**Academic Half Day: Acute Respiratory Failure**

Facilitator Guide

August 26, 2021

Case 1

Ms. Ersats is a 25-year-old female with past medical history of asthma and allergic rhinitis, who presents to the hospital with 1 hour of shortness of breath and wheezing. She was cleaning her new apartment prior to sudden onset of shortness of breath. She notes chest tightness as well. She used her home Albuterol inhaler with little improvement. She denies chest pain, fever, chills, and sputum production. She does not take any medications regularly but does take fexofenadine occasionally for seasonal allergies. She has never smoked or vaped but does drink alcohol on the weekends.

Vitals:
T 98.6. HR 105. BP 130/70. RR 29. SpO2 92% on 2 L oxygen

Exam:
General: Appears uncomfortable and tachypneic. She is awake, alert, and oriented to person, place, and time.
HEENT: Normocephalic, atraumatic. Mucus membranes are moist. No cervical lymphadenopathy.
Cardiac: Regular rhythm, slightly tachycardic. Well-perfused in extremities. No murmurs appreciated. No edema noted.
Lungs: Diffuses wheezes throughout all lung fields. Decreased air movement throughout all lung fields.
Abdomen: Soft, non-tender to palpation. Normoactive bowel sounds.
Neuro: AAOx3. Moving all 4 extremities spontaneously with 5/5 motor strength. No sensory deficits. Answering questions appropriately.

Labs:
CBC: WBC 7.3, Hgb 13.2, Plt: 320
Renal: Na 137, K 4.1, Cl 101, HCO3 24, BUN 14, Cr 1.0 (baseline 0.9)
ABG: pH 7.48, pCO2 32, pO2 72
CXR: No cardiopulmonary abnormalities

1. What is the diagnosis and what are the next steps in management?
	1. Acute hypoxic respiratory failure due to asthma exacerbation
	2. Supplemental oxygen
		1. Nasal cannula – up to 6 L
		2. How much FiO2 are we able to provide? (40-45%)
	3. Albuterol nebulizers
		1. 2.5 to 5 mg every 20 minutes for 3 doses, then 2.5 to 5 mg every 1-4 hours as needed
		2. Can also use MDI with spacer, or continuous nebulization
	4. Steroids
		1. Prednisone 40 to 60 mg
		2. Solumedrol 125 mg
	5. Magnesium sulfate
		1. Recommended in patients with life-threatening exacerbation or who do not respond to initial therapy

The patient was admitted to medicine. You get a page from the nurse for a diet order. Repeat labs resulted while you were opening the patient’s chart and repeat vitals were obtained:

Vitals:
T 99.1. HR 112. BP 126/70. RR 26. SpO2 92% on 3L oxygen

VBG: 7.38, pCO2 44

You reassess the patient at bedside and the patient continues to appear uncomfortable. She has intercostal retractions. Her nasal cannula is in proper position. She is taking breaths every 3-4 words when answering questions.

1. What is happening with this patient?
	1. Patient is starting to tire out. The blood gas appears to be normalizing, but when you look at it compared to the prior, she is becoming more hypercapnic.
2. What are your next steps?
	1. Intubate the patient vs. NIPPV? Make the learners commit to a side and defend their answer.
		1. The patient is a good candidate for intubation. However, there are new studies (without official recommendations) that indicate that NIPPV in these settings for a short duration of time (~30 minutes) can treat the problem and avoid invasive treatment such as intubation.
		2. Official ATS 2016 Guidelines: Given the uncertainty of evidence we are unable to offer a recommendation on the use of NIV due to asthma
		3. Althoff, M. D. et al. In 2020 indicated a decrease in rates of intubation and in-hospital mortality.
3. What are the indications for intubation?
	1. Unable to maintain airway (AMS, trauma, hematemesis, swelling)
	2. Upper airway obstruction (epiglottitis, swelling)
	3. Suctioning / mucus plugging
	4. Respiratory failure
4. What are contraindications to NIPPV?
	1. Not able to cooperate, protect airway, or clear secretions (never in restraints!)
	2. Decreased level of consciousness
	3. Excess respiratory secretions and risk of vomiting and aspiration
	4. Facial deformity that prevents proper fitting
	5. Not able to be closely monitored (nursing ratios)
5. Where will you treat this patient?
	1. Transfer to the ICU vs. MSD

Ms. Ersats is transferred to the ICU and intubated. Initial vent settings: AC/VC, FiO2 100%, PEEP 8, RR 16, Vt 6 cc/kg IBW.

Case 2

Mr. Proseva is a 66 year old male with past medical history of hypertension, hyperlipidemia, and CKD stage 1. He presented to the hospital 3 days ago for fever, cough, nausea, and vomiting. Initial evaluation was significant for hypoxia requiring 2L NC and otherwise normal vital signs. Workup revealed a positive PCR for Influenza A with a negative COVID test. He was started on Tamiflu and admitted to the wards due to his hypoxia. He was also placed on maintenance fluids due to his recent decreased PO intake. You are cross-covering overnight when a rapid response is called to his room for an SpO2 of 80% on 5L NC.

You arrive to find him in bed, lethargic but arousable. He says his breathing has been getting worse throughout the day.

Vitals: HR 110, BP 100/60, RR 28, SpO2 85% on 6L NC, T 38.5

Exam: General: Lethargic but arousable, tachypneic, middle aged male in mild distress.
HEENT: Normocephalic, atraumatic. Mucus membranes are moist. No cervical lymphadenopathy.
Cardiac: Regular rhythm, slightly tachycardic. Well-perfused in extremities. No murmurs appreciated.
Lungs: Bibasilar crackling with coarse rhonchi R>L
Abdomen: Soft, non-tender to palpation. Normoactive bowel sounds.
Extremities: Pitting edema bilateral LE to mid-shin, edema appears worse on the right. No overt calf tenderness.
Neuro: AAOx3. Moving all 4 extremities spontaneously with 5/5 motor strength. No sensory deficits. Answering questions appropriately.

Labs:
CBC: WBC 19, Hb 13, Plt 330
Renal: Na 136, K 4.8, Cl 103, HCO3 22, BUN 35, Cr 1.9, Glucose 188

Your intrepid co-intern looks up his admission labs and notes they are largely unchanged to now.

ABG: 7.49/32/59 on 5L NC (pH/pCO2/pO2)

1. What are the five causes of hypoxemia?
	1. Hypoventilation (opiates, MG, hypophosphatemia, etc.)
	2. V/Q mismatch (COPD, pulmonary hypertension, interstitial lung disease)
	3. Right-to-left shunting
		1. Anatomic shunts: pulmonary AVMs, intracardiac shunts
		2. Physiologic shunts: atelectasis, pneumonia, ARDS
	4. Diffusion restriction (pulmonary fibrosis)
	5. Reduced inspired oxygen tension (high altitudes)
2. What is the A-a gradient and how is it impacted by each cause of hypoxemia?
	1. It is the difference between oxygen concentration in the Alveoli and the arterial system. It assesses the degree of shunting and mismatch.
	2. Elevated in hypoxemic respiratory failure due to diffusion restriction, V/Q mismatch, and shunting. It is normal in low FiO2 states and hypoventilation
	3. PAO2 is calculated, paO2 is measured
		1. PAO2 = FiO2 x (Patm – 47) - (PaCO2/0.8) = FiO2 x 713 - (PaCO2/0.8)

Learners will have this chart to fill out for these questions:

|  |  |  |  |
| --- | --- | --- | --- |
| Cause of Hypoxemia | Examples | Effect on A-a gradient | Corrects with supplemental O2? |
| Hypoventilation | Opiate overdose | Normal | Yes |
| V/Q Mismatch | COPD | Elevated | Yes |
| Shunting | ARDS | Elevated | No |
| Diffusion Restriction | Pulmonary Fibrosis | Elevated | Yes |
| Reduced FiO2 | High Altitudes | Normal | Yes |

1. What is on your differential?
	1. ARDS
		1. Berlin criteria:
			1. Timing: Within 1 week
			2. Imaging: Bilateral diffuse parenchymal opacities
			3. Etiology: NOT secondary to heart failure
			4. Hypoxia: P/F ratio <300
		2. Most likely given known diagnosis of influenza
		3. Could be related to progression of influenza or bacterial superinfection
	2. Cardiogenic pulmonary edema (be sure to specify)
		1. No known history of heart failure
		2. Did receive fluids as part of resuscitation
	3. Pulmonary embolism
		1. Asymmetric calf swelling- no pain, but certainly possible
		2. Always want to make sure patient is getting appropriate DVT prophylaxis
2. What do you want to order?
	1. Oxygen
		1. Ambient air is 21% oxygen (FiO2 0.21). For every increase in 1 L oxygen, FiO2 increases by ~4%.
		2. Nasal cannula: max 6 L (40-45%). Anything above 6L, there is questionable incremental benefit compared to increase in drying and discomfort that goes along with it. This can be used on the floor (max for floor is 6 L ABOVE BASELINE O2 REQUIREMENT).
		3. High flow nasal cannula (green tubing): max 15 L (60-70%). Will be used in step down unit if 6 L above patient’s baseline.
		4. Simple Face Mask: 0-15 L (60-70%). Used mostly in the OR weaning oxygen post-procedure
		5. Venturi Mask: 0-15 L (24-50%). Has side ports that allows for mixing of oxygen and ambient air, which is why the FiO2 caps at 50%. Requires step down level of care.
		6. Non-rebreather: 15L (~85%). Has a one-way valve from reservoir, one way exhalation valve, and one open side port. Used for acute desaturations and rapid responses.
		7. Optiflow/Vapotherm: 15-80 L and 21-100%. Flow and FiO2 are set separately. Require step down or ICU. Use for severe hypoxia. Improves PEEP to help with increased work of breathing. Useful in cases of RV failure and pulmonary hypertension. Used as a bridge to intubation for COVID patients. Keep in mind that in the literature (specifically with COVID guidelines) high-flow nasal cannula typically means Optiflow/Vapotherm.
	2. CXR vs. CT chest. Is your clinical suspicion for PE high enough for CTPA? Do you empirically start anticoagulation? If so, what would you start? Guide the learners though this thought process.
	3. Antibiotics
		1. Which microbes are you covering?
			1. Hospital-acquired pneumonia --> Staph, Gram negatives (including Pseudomonas)
		2. Which antibiotics to cover these microbes?
			1. MRSA coverage? Yes, due to >20% incidence in our hospital. Vancomycin vs. Linezolid
			2. Gram negatives --> Cefepime, Meropenem, Piperacillin-Tazobactam, etc.
	4. ECG? Troponin? BNP?
	5. Lasix? Do you think he is volume-overloaded?
3. Where would you send this patient?
	1. MSD vs. ICU

Imaging:



Mr. Proseva is subsequently transferred to the ICU and intubated.

**Take a break and ask the ICU attending any questions while the other ICU attending, Dr. Strange, is helping the fellow intubate your patient.**

Initial vent settings: AC/VC, FiO2 100%, PEEP 8, RR 12, Vt 6 cc/kg IBW. Now that the multiverse has been unraveled by Dr. Strange, you find yourself in an alternate reality in which you are the ICU resident taking care of both Ms. Ersats and Mr. Proseva. Mr. Proseva’s ABG shortly after intubation results: 7.15/90/70.

1. What changes to the vent settings do you want to make and why?
	1. Increase the respiratory rate!
	2. Respiratory rate and tidal volume impact ventilation (removing CO2) whereas PEEP and FiO2 impact oxygenation.
2. What is the pathophysiology of this patient’s lungs? What are the important aspects of oxygenating this patient?
	1. 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.*
	2. Increase Oxygen Exchange Surface Area
		1. 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)*
		2. 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. Ask the learner when they would consider proning for this patient
				1. PEEP 10-12
				2. FiO2 --> no strict cutoff, but thinking 50-60%
				3. P:F ratio <150
				4. How long would you prone the patient?

At least 16 hours per the Proseva trial

* 1. 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.
		1. 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.
	2. Bonus – inhaled nitric oxide can be used (expert opinion)– how would this help in ARDS? (expert question)
1. 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?
	1. Permissive hypercapnia: With low TV ventilation, high CO2 is okay, goal is to keep pH>7.25
	2. Oxygenation has improved, can lower O2 and see what the minimum O2 is to maintain oxygenation. Try increasing PEEP to improved oxygenation.

The respiratory therapist alerts you that both patients (conveniently located in adjacent beds) are having peak pressure alarms on their ventilators.

1. Why do airway pressures change on volume control ventilation?
	1. The ventilator is being told to deliver a set tidal volume. As the patient’s respiratory mechanics change, the pressure required to deliver the volume changes
2. What components of respiratory mechanics comprise a peak airway pressure?
	1. Airway resistance (the ET tube, ventilator circuit, trachea, and bronchi)
	2. Lung compliance (the patient’s lung parenchyma/chest wall)
	3. PEEP (set by you, the astute physician)
3. What maneuver on the ventilator can be done to help differentiate between the above changes?
	1. You can perform an inspiratory hold. Ask the learners how to do this --> Ask the RT to perform an inspiratory hold by pressing the inspiratory hold button on the ventilator.
	2. Increase in airway resistance increases PEAK pressure only
	3. Drop in lung compliance increases PLATEAU and PEAK both
	4. Increase in PEEP will increase all three pressures

Case 1:  Case 2: 

1. What is the differential for a high-pressure alarm with a wide delta?
	1. Biting the tube
	2. Mucus plugging, airway secretions
	3. Bronchospasm
	4. Foreign body
2. What is the differential for a high-pressure alarm with a narrow delta?
	1. Edema
	2. Atelectasis
	3. Pneumonia
	4. Pneumothorax
	5. Mainstem intubation
3. Why does Case 1 have a different pattern than Case 2?
	1. Case 1 reflects a severe asthma exacerbation progressing into status asthmaticus and the high-pressure alarm likely represents bronchospasm- an airway RESISTANCE problem. Her lungs and chest wall are otherwise fine.
	2. Case 2 reflects ARDS, which would impact COMPLIANCE of the lungs. His airway resistance should be normal, so the gap between his peak and plateau pressures are normal.
4. What adjustments do you want to make to Case 1?
	1. Decrease Vt (if pH/pCO2 allow)
	2. Albuterol
	3. Magnesium
	4. Ketamine?
	5. Paralysis (as little as possible, ALWAYS with sedation!)
	6. ECMO consultation --> transfer to CVICU
5. What adjustments do you want to make for Case 2?
	1. Decrease Vt (if pH/pCO2 allow)
		1. 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.
		2. 4-6cc/kg of Ideal Body Weight
	2. Increase inspiratory time (same volume over longer should take less pressure)

You sign out and go home for the day.

Case 3

Mr. Gold 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:
T 99.3. BP 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, HCO3- 29, BUN- 14, Cr- 1.0

ABG - 7.23/60/62 on 2L  (pH/CO2/O2)
CXR: No acute cardiopulmonary process

1. What is the diagnosis and what are the next steps in management?
	1. Acute on chronic hypercapnic respiratory failure due to COPD exacerbation (trigger of dust exposure?)
	2. Diagnostic criteria = PaCO2>45/50 mmHg & pH≤7.30; associated hypoxemia is common
	3. Start duonebs q4-6h and albuterol q2h PRN, azithromycin, and prednisone 40 mg qday
	4. Do you continue home long-acting Anticholinergics? No evidence to show that they help – okay to stop.
	5. NIPPV
	6. What is your SpO2 goal? ,
		1. Discuss why the patient should not be 96-100% (decrease the respiratory drive, but more importantly worsens V/Q mismatching)
2. You start Mr. Gold 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?
	1. Discuss minute ventilation as the issue here, need to maximize this!
	2. 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
	3. PEEP is to help splint open the alveoli to increa-se the surface area of the gas exchange but will NOT help with ventilation as the issue here is with minute ventilation NOT surface area
	4. Bi-level has been shown to improve mortality and decrease intubation rates in acute hypercarbic respiratory failure and acute pulmonary edema from heart failure
		1. 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.
3. How soon after initiating this therapy should you check an ABG? What parameters are you hoping to see?
	1. 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
	2. Want to see pH coming up, PCO2 coming down.
	3. 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.
	4. If adjusting, don’t forget to check another gas in 20 minutes.