(1) **Upper**: Obstruction tends to manifest by impaired inspiration
(2) **Lower**: Obstruction suggested by alterations in inspiration or expiration, but usually expiration first

c. **Inspect:** If responsive: are they crying or talking w/o difficulty or noise?

(1) YES → Assess breathing
(2) NO → Continue to assess airway

d. **Etiologies of airway obstruction**

(1) Tongue: Most common cause of obstruction with AMS
(2) Secretions, blood, vomitus
(3) Foreign body, teeth, dentures, debris
(4) Inflammation, edema
(5) Tissue trauma: airway, facial, laryngeal, inhalation
(6) Laryngeal or bronchial spasm, tracheal compression
(7) Prolonged, severe, compression to chest
(8) Malpositioned oropharyngeal airway

e. **Airway inspection**

(1) Observe for spontaneous ventilatory efforts

(2) Face and neck for symmetry, wounds, edema, foreign bodies, secretions in the mouth, drooling. There should be no foreign matter visible in the upper airway.

(3) Look for classic signs of upper airway obstruction (hand to throat)

(4) Ventilations should be quiet without any harsh or added sounds. **Audible sounds heard without a stethoscope**

(a) Snoring; gurgling
(b) Hoarseness
(c) Stridor, choking sounds
(d) Wheezing - inspiratory or expiratory
(e) Crackles heard through mouth
(f) Expiratory grunting

f. **Signs and symptoms of airway impairment**

(1) Secretions/debris in airway
(2) Inspiratory/expiratory stridor, snoring, gurgling, grunting
(3) Restlessness, anxiety, dyspnea, unresponsiveness
(4) Apnea, agonal respirations
(5) Use of accessory muscles, nasal flaring/head bobbing in infants; unable to breathe without violent respiratory effort
(6) Substernal/intercostal retractions; tracheal tugging
(7) Rocking chest motion
(8) Evidence of hypoxia, hypercarbia; cyanosis or ashen discoloration of skin or mucous membranes
(9) Unable to speak/make sounds approp. for age; change in voice
(10) Faint or absent breath sounds

g. **Expected outcome: patent airway**

(1) Ventilations quiet without snoring, gurgling, stridor or retractions
(2) Patient speaks or makes appropriate sounds for age
(3) Chest rises and falls easily with spontaneous/assisted ventilations
(4) Foreign material not visible in upper airways

2. **B = Breathing/ventilatory status/adequacy of gas exchange**:

a. A patent airway does not ensure adequate ventilations or gas exchange; assess general respiratory rate, depth

b. **Causes of inadequate ventilations to consider**

(1) Airway obstruction (should be corrected by now)
(2) Chest wall injury (fractured ribs, open pneumothorax)
(3) CNS dysfunction (high SCI, stroke, head trauma, neuro diseases)
(4) Diaphragmatic injuries
(5) Parenchymal injuries (pulmonary contusion)
(6) Pleural collections (pneumo/hemothorax, effusion)

c. **Inspection** (Expose the chest)

(1) **Ventilatory attempts**: Present/absent. If present, are they generally fast or slow. Don't count actual rate yet. Assess ventilatory depth (should be at least 500 cc - a 40% decrease in tidal volume = a 60% decrease in inspired air exposed to alveoli and circulating blood)

(2) **Life-threatening injuries to find/resuscitate at this stage:**

(a) **Tension pneumothorax**
(b) **Open pneumothorax** (communicating pneumothorax)
(c) **Flail chest:**

(3) **Symmetry of chest expansion**: Assess for unequal expansion (hyperinflation), flail segments with paradoxical motion or unequal movements, and retractions. Air accumulating under pressure in the pleural space resulting in a tension pneumothorax causes collapse of the vena cavae and right atrium, a mediastinal shift, impaired venous return to the right heart and cardiovascular collapse. Can be due to trauma or medical causes.

S&S tension pneumo: **Severe dyspnea**, asymmetric chest expansion increased work of breathing, hypoxia/cyanosis, **unilateral absence of breath sounds**, ↓ **BP** with narrowed pulse pressure, **JVD**, anxiety/apprehension, hyper-resonance to percussion, distant heart sounds, resistance to BVM ventilations, tracheal deviation to unaffected side (late). If tension pneumothorax (must be hypotensive!): Needle decompress affected side.

S&S Flail chest: Two or more adjacent ribs fractured in two or more places create a freely moving chest wall segment. Lethal complication is a pulmonary contusion. Severe chest pain on inspiration (pleuritic), dyspnea, asymmetrical chest expansion (paradoxical movement), crepitus, shallow, rapid respirations, and cyanosis/persistent hypoxia.

Retractions: Try to determine etiology; support ventilations ASAP

(4) **Chest wall integrity**; inspect for wounds – blunt or penetrating. An open pneumothorax becomes an immediate life threat because air will preferentially flow through the wound in the chest wall rather than go through the tracheobronchial tree if size of wound approximates 2/3 the diameter of the trachea. Assess for frothy/bubbly blood at site, air heard leaking from wound, anxiety, air hunger, pain, inability to speak. Breath sounds may be absent bilaterally as negative intrathoracic pressures during inspiration cause movement of air in and out of the wound rather than through the tracheobronchial tree. Resuscitate when found by converting it to a closed pneumo with an occlusive dressing.

(5) **Work of breathing/muscles used to ventilate**. Use of accessory muscles indicates airway resistance or ventilatory distress and is a good clinical indicator of work of breathing. May need abdominal muscles to help exhale.

(6) **Diaphragmatic, See Saw, Apical breathing?** Diaphragmatic ventilations may be associated with a spinal cord injury – BELOW C5 – as C3-4-5 keep the diaphragm alive. Splinting may suggest a fractured rib.

(7) Breathing with **pursed lips**: patient is adding their own PEEP to keep distal airways open longer and prolong oxygen diffusion time. Helps keep a patient with COPD out of ventilatory failure. Consider need for CPAP.

(8) **Ability to talk**: Patient should speak or make age-appropriate sounds

(a) Length of sentences (word clusters/syllables). If short of breath ask questions that lend themselves to yes and no nods of the head and/or short answers.

(b) Quality of voice; hoarse or raspy?

(c) **Stuttering.** With any voice change, consider the possibility of an aortic disaster causing compression of the recurrent laryngeal nerve or clot with embolism to the brain with stuttering an expression of a possible stroke.

(9) **Chest/abdominal contour**: Abdominal distention may impair ventilations, especially in children. **Muscle insertion angle:** The diaphragm can only pull in the direction the fibers run. Gastric distention causes the angle to be more pronounced, causing dependent portions to pull inward when contracting. This causes the dome to elevate rather than flatten in a phenomenon known as **paradoxical ventilation**. This dysfunction also occurs when the diaphragm is paralyzed (high C-spine injury). It moves up with inspiration due to decreased intrathoracic pressure.

A barrel chest, seen in emphysema, pulls the diaphragm inward on inspiration so the chest contracts rather than expands.

(10) **Obese patients**

(a) **Pulmonary changes:** Lungs 35% less compliant; weight of chest makes breathing difficult – ↑ chest wall resistance, ↑ airway resistance, abnormal diaphragmatic position, ↑ upper airway resistance contribute to ↑ WOB

(b) Breath sounds may be difficult to hear

(c) **SpO_2 monitoring**: **Can desaturate quickly if supine** and be more difficult to monitor - consider use of earlobe (central) sensor to better detect perfusion; Expect SpO_2 of 88% – 92% on 6L O_2.

(d) O_2 by NRM or **CPAP** w/ PEEP 5 – 10 cm H_2O. Assist w/ BVM if hypoxia or hypercarbia persists. If ventilated: give a tidal volume (V_T) of 8 – 10 mL/kg of ideal body weight.

(e) **CO_2 retention probable** (46-52 mEq/L); monitor capnography

(11) **Assess gas exchange/evidence of hypoxia**

(a) **Causes of hypoxemia** to consider and rule out

(i) Atmospheric deficiency of O_2
(ii) Ventilatory deficiency: ↓ alveolar oxygen tension
(iii) Diffusion deficiency: ↓ central arterial O_2 content
(iv) Perfusion deficiency: systemic or regional
(v) Ventilation/perfusion mismatch
(vi) Hemoglobin/red blood cell (RBC) deficiency or abnormality (anemia, Sickle cell disease)
(vii) Cellular inability to use O_2: (cyanide poisoning)

(b) **Skin** (Correlate with other signs of hypoxia)

(i) Adequacy of oxygenation
(ii) Adequacy of peripheral perfusion
(iii) Evidence of SNS stimulation/compensation
(iv) Cool, hot, pale, flushed, mottled, ashen, cyanotic, & diaphoretic skin must be explored for cause

(v) **Factors that influence detection of cyanosis**

(a) Rate of blood flow
(b) Degree of desaturation (Need at least 5 Gm of desaturated hemoglobin for cyanosis to occur)
(c) Type of light
(d) Observer skill
(e) Thickness and color of skin

(vi) Cyanosis is divided into two categories

(a) **Central**: Lips, mucous membranes, conjunctiva

(b) **Peripheral**: nail beds

(vii) Skin color unreliable in the presence of anemia & peripheral vasoconstriction

(c) **Subjective (non-numeric) S&S of hypoxia**

(i) Restlessness, anxiety
(ii) Disorientation, confusion, combativeness
(iii) Dyspnea, air hunger, hypoventilation
(iv) Cyanosis

d. **Problem: Many have unrecognized hypoxia by physical exam alone**

If in distress: Apply **pulse oximetry** monitor immediately and observe the room air (RA) or baseline reading

(1) The SpO_2 artificially elevates when a patient is hyperventilating. For each 1 Torr pt ↓ pCO_2, the pO_2 ↑ by 1 Torr. Extremely low SpO_2 readings (< 90%) are a predictor of poor outcomes. Differentials: *Hypoxia common in HF & COPD; means severe distress in asthma.*

(2) When deciding which monitor to use – remember their function

(a) Pulse ox measures oxygenation
(b) $EtCO_2$ measures *ventilation, perfusion + host of other things…hold that thought until next month*

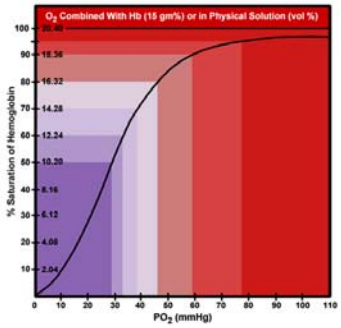
(3) **Blood gas PaO_2** is a function of the ventilation/perfusion ratio, the thickness of alveolar membranes, the *diffusion of oxygen in the lungs, and the adequacy of oxygen transport in the blood*. It is a measure of the amount of oxygen dissolved in the plasma.

(4) **SpO_2** provides information about O_2 transfer at the alveolar-capillary interface and the percentage of hemoglobin that is saturated with a gas in the RBCs. It provides an objective, non-invasive, continuous detection of pulsatile arterial blood in tissue beds and compares bound Hb as a ratio to the amount of free Hb available for binding to objectively detect hypoxia.

While it reports a HR, the SpO_2 monitor **IS NOT** a substitute for assessing the quality and rhythmicity of peripheral pulses. "Electronic vital signs" are not acceptable as baselines.

However, it can be used to detect perfusion distal to a limb injury when pulses are difficult to palpate.

(5) **It is important to remember that SpO_2 is NOT the same as the pO_2!** However, there is a correlation known as P values plotted on the oxyhemoglobin dissociation curve.



(a) **P90: SpO_2 90 = pO_2 60**
(b) P75: SpO_2 75 = pO_2 40
(c) P50: SpO_2 50 = pO_2 27

(6) **Pulse ox range guidelines**

(a) Ideal: 96%-100%
(b) Mild-mod hypoxemia: 90%-95%
(c) Severe hypoxia: < 90%

(d) Patients may be at risk when the PaO_2 drops to 60 or less (pulse ox < 90%) because the ability of hemoglobin to bind to oxygen becomes impaired.

(7) **Pulse ox limitations:** Function may be affected by the following:

(a) Motion artifact (shivering, tremors) – most common
(b) Ambient light, anemia
(c) Poor peripheral perfusion due to hypotension, vasoconstriction, age, hypothermia
(d) Electrical noise
(e) Dark skin color, nail polish

Unfortunately, no bedside device measures the oxygenation status of the tissues or cells. The lack of sensitivity must be offset by careful clinical correlation.

(8) **Use the right tool for the job**! Use an appropriate site and sensor (finger vs. central site). If low, validate on another site. If cold, tremoring, vasoconstricted, or poor peripheral perfusion - use a central sensor.

(9) The affinity of hemoglobin for O_2 is altered by conditions in the tissues the blood is flowing through. **Acid-base status, body temperature, and the amount of hemoglobin will all influence the amount of O_2 delivered to cells.** Thus, pulse ox readings need to be interpreted in context.

(a) In hot, acidotic tissues with high CO_2 levels (exercising muscle), the O_2 affinity to hemoglobin is poor (bonds are loose), allowing more oxygen to be offloaded to the tissues.

(b) In cold, alkalotic blood with low CO_2 levels or high carbon monoxide levels, the hemoglobin binds gasses more tightly but does not release it to tissues. Thus the pulse ox reading may be deceptively high.

(c) The pulse ox monitor does not distinguish which gas is bound to the hemoglobin. Patients with high carboxyhemoglobin levels may have a pulse ox reading of 98%, but be near death due to hypoxia.

e. **Quantitative waveform capnography:** Extremely valuable. Indicates adequacy of ventilations, perfusion, & dead space by detecting how much CO_2 is being exhaled and giving a numeric value & a graphic waveform.

(1) HF should have normal, squared off waveform

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

(3) Reading > 45 may indicate hypoventilation with impending ventilatory failure.

f. **Palpation if in distress**

(1) Amount of air movement; symmetry of chest wall expansion
(2) Tenderness, instability, crepitus, deformity; sub-q emphysema
(3) Tracheal position in neck (Extrathoracic tracheal position will almost always be midline as it is tethered securely to midline structures. In a tension pneumothorax with mediastinal shift, the intrathoracic trachea will deviate, evident on chest X-ray.)

g. **Auscultate** immediately if patient appears to be in ventilatory distress. May defer to secondary assessment if in no distress.

Most ominous finding in a spontaneously ventilating patient is silence due to tissue mismatch that reflects sound away from the chest wall (pneumothorax, hemothorax) or severe bronchial obstruction/spasm.

h. **Signs of compromised ventilations/inadequate gas exchange**

(1) Apnea
(2) S&S hypoxia: Agitation, restlessness, disorientation, confusion, stupor, coma; cyanosis
(3) Dyspnea (exertional or non-exertional)
(4) Use of accessory muscles
(5) Upright, tripoding position; orthopnea
(6) Ventilatory efforts that are weak, shallow, labored, or retracting
(7) Adult RR < 10 or ≥ 24/min
(8) SpO_2 < 94%; $EtCO_2$ > 45
(9) Diminished or absent breath sounds
(10) Paradoxical or unequal chest wall movements
(11) External trauma to chest; open wounds, contusions, abrasions
(12) Subcutaneous emphysema
(13) Hemoptysis; frothy or mucopurulent sputum
(14) Tachycardia → bradycardia
(15) Dysrhythmia, hypotension

i. **General interventions for impaired gas exchange/ineffective breathing**

(1) **Oxygen therapy** - Goals:

(a) Decrease work of breathing
(b) Decrease myocardial work
(c) Reverse or prevent tissue hypoxia

(2) **Evolving science regarding oxygen delivery:** 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.

(a) Oxygen delivery to cells depends on O_2 content + perfusion

(b) In most tissues, **hyperoxia causes vasoconstriction**, like norepinephrine, directly or through hyperoxia-induced hypocapnia. This response is part of the autoregulation process, where organs alter their own perfusion in response to oxygen and CO_2 levels, acidity, potassium, and lactate levels in the blood. **Exception: lungs – where hypoxic pulmonary vasoconstriction** lets lung tissues that are poorly ventilated decrease their blood supply to rectify the ventilation/perfusion mismatch.

(c) If local tissue perfusion drops more than arterial O_2 content increases during hyperoxia, regional oxygen delivery decreases.

(3) **Patients at risk if subjected to <u>hyper</u>oxia**

(a) **MI**: Cochrane review (2010) concluded that there is no benefit and potential harm from oxygen use in these patients. AHA (2010 guidelines) recommended that O2 not be given to patients with uncomplicated cardiac chest pain who have an O2 sat greater than 94% due to potential for coronary artery vasoconstriction.

Recommendation given when new SOPs were introduced in 2011: If dyspneic, hypoxemic, or obvious signs of HF, titrate O_2 to achieve SpO_2 ≥ 94%. If SpO_2 is ≥ 94% and no S&S as above - no supplemental O_2 may be needed. If 92%-94% apply a nasal cannula at 1-6 L and if < 90% use a NRM at 12-15 L.

(b) **Cardiac arrest:** Hyperoxia may be more harmful than hypoxia in adults during and immediately following cardiac arrest. A recent study enrolled 6,326 pts over 5 years (Kilgannon et al, 2010). Parameters of study: hypoxia (PaO_2 < 60 mmHg (O_2 sat < 90); normoxia (PaO_2 61-299 mmHg); hyperoxia (PaO_2 > 300 mmHg). Hyperoxia was a significant independent risk factor for in-hospital mortality. While correlation does not imply causation – researchers concluded that **O_2 should be limited to sufficient amounts to keep O_2 sats between 94%-96%**. Exception: Just prior to intubation – use 95%-100% O_2 via NRM or BVM to "wash out" nitrogen and prevent rapid onset of hypoxia during intubation attempt (AHA, 2010): **Target SpO_2 94% following successful resuscitation.**

(c) **COPD:** Recent study conducted in Australia indicated that giving high-flow O2 to patients with an exacerbation of COPD, and possibly patients with undifferentiated shortness of breath who don't have a diagnosis of COPD can significantly increase mortality (Austin et al, 2010). Study titrated pulse ox values in the intervention arm to 88-92% to achieve the best outcomes.

(d) **Stroke:** Studies suggest no benefit and potential harm when given to patients with ischemic stroke – similarly to patients with AMI. Hyperoxia causes free radical intracellular damage and cell destruction (like pouring hydrogen peroxide into an open wound). Ischemic tissues are very sensitive to free-radical damage. Plus, hyperoxia causes vasoconstriction which can impair perfusion to the ischemic penumbra as well as to the rest of the brain.

(e) **Neonatal resuscitation**: It has long been known that high concentrations of oxygen given to premature newborns for a prolonged period (>24 h) can be associated with retinopathy of prematurity that causes blindness. A relatively recent finding is that newborns resuscitated with air, rather than 100% oxygen had a decrease in mortality (Davis et al, 2004). AHA guidelines recommend initial resuscitation of newborn infants with air, switching only to oxygen when there is clear evidence of significant hypoxemia following resusciatation with air.

(f) **Give O_2 to the above pts only if evidence of hypoxia and titrate to dose that relieves hypoxemia without causing hyperoxia (SpO_2 94%)** (Iscor et al, 2011).

(4) Profound, prolonged hypoxia is **BAD** and almost universally fatal. **The following continue to need oxygen therapy per SOP:**

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

(b) Actual or potential **airway compromise** (epiglottitis, airway burns)

(c) **DAI** (to increase O2 reserves): Reminder from SOP roll-out: **Preoxygenation tip:** Can be used with the morbidly obese or others in respiratory distress prior to DAI – place a NC under the O_2 face mask run at 5 L. When the mask is removed to intubate, the NC is still in place and may help prevent hypoxia during the procedure. Levitan, R. (Dec, 2010). NO DESAT: Nasal oxygen during efforts securing a tube. Emerg. Physicians Monthly

(d) Globally poor tissue oxygenation and perfusion (**shock**)

SOP

Breathing/gas exchange: Provide O_2 and assist ventilations as needed UNLESS CONTRAINDICATED

Oxygen 1-6 L/NC: Adequate rate/depth; minimal distress, mild hypoxia (target SpO_2 94-98%)
Oxygen 12-15 L/NRM: Adequate rate/depth: moderate/severe distress; $SpO_2 \leq 94\%$ or per protocol
Oxygen 15 L/ BVM: Inadequate rate/depth: moderate/severe distress; unstable

- Before advanced airway: Adults: 1 breath every 5 to 6 sec (10-12 breaths/minute) (Asthma: 6-12 BPM)
- After advanced airway: Adults: 1 breath every 6 to 8 sec (8-10 BPM)

A common mistake is using a high respiratory rate after intubation. A pt w/ COPD is most likely acidotic and has a marginally normal or low potassium level due to diuretic and bronchodilator therapy. With a too-rapid respiratory rate, the patient will become alkalotic, causing an intracellular shift in K with potentially dangerous hypokalemia as a result.

CPAP: Indications: COPD/asthma w/ severe distress, pulmonary edema, flail chest w/o pneumothorax, near drowning, pneumonia (?), palliative care (?), diaphragmatic weakness, post-extubation rescue

What do these conditions have in common? Severe dyspnea & refractory hypoxia, poorly expanded lung fields, ↑ WOB (↑ inspiratory muscle work), ↓ minute ventilation [size of each breath (tidal volume) X RR per minute], inability to remove CO_2 from body, hypercarbic ventilatory failure, narcotic effect on brain → ↓ RR, fatigue + ↓ RR = respiratory arrest

j. **Expected outcomes**

(1) Respirations are spontaneous and unlabored
(2) Chest expansion is equal bilaterally
(3) Breath sounds are present and normal bilaterally
(4) Speech pattern is normal; patient phonates well
(5) Gas exchange is adequate with no signs of hypoxia, hypo or hypercarbia

3. **C = Adequacy of cardiac output, rhythm, fluid volume, perfusion**

a. **Pulse**: Presence/absence; location, general rate (fast/slow - don't count yet), quality (strong/weak/thready), rhythmicity

(1) A weak or absent radial pulse and presyncopal symptoms do not appear until just prior to the point of circulatory decompensation and cardiovascular collapse. Worsening radial pulse character is associated with decreases in systolic BP (Ryan et al, 2008). Rapid, thready pulses may be early signs of hypovolemia, but may have other causes as well, i.e., pain, sympathetic nervous system activation, overdose, fever. A sustained HR faster than 100 suggests hypovolemic shock in a trauma patient.

(2) Central pulses absent at more than one site, without obvious cause, necessitate immediate resuscitative measures to replace blood volume and restore effective cardiac output.

(3) Be alert to patients with pacemakers, the elderly, or those on medications (beta/calcium blockers, digoxin) that prevent the usual tachycardic response to dehydration or shock. Assess LOC carefully.

(4) An irregular pulse is usually a warning of cardiac dysrhythmia

b. **Perfusion:** A BP can be misleading in evaluating a patient for shock as significant deficits can occur before the pressure drops depending on the patient's age and ability to compensate. .DON'T TAKE BP during primary assessment. Be alert to those whose actual condition may be masked by physiologic reserves or robust compensatory mechanisms, i.e., athlete, child, or pregnant woman (ACS, 2008). In addition to pulses, perfusion can be rapidly assessed by evaluating mental status (central perfusion) and observing skin color and temperature of extremities (peripheral perfusion).

c. **Cardiac rhythm: monitoring** If the pulse is weak, very fast, very slow, irregular, central pulses are absent or there is evidence of cardiovascular, neurological, or respiratory compromise, monitor ECG immediately. Always look for underlying cardiac ischemia with acute exacerbations of COPD. With hypoxia and distress, many of these patients can have unrecognized underlying ischemia.

Obtain a 12 lead ECG if indicated:

(1) Discomfort (nose to navel, shoulder, arm, back)
(2) **SOB**/HF
(3) Palpitations
(4) Dysrhythmia (VT/SVT)
(5) GI complaint (nausea, indigestion)
(6) Diaphoresis
(7) Dizziness/syncope
(8) Weak/tired/fatigued

d. Ventricular hypertrophy may be evident in leads that overlie the affected ventricle. The 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.

IV. **Secondary assessment**

A. Performed to detect non-life threatening illnesses/injuries and to provide care for those conditions/injuries. Usually performed enroute in a time-sensitive patient.

B. **Patient history** (acquire during/incorporate into physical exam)

1. History may be obtained from the patient, family, significant others, bystanders, or personal belongings such as wallet, medic-alert tags, tattoos, or jewelry.

2. **S = Signs & Symptoms**: Chief and associated complaints. Will be different for medical vs. trauma patients. What is the main problem? Why was EMS called today? Assess the circumstances of the present illness, sometimes called the history of present illness or **HPI**. A typical chief complaint of a pulmonary problem would be dyspnea, shortness of breath, trouble breathing, cough, or chest pain.

a. **O = Onset**: What were you doing when the problem began? How did it begin, abruptly or gradually?

b. **P = Provocation**/palliation

(1) What, if anything, precipitated the complaint?

(a) Contributing factors? Exertional or non-exertional onset?
(b) Can the problem be related to the patient's activity or environment? DOE suggests that the patient is unable to meet metabolic or oxygen demands as a result of impaired cardiac output or decreased alveolar diffusion.
(c) Associated factors: toxic inhalants, drugs, alcohol

(2) What makes it worse?
(3) What makes it better?
(4) What have they done to relieve the symptoms?

c. **Q = Quality of discomfort**: Have patient describe the problem without prompting. Ask pt to take deep breath & ask about pain. The most common and prominent of all the symptoms of **pleuritic chest pain** is the stabbing ache experienced in the chest that is aggravated when the patient breathes deeply or coughs.

(1) Other common symptoms in patients with pleuritic chest pain are fever, shoulder pain, worsening of the pain due to chest movement, cough and deep breathing, cough, cyanosis, & fluid accumulation in the pleuritic region that leads to further chest pain.

(2) **Pleuritic chest pain may suggest the following:**

(a) Pleuritis, pleurisy, pleural effusion
(b) Pneumonia (ask about chills or fever)
(c) Inflammation of lungs from TB
(d) Pulmonary thromboembolism
(e) Pulmonary HTN: there is a lot of pain along with difficulty in breathing, edema and cough
(f) Costochondritis
(g) Pericarditis: This is a condition associated with substernal pain due to inflammation of the pericardium. The pain is relieved by leaning forward or simply sitting up.
(h) Rib fracture
(i) Lung cancer

d. **R = Region**, radiation

(1) Where is it most acute; does it radiate? Where?
(2) Under what circumstances?

e. **S = Severity**, intensity

(1) Quantify degree of severity
(2) Trend subjective perception of pain using an appropriate tool

f. **T = Time**

(1) Duration - how long has it lasted?

(2) Frequency

(a) Have they ever had this before?
(b) How often does it occur?
(c) If more than one sign/symptom is present, determine their relationship; which came first?

g. **Coughing?**

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

(2) Differential

(a) Aspiration
(b) Smoke inhalation
(c) Secretions
(d) Irritation
(e) Hyperreactive airways
(f) Bronchospasm

(3) Determine if productive or non-productive. If productive, describe sputum in terms of color, consistency, amount, and odor.

(a) Clear, mucoid
(b) Yellow/Green: infection –bronchitis, pneumonia, COPD.
(c) Rusty/bloody: Some forms of pneumonia

(d) Frothy: (occasionally pinked-tinged from surfactant washing out) due to acute pulmonary edema. Listen for S3 heart sound to confirm heart failure.

3. **A = Allergies**: To medications, foods, and/or environment

4. **M = Medications:** Legal and illegal

Important questions to ask:

a. **"What medications do you take?"** Avoid asking only about medications taken every day - some are taken every other day, some are injected every other week, etc. Also avoid asking if they take any prescription medications. These questions suggest that you don't want to hear about the aspirin they take every day or the numerous over-the-counter antihistamines they have been taking.

b. **"What other medications, drugs, vitamins, etc. do you take?"** No matter how many medications a patient initially says they take, ask "What else?" until the patient says, "That's all." Patients often forget some or get interrupted and lose their train of thought.

c. **"What medications are you supposed to be taking?"** and **"What medications have you been on before?"** These are open-ended questions that should be asked if a patient denies taking any medications. They often reveal something about the past medical history or the fact that a patient may not be taking or have stopped taking a medication without consulting with their physician.

d. **"Have you been taking your medicines the way you were instructed to?"** Patients often decide to alter their medication routine for various and strange reasons. They may or may not admit to it.

e. **"When was your last dose?" and "How much did you take?"**

f. **"Has there been any recent change in your medications?"** Any NEW medications? Increases or decreases in types of meds or doses may shed light on their current condition.

g. Prescribed; check bottles for dates. Bring to hospital. See SOPs page 19.

h. **Ace inhibitors ("Prils")**: All pts with LV systolic dysfunction and type 1 diabetes should be treated with an ACEI unless contraindicated or not tolerated. First-choice drug therapy for HF (proven to slow progression). They prevent the conversion of angiotensin I to angiotensin II which is a potent vasoconstrictor. Thus they **vasodilate the patient, lower the BP, decrease the heart's workload, and restore the balance between myocardial O_2 supply and demand.** They reduce LV mass, prevent remodeling, reduce sympathetic stimulation and have a direct antiatherogenic effect. Blunts aldosterone release so prevents salt and water retention.

   SE: Some develop a **persistent cough**. Weakness and dizziness common due to reduction in BP and inability to compensate rapidly for changes in position. Other SE: Skin rashes, altered sense of taste, hyperkalemia. Pts may need K levels checked regularly, especially if they also take diuretics or K supplements. Nausea and headache possible.

i. **Angiotensin Receptor Blockers (ARBs)**: "Sartans" – candesartan (Atacand), eprosartan (Teveten), irbesartan (Avapro), losartan (Cozaar), olmesartan (Benicar), telmisartan (Micardis), valsartan (Diovan)

j. Antiarrhythmics: See chart at end of handout

k. **Beta Blockers "lols"**: Beta blockers, also known as beta-adrenergic blocking agents, are drugs that block norepinephrine and epinephrine from binding to beta receptors on nerves.

   (1) There are three types of beta receptors and they control several functions based on their location in the body.

      (a) Beta-1 (β1) receptors located in heart, eye, and kidneys

      (b) Beta (β2) receptors are found in the lungs, GI track, liver, uterus, blood vessels, and skeletal muscle

      (c) Beta (β3) receptors are located in fat cells

(2) Beta blockers primarily block β1 and β2 receptors. By blocking the effect of norepinephrine and epinephrine, beta blockers reduce HR, reduce BP by dilating blood vessels; and may constrict bronchioles by stimulating smooth muscles surrounding them to contract.

(3) Beta blockers are used to treat abnormal heart rhythm, high BP, HF, angina, tremor, pheochromocytoma (adrenal tumor), and prevention of migraines. They also prevent further heart attacks and death after a heart attack. Other uses: treatment of hyperthyroidism, akathisia (restlessness or inability to sit still), and anxiety. Some reduce the production of aqueous humor in the eye and are used to reduce intraocular pressure caused by glaucoma.

l. **Ca channel blockers**: Amlodipine/Norvasc, **f**elodipine (Plendil), nicardipine (Cardene), verapamil (Calan, Verelan)

m. **Diuretics:** amilor***ide*** (Midamor), bumetanide (Bumex), chlorothiazide (Diuril, Diazide), furosemide (Lasix), hydrochlorothiazide (Hydrodiuril), indapamide (Lozol), metolazone (Zaroxolyn), Polythiazide, spironolactone (Aldactone), Torsemide, trianterene (Dyrenium)

n. **Vasodilators:** Hydralazine (Apresoline), isosorbide (Isordil), minoxidil (Loniten), nesiride (Natrecor), nitrates/NTG

o. **Anticoagulants**: Anticoagulants reduce blood clotting to prevent deep vein thrombosis, pulmonary embolism, myocardial infarction and stroke.

(1) Heparin [activates antithrombin III, which blocks thrombin from clotting blood], low molecular weight heparin

(2) warfarin (Coumadin) [Vitamin K antagonist]

(3) rivaroxaban (Xarelto) [Direct factor Xa inhibitors ('xabans') act directly on Factor X in the coagulation cascade, without using antithrombin as a mediator]

(4) dabigatran (Pradaxa) approved by the FDA to treat Atrial Fib in Nov. 2010. [Direct thrombin inhibitors – Examples: bivalent drugs - hirudin, lepirudin, and bivalirudin; monovalent drugs - argatroban and dabigatran]

(5) clopidogrel (Plavix): Prevents platelets from clumping

(6) ASA

p. **Drugs to treat high cholesterol:**

(1) **Statins:** atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor), pravastatin (Pravachol), simvastatin (Zocor), rosuvastatin (Crestor)

(2) **Niacin:** Nicotinic acid is a B-complex vitamin found in food, but is also available at high doses by prescription. It lowers LDL cholesterol and raises HDL cholesterol. Main SE are flushing, itching, tingling and headache. A recent research study suggested that adding niacin to statin therapy was not associated with a lower risk of heart disease. Examples: Nicolar and Niaspan.

(3) **Bile Acid Resins:** Work inside the intestine, where they bind to bile from the liver and prevent it from being reabsorbed into the circulatory system. Bile is made largely from cholesterol, so these drugs work by depleting the body's supply of cholesterol. Main side effects are constipation, gas and upset stomach. Examples: Questran and Questran Light, Colestid, WelChol

(4) **Fibrates** reduce the production of triglycerides and can increase HDL cholesterol. Examples: Atromid, Tricor, Lopid

q. **Meds for pulmonary problems**

(1) **Short acting beta agonists**: Albuterol 2.5 mg/3 ml NS (Proventil, Ventolin); terbutaline sulfate (Brethaire, Brethine, Bricanyl); metaproterenol sulfate (Alupent, Metaprel) 15 mg/3 ml, pirbuterol (Maxair); isoetharine (Bronkosol, Bronkometer); bitolterol (Tornalate); levalbuterol (Xopenex). Regular use of inhaled beta-agonists show an association with risk of hospitalization of death from asthma (O'Hollaren, 2005)

(2) ***Long-acting beta2-agonists (LABAs)***— salmeterol xinafoate (Serevent) and formoterol are bronchodilators, with action lasting up to 12 hours, that may be used in combination with inhaled corticosteroids (they shouldn't be used as monotherapy); note the new black box warning about asthma-related deaths on all preparations containing a LABA (www.NursingMadeIncrediblyEasy.com , March/April 2010).

(3) **Anticholinergics:** Ipratropium bromide (Atrovent), ipratropium with albuterol, tiotropium (Spiriva). Less potent, slower onset, & longer acting bronchodilator than a beta-2 agonist. Vagal blocking action decreases airway constriction. May have an additive effect when used simultaneously (shown more for COPD than asthma).

(4) **MAST cell inhibitors** block histamine and prostaglandin release: cromolyn sodium (Nasalcrom, Intal; Opticrom); nedocromil sodium (Tilade).

(5) **Leukotriene modifiers**: Leukotrienes promote the inflammatory response caused by exposure to allergens. Less leukotriene = less inflammation = fewer symptoms. Examples: Singulair^{R} (montelukast); Accolate (zafirlukast); Zyflo (zileuton):

(6) **Immunomodulators:** Monoclonal antibodies - Newer asthma medication: omalizumab (Xolair) – prevents the binding of IgE antibodies to the receptors on basophils and mast cells. Black box warning to be ready to treat anaphylaxis.

(7) **Steroids**: Steroids block the late phase reaction to allergens, reduce airway hyperresponsiveness. interfere with the synthesis of inflammatory agents, and prevent migration and activation of inflammatory cells. Steroids treat the disease, beta-agonists only treat the symptoms in diseases like asthma. Examples inhaled steroids: triamcinolone acetonide (Azmacort), beclomethasone (Vanceril, Beclovent, Beconase), budesonide (Pulmicort Turbuhaler), budesonide with formoterol, flunisolide (AeroBid), fluticasone propionate (Flovent); fluticasone with salmeterol, and mometasone.

(8) **Methylxanthines:** sustained-release theophylline is a mild-to-moderate bronchodilator that's used as an alternative, but not preferred, therapy

(9) **Erectile dysfunction drugs** – Cialis and Viagra sometimes prescribed to treat pulmonary hypertension

(10) On home oxygen?

5. **P = Pertinent past medical history**

a. **Existing conditions**

(1) **Respiratory diseases**

(a) COPD: Asthma, emphysema, chronic bronchitis, cystic fibrosis, bronchiectasis

(b) Restrictive lung diseases Sarcoidosis, tuberculosis, pneumonia, ARDS, Valley Fever

(c) Occupational lung diseases: Psiticosis, CWP – black lung, Byssinosis - white lung, Silicosis, Asbestosis, Pidgeon breeders disease

(2) **Cardiovascular diseases/conditions** (CVD), coronary artery disease (CAD), MI, hypertension (HTN); acute coronary syndrome (ACS); heart failure (HF), cardiomyopathy, high cholesterol, internal cardioverter defibrillator (ICD), pacemaker, dysrhythmias, peripheral vascular disease (PVD); valve disease

(3) Endocrine diseases (diabetes)
(4) Neurological diseases/conditions: stroke/TIA
(5) Previous surgeries
(6) Previous/current infectious diseases

b. Are they being seen by a doctor? Who and how long ago?

c. **Social history**: Ask about cigarette and tobacco use. Report in pack years -# of packs smoked/day X # of years they've smoked. Problems when pack years surpass 20.

6. **L**: **Last oral intake** (liquid/food ingested - Change in eating or drinking habits)

7. **LMP**: Females 10-55: last menstrual period; sexually active?

C. **Full set of vital signs**

1. **Respirations**

a. **Technique**: Observe the chest rise and fall or place one hand over patient's chest and count the number of breaths. Try to be discrete as pt may control their rate if they think you are counting. Count RR while pretending to count pulse rate OR get rate from the capnography monitor.

b. **Rate**: Number of breaths per minute

(1) Adult RR should be 12-20/min; faster for children and infants
(2) **Tachypnea**: Over 20
(3) **Bradypnea:** Less than 12

Causes of Tachypnea	**Causes of Bradypnea**
↑ Metabolic rate	Poisoning
Fever, infection, exertion	Sedatives
Stress, pain	Narcotics
Cardiac dysfunction	Tranquilizers
Abdominal distention	Alcohol
Obesity, ascites, pregnancy	Barbiturates
Metabolic acidosis	Endocrine
Uremia	COPD
Poisoning	with O_2 retention
Salicylates	Diuretic therapy
Encephalopathies	Metabolic alkalosis
CNS dysfunction	CNS dysfunction
Psychogenic, anxiety	Sleep
Fear	Severe hypoxia
Anemia, shock	Hypoxia

2. **Pattern**: The normal pattern of ventilations is called ***eupnea***

3. **Abnormal respiratory patterns**

a. **Cheyne-Stokes**: Crescendo/decrescendo respirations (waxing and waning depth and rate) with periods of apnea up to 20 seconds; seen with ↑ ICP. Due to increased sensitivity to CO_2 that results in the change in depth and rate. Decreased stimulation from respiratory centers results in apnea. Lesions are most often located bilaterally deep within the cerebral hemispheres, diencephalon (thalamus and/or hypothalamus), or basal ganglia.

b. **Kussmaul**: deep, hyperpnea; seen in metabolic acidosis

c. **Central neurogenic hyperventilation**: Increased rate and depth of respirations. Thought to be due to release of the reflex mechanisms for respiratory control in the lower brain stem. Results in a decreased CO_2 and an alkaline pH. Giving oxygen does not change the pattern. Lesion location is unclear, often in the midbrain and upper pons.

d. **Apneustic**: A pause of 2-3 seconds noted after a full or prolonged inspiration. May alternate with an expiratory pause. Lesion located in the lower pons, usually due to a basilar artery occlusion.

e. **Cluster:** Clusters of slow irregular breaths with periods of apnea at irregular intervals (gasping breathing has features similar to cluster breathing). Lesion located in the lower pons or upper medulla.

f. **Ataxic** (Biot's) breathing: Completely irregular, unpredictable pattern with deep and shallow random breaths and pauses. Lesion located in the medulla.

D. **Maneuvers of assessment**

1. **Inspection- Process of informed observation** Anything you can see, hear without amplification through a stethoscope, or smell
2. **Palpation**- Use of touch to gather information
3. **Percussion**-percussion note(s); assess for diaphragmatic excursion
4. **Auscultation**-breath sounds (normal, abnormal), adventitious

E. **Pulmonary inspection**

1. **Contour and integrity of chest wall.** Should be symmetrical with 2:1 lateral to anterior-posterior (AP) diameter. Abnormalities to note:
   a. Barrel chest: Increased A-P diameter (barrel chest) with diaphragmatic flattening associated with emphysema and lung hyperinflation
   b. Pectus excavatum: Congenital posterior displacement of lower sternum
   c. Kyphosis
   d. Scoliosis
   e. Chest wall lesions to note: old wounds, surgical scars
2. **Assess for JVD**
   a. Best measured with patient supine and head elevated 45° (range: 20°-60°). Adjust angle so top of jugular vein is in the middle of the neck. Measure height of venous column from angle of Louis; add 5 cm to determine measurement from RA.
   b. Results from elevated RA pressure or inability to drain blood into RA so may not be present with acute LV failure. May be present if chronic right HF and fluid retention.
   c. Causes of R heart failure – all can be associated with dyspnea
      (1) Cor Pulmonale: Increased RV strain = cardiac hypertrophy, venous congestion, HTN
      (2) Pulmonary embolism (large) can cause acute R heart failure
3. **If history of COPD** – clues to distinguish between patients with chronic bronchitis and those with emphysema
   a. **Chronic Bronchitis - Blue bloater:**
      (1) Patients produce at least 10 mL green or yellow sputum daily. Productive cough and dyspnea increase with disease severity. The cilia are damaged causing decreased removal of mucous. These secretions cause **airway obstruction from mucus plugging** and provide an excellent growth medium for microorganisms. Patient experiences frequent infections.

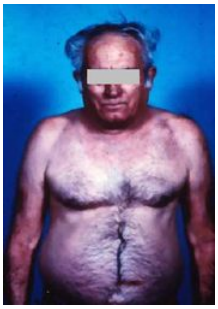
(2) Thickening and rigidity of the bronchial mucosa results from vasodilation, congestion and edema. Increased resistance can be with or without emphysemic changes. Unlike emphysema, the alveolar walls are not severely affected in chronic bronchitis and diffusion across the respiratory membrane remains normal.

(3) Alveolar hypoventilation adversely affects gas exchange leading to arterial hypoxia and CO_2 retention (hypercarbia) sometimes giving the patient a bluish complexion. Increased $PaCO_2$ may lead to irritability, somnolence, decreased intellectual abilities, headaches, and personality changes.

(4) **Pathogenesis of Cor Pulmonale (primary right heart failure)**

(a) Bronchial obstruction leads to ↓ alveolar oxygen and hypoxia. Hypoxia, acidosis and hypercarbia cause vasoconstriction of small, midsized arteries linking the right heart and lungs (hypoxic vasoconstriction) resulting in **pulmonary hypertension**.

(b) This generates an ↑ cardiac workload for the right ventricle (↑ RV afterload) resulting in RV hypertrophy and failure. If the RV does not send blood forward to the lungs, blood cannot flow into it from the peripheral veins resulting in systemic venous congestion (JVD, peripheral edema) and HTN.



(c) Patients more likely than those with emphysema to adjust to chronically elevated CO_2 levels by switching to an oxygen-dependent ventilatory drive.

(5) May be obese
(6) Accessory muscle use common
(7) Poor peak flow (<150-200 mL)

b. **Emphysema (Pink puffer)**

(1) BP: Pulsus paradoxus
(2) P: Tachycardia
(3) RR: Tachypnea: 1-2 word dyspnea
(4) Increased WOB; pursed lip breathing
(5) Little/no cough
(6) With low cardiac output - rest of body suffers hypoxia & pulmonary cachexia resulting in muscle wasting & weight loss

F. **Palpate**: ribs, clavicles, sternum, scapulae

1. Palpation plays minor role in exam of a normal chest; lungs are covered by ribs & aren't palpable
2. Palpate by compressing downward on sternum and inward on lateral chest wall
3. Crepitus: Bony/sub-q
4. Tracheal position
5. Assess for equal bilateral **chest excursion** (movement) by placing your hands on each side of the chest with thumbs at the xiphoid process. Must have significant disease before asymmetry can be identified on exam
6. Ratio of inspiration to expiration. Prolonged expiration indicates distress.
7. Point tenderness: Investigate painful areas
8. Deformity, instability
9. Point of maximum impulse (apex of heart)
10. Elderly at risk for rib fractures due to bone brittleness

11. **Tactile fremitus** - Normal lung transmits palpable vibration to chest wall
    a. May use a one or two hand method. Place **palm** on either side of chest. Start at apex (top) of lungs and proceed down to bases (bottom) and then move to the lateral chest.
    b. Ask patient to say "99" each time you move your hands. Vibrations palpated will be loudest at the apex and will decrease in intensity as you move down the back.
    c. Normal finding - symmetrical; increased over areas of consolidation; decreased over a pleural effusion

G. **Percussion**: Striking a part of the body with short, sharp blows in order to produce a sound

1. Place the 3rd finger of your non-dominant hand on the area you want to percuss. Always percuss over soft tissue (between ribs -never over bone). Elevate the rest of the fingers and palm to avoid contact with the skin. Pressure from other fingers and palm on adjacent skin surface will muffle the sound.
2. Rapidly strike that finger just proximal to the nailbed (not on the nail) with the tip of the 3rd finger of your dominant hand (do not use the finger pad). This creates vibrations that produce sound waves from four to six centimeters deep into underlying body tissue. Use a sharp, crisp, rapid striking movement from a relaxed wrist. Draw the striking finger back quickly to avoid dampening the sound.
3. Percuss in a systematic manner, from side to side from apex of lungs to base, listening to changes in tone from one area to another. Percuss two to three times at each site.
4. The sounds produced are **percussion notes** that are generally classified as *resonant, hyperresonant, flat or dull*. The tone's resonance or lack of resonance indicates whether the underlying region is filled with air, air under pressure, fluid, or normal tissue. The denser the tissue, the quieter the tone.
    a. Resonance: normal aerated lung tissue
    b. Tympany (hyper-resonance): pneumothorax
    c. Flatness/dullness: hemothorax

Sound	Description	Intensity	Pitch	Duration	Location
Tympany	Drum-like	Loud	High	Medium	Stomach
Hyperresonance	Booming	Loud	Low	Long	Hyperinflated lung
Resonance	Hollow	Loud	Low	Long	Normal lung
Dull	Thud	Medium	Medium	Medium	Solid organs
Flat	Extremely dull	Soft	High	Short	Muscle, atelectasis

5. Percussion is rarely done in the field due to the levels of ambient noise and EMS personnel's' lack of familiarity with the process, but can be valuable in distinguishing between a pneumo and hemothorax.

V. **Chest auscultation:** Presence/absence and equality of normal/abnormal and adventitious breath sounds and heart sounds

A. **Purpose of chest auscultation is to**

1. assess the patient's pulmonary status and function.
2. permit assessment of airflow through and patency of the airways.
3. detect airway obstruction
4. and assess the condition of the lung parenchyma and pleura.

B. **Breath sound production**

1. Studies of gas flow through the lungs have made it possible to make certain assumptions as to how breath sounds are produced and where they originate.

2. Breath sounds are produced by airflow patterns, by associated pressure changes within the airways, and by solid tissue vibrations. The amplitude and intensity are affected by airflow patterns, regional lung volumes or distribution of ventilation, body position, and the sound production site.

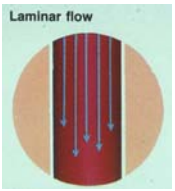
3. The sounds are normally diminished and filtered when they are transmitted through air-filled alveoli, fluid accumulations in the pleurae, and solid structures such as bone.

C. **Airflow patterns within the tracheobronchial tree**

1. Breath sounds originate in the large airways where air velocity and turbulence induce vibrations in the airway walls. Normal breath sound production is directly related to air flow velocity and airway lumen architecture. Air flow velocity is primarily determined by pulmonary ventilation and TOTAL cross sectional airway area. The passage of air through the tracheobronchial tree creates a characteristic set of noises audible through the chest wall.

2. **Turbulence** is created in the **TRACHEA** and **LARGER BRONCHI** from the random movement of air molecules that collide against airway walls and each other. The colliding molecules produce rapid pressure changes within the airway. These pressure variations produce audible sounds that are loud and harsh.

3. **Transitional flow** at airflow vortices separates the airstream into layers that move at different velocities. At the bronchial bifurcations, swirling airflow patters are created in more narrow airways. Energy is transferred between colliding gas molecules. This results in transient pressure variations capable of producing audible sound waves. Transitional flow creates normal vesicular sounds.



4. **Laminar flow**: Terminal bronchioles (airways with a diameter <2 mm) and alveoli do not contribute to breath sounds as the air flow is laminar and the velocity at this level and too slow to produce significant turbulence and sound waves. By the alveoli, gas movement is by diffusion not gas flow. However, terminal airway and alveolar disease does modify the breath sounds heard at the surface by either increasing or decreasing the sound transmission through the diseased tissue. Thus, the sounds that are heard at the periphery of the lung are produced in more central (hilar) regions and are altered in intensity and tonal quality as they pass through pulmonary tissue to the periphery.

5. Breath sounds heard through the chest wall come mainly from the larger bronchi as they are transmitted through the lung tissues.

D. **Stethoscope**

1. Used to auscultate sounds. Transmits sound waves from the source through a diaphragm or bell, along a rubber tube to ear pieces

   a. Diaphragm best transmits high-pitched sounds like lung and BP sounds. It screens out lower-pitched sounds like heart and bowel sounds.

   b. The bell uses skin as the diaphragm. Sounds vary based on the tension placed on the skin. Best side for listening to heart and bowel sounds.

2. **Characteristics of a good stethoscope** to optimize auscultation

   a. Rigid diaphragm cover
   b. Thick, heavy tubing that conducts sound better than thin, flexible tubing
   c. Short tubing (30-40 cm) to minimize distortion
   d. Ear pieces that fit snugly - large enough to occlude ear canal; angled toward nose to project sound toward the ear drum
   e. A bell with a rubber-ring edge to ensure good contact with the skin

E. **Auscultatory sites and sequencing**

1. Accurate interpretation of breath sounds requires the ability to describe the location of abnormal findings in relation to bony structure and anatomic landmark lines. Breath sounds should be auscultated over the anterior, lateral and posterior chest wall surfaces.

   a. **Anterior chest wall**

      (1) Apex: The lung s extend ¼" to 1½" (2-4 cm) above the clavicles (Isthmus of Koenig)

      (2) The trachea bifurcates under the sternal angle (Angle of Louis) at the junction between the manubrium and the body of the sternum

      (3) The ribs and intercostal spaces provide horizontal landmarks used to describe the location of breath sounds; the second rib and second intercostal spaces serve as reference points

      (4) **Vertical landmark lines**

         (a) Right and left midclavicular lines extend downward from the center of each clavicle

         (b) Midsternal line: Bisects the sternum

   b. **Lateral chest wall**

      (1) Anterior axillary line
      (2) Midaxillary line
      (3) Posterior axillary line

   c. **Posterior chest wall surface**

      (1) Bony structures

         (a) Scapulae: Inferior borders are at the same level as the 7th rib and interface

         (b) Thoracic vertebrae

      (2) Landmark lines

         (a) Midscapular line extends downward from the inferior angle of each scapula

         (b) Vertebral line extends downward over the vertebrae

2. **Listening to breath sounds**

   a. Many sounds have frequencies near the lower threshold of human hearing. The environment needs to be as quiet as possible to hear sounds clearly and distinctly.

   b. Warm stethoscope head; hold between 2nd & 3rd fingers

   c. Press the diaphragm head firmly against the chest wall over an intercostal space. Do not listen directly over bone. Whenever possible, do not listen over clothing, which impedes or alters sound transmission. Avoid extra sounds caused by the stethoscope tubing rubbing against an object.

   d. If the patient has a very hairy chest, hold the stethoscope firmly against the skin to minimize the crackling noises produced by the hair.

   e. Concentrate first on inspiration location, intensity, pitch, duration, quality, timing, normal and adventitious components; then note expiration

   f. Close eyes to focus on sounds

   g. If alert and hemodynamically stable, begin auscultation with the patient sitting up and leaning slightly forward.

   h. If patient is comatose or critically ill, they may be rolled from side to side to auscultate dependent lung regions. Listen initially to the dependent site as gravity-dependent secretions or fluids may produce abnormal sounds that disappear when the patient is turned, breathes deeply or coughs.

i. Have the patient take one **breath through the mouth** for each site examined. Monitor patient's breathing throughout the examination. Offer times for the person to rest and breathe normally.

j. **Where to listen?** Assess lung sounds in all lobes (front and back): Lower lobes occupy ¾ of posterior fields; RML right axilla; Lingula in L axilla (the lateral portions of the lungs are usually assessed along with the posterior lobes); upper lobes are assessed over the anterior chest and top ¼ of posterior fields.

3. **Auscultatory sequencing – Where to start**

a. **Posterior chest:** Start at **auscultatory triangle.** Have pt lean forward slightly and hold elbows with the hands to help separate the scapulae. Listen over the space formed by the lateral edge of the Trapezius muscle, superior rim of the Lasissimus dorsi muscle and medial/ lower scapula. Less muscle mass at that point makes it easy to hear sounds. Diaphragm has a lower attachment in back, so fluid accumulates there first.

b. **Move up from posterior bases** - follow a systematic sequence to compare sounds at the same sites on right and left sides. Use patient as their own control. Sounds should be well matched at symmetrical sites. Have pt take one breath through the mouth at each site.

c. Have patient cough if sounds are difficult to hear. Have them exhale forcefully to accentuate wheezing if faint when breathing normally. If crackles are present, determine level, i.e., ¼, ⅓, ½, ¾ of the way up.

d. **Anterior chest**: Anteriorly, start at apex and move down to bases comparing side to side. Listen to apex (above clavicle) once on each side.

e. Position affects sound characteristics. Try to auscultate with the patient in the same position each time so valid comparisons can be made.

F. **Contributors to incorrect prehospital impression or missed diagnoses**

1. Poor equipment that does not amply sounds well
2. Ambient noise
3. Poor auscultation technique
4. Lack of adequate history
5. Wheezes instead of crackles (HF)
6. Crackles isolated to 1 lobe (pneumonia) assumed to be heart failure

G. **Normal breath sounds**

1. **Definition:** Breathing heard through the chest wall of a healthy individual.

2. **Factors which influence normal breath sounds**

a. Distance between the source of the sounds and the chest wall. Ex: Obesity

b. Path of sound transmission. Example: Consolidation

3. **Location**

a. Describe in terms of relationship to bony structures and anatomic landmarks

b. Should hear normal vesicular breath sounds over most of lung periphery

4. **Intensity**

a. Quiet, wispy inspiratory phase followed by a short, almost silent expiratory phase (because of decreasing airflow). These sounds are NOT produced by air moving through terminal bronchioles or alveoli but are caused by transitional and more turbulent flow in the larger airways. Alveoli are a silent zone.

b. **Regional airflow and breath sounds** - Sounds correlate with rate of air flow and are affected by position and auscultatory site. In the upright position, airways at the apex fill earlier than those at the bases. This results in decreased apical intensity as inspiration proceeds. The bases fill later which results in increased intensity in later inspiration.

c. Expiration is much quieter than inspiration, almost inaudible. There should normally be no pause between inspiration and expiration.

5. **Duration**

a. Describe as long or short; continuous or interrupted

b. Normally should hear sound throughout inspiration

c. Inspiration should sound two to three times as long as expiration (I>E) during auscultation. Expiration is shorter due to the reverse in airflow direction and drop in airflow rate and, therefore, sound intensity. The trachea offers little resistance to passage of sound waves. Over the sternum, you should hear equal duration during inspiration and expiration.

6. **Pitch**

a. Definition: Acoustic frequency of sounds that reach the ears, like the pitch of a musical note

b. Usually described as high or low

(1) **Decreased frequency** = decreased pitch
(2) **Increased frequency** = increased pitch

c. Normal breath sounds have a low pitch on inspiration and expiration due to the muffling of high-pitched sounds by the lungs and chest wall tissue. They are described as sighing or gentle rustling.

H. **"Sound matching"**: When two lung media are well matched acoustically, sounds are transmitted readily between them

1. Normal sounds may be modified by obstruction within the respiratory passageways or by changes in the lung parenchyma, the pleura, or the chest wall. Ex.: consolidated lung tissue enhances the transmission of sounds generated in the trachea and bronchi. Sounds are louder than normal with higher frequencies preserved and can be heard throughout the respiratory cycle.

2. Reflection of sounds away from the chest wall is due to a mismatch of acoustical properties of diseased areas and the chest wall. When air or fluid separates the sound source from the chest wall, the sound is reflected inward rather than transmitted to the surface and breath sounds will be diminished or absent at that site. Examples: hyperinflated lungs, pneumothorax, hemothorax, or pleural effusion. Even a shallow pneumothorax will reflect sounds. Sounds will not return until all air is absorbed and pleurae are in contact again.

I. **Abnormal breath sounds**

1. **Bronchial breath sounds**

a. Sounds with little filtered loss; resemble tracheal breath sounds; or air blowing through a hollow pipe

(1) Loud; harsh; high-pitched; increased frequencies preserved

(2) Normally present over trachea and manubrium generated by turbulent air flow in large airways. Remain audible through inspiration and expiration. The inspiratory phase is usually louder and the expiration phase is longer (I<E) (1:2). A distinct pause can be heard between inspiration and expiration.

(3) Not normally heard over the thorax in those at rest (can be heard after exercise). Always considered abnormal if heard over peripheral lung fields - indicates abnormal sound transmission.

b. May be heard over an airless upper lobe whether the bronchus is patent or obstructed. This is because the mediastinal surface of the upper lobe is in contact with the trachea, which permits sound transmission directly to lung tissue. Conversely, there is no direct path of sound transmission to the lower lobes, so tracheal sounds are not heard in the lower lobes unless the intervening bronchi are patent. Thus, bronchial breathing is absent if lower lobe is consolidated or atelectatic as a result of bronchial obstruction.

c. Suggest the following conditions:

(1) Consolidation
(2) Atelectasis
(3) Pulmonary fibrosis
(4) Pneumonia
(5) Top of a pleural effusion

2. **Diminished and/or absent breath sounds**: Most sinister of all

a. Usually associated with decreased air flow as occurs in poorly expanded lung fields (basal lung collapse) seen in COPD, severe airway obstruction or atelectasis or in the presence of air-filled or fluid-filled pleural spaces such as in a pneumothorax, hemothorax or pleural effusion. Reflection of sounds away from the chest wall is due to a mismatch of acoustical properties of diseased areas and the chest wall. Even a shallow pneumothorax will reflect sounds. Sounds will not return until all air is absorbed and the pleurae are in contact again.

b. **Localized**: pneumo/hemothorax, pleural effusion, occluding lung cancer, airway obstruction

c. **Generalized**: obesity, thick chest wall, COPD, severe asthma attack

3. **Abnormalities in transmitted voice sounds (Nice to know only)**

a. **Bronchophony** - Heard over areas of consolidation or atelectasis. To assess for bronchophony, ask the patient to repeat the words, "99" several times. Over normal lung tissue, the transmitted sounds will be unintelligible but over a consolidated area, the voice sounds will be clear because the consolidated lung tissue enhances the sound rather than muffles it.

b. **Whispered pectoriloquy**: Another of consolidation or atelectasis. To access for this, ask the patient to whisper the words "1-2-3". Over normal lung tissue the transmitted words will be unintelligible, but over a consolidated area, the words will be clear because higher frequency vowel sounds are being transmitted. The opposite effect or diminishing of voice sounds is expected when fluid or air separates the lung from the chest wall. Ex: COPD, pneumothorax or pleural effusion.

c. **Egophony**: Another sign of pulmonary consolidation as in lobar pneumonia. To access, ask the patient to repeat the letter "E" several times. Over normal lung tissue, the transmitted letter will sound as it usually does; but over a consolidated area, the sound will change to "A" and have a bleating or nasal quality.

J. **Adventitious sounds**: Sounds that are super-imposed on normal breath sounds

1. **Crackles** (formerly called rales)

a. The pattern of crackles typically changes over different areas of the lung depending on the cause. Described according to pitch, intensity, timing and location which give clues to the underlying pathology.

(1) **Fine crackles** (formerly called rales): **Etiology:** inhaled air collides with a deflated or obstructed airway and the airway suddenly pops open, equilibrating the pressures on either side of the obstruction resulting in transient vibrations in the airway wall. This creates a short explosive discontinuous sound when the

airway bursts open. The dynamic airway obstruction can be caused by accumulation of secretions within the airway lumen or by airway collapse caused by pressure from inflammation or edema in surrounding pulmonary tissue.

(2) **Coarse crackles -** Loud, low pitched, bubbling or gurgling sounds that starts in early inspiration and may extend into the first part of expiration. They sound like the opening of a Velcro fastener. The inhaled air bubbles through secretions in the trachea and large bronchi, dilated bronchi as in bronchiectasis or in pulmonary cavities. These sounds occur in such conditions as pulmonary edema, pneumonia, pulmonary fibrosis, and terminally ill with a depressed cough reflex.

(3) **Crackles are more common during inspiration than expiration**

(a) Crackles can be heard during inspiration when intrathoracic negative pressure results in opening of the airways or on expiration when thoracic positive pressure forces collapsed or blocked airways open.

(b) **Origin of crackles correlated to phase of inspiration**

(i) Early: Small airway disease as in bronchiolitis

(ii) Middle: Pulmonary edema

(iii) Late: Variable intensity, basilar and symmetrical caused by gravity-related traction on small peripheral airways that produce sudden opening of collapsed peripheral airways and adjoining alveoli. Characteristic of fibrosing alveolitis (fine-rubbing hair between the fingers), interstitial pulmonary edema (medium), atelectasis in poorly ventilated areas; or bronchial secretions in COPD, pneumonia, lung abscess, and tubercular lung cavities (coarse).

(4) No change from coughing; secondary to LV heart failure, early pneumonia or pulmonary fibrosis. Crackles secondary to atelectasis or secretions in the large airways usually clear with coughing or suctioning, but will often reappear.

(5) **Conditions that may present with crackles**

(a) LV failure w/ pulmonary edema (generalized & symmetrical)
(b) Poorly ventilated areas of atelectasis (localized or generalized)
(c) Localized over early or non-consolidating pneumonia
(d) Pulmonary fibrosis, tubercular lung cavities, lung abscess
(e) Terminally ill w/ depressed cough reflex

Posted by Dr. Inthush Kavisha on Friday, March 19, 2010

2. **Wheezes**

a. **Definition:** Continuous, harmonic, musical sounds that are most commonly heard at the end of inspiration or early expiration.

b. **Pathology:** Wheezes imply decreased airway diameter either due to smooth muscle spasm and/or swelling of reactive airway walls or collapse of airways due to pressure from surrounding pulmonary disease.

(1) Wheezes are produced when air passes rapidly through a bronchus so narrowed that it is almost closed. They result as a collapsed airway lumen gradually opens during inspiration or gradually closes during expiration. As the airway lumen becomes smaller, the air flow velocity increases resulting in harmonic vibration of the airway wall.

(2) Bronchioles are sensitive to any type of irritation. When interstitial water collects around terminal bronchioles and irritation occurs (as in HF), bronchioles constrict in response to irritation as well as external airway compression from interstitial water.

c. **Described according to pitch, duration, location, timing, complexity**

(1) **Wheezes (Sibilant)-**a high-pitched, musical sound heard in narrowed airway diseases (asthma and chronic emphysema).

(2) **Wheezes (Sonorous)-**(formerly called rhonchi): Low-pitched, coarse, loud, low snoring or moaning sound. Heard primarily during expiration, but may also be heard during inspiration. Mechanism: narrowing of lg airways or obstruction of bronchus.

(3) **Location:** Wheezes produced in the large central airways are usually audible without a stethoscope at the mouth. Those produced at the peripheral airways are audible with a stethoscope only. A fixed bronchial obstruction, most commonly due to an aspirated FB or lung cancer, may cause localized wheeze with a single musical note that does not clear on coughing.

(4) **Timing:** Tend to be louder on expiration because lower airways normally dilate during inspiration and narrow on expiration.

Expiratory polyphonic wheezes: Typically associated with widespread airflow obstruction; characteristic of collapsed and weakened bronchi. Example: COPD. During expiration, recoil pressure falls and resistance in smaller airways increases. Bronchial walls fluctuate due to rapid airflow. Each affected bronchus contributes a differently pitched sound that continues until the end of expiration. If such wheezes disappear suddenly in a patient with COPD, suspect ventilatory failure.

d. **Disappearance of wheezes:** Severity of wheezes **does not correlate well w/ degree of airway obstruction.** Assess capnography to determine adequacy of ventilations. Wheezes in asthma usually dissipate with bronchodilator therapy. But they may also disappear if bronchoconstriction becomes so severe that airflow velocity diminishes below level necessary to produce audible sounds. Correlate the disappearance of wheezes with a patient's ability to move air by assessing their ability to speak in complete sentences without gasping for breath.

e. **All that wheezes is not asthma. Wheezing differential**

(1) **A**: Asthma
(2) **S**: Stasis: Pulmonary embolism
(3) **T**: Toxins/inhaled irritants
(4) **H**: Heart: HF; “cardiac asthma”
(5) **M**: Mechanical obstruction, FB, cancer
(6) **A**: Allergy/aspiration
(7) **TIC**: Trauma, infection, chronic (COPD)

f. **Significance:** Important to distinguish wheeze from inspiratory stridor.

3. **Pleural friction rub**

a. Produced when inflamed pleurae move over one another - associated with pleuritic pain. Sounds like creaking leather or like walking on snow.

b. Best heard with the stethoscope diaphragm over areas of inflamed pleura in pulmonary thromboembolism, pneumonia and pulmonary vasculitis. May be heard only on deep breathing at the end of inspiration and beginning of expiration.

c. If the pleura adjacent to the pericardium is involved a pleuropericardial rub may also be heard. Pleural friction rubs disappear if an effusion separates the pleural surfaces.

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

4. **Stridor**

   a. Intense continuous monophonic crowing sound heard loudest over extrathoracic airways. Indicates significant partial upper airway obstruction. They tend to be accentuated during inspiration when extrathoracic airways collapse due to negative internal lumen pressure. (Upper airways tend to collapse on inspiration. Lower airways tend to collapse on expiration).

   b. Can often be heard without the a stethoscope. Careful auscultation with a stethoscope can usually identify an area of maximum intensity associated with airway obstruction. Typically at the larynx or at the thoracic inlet.

   c. Extrathoracic sounds can be referred down the airways and can be heard over the thorax and are often mistaken as pulmonary wheezes.

5. **Gurgling oropharygeal secretions**

   a. "Although the exact origins of the term "death rattle" are not clear, in 1853, Barclay attributed it to Laennec, who also described "gargouillement" (gurgling). Laennec initially used the term "rale" (French for rattle) in his writings but recognized it might frighten patients familiar with the term "death rattle."

   b. In 1859, Thomas Inman indicated that "…an abundant perspiration, and the 'death rattle' in the throat, have long been recognized in many diseases as immediate harbingers of death."

   c. Gurgling does not fit authoritative descriptions of crackles or wheezes. While gurgling has been appreciated by clinicians for centuries, its technical features and pathophysiology have not been well studied.

   d. Not all seriously ill patients who gurgle die; that this finding can be associated with hospital acquired pneumonia (HAP)" (Vasquez et al, 2010).

VI. **Accurate documentation**: Lung sounds must be documented in all patients with a chief complaint or physical exam evidence of ventilatory, respiratory, or cardiovascular disease or distress. Note the underlying sounds as normal, diminished, or absent for the both the left and right sides and the location and timing of any adventitious sounds.

References

Aehlert B. (2010). Patient assessment. Paramedic Practice Today Above and Beyond Vol. 1 (578-630). St. Louis: Mosby JEMS.

Alexander, M.A., Mistovich, J.J. & Karren, K.J. (2010). Assessment. In Alexander et al, Prehospital Emergency Care (9th ed, Instructor's wraparound edition) (320-345; 373-405). Boston: Brady.

Anderson, S. (2011). Behind the mask; is oxygen harming your patient? JEMS, 36(7), 51-66

Austin, M., Wills, K., Blizzard L. et al. (2010). Effect of high flow osygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting randomized controlled trial. *British Medical Journal*, 341:c5462.

Cabello, J., Burls, A., Emparanza, J. et al. (2010). Oxygen therapy for acute myocardial infarction. Cochrane Database of Syst. Rev., 6: CD007160.

Davis, P., Tan, A., O'Donnel, C., et al. (2004). Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. *Lancet*, 364(9442), 1329-1333.

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

Kattwinkel, J., Perlman, J., Aziz, K. et al. (2010). Neonatal Resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*, 122(18 Suppl 3), S909-919.

Kilgannon, J., Jones, A., Shapiro, N. et al. (2010). Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality. *JAMA*, 303(21), 2165-2171.

Kleinschmidt, P. (2011). Chronic obstructive pulmonary disease and emphysema. Accessed on line: www.medscape.com, Updated: Jan 4, 2011.

Miller, C. (Dec. 8, 2011). P.E.N.M.A.N.: An EMS acronym you should know. Accessed on line: www.EMS1.com.

O'Connor, R., Brady, W., Brooks, S. et al. (2010). Part 10: acute coronary syndromes: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*, 122(18 Suppl 3), S787-817.

Peberdy, M., Callaway, C., Neumar, R. et al. (2010). Part 9: Post-cardiac arrest care. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*, 122(18 Suppl 3), S768-786.

Sanders, M.J., McKenna, K. (2012). History taking. In Sanders et al, *Mosby's Paramedic Textbook* (4th ed) (491-502). St. Louis: Elsevier Mosby JEMS.

Sanders, M.J., McKenna, K. (2012). Primary assessment. In Sanders et al, *Mosby's Paramedic Textbook* (4th ed) (503-511). St. Louis: Elsevier Mosby JEMS.

Sanders, M.J., McKenna, K. (2012). Secondary assessment. In Sanders et al, *Mosby's Paramedic Textbook* (4th ed) (512-559). St. Louis: Elsevier Mosby JEMS.

Sanders, M.J., McKenna, K. (2012). Clinical decision making. In Sanders et al, *Mosby's Paramedic Textbook* (4th ed) (560-567). St. Louis: Elsevier Mosby JEMS.

Vazquez, R., Gheorghe, C., Ramos, F., Dadu, R., Amoateng-Adjepong, Y., & Manthous, C. (2010). Gurgling breath sounds may predict hospital-acquired pneumonia *Chest, 138* (2), 284-288 DOI: 10.1378/chest.09-2713

Differentials to consider	
CARDIAC	**PULMONARY**
PMH	
Cardiovascular disease (CVD), CAD, MI, hypertension (HTN); acute coronary syndrome (ACS); heart failure (HF), cardiomyopathy, high cholesterol, ICD, pacemaker, dysrhythmias Peripheral vascular disease (PVD); valve disease Stroke or TIA ; diabetes; renal disease No Hx respiratory problem + cardiac risk factors, drug abuse; alcoholism	Asthma/COPD (emphysema, chronic bronchitis) Hx allergies; exposure to trigger or known allergen Pulmonary embolus risk factors: venous stasis, damage to vessels, prone to clotting (hypercoagulability) Pneumothorax; Pulmonary effusion; Pulmonary hypertension Lung cancer; Tuberculosis Smoking, work-related exposures
Medications – see full chart below	
ACEIs: “prils” ; ARBs: “sartans” Beta blockers: “lols” ; Ca Blockers; antiarrhythmics Drugs for high cholesterol: “statins” Diuretics; Vasodilators; Anticoagulants	Short or long-acting beta agonists Anticholinergics; Leukotriene modifiers Monoclonal antibodies; Steroids Methylxanthines; erectile dysfunction drugs; home oxygen
Clinical differentials	
Fatigue; DOE to dyspnea at rest; upright, orthopnea, PND; frequent urination at night	DOE to dyspnea at rest; upright, orthopnea, tripoding, use of accessory muscles, pursed lip breathing
Crude bedside test for distinguishing COPD from HF is peak expiratory flow rate. Peak flows > 200 mL indicate a probable HF exacerbation.	If patients blows 150-200 mL or less, they are probably having a COPD exacerbation. Pts with asthma often have peak flow monitors at home.
Capnograph: square waveform	Sharkfin waveform
12 L ECG: Abnormal (acute MI, LVH, ischemia, BBB, “age-undetermined infarct)	P pulmonale pattern
Heart sounds: S3; S4 heart sounds suggests HF	

Differential for SOB				
S&S	**HF/PE**	**AMI**	**COPD**	**Pneumonia**
SOB	+	+	+	+
Cough	-/+	-	+ / early am	+
Sputum	Clear/white; frothy (pink)	-	Chronic bronchitis: 10 mL green or yellow sputum daily	Yellow/green/rusty
Fever	-	-	-	+/ chills
Skin	+ Cold/moist	+ Cold/moist	-	+ / Hot
JVD/Peripheral edema	+ (RH)	-	+ Cor Pulmonale	-
Chest pain	-	+/-	-	+/-
Chest pain nature	-	Heavy, tight	-	Sharp, pleuritic
Chest pain duration	-	Varies; usually > 20 min	-	Gradually worsening, then constant
Smoking Hx	+ Risk	+ Risk	Almost always	+/-
Hx HTN	+ Risk	+ Risk	-	-
Cyanosis	+/-	+/-	+/-	+/-
Air entry to lungs	Good upper/ worse at bases	Good	Poor	Patchy
Wheezing	+/-	+/-	Must have air entry to wheeze	+/- patchy
Crackles	+	+ with HF/ otherwise clear	+ chronic bronchitis	+ patchy; isolated to infected lobes
Hemodynamics: BP	Hyper/hypodynamic ↑ is a risk factor; ↓ if poor CO	↑ is a risk factor; ↓ if poor cardiac output	Usually unaffected; ↓ if severe air trapping Pulsus paradoxus	Usually unaffected unless dehydrated
Pulse	Pulse deficits if v. fast or v. slow HR or ectopics Weak w/ poor CO	Pulse deficits if v. fast or v. slow HR, dysrhythmias or ectopics	Tachycardia	Tachycardia

Cardiac meds	Respiratory meds
Ace inhibitors ("Prils"): benza***pril*** (Lotensin) captopril (Capoten) enalapril (Vasotec) fosinopril / monopril lisinopril / Prinivil / Zestril moesipril / Univasc perindopril / Aceon quinapril / Accupril ramipril / Altace trandolapril / Mavik	**Short acting beta agonists**: albuterol (Proventil, Ventolin) bitolterol (Tornalate) isoetharine (Bronkosol, Bronkometer) levalbuterol (Xopenex) metaproterenol (Alupent, Metaprel) pirbuterol (Maxair) terbutaline (Brethaire, Brethine, Bricanyl)
Angiotensin Receptor Blockers (ARBs): "Sartans" cande***sartan***/Atacand eprosartan/Teveten irbesartan/Avapro losartan/Cozaar olmesartan/Benicar telmisartan/Micardis valsartan/Diovan	***Long-acting beta2-agonists (LABAs)*** salmeterol xinafoate (Serevent) formoterol (Foradil/Foradile)
Antiarrhythmics (See chart below): Quinidine, procainamide, disopyramide, lidocaine, phenytoin, mexiletine, tocainide, flecainide, propafenone, moricizine, adenosine, amiodarone ibutilide, dofetilide, dronedarone, magnesium sulfate, digoxin	**Anticholinergics** ipratropium bromide (Atrovent), ipratropium with albuterol, tiotropium (Spiriva)
Beta Blockers "lols" acebutolol (Sectral) atenolol (Tenormin) betaxolol (Kerlone) bisoprolol (Zebeta) carvedilol (Coreg) labetalol (Normodyne, Trandate) metoprolol (Lopressor/Toprol) nadolol (Corgard) pembutolol pindolol propranolol (Inderal) timolol (Blocadren) sotalol (Betapace)	**Steroids:** triamcinolone acetonide (Azmacort), beclomethasone (Vanceril, Beclovent, Beconase), budesonide (Pulmicort Turbuhaler)), budesonide with formoterol, flunisolide (AeroBid), fluticasone propionate (Flovent); fluticasone with salmeterol, mometasone.
Ca channel blockers: amlodipine/Norvasc felodipine (Plendil) nicardipine (Cardene) verapamil (Calan, Verelan)	**MAST cell inhibitors** cromolyn sodium (Nasalcrom, Intal; Opticrom) nedocromil sodium (Tilade)
Diuretics: amilor***ide***/Midamor bumetanide/Bumex chlorothiazide/Diuril/Diazide furosemide/Lasix hydrochlorothiazide/Hydrodiuril indapamide/Lozol metolazone/Zaroxolyn Polythiazide spironolactone/Aldactone Torsemide trianterene/Dyrenium	**Leukotriene modifiers**: Singulair (montelukast); Accolate (zafirlukast); Zyflo (zileuton)
Statin drugs for high cholesterol: atorvastatin (Lipitor) fluvastatin (Lescol) lovastatin (Mevacor) pravastatin (Pravachol) simvastatin (Zocor) rosuvastatin (Crestor)	**Immunomodulators:** Monoclonal antibodies - omalizumab (Xolair)
Vasodilators: hydralazine/Apresoline, isosorbide/Isordil, minoxidil/Loniten, nesiride/Natrecor, nitrates/NTG	**Methylxanthines:** aminophylline
Anticoagulants: Heparin, low molecular weight heparin, clopidogrel (Plavix), warfarin (Coumadin), rivaroxaban (Xarelto), dabigatran (Pradaxa), ASA	**Erectile dysfunction drugs** – sometimes prescribed to treat pulmonary hypertension: Cialis and Viagra

Singh Vaughan Williams classification system of antidysrhythmic agents

The Singh Vaughan Williams classification, introduced in 1970, is based on the work of Bramah N. Singh in his doctoral thesis at Oxford where Vaughan Williams was his advisor and on subsequent work by Singh and his colleagues in the United States. Its dependence on primary drug mechanism is one of the limitations since many antiarrhythmic agents have multiple actions. Example: Amiodarone has effects consistent with all of the first four classes. Another limitation is the lack of consideration for the effects of drug metabolites. Procainamide, a class Ia agent, has a metabolite, N-acetyl procainamide (NAPA, with a class III action. Originally, drugs such as digoxin and adenosine – important antiarrhythmic agents – were not included This has since been rectified by the inclusion of class V.

Class I agents interfere with the sodium (Na^+) channel
Class II agents are anti-sympathetic nervous system agents. Most agents in this class are beta blockers.
Class III agents affect potassium (K^+) efflux
Class IV agents affect calcium channels and the AV node
Class V agents work by other or unknown mechanisms

Class	Known as	Examples	Mechanism	Clinical uses
Ia	Fast-channel blockers-affect QRS complex	Quinidine Procainamide Disopyramide	(Na^+) channel blocker (intermediate association/dissociation)	• Ventricular arrhythmias • Prevention of paroxysmal recurrent atrial fibrillation (triggered by vagal overactivity) • Procainamide in Wolff-Parkinson-White syndrome
Ib- Do not affect QRS complex		Lidocaine Phenytoin Mexiletine Tocainide	(Na^+) channel block (fast association/dissociation)	• Treatment and prevention during and immediately after myocardial infarction, though this practice is now discouraged given the increased risk of asystole • Ventricular tachycardia • Atrial fibrillation
Ic		Flecainide Propafenone Moricizine	(Na^+) channel block (slow association/dissociation)	• Prevents paroxysmal atrial fibrillation (AF) • Treats recurrent tachyarrhythmias of abnormal conduction system. • Contraindicated immediately post-MI
II	Beta-blockers	Propranolol Esmolol Timolol Metoprolol Atenolol Bisoprolol	Beta blocking Propranolol also shows some class I action	• Decrease myocardial infarction mortality • Prevent recurrence of tachyarrhythmias
III		Amiodarone Sotalol Ibutilide Dofetilide Dronedarone E-4031	K^+ channel blocker Sotalol is also a beta blocker[2] Amiodarone has Class I, II, and III activity	• Wolff-Parkinson-White syndrome • (sotalol:) ventricular tachycardias and AF • (Ibutilide:) atrial flutter and AF
IV	Slow-channel blockers	Verapamil Diltiazem	Ca^{2+} channel blocker	• Prevent recurrence of PSVT • Reduce ventricular rate in pts with AF
V		Adenosine Digoxin Magnesium sulfate	Work by other mechanisms (Direct nodal inhibition, replace deficient electrolyte)	Used in supraventricular arrhythmias, especially in heart failure with AF, contraindicated in ventricular arrhythmias. Magnesium used in Torsades de Pointe.

For more information see: http://en.wikipedia.org/wiki/Antiarrhythmic_agent

<table>
<tr><th>Emphysema</th><th>Chronic bronchitis</th></tr>
<tr><td>Chief complaint: Dyspnea on exertion (early stages); at rest (late). Severe exercise intolerance. The patient's subjective feeling of distress may be the most accurate indication of their illness severity as the other S&S may be always present. Weight loss and chronic fatigue from increased energy demands for respiration and pulmonary cachexia.</td><td>Copious production of sputum; usually most plentiful during the morning, but may continue all day.
Early disease: Dyspnea on exertion (DOE) due to airflow obstruction and cough with frequent infections.
Advanced disease: Dyspnea at rest and continuous cough; shortness of breath (SOB). Severe exercise intolerance.</td></tr>
<tr><td colspan="2">PMH: Ask about tobacco use. Report in pack years: # of packs smoked per day times the # of years they have smoked. Medical problems related to smoking (emphysema, chronic bronchitis, lung cancer) usually begin after pt surpasses a 20 pack years, although that can vary significantly.</td></tr>
<tr><td colspan="2">Vital signs
BP: Usually WNL; may progress to pulsus paradoxus
P: Tachycardia; ECG: dysrhythmias; tall peaked P waves: "P Pulmonale" pattern
RR: Tachypnea
1-2 word dyspnea is normal; respiratory effort is markedly increased using accessory muscles. Prolonged expiratory cycle with pursed lip breathing. This creates positive pressure similar to PEEP which prevents collapse of the lower airways by elevating intra-airway pressures allowing more gas exchange to occur and facilitates exhalation of CO_2.
SpO_2: Depends on state of disease; often decreased. Chronic hypoxemia is often severe in chronic bronchitis.
pCO_2: Hypercapnia is common to moderate degree - results from alveolar hypoventilation with ventilation/perfusion mismatch. Increased carbon dioxide levels lead to ↑ ventilation.
Ventilatory drives: When hypercapnia becomes chronic, central chemoreceptors are depressed and peripheral chemoreceptors on aortic arch and carotid bodies (sensitive to ↓ O_2 levels) may take over the ventilatory drive function. Only a small percentage of these patients switch to an oxygen dependent ventilatory drive.
Late in disease: Mental status changes: restlessness, impaired judgment, confusion, stupor, coma</td></tr>
<tr><td colspan="2">HEENT: Usually normal; JVD if Cor Pulmonale
Pulmonary/CV
Barrel chest due to well developed accessory muscles and chronic hyperinflation of the lungs.
Orthopnea: Cannot lie flat, must sit up to breathe. Charted as two pillow or three pillow orthopnea.
Prolonged expiratory phase; suprasternal retractions</td></tr>
<tr><td>Breath sounds: Decreased or diminished in all lung fields unless infection present; then may or may not hear wheezes or crackles depending on the degree of air flow obstruction.</td><td>Coarse crackles due to occlusion of the larger airways with mucus plugs; wheezing.</td></tr>
<tr><td>Occasional cough with small amounts of whitish-gray mucus in the morning.</td><td>Chronic cough; changes in sputum color suggest purulence or respiratory infection.</td></tr>
<tr><td colspan="2">GI/GU: Hepatic congestion if Cor Pulmonale. Assess for hepato-jugular reflux.
Clubbing of the nail beds: The fingertip will enlarge and will lose the normal angle at the nail bed. Normal nail exits bed at a 160° angle. Clubbing causes a 180° + angle. Seen with polycythemia and chronic hypoxemia. Etiology unclear. Thought that capillaries dilate in an effort to bring oxygen to tissues.</td></tr>
<tr><td>Skin: Pink puffer: Polycythemia causes ↑ blood viscosity = ↓ O_2 to tissues. Cyanosis is a late and unreliable sign. Need > 5 Gm desaturated Hb or pO_2 < 40 mmHg.</td><td>Skin: Blue Bloater". If Cor Pulmonale present, patient has right HF with peripheral fluid retention (dependent edema) and is more likely to appear cyanotic.</td></tr>
</table>