Foundations of Emergency Medicine
Foundations III: Guided Small Group Experience

Critical Care
Session 14: "The Physiologically Unstable Intubation"

- **Agenda and Learning Objectives**
  - Case Part I – Hypoxic and Hypotensive Intubations (20 min)
    - Discuss management of hypotension prior to intubation
    - Discuss options for pre-oxygenation prior to intubation in hypoxic patient
  - Case Part II – Metabolic Acidosis intubations (20 min)
    - Tools to try an avoid intubation in the acidotic patient
    - Discuss ways to prevent deterioration of acidotic patient during and after intubation
  - Case Concludes (10 min)
    - Review Session Teaching Points

- **Case Begins –** Hypoxic and Hypotensive Intubations (20 min)

  A 78 y/o F with PMH of HTN, DM and CAD comes in with shortness of breath for the past 5 days. On arrival the patient's vital signs are T: 103 F, BP 80/50, HR 132, RR 27 and O2 81%. The patient is clearly in respiratory distress on your exam and a quick bedside chest x-ray reveals a multi-focal pneumonia. You start to prepare for intubation.

- **Discussion Questions with Teaching Points**
  - **Why is this an anticipated difficult airway?**
    - There are two categories of difficult airways, physiologic and anatomical
      - Anatomical features that can make an intubation difficult include factors such as the patient's body habitus and trauma
      - This patient is an example of a physiological difficult airway.
    - Physiologically difficult airways are caused from one of the 'HOP' killers → **H**ypotension, **O**xygenation and **Ph**/ventilation
    - These are the factors that if deranged will put your patient at higher risk of peri-intubation arrest → need to be addressed prior to intubation
    - Trials looking at peri-intubation hemodynamic decline and cardiac arrest have found both hypotension (SBP <90) and hypoxia to be risk factors for deterioration

  - **How will you address the hypoxia in this patient prior to intubation?**
    - Consider not intubating them at all → you can start to DSI (see below) and assess whether NIV is enough to improve their oxygenation, the increased PEEP of NIV often is
    - Nasal cannula at flush rate (15LPM) plus a NRB at flush rate (at least 40 LPM → turn it all the way up on the wall!)

- If the above doesn't improve oxygen the patient likely needs increased PEEP, consider shunt pathology such as PNA or pulmonary edema
    - **Option 1:** add PEEP valve to BVM mask to help recruit more alveoli. These patients don't need to be bagged, rather just a tight mask seal and a jaw thrust (make sure the flow rate is at least 40 LMP or you will suffocate your patient!). You are performing apneic CPAP recruitment. This is especially helpful in critically ill patients when you can't get the spO2 above 95%
    - **Option 2:** Delayed Sequence Intubation. This is helpful in either combative or uncooperative patients that will not allow proper preoxygenation. Essentially you are giving procedural sedation in order to provide oxygenation. Give 1mg/kg of ketamine → preoxygenate as needed → paralyze → apneic oxygenation (NC+BVM+PEEP valve) → intubate
- As far as positioning is concerned, the most important consideration is aligning the ear to the sternal notch → this may require ramping or other bolsters
    - A recent article examined using a ramp position of the bed in ICU intubations and found ***worse*** outcomes that those intubated using conventional positioning → this may be 2/2 using the bed as a ramp rather than pillows/blankets etc which allow ear to sternal notch alignment while still placing the patient in ramp (Semler et al.)
- Positioning, and perhaps consideration of some ramping, is particularly important in obese patients and/or those with OSA where lying flat severely compresses their airway either due to obstruction or weight from their chest/abdomen
- Remember, once you have your patient positioned to look at them from the side to ensure their ears are lined up with their sternal notch, this allows optimal visualization of the airway structures
- Many of these interventions can be done (and should be done) simultaneously to help improve the oxygenation of your patient

- **What can be done to improve patient's hypotension prior to intubation?**
    - Hypotension is thought to be the biggest predictor of peri-intubation arrest and should be intervened upon immediately → a pre-intubation shock index (SI) of >0.9 is an independent risk factor for post-intubation arrest
        - SI = heart rate/systolic blood pressure
    - Adequate vascular access is paramount to both improve perfusion and to deliver intubation drugs effectively → two large bore peripheral IVs should be placed and if unable to obtain PIVs, an IO should be placed in the humeral head (ideally) for maximal flow rate and intubation drug delivery
    - Generally, can start 1-2L of crystalloid fluid wide open in order to fluid load the right heart as we anticipate a post-intubation drop in RV preload due to positive pressure ventilation

    - Remember your patients with right heart failure are different! They cannot tolerate IVF well so use pressors instead!
- The goal is to obtain a near normal blood pressure with IVF and pressors (see below) as needed prior to intubation
- Push dose pressors can be used to quickly increase the patient's BP while not having to wait for other interventions preventing a delay in intubation and possible further morbidity/mortality → epinephrine is often the pressor of choice given both the alpha and beta agonism though can also consider phenylephrine (50-100 mcg) if available
    - Mix push dose epinephrine by taking a 10cc NS syringe and emptying out 1cc, draw up 1cc of code does epi (100mcg/mL) thereby creating a syringe of 10mcg/mL of epi
    - Give 0.5mL-2mL (5-20mcg) every 2-5 minutes as needed
    - This is a temporizing measure to stabilize BP peri-intubation → work on getting an pressor drip to bedside (or wait if you can)
    - If you choose to mix your own pressors, it is extremely important to be meticulous in your safe handling!
        - Label your syringe
        - Take individual responsibility for your medication and consider administering the medication yourself
        - Dispose of the syringe when you are done → leaving an unlabeled syringe of epinephrine around that someone may mistake as saline is ***extremely*** dangerous!
- If possible, consider waiting until you have an appropriate pressor drip available prior to intubating → premixed pressors are usually dopamine and this is usually the wrong choice
- Peripheral pressors are safe in the antecubital fossa or more proximal for at least 4hrs → small IV's distal to the AC fossa and/or running pressors longer than 4 hours increases changes of extravasation and the ensuing complications
- If you run peripheral pressors, inform your nursing staff and ask them to keep a close eye on the IV site
- An awake intubation can be performed in patients that have tenuous hemodynamics allowing the patients endogenous catecholamine rush to improve their hemodynamics → if time permits this is a good option to help preserve the patient's native compensatory mechanisms
    - Be aware that post-intubation hypotension will still occur in awake intubations it will just occur later so be prepared
    - Hypotension in these cases is generally not due primarily to induction, but to the loss of sympathetic tone once you place a patient on positive-pressure ventilation

- **How should intubation drug choices/dose change in a patient with hypotension?**
  - The dose of your paralytic and sedative should be adjusted to the patient's pre-induction physiology
  - The paralytic dose should be increased → in a patient with shock paralytics take longer to work as they are cardiac output dependent
  - The induction agent dose should be reduced as they can cause a further drop in BP by decreasing the vascular tone, sympathetic tone and reducing the venous return
  - In addition, positive pressure will further decrease the venous return
  - Appropriate induction agents include etomidate and ketamine
    - Consider administering a half dose induction agent (ie 0.5 mg/kg ketamine or 0.15 mg/kg etomidate) and assess sedation prior to paralytic administration rather than giving a full dose initially to decrease potential medication side effects
    - Be aware of the potential negative effects of each of these agents → basically there is no perfect choice!
      - Etomidate has been shown to suppress adrenal function though it is unclear if this is clinically relevant for a one-time induction dose
      - Some will argue that in patient in shock, ketamine may cause a paradoxical negative ionotropic effect (and worsening hypotension) as the patient's sympathetic stores have already been maxed out though there is no data to date to support this
  - Many critical care MDs prefer rocuronium (1.5 mg/kg) to succinylcholine as when give in the larger dosage it has the same onset of action as succinylcholine but may have a longer safe apnea time (not data based)
  - If you want to use succinylcholine the dose should be 2mg/kg

❖ **Case Part II –** Metabolic Acidosis Intubations (20 min)

Shortly after sending your previous patient up to the ICU, EMS calls bringing in an obtunded 42 y/o diabetic male found at home. The patient's wife says he hasn't taken his meds in a couple weeks and he stayed home from work as he hadn't been feeling well. EMS states his vitals were stable in route and his accucheck was >500.

❖ **Discussion Questions with Teaching Points**

- **Your initial labs come back and suggest your patient is in DKA with a pH of 6.9 and he is no longer protecting his airway. What concerns do you have about intubating this patient?**
  - Intubating a severely metabolic acidotic patient puts him at risk of decompensation if you are not able to match the physiologic respiratory response to blow off CO2
  - If the proper precautions are not taken during and after induction, intubation could worsen the patient's acidosis and lead to cardiac arrest

- Make attempts to try and not intubate these patients (usually with early, aggressive NIV facilitated with medications as needed) → when intubation is inevitable attempt to match the pre-intubation physiology as much as possible

- **What can be done to try and avoid intubation in the severely acidotic patient?**
  - Bicarbonate infusion has been shown not to help these patients! The pH might be better but multiple studies have shown no improvement in hemodynamics or intracellular pH
  - The bicarb will eventually be converted to CO2 → putting more stress on a patient who is already tachypneic and thereby worsening the acidosis
    - Bicarb will only work if the patient (or us with the ventilator) can increase minute ventilation and most of these patients have already maxed out their minute ventilation as an attempt to compensate for their acidosis
  - Can attempt a very short, very well monitored trial of non-invasive ventilation to stave off intubation while trying to fix the metabolic acidosis
    - This will likely work better in hypercarbeic respiratory failure with 2/2 AMS than profound metabolic acidosis
  - Can also use ventilator-assisted pre-oxygenation. Place the patient on a ventilator with a NIV mask to set up a pseudo-NIV. Use following ventilator settings:
    - Mode: SIMV (volume control)
    - Tidal Volume: 550 ml (8cc/kg)
    - FiO2: 100%
    - Flow Rate: 30 lmp
    - PSV: 5-10
    - PEEP: 5
    - RR: 0 initially (assuming pt breathing adequately on their own) but will need to increase the rate as you begin to sedate the patient and set it at the final rate once paralytics are pushed
  - Whether using the above oxygenation setup or another, attach an EtCO2 detector and measure the pre-intubation value → write this value down and if you intubate, you should attempt to at least match this number post-intubation with your ventilator settings!
  - Matching their pre-intubation EtC02 will allow you to ensure you do not make your patient more acidotic when you place them on the ventilator → can be difficult!

- **The patient does not improve on NIV and is truly not protecting his airway. What can you do prior to intubation to decrease his risk of cardiac arrest during intubation? Does pushing bicarb help?**
  - Anything you can do to be as quick as possible in your intubation!
  - The danger is an apneic period during which your patients PaC02 increases and makes them more acidotic → this can lead to arrest
  - Keep the patient on NIV (or ventilator assisted oxygenation as above) as long as possible

- Increase the RR just prior to intubation (watch for breath stacking!) if using vent-assisted ventilation
- This is a time to not use our normal RSI techniques and ventilate the patient as soon as their RR begins to drop → don't allow them to have apneic (or even hypoventilation) time!
- Can also consider a partially awake or DSI intubation where you paralyze at the last minute only after you have visualized the cords
    - You can often facilitate this with sedation doses of ketamine (1 mg/kg) which will allow you to perform DL (video assisted is often easier in this setting)
    - Once you see the cords, push the paralytics and intubate as quickly as possible
- Make sure to bag aggressively after intubation and place on vent (settings discussed below) as quickly as possible → remember, the goal is NO apneic time!
- The most experienced person should intubate this patient → this is truly not a good intern case!
- Pushing bicarb does not help your overall pH in this setting because remember, their minute ventilation is already maxed out!

- **After intubation, what should your ventilator settings be?**
    - Our goal is to match the pre-intubation respiratory physiology after you put the patient on the vent → if not properly done, the patient will continue to become more acidotic and likely arrest from a cardiac dysrhythmia
    - Turn the respiratory rate up to **30** following the intubation to help this patient blow off the CO2 → check the EtCO2 and ensure it is at least as low as the pre-intubation value!
    - If not, increase the respiratory rate until the desired number is reached → make sure to check an ABG after 5 min to ensure no change or at least improvement in the acidosis with your current settings
    - Watch for breath stacking and auto-PEEP at high RR!
    - The flow rate should be 60 lmp (standard vent setting) and tidal volume should be 8cc/kg of their predicted IBW

❖ **Case Concludes (10 min)**

- *You are able to intubate your patient successfully, change the ventilator settings to match the pre-intubation minute ventilation and he stays stable following the intubation. You continue your standard DKA management and the patient is accepted to the ICU for further care.*

❖ **Case Teaching Points Summary**

- Resuscitate Before you Intubate!
  - When preparing to intubate a patient ask yourself if you have fully resuscitated your patient prior to intubation in order to decrease chances of peri-intubation decompensation
  - Hypotension, hypoxia and acidosis(pH) (aka HOp killers) are all risk factors for peri-intubation cardiac arrest
- Hypotensive intubation
  - Calculate the shock index (HR/SBP) prior to intubation → if above 0.9 you should resuscitate the patient prior to intubation
  - Strategies to improve hemodynamics prior to intubation include ensuring adequate IV access, IVF boluses (careful in RHF), push dose pressors and starting pressor drips in a good PIV prior to intubation if needed
  - Change your induction doses appropriately for the hypotensive patient
    - Double your paralytic dose and half your sedative doses
    - When the proper doses are used, rocuronium and ketamine or etomidate seem to be the ideal induction medications
- Hypoxic Intubation
  - Place your patient on a flush rate nasal cannula (at least 15 LMP) and a NRB (at least 40LMP) to achieve adequate oxygenation or consider using a BVM w/ a PEEP valve
  - If your patient continues to be hypoxic (pO2 <95% though some argue < 100%) you can add PEEP in two different ways:
    - Add a PEEP valve on a BVM to recruit more alveoli
    - Perform Delayed-Sequence Intubation (DSI) by giving the patient a sedative (ketamine) and place them on NIV to deliver adequate PEEP
  - Remember ramping may or may not help you and if you do ramp, consider using bolsters/blankets etc instead of the bed to ramp
  - Finally, look at your patient from the side once positioned to ensure their ear and sternal notch line up → this is the most important consideration in positioning!
- Metabolic Acidosis Intubation
  - The goal is to NOT intubate these patients as it is difficult to match the pulmonary compensation physiology on a ventilator
  - Intubation can be delayed by using a trial of NIV or 'psuedo-NIV'
  - Bicarbonate therapy is unlikely to assist and might hurt these patients
  - If you intubate a metabolically acidotic patient ensure to match their pre-intubation EtCO2 when on the ventilator
  - Bag them as soon as their RR begins to drop! Goal is **no** apneic time!
  - Ensure to set the ventilator TV to 8cc/kg of their IBW and turn up RR to at least 30
  - Check an ABG quickly after putting these patients on the vent to ensure there is no worsening of their acidosis

❖ **Facilitator Background Information**

It is imperative that we resuscitate before we intubate and we recognize the risk factors for peri-intubation decompensation/cardiac arrest. The most important risk factor are hypotension, hypoxia and metabolic acidosis/pH (HOp killers). If you are preparing to intubate a patient and notice they are tachypneic make sure they are not acidotic as this could worsen their clinical course. By not recognizing and addressing these risk factors we are putting our patients at increased risk.

Hypotension prior to and following an intubation puts your patient at the greatest risk for a cardiac arrest. Make sure to have adequate IV access prior to intubation and be familiar with what vasopressor options are accessible in your department. If you have premixed pressors, consider hanging them prior to intubation to help improve the hemodynamics. If you have to wait for pharmacy to mix a pressor for you consider making push dose pressors to help bridge your patient until a proper drip can be made (epinephrine or phenylephrine). In addition, do not wait to place a central line to start vasopressors. They can safely be administered through a PIV at the AC fossa or more proximally for around 4 hours (at least). A central line should be placed after intubation or in the ICU if it can be done in a timely manner. Medication doses need to be altered to match the patients altered physiology as well. As paralytics are dependent on cardiac output and take longer to work in shock states, we need to close to double the dose to ensure adequate drug delivery. Sedative doses should be generally be halved as the patient is already hypotensive and some sedatives will cause further hypotension through loss of vascular tone. Etomidate and Ketamine are likely both appropriate induction agent choices for a hypotensive patient and many intensivists prefer rocuronium (at the 1.5 mg/kg dosing) to succinylcholine in these setting.

There are a couple ways to improve hypoxia in the peri-intubation period. After the decision has been made to intubate, every patient should have a nasal cannula placed for pre-oxygenation and apneic oxygenation at a flush rate (15 LMP). Add a NRB at at least 40LMP if a spO2 of at least 95% is not achieved. If this is not enough, your patient likely has shunt physiology and needs alveolar recruitment. A PEEP valve can be added onto a BVM mask and used to help recruit alveoli. You can also use NIV to achieve this goal. If your patient will not tolerate NIV, can administer a sedative (often ketamine 0.5-1 mg/kg) and place them on non-invasive ventilation, also known as Delayed Sequence Intubation (DSI). This is helpful for combative patients as it sedates them and gives you time to effectively pre-oxygenate.

The final HOp killer and the most difficult to correct is metabolic acidosis. These patients are challenging if they are intubated as it is difficult to match the natural pre-intubation pulmonary physiology when on the ventilator. These patients are dependent on respiratory compensation by blowing off their CO2 and if this process is hindered, eg by being on a ventilator or having a prolonged apneic period while being intubated, if can worsen their acidotic state and lead to cardiac arrest. Ideally, we are able to avoid intubation by using other measures (namely NIV with increased Tv). When this is not possible, we want to ensure adequate ventilation throughout and after the intubation. Measuring pre-intubation EtC02 can help ensure we are matching their pre-intubation pulmonary physiology after intubation with our ventilator settings. We also need to minimize increases in PaC02 during the intubation period. This can be achieved through performing an awake intubation, minimizing their apneic time by ventilating once

induction drugs are given and setting the post-intubation respiratory rate high enough to keep up with the pre-intubation respiratory compensation. Post-intubation ventilator settings should be aggressive in terms of RR (often near 30) and Tv of 8cc/kg. Compare the pre-intubation EtC02 to the post-intubation EtC02 to ensure you are at least matching this number, it should be the same or lower (confirm this with an ABG quickly after intubation). Matching the PaC02 can be extremely difficult! At aggressive respiratory rates, it is important to watch for breath stacking and auto-PEEP as this is a complication and can additionally lead to death if not recognized. Make sure you check frequent ABGs on these patients while they are in the ED to ensure there is no worsening of the acidosis. Finally, make sure you are treating the underlying cause of the acidosis as this is ultimately what will stabilize your patient!

- **References**
  - **Author:** Dr. Sean Dyer
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