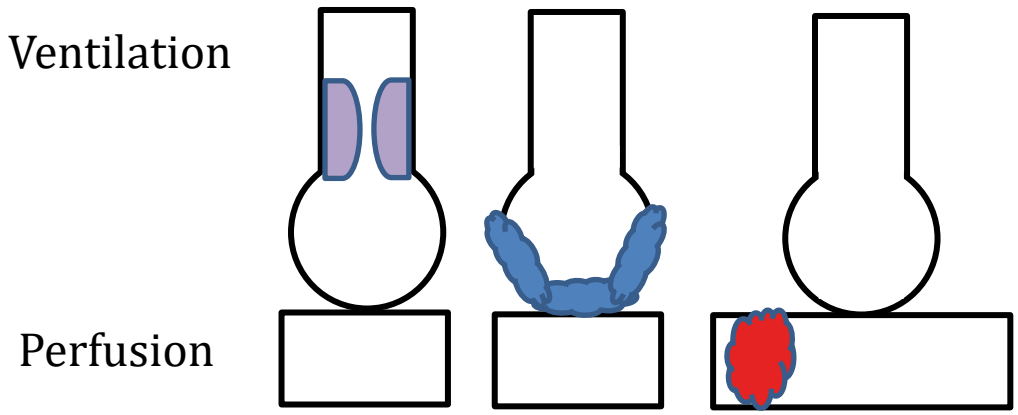
Academic Half Day - Acute Respiratory Failure

Facilitator Guide

Physiology Exercise



	V	Q	Airway Resistance	Compliance	Mv	SA	PCO2	PO2
Asthma	Down	nl	High	Normal	Down	Normal	Up	Down
CHF	nl then down	nl	Normal	Initially normal, then down	Up then down	Down	Down then Up	Down
PE	High	Down	Normal	Normal	Up	Down	Down	Down

Discussion Questions

1. **What is minute ventilation?** Respiratory Rate x Tidal Volume = Minute Ventilation
    a. Normal is 5-8L/min, increases with increased metabolic demand (e.g. sepsis)
2. **What is lung compliance?** Change in volume/change is pressure
    a. This is a measure of how much pressure is needed to change the size of the lung. Wet/edematous lungs have decreased compliance meaning that more pressure is needed to get the same volume expansion as normal lungs.

3. **Why do you not become hypercapnic in CHF exacerbations initially?**
    a. With pulmonary edema, you decrease the ability for gas to diffuse across the membrane – causing both hypoxia and hypercarbia. However, ventilation is dependent on minute ventilation and as your RR increases you will blow off the extra CO2. Oxygenation is dependent on FiO2 and Surface area which do not increase with increased RR. If more edema develops, eventually this will cause decreased lung compliance -> decreased minute ventilation -> hypercarbia.
4. **In which of these pathologies will PEEP help? What is PEEP?**
    a. PEEP is positive end-expiratory pressure. This will maintain pressure in the airways at the end of a breath, stenting open alveoli that have decreased surface tension due to edema.
    b. PEEP will help with the second case (pulm edema/ARDS) but not as much for asthma/COPD or PE.
    c. What is plateau pressure?
        i. This is a pressure measurement made on a vent during a paused breath, when no air is moving. This pressure is entirely dependent on the pressure in the alveoli and the compliance of the lung.
5. **In which of these pathologies with inspiratory pressure support help?**
    a. Pressure support is additional pressure given during a breath to aid with air flow to achieve adequate tidal volumes and minute ventilation. This is helpful in conditions with increased resistance to airflow – obstructive processes such as asthma/COPD.
    b. What is peak inspiratory pressure?
        i. This is a pressure measurement made on a vent during a breath, when air is moving. This pressure is dependent on both the resistance in the airways and the plateau pressure.

Case 1

Mr. Ahtdyshka is a 62 yo male with a history of COPD who presents to the ED with a 2-day history of gradually progressive dyspnea on exertion and increased frequency of his chronic cough which is productive for clear sputum. He denies fevers. He is adherent to prescribed albuterol MDI, salmeterol BID, and tiotropium QD. His albuterol helps his symptoms some. He has a 30-pack year smoking history and currently smokes 0.5 PPD. His apartment complex has been cleaning the AC vents recently.

Vitals: 99.3, 156/87 HR105 RR 26, 91% on 2L (new requirement)

Gen - Uncomfortable, AAOx3
HEENT- PERRL, moist mucous membranes, no oral cavity lesions
CV - Heart sounds distant. Tachycardic, regular, no murmurs, no JVD
Resp - Labored, no wheezing, +accessory muscle use. Able to speak in partial sentences

Abd - soft/nontender/nondistended, +BS
Ext - 2+ radial and DP pulses. No LE edema

Labs:
WBC - 7.3 (normal diff); Hgb- 14.3; Plt- 320
Na - 137, K- 4.1, Cl- 101, HCO_3- 29, BUN- 14, Cr- 1.0
ABG - 7.23/60/62 on 2L (pH/CO2/O2)
CXR: **get CXR here** – have them read it (hyperinflation)

1. **What is the diagnosis and what are the next steps in management?**
    a. Acute on chronic hypercapneic respiratory failure due to COPD exacerbation (trigger of dust exposure?)
    b. Diagnostic criteria = PaCO2>45/50 mmHg & pH≤7.30; associated hypoxemia is common
    c. Start duonebs q4-6h and albuterol q2h PRN, azithromycin, and prednisone 40 mg qday
    d. Do you continue home long-acting Anticholinergics? No evidence to show that they help – okay to stop.
2. **Would you place this patient on NIPPV (non-Invasive positive pressure ventilation)?**
    a. Somewhat of a clinical decision on if you should start NIPPV right away
    b. ATS/ERS 2017 guidelines
        i. Recommend considering bilevel NIV for COPD exacerbation with pH pH≤7.35, PaCO2>45 mm Hg, and RR>20-24 breaths/min when not improving with medical tx
    c. Acuity of respiratory distress & determination of fatigue help decide if try medical tx alone first
    d. What are contraindications to NIPPV? (specifically, in this patient what may be an issue – hypercarbia and AMS)
        i. Cardiac or Respiratory arrest
        ii. Inability to fit or safely place mask (Facial surgery, trauma or deformity, etc.)
        iii. Inability to cooperate or protect airway (Encephalopathy/GCS<10, requiring restraints)
        iv. Inability to clear respiratory secretions
        v. Hemodynamic instability or unstable cardiac arrhythmia
        vi. Severe upper GI bleed
        vii. High aspiration risk (ex: swallowing impairment)
3. **You start Mr. Ahtdyshka on bilevel NIPPV. What are you trying to improve in this patient from a respiratory perspective? Return to physiology - where is the defect in the lungs, how do we use NIPPV to overcome this defect?**
    a. Discuss minute ventilation as the issue here, need to maximize this!

b. Resistance in this patient is coming from the AIRWAYS - need to increase in the inspiratory pressure to maintain an adequate tidal volume to maintain an adequate minute ventilation
c. PEEP is to help splint open the alveoli to increase the surface area of the gas exchange but will NOT help with ventilation as the issue here is with minute ventilation NOT surface area
d. Bi-level has been shown to improve mortality and decrease intubation rates in acute hypercarbic respiratory failure and acute pulmonary edema from heart failure
    i. Physiology: CPAP/BiPAP will increase in the intrathoracic pressure, reducing the venous return to the heart – decreasing preload. In states where a patient is hypervolemic this is akin to giving insta-lasix (copyrighted Dr. Reza) - decreasing preload and getting them closer to the Starling Curve. (in patient's who are pre-load dependent (i.e. inferior MI, right sided heart failure) positive pressure will make them worse!) Additionally, the PEEP will increase surface area for gas exhchange, improving oxygenation.

4. **How soon after initiating this therapy should you check an ABG? What parameters are you hoping to see?**
    a. About 20 minutes. An hour is too long. PubMed for a reference. "This study shows that oxygen equilibration relevant for clinical interpretation requires only 10 minutes following an increase and 16 minutes following a decrease in FIO2" PMID: 23537296
    b. Want to see pH coming up, PCO2 coming down.
    c. If small improvement, pay attention to the minute ventilation in this patient – if the Mv is low, can consider changing the pressure support to get better ventilation prior to considering this a failure if they are clinically stable.
    d. If adjusting, don't forget to check another gas in 20 minutes. ABG vs VBG (expert question)

5. **How long is "too long" in terms of trialing non-invasive ventilation before considering intubation?**
    a. It depends on the patient situation and pathology but in general, significant clinical improvement should be seen by 2 hours. If deteriorating at any time, escalate therapy.
    b. Can use HACOR score - Heart Rate, Acidosis, Consciousness, Oxygenation, Respiratory Rate – at 2 hours after BiPAP started.
        i. In total, 46% of patients ultimately failed NPPV. For predicting need for intubation, a HACOR score of 6 or more was highly predictive of NPPV failure. **Overall, only 18% of patients with a HACOR score ≤5 failed NPPV, compared to 78% with a score of 6 or more.** (https://doi.org/10.1007/s00134-016-4601-3)

His nurse calls you about 25 minutes later because he is having a hard time arousing he. You ask for an ABG as you make your way to the room. Upon arriving at bedside, you find that Mr. Ahtdyshka requires a sternal rub to open his eyes. When awake, he responds but often drifts off. He withdraws to pain in all extremities. The ABG returns with 7.18/70/63 (pH/CO2/O2).

6. **What is your next step?**
    a. This patient is failing BiPAP and now has a contraindication to BiPAP (AMS)
    b. Talk to the MICU fellow. He needs to be intubated and moved to the ICU

Decision to Intubate

What are the Indications for Intubation?

- Respiratory (Hypoxic/Hypercarbic) Failure
- Failure to protect airway – mental status
- Anticipated clinical course

1. Patient is a 76 y/o with a history of cirrhosis, CHF who presented with severe CAP. Initial vitals: 101F, 112, 92/76, RR 32, 82% on NRB.
    a. What is going on with this patient? What are next steps?
    b. Acute Hypoxic Respiratory Failure! One of the indications for intubation
2. Patient is a 23-year-old with a history of severe asthma who presented with an acute exacerbation. On admission patient was AF 130/70 HR 105 RR 29 92% on 2L. VBG 7.36/42. Repeat labs and vitals show VBG 7.32/53 and RR 26.
    a. What is going on with this patient? What are next steps?
    b. Asthma Exacerbation, gasses are not terrible but patient is tiring out, clinical course anticipated -> hypercarbic respiratory failure -> intubation
3. Patient is a 56 y/o with a history of cirrhosis who presented with AMS. Lactulose was ordered but he did not receive any as he was not tolerating PO. In the am you get the following exam: somnolent, not following commands, moans with physical stimulus, withdraws to pain.
    a. What is going on with this patient? What are next steps?
    b. Slightly vague – would definitely evaluate with ICU fellow. But this patient is on the verge of not protecting their airway. Could probably intubate at this time, however this is a eminently treatable process - could also be reasonable to place an NG and give lactulose + lactulose enema to see if there is any improvement.
4. Patient is a 95-year-old with a history of MDS, HFrEF, HTN, HLD, Dementia who presents with respiratory distress, pulmonary edema on CXR, tachypneic to 26, hypoxic and hypercarbic.
    a. What is going on with this patient? What are next steps?

   b. Hypercarbic/Hypoxic respiratory failure – which begs for intubation - however this patient is old with many co-morbidities…. A good goals of care discussion and code discussion is in order.
5. Patient is a 53 y/o with a history of moderate COPD who presented with cough, sputum production, and shortness of breath. Initial VBG showed 7.28/72. Vitals: AF 82 120/70 91% on 4L RR24. Patient's work of breathing is increased.
   a. What is going on with this patient? What are next steps?
   b. This is borderline. It would be reasonable to try BiPAP and follow VBGs/Clinical Picture over the next few hours prior to making that decision.
6. Patient is a 62 y/o with a history of COPD, CAD, HLD, DMII, and HTN who presented with E.Coli bacteremia, with worsening hypotension with a lactic acidosis (AGMA, bicarb 12), and tachypneic to 33, satting 98% on RA.
   a. What are some of the consequences of intubating this patient?
   b. This is cool physiology guys… this patient's minute ventilation is sky high!! He is compensating for the metabolic acidosis, but he is decompensating hemodynamically and needs intubation. Once you intubate, you cannot maintain the same minute ventilation and this patient will get more acidotic! Bicarb drip here you come.

Case 2

Mr. Tenghianafes with a past medical history of MDS, CHF, COPD and distant provoked DVT/PE who presented to the ED and was admitted this afternoon with mild exertional shortness of breath, slowly progressive over the last two weeks and noted to be anemic to Hgb 6.5 from a baseline of 9-10 (6 months ago), no signs of bleeding. *A Rapid Response was called on the floor for hypoxia.*

Vitals: 100.6, 104, 95/65, 72% on 2L RR 25
He is 5'10" (178cm) and 100kg
CXR: **QR**

1. **You are the first senior to respond, what do you do?**
   a. **Ambient air** is 21% oxygen
   b. **Nasal cannula** - for every liter O2/min, add 3-4% to ambient air. Max 6L, at anything above 6L (39-45%), questionable incremental benefit compared to increase in drying and discomfort that goes with it
   c. **Simple Face Mask** - 6-10 L/min, 35-50% FiO2
   d. Venturi Mask (Venti-Mask) – valve on mask control O2 flow delivery, 24% to 60% FiO2
   e. **Non-rebreather mask** – bag reservoir to face mask, 50% up to 100%
   f. **Optiflow** – 21 to 100% FiO2, with flow rate 0-70 L/min. Provides small amount of PEEP

i. Some studies show decreased 90-day mortality and intubation with Optiflow in acute hypoxemic respiratory failure when compared to standard O2 (continuous non-rebreather, etc.)

g. **CPAP / BiPAP** – up to 100% FiO2 (assuming perfect seal)
h. **Mechanical Ventilation** – up to a true 100% FiO2

2. **What could be going on with this patient?**
   a. The CXR has bilateral patchy opacities, pulmonary edema appearing.
   b. No history of heart failure
   c. The patient is in the process of receiving 1u pRBC
   d. ARDSy picture – aspiration, **TRALI**, TACO (less likely given 1u pRBC transfused), HTN urgency with pulm edema?!, MI with ruptured paps?!

Patient is persistently hypoxic on NRB 81%. Mr. Tenghianafes is intubated and started on volume-control continuous mechanical ventilation with a TV of 600 cc, RR 12, FiO2 of 1.0, and PEEP of 5 cm H2O. An ABG shortly after intubation: 7.10/16/73 (pH/CO2/O2)

3. **Does this patient meet criteria for TRALI/ARDS?**
   a. Respiratory symptoms must have begun within one week of a known clinical insult (likely TRALI in this case) **(yes)**
   b. Bilateral opacities consistent with pulmonary edema **(yes)**
      i. Must not be fully explained by pleural effusions, lobar collapse, lung collapse, or pulmonary nodules
   c. Non-cardiac cause **(yes)** (if asked, he got an Echo that was normal)
   d. A moderate to severe impairment of oxygenation must be present, as defined by the ratio of arterial oxygen tension to fraction of inspired oxygen (PaO2/FiO2)
      i. Mild ARDS – PaO2/FiO2 is 200-300 mmHg, with (PEEP) or (CPAP) ≥5 cm H2O.
      ii. Moderate ARDS – PaO2/FiO2 is 100 - 200 mmHg
      iii. Severe ARDS – The PaO2/FiO2 is ≤100 mmHg **(Our patient's is 73!!)**
4. **What changes to the vent settings do you want to make and why?**
   a. **Lower the tidal volume!!!** Low Tidal volume ventilation is to prevent ventilator associated lung injury
   b. The lung is full of fluid non-cardiogenic edema from inflammation, this severely decreases the compliance of the lungs. "Normal" lung volumes would necessitate high filling pressures to inflate the diseased alveoli. However, ARDS is not a homogenous process and some alveoli are normal – when subjected to high pressures they get barotrauma.
   c. 4-6cc/kg of Ideal Body Weight = need to decrease TV to ~440cc
5. **What is the pathophysiology of this person's lungs? What are the important aspects of oxygenating this patient?**
   a. At a normal V/Q, hemoglobin is nearly 100% oxygenated – increase respiratory rate or tidal volume will not change the amount of oxygenation that occurs over edematous alveolus/capillary surfaces. *Your goal is to*

increase the surface area for gas exchange and the O2 partial pressure gradient between the alveolus and capillary bed.

b. Increase Oxygen Exchange Surface Area
    i. **PEEP** - This is to stent open the alveoli, increasing the surface area for gas exchange. High PEEP – but was is too high? Guidelines state above 5 at least, use the SpO2 as a guide – if the patient is hypoxic with an FiO2 of 100%, increase the PEEP, try to get the FiO2 low (as discussed below). *Watch the Plateau pressure (keep <30cmH2O)*
    ii. **Proning** – ARDS lungs are sponges laden with water, gravity will pull the fluid and blood (perfusion) to the bottom worsening the V/Q mismatch. Proning will help decrease the V/Q mismatch temporarily while the edema shifts
        1. Indicated in ARDS patients with PaO2/FiO2 <150
c. **FiO2** – Start at 100% and titrate down. Oxygen is *not* benign and contributes to lung injury by promoting free radicals. People with normal lungs develop tracheobronchitis and decreased vital capacity after breathing 100% FiO2 for 6-12h. Lower the FiO2 below 60% to limit this damage as soon as possible.
    i. This is a good place to discuss shunts again. Given that severe ARDS is essentially a high-shunt state (those alveoli are so full of water that they are not functioning in gas exchange) - increasing FiO2 will have minimal effect on oxygenation without increasing the gas exchange surface area.
d. **Bonus** – inhaled nitric oxide can be used (expert opinion)– how would this help in ARDS? (expert question)

6. **After 30 minutes on the above settings with FiO2 at 1.0, his ABG is 7.28/60/95 (pH/CO2/O2). What adjustments should be made at this time?**
    a. Permissive hypercapnia: With low TV ventilation, high CO2 is okay, goal is to keep pH>7.25
    b. Oxygenation has improved, can lower O2 and see what the minimum O2 is to maintain oxygenation. Try increasing PEEP to improved oxygenation.

Vent Exercise

Objectives:

1. Define the difference between Pressures and Volume Control
2. Go over the different modes of ventilation
3. Define the difference between PEEP vs Inspiratory Pressure
4. Knob-ology