Press Before the Tube! Intubating Hypotensive Patients

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Learning Outcomes

1. Describe the risks in utilizing rapid sequence intubation and positive pressure ventilation in the hypotensive patient.
2. Verbalize the preparation and sequencing of medications to manage intubation of the hypotensive patient.
3. Select appropriate drugs and dosing in managing a clinical scenario.

Life is a hard teacher,
first comes the exam,
then the lesson.

-M. Bromiley[1]

7 P's of RSI
Preparation
Preoxygenation
Premedication/Sedation
Paralysis
Protect and Placement
Placement confirmation
Post-intubation management

There is no one *single* approach to all airways: *always* expect the unexpected

Critically ill patients[2]

- "For critically ill patients who develop respiratory distress and undergo emergent intubation…rapid sequence induction of sedation, analgesia, and paralysis are often used"
- "Induction agents…each have a different side effect profile which should engender careful consideration of a compilation of various drug and patient related factors prior to their use"
- "9% of patients suffered severe hypotension following induction, the majority in the 6 to 10-min timeframe, Statistically signficant predictors of post-induction hypotension included ASA III or IV, baseline MAP <70 mmHg, age >= 50years, the use of propofol, and increasing doses of fentanyl"
- "If patients developed hypotension following endotracheal intubation, the odds of death increased by nearly 2-fold."

Indications

The need for an endotracheal tube falls into three broad categories:

- Failed airway
- Failed ventillation
- Clinical course

Do we approach this as an algorithim, or clinical gestalt, or a hybrid of the two?

Indications for Intubation[3]

Medical encounters 84%
Cardiac & traumatic arrest 38%
AMS 25%
Trauma 16%
Respiratory failure 15%
Shock 11%

Categories of possible "crash airways"[3]

Airway obstruction 3%
Facial/neck trauma 1%
Burn/inhalation 1%

In the rare event of a true “crash” airway, establishment of the airway precedes all other efforts, unless they can be attempted simultaneously without diverting focus.

HOp Killers[4]

Hemodynamics : PreSuscitation
Oxygenation : PreOx prevents DeOx
pH : Match Respiratory Rate

RSI is NOT Benign

Single-center retrospective cohort of 542 emergency intubations[5]

- 12% suffered Cardiac Arrest
- SI >/= to 0.9 is associated with adverse outcomes (requires further study)
- Pre-intubation SPO2 <92% more common but not independently associated with CA
- CA odds increased 1.37 times for every 10kg increase in weight.

1395 patients intubated for vascular surgery[6]

- “A reduction in blood pressure related to the administration of medications and the physiologic effect of positive pressure ventilation in conjunction with other factors after intubation is a common adverse event during ETI.”
- “This reduction in blood pressure, known as post-intubation hypotension (PIH), is frequently anticipated by anesthesiologists and may be monitored without intervention

or alternatively managed with intravenous volume expansion or vasopressor administration."

479 ICU intubations[7]

- "Medications used to facilitate ETI and PPV have potential to impact patient hemodynamics directly"
- "In critically ill patients, sympathetic outflow is commonly increased due to hypercarbia, hypoxemia, and respiratory distress; this results in relative "hemodynamic stability". After induction, however, hypercarbia and hypoxemia generally improve, the work of breathing is diminished, and sympathetic outflow dramatically decreases, often resulting in hypotension in patients who require ETI."

Multi-center prospective study of 3,872 ED intubations[8]

- Age of 65 or greater had higher rate of PIH (3% v 1%, OR 2.7)
- "Diminished overall cardiovascular re- serve in elderly patient results in heightened sensitivity to the negative inotropy and vasodilatory effects of induction agents and other vasoactive drugs."
  "In elderly patients, the goals of airway management are not only to provide appropriate intubation condition, but also to preserve myocardial and haemodynamic function, control for the effects of pre-existing disease and avoid adverse events such as hypotension or dysrhythmia."

Increased Risk Factors

Single-center retrospective cohort study of 542 emergency intubation patients[5]

- Pre-intubation hypotension found in 16% of all patients, and in 35% of patients who arrested
- CA occurred in 12% of pt with hypotension which "…highlights the risk of emergency management in patients with uncompensated shock."
- Peri-intubation arrest occurred in 4.2% at a mean of 6 minutes post intubation
- Reasons for intubation: Airway protection 72%, Respiratory failure 20%
- Pre-intubation and peri-intubation factors predict the complication of PIH. Elevated SI strongly and independently forewarned of cardiovascular deterioration after emergency intubation with pre-RSI SI 0.8 or higher as the optimal threshold to identify patients at risk. [9]

Post-Intubation Hypotension (PIH) Defined[6]

- Decrease in SBP to ≤80mmHg or any decrease in SBP ≥20% from baseline within 15 minutes of intubation
- MAP ≤60mmHg within 15 minutes of intubation
- For patients hypotensive prior to intubation, an additional decrease in SBP of >5mmHg within 15 minutes of intubation
- Administration/Increase in vasopressor within 15 minutes of intubation

Peri-Intubation Cardiac Arrest

- Peri-intubation arrest occurred (overall) in 4.2% of emergency ETI at a mean of 6 minutes post ETI[5]
- Arrest more common with pre-ETI hypotension (12% v 3%)[5]
- Peri-ETI CA associated with 14-fold increase in odds of hospital death[5]
- Intubation-related cardiac arrest 2.7% (49/1847) or 1 in 40 intubations[10]
- Comparison: cardiac arrest for elective surgery 0.019%[10]
- Immediate mortality 28.5% 28-day mortality 71.4%[10]
- 5 risk factors[10]
    - Hypoxemia prior to intubation
    - Hemodynamic failure prior to intubation
    - Absence of preoxygenation
    - BMI>25kg/m2
    - Age>75
- Incidence of PIH to be 60% in pt intubated for vascular surgery, and were more likely to die in-hospital. [6]
- "our data support the opinion that PIH occurs at a rate that has been underreported in the literature, and it's importance has been underappreciated." [6]
- "In an investigation of 218 adult patients intubated in an ED setting…the incidence of PIH was 44%.[6]

Trauma Patients[11]

In trauma patients, PIH incidence 36.3%
In hospital mortality was 29.8% in PIH pt (vs 15.9%)

Overview of the plan:

- High-risk patients, especially those exhibiting pre-RSI SI of 0.8 or higher should undergo **pre-intubation hemodynamic optimization as time permits**.[9]
- **Empiric volume loading, blood transfusion, and peri-RSI catecholamine support may be indicated depending on the clinical circumstances.** [9]
- Attention to **slow, low-tidal volume ventilation** aims to limit intrathoracic pressure and its cardiovascular consequences. [9]

The Plan

- (P)Resuscitate
- Push-Dose Vasopressors
- Vasopressor Infusions
- Shock as Sedation
- Dosing Changes
- Drugs to Avoid
- H's & T's at ALL TIMES!!!

(P)Resucitate

We can no longer blindly follow the alphabet…

- While airway and ventillation management IS a fundamental part of our primary resucitation, intubation does not *necessarily* come first...
- Utilize fluids as the initial part of the resucitation strategy, but frequently assess patient response to fluids (via US or invasive monitoring), and be mindful of the pH of normal saline (5.6).
- Initiate colloids (MTP/balanced resucitation) in patients who are hypotensive secondary to bleeding/trauma

Push Dose Vasopressors

Push-Dose Epinephrine[12]

- Draw 1ml(100mcg) of 1:10,000 epinephrine ("code cart epi") into
- 9ml of normal saline, shake and label
- 10mcg/mL final concentration
- Push dosing is 0.5-2mL (5-20mcg)

Push-Dose Phenylephrine[12,13]

- Draw 1mL (10mg) and inject into mini-bag
- 100mL of normal saline, shake and label
- 100mcg/mL final concentration
- Push dosing is 0.5-2mL

Calcium Chloride[14]

- 10mg/kg over 8-10 seconds, may repeat in 6 minutes
- With the clinical conditions employed in this study, injection of CaCl exerted two substantial effects-an immediate enhancement of myocardial contractile state and a gradual increase in SVR.
- Experimental evidence suggests that the primary source of contractile calcium is located in the sarcoplasmic reticulum; this is released by an influx of external calcium. The early response (starting at 4 seconds and peaking at 23 seconds after calcium injection) observed in our study consisted of an increase in myocardial contractile state, CI, and SVI, and a decrease in SVR. This may represent the direct effect of Ca++.

Note: Common practice [my experience/anecdotal] is 0.5-1gram IV Push

Vasopressin

- In those whose sympathetic and renin- angiotensin systems have been blunted by general anesthesia and ACEIs/AIIRAs, TP (IV 1–2 mg) or AVP (IV 2–3 U) is an effective vasopressor if they are refractory to common adrenergic vasopressors.[15]
- Although vasopressin infusions have been used in a variety of other situations, there are limited data to guide bolus dosing. Others report lower doses of vasopressin bolus. A 2-U bolus dose was used to treat anaphylactic shock.5 As in this case, two 0.4-U boluses

successfully treated hypotension secondary to both bowel retraction in patients having abdominal aortic resection repair and postreperfusion syndrome during liver transplantation.[16]

Vasopressin Mixing/Administration

- Draw 1mL (20 units) into
- 9mL of normal saline, shake and label
- Dose is 0.5-1mL (1-2 units)

Norepinephrine

- The use of intermittent IV norepinephrine boluses to prevent spinal-induced hypotension in elective CD seems feasible and was not observed to be associated with adverse outcomes.[17]
- Practically, we suggest an ED90 dose of 6 μg. Further work is warranted to elucidate the comparative effects of intermittent IV bolus doses of phenylephrine and norepinephrine, in terms of efficacy and safety.[17]

Norepinephrine Mixing/Administration

- Draw 4mL (4mg) into
- 500mL of normal saline, shake and label
- 8mcg/mL final concentration
- Dose is 0.5-1mL (4-8mcg)

Note: Norepinephrine blousing might be more useful in the context of your institutions infusion concentration. For example, 4mg norepinephrine in a 250mL mini-bag is a common concentration. In this setting, mixing for a drip and then utilizing 0.25-0.5mL pushes as needed to temporize low BP/MAP while the drip is titrated up could provide a clinical advantage.

You will be doing this under stress!!!
You must plan, practice, and provide tools to increase success and safety!!!

Vasopressor Infusions

- Know your facility's standard mixing, starting ranges, and titration protocols for epinephrine, norepinephrine, phenylephrine, vasopressin, etc.
- Have reference materials (mixing guides, titration guides, etc.) **readily available** in your resuscitation areas. A common failure point is the assumption that this information is "readily" available online, in the MAR, etc. Be aware that under stress and moving quickly, this information can be more challenging to access.
- KNOW YOUR PUMP, and know where to get one!!! Do you always have a pump (and channels) physically available in your resuscitation area?
- TIME YOURSELF mixing various drips, physically accessing the meds, drawing up, locating a pump. A fun experiment is to drill this randomly (using expired meds or saline/water) to get a sense of how long it ***truly*** takes to accomplish this. The results are often surprising!!! Then decide if you patient will have that much time to wait in the context of emergent RSI!!!

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Shock as Sedation

- Consider the three components of RSI: analgesia, sedation, and paralysis.
- Patients who are hypotensive (shock state) are inherently sedated, and therefore it is reasonable (if not imperative) to consider changing (lowering) the amount of sedation medication used in RSI.
- "Patients with a shock index (defined as heart rate divided by SBP) of greater than 0.8 are at particular risk of developing significant hypotension in the postintubation period."[18]

Use *EXTREME* caution with any medication that can lower blood pressure!!!
Examples include benzodiazepines (especially at induction doses), propofol, and some opioids.

Ketamine

- Ketamine is frequently used in this situation due to its' sympathomimetic actions (elevated heart rate and blood pressure).
- Be mindful that ketamine *can* cause hypotension in select cases (e.g. uncompensated cardiogenic shock) through a negative inotropic effect.
- "Clinical hypotension was less prevalent in septic patients who received ketamine versus etomidate" (51.3 v 73%), and patients receiving etomidate had significantly lower MAP in the first 24 hours after intubation."[19]

The "3-2-1" RSI[20]

- **3mcg/kg fentanyl**
- **2mg/kg ketamine**
- **1.2-1.5mg/kg rocuronium**

The Overall Approach

- **A "script" discussed in advance**. Should include personnel, equipment, procedure for how a typical RSI will occur. A discussion of typical drugs and dosing ranges should also occur in advance, and ideally be included in a standard operating procedure for RSI. The SOP could address pre-oxygenation, positioning, premedication (including push-dose pressors), and optimal selection/sequencing of RSI medications.
- **Provider**. This will be facility dependent. Should anesthesia be involved? ENT? Is there a specific phone number to call? What's the response time? What is the providers focus? The tube, the resuscitation, or both? Can any of this be offloaded into SOPs and/or nursing? Once paralytics have been pushed, the providers primary goal is rapid first-pass success passing the tube through the cords.

- **Respiratory Therapist**. Do any respond or do too many respond? Again, resources and scope of practice will vary among facilities. PRE-PLAN who will do what, and have 24/7/365 accountability in mind.
- **RN for RSI**. Ideally, the specific meds, doses, and sequence will be discussed, as well as the parameters to withhold and/or modify the dosing. This RN will be empowered "stop the line" for poor hemodynamics, poor patient response, etc.
- **RN for Hemodynamics**. Ideally, fluids, meds, doses, and sequence will be discussed and/or outlined in an approved SOP. This RN will focus on the obtaining an appropriate BP/MAP to allow safe RSI. This RN should also be empowered to "stop the line", and should be responsible for independently administering PRN pressors/adjunctive medications from the pre-determined SOP or patient/provider/situation specific plan.

Questions are welcome!!!

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