DON'T CRASH YOUR PATIENT!

MITIGATING THE PHYSIOLOGICALLY DIFFICULT AIRWAY

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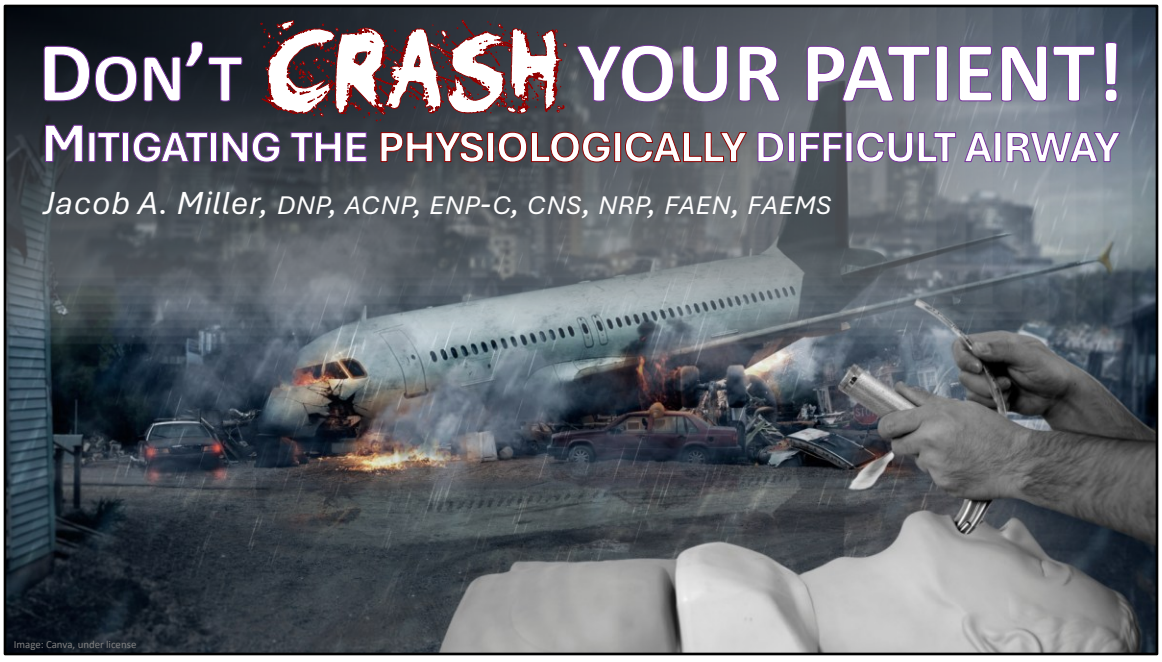

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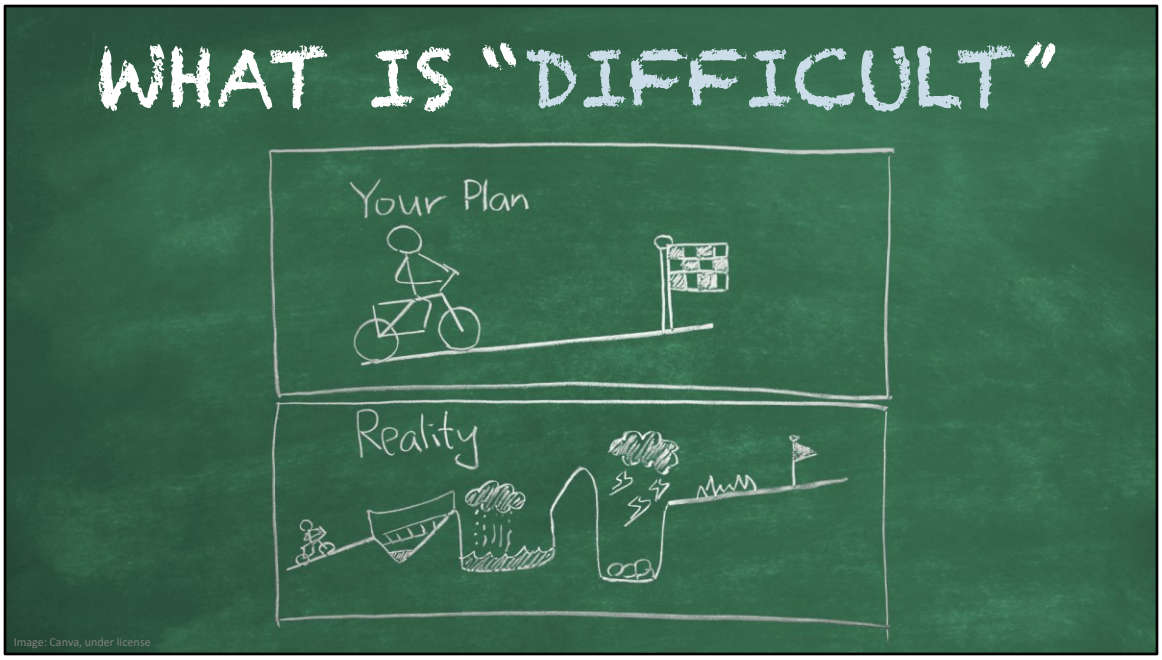
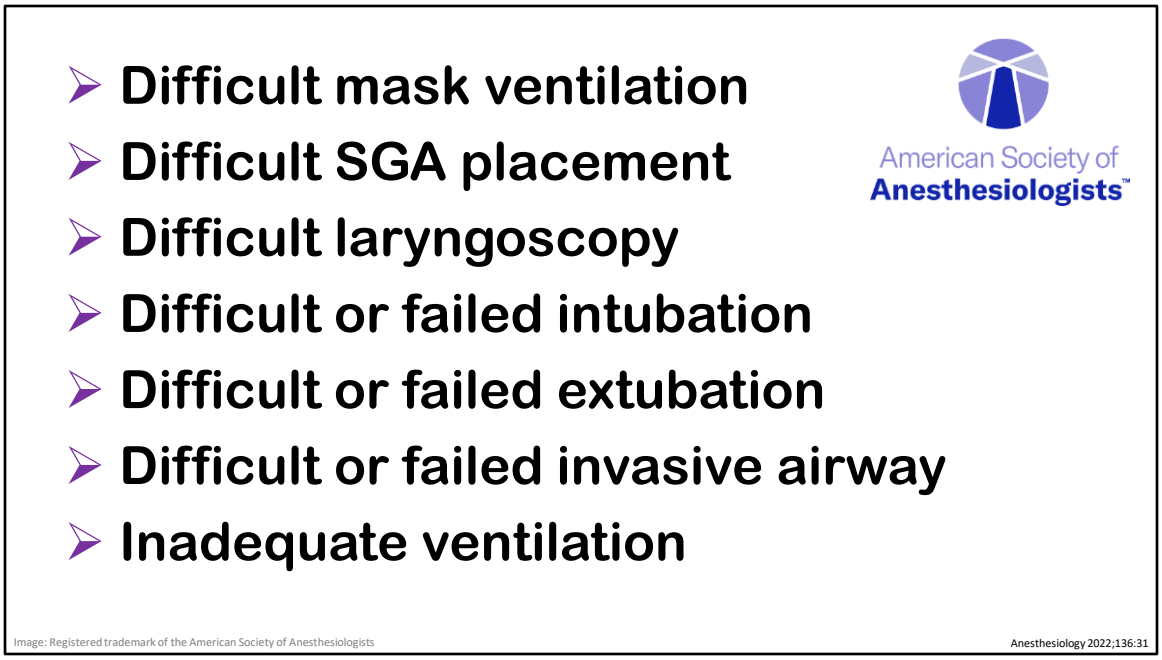
- Views/opinions are entirely my own and may not be reflective of my employers, ENA, or others.
- This presentation is **not** a substitute for sound medical judgment or common sense. Seek expert consultation as indicated.

Disclosures

- Abiomed: Travel/honorarium for consultation

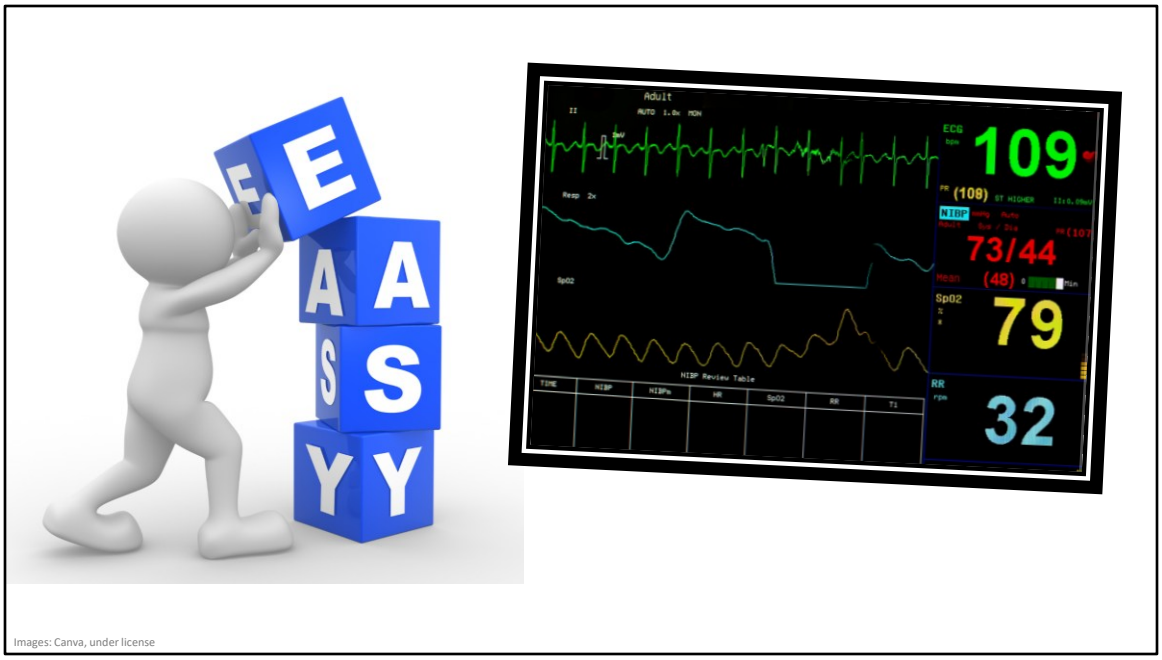


What defines a “Difficult airway”



The American Society of Anesthesiologists break “difficult airway” into many classes, including these

This, however, only speaks to the technical and anatomic predictors of a difficult *procedure*.



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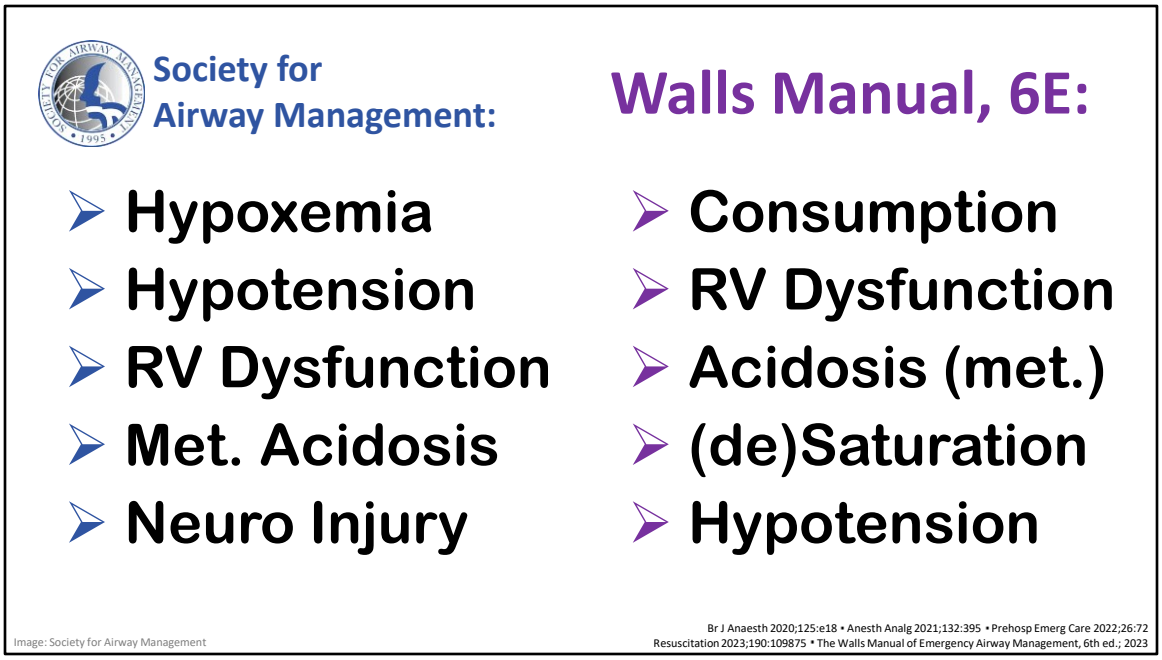
More recently, attention has been brought to the *physiologically* difficult airway, and the risk for peri-intubation deterioration.

There are plenty of patients—and if you've been in practice long enough, you've likely encountered at least one—in whom the airway management *procedure* was technically easy, but because the patient had *physiologic* distress, their condition acutely worsened (or the patient arrested) during or shortly after their intubation.



Of note, the concept of a physiologically difficult airway has been around for well over a decade; Scott Weingart from EMCrit introduced the concept of the "HOp killers" (Hypotension, Oxygenation, and pH) back in 2012. The original post is here (although sadly now appears to be paywalled): https://emcrit.org/emcrit/hop-mnemonic/

The first national position paper formally exploring the concept of the "physiologically difficult airway" was only recently published by the Society for Airway Management in 2021. They identified the factors above as those with the highest risk factors for peri-intubation complications, including cardiac arrest and death.

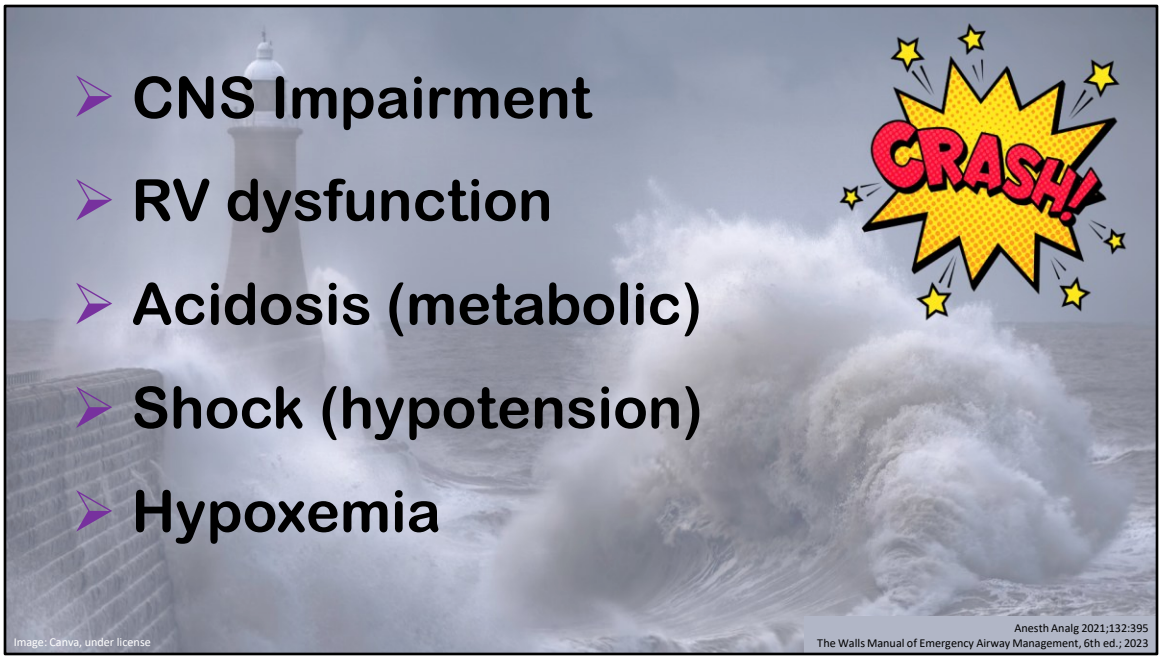
A multicenter observational study looking at pediatric patients validated most of these high-risk factors for pediatric patients as well—in the pediatric population, physiologic difficulty was predicted by:

- Hypotension
- Hypoxemia
- Severe metabolic acidosis
- Concern for cardiac dysfunction
- Post-ROSC patients
- Status asthmaticus

The National Association of EMS Physicians has also issued guidelines on optimizing physiology in their 2022 Airway Compendium series published in Prehospital Emergency Care.

Most recently, the "CRASH" mnemonic was introduced in the latest edition of the "Airway Bible" (the Walls Manual of Emergency Airway Management).

While these lists are similar in many regards, they're not identical.



After reconciling the similarities and differences between the two lists, I would like to propose *my* version of the CRASH mnemonic:

- CNS Impairment
- RV Dysfunction
- Acidosis
- Shock, and
- Hypoxemia

Changing the “S” from “Saturation” to “Shock” was intentional → not all shock is necessarily hypotensive! This will be explained later.

Why is this important? What's the risk?

Surely, if your patient needs an airway **now**, they need an airway, well, **NOW**, right?

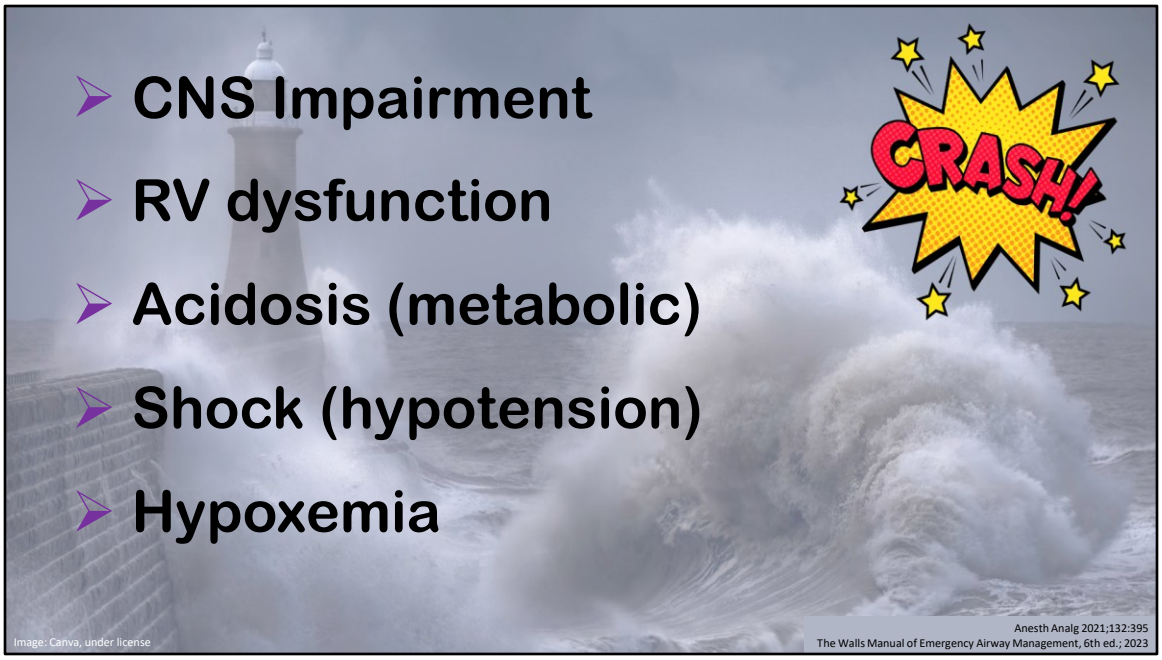
Turns out, if your patient experiences a peri-RSI arrest, even with successful resuscitation, odds of in-hospital mortality are increased by between **4** and **14 TIMES** (depending on study)

AND, most RSI arrests occur within the first **10 to 15 minutes** of the induction and intubation.

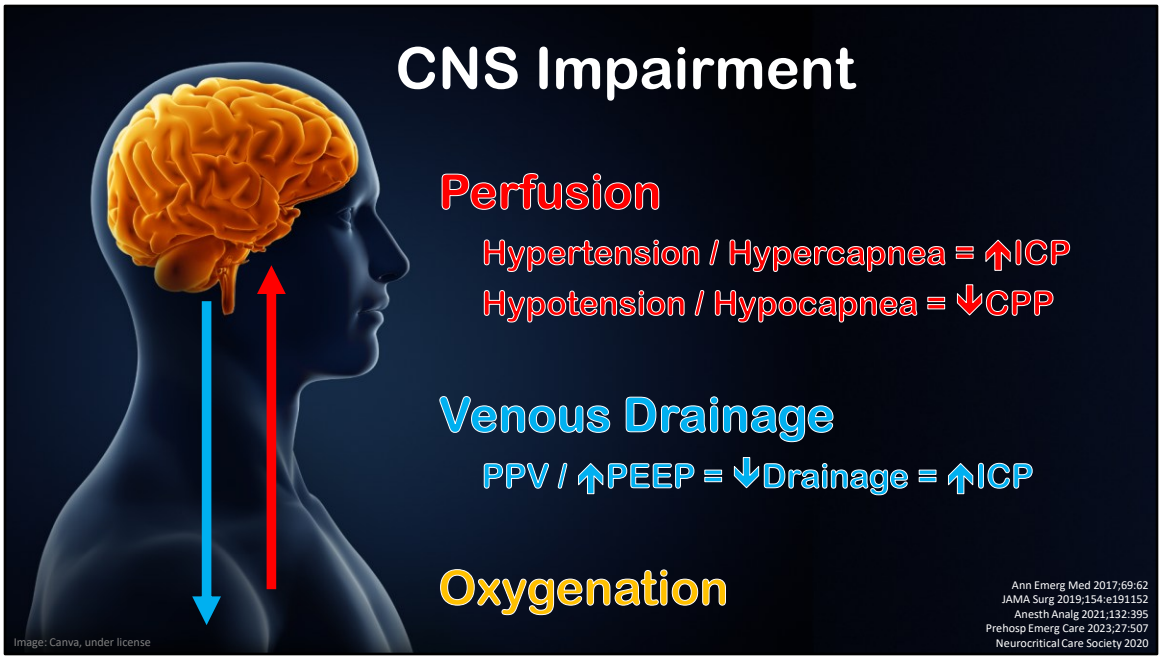
Think about your clinical practice:
What are you usually doing in the 15 minutes after you perform an RSI intubation? And how vigilantly are you monitoring the patient's hemodynamics during that time?

Often, we become task-saturated, focus on transitioning the patient to "the next thing on the list"

Also, many hospitals and EMS agencies alike default their cardiac monitor NIBP cycles to *maybe* every 5 minutes, but usually longer intervals. It's *critical* that, if your patient does not have invasive hemodynamic monitoring, your NIBP cycle be set to as frequently as possible for the first several minutes after induction.



So now let's get into each of the CRASH killers!



CNS Impairment—specifically those patients with, or at risk for, increased ICP—can be sensitive to the hemodynamic and physiologic changes that accompany RSI.

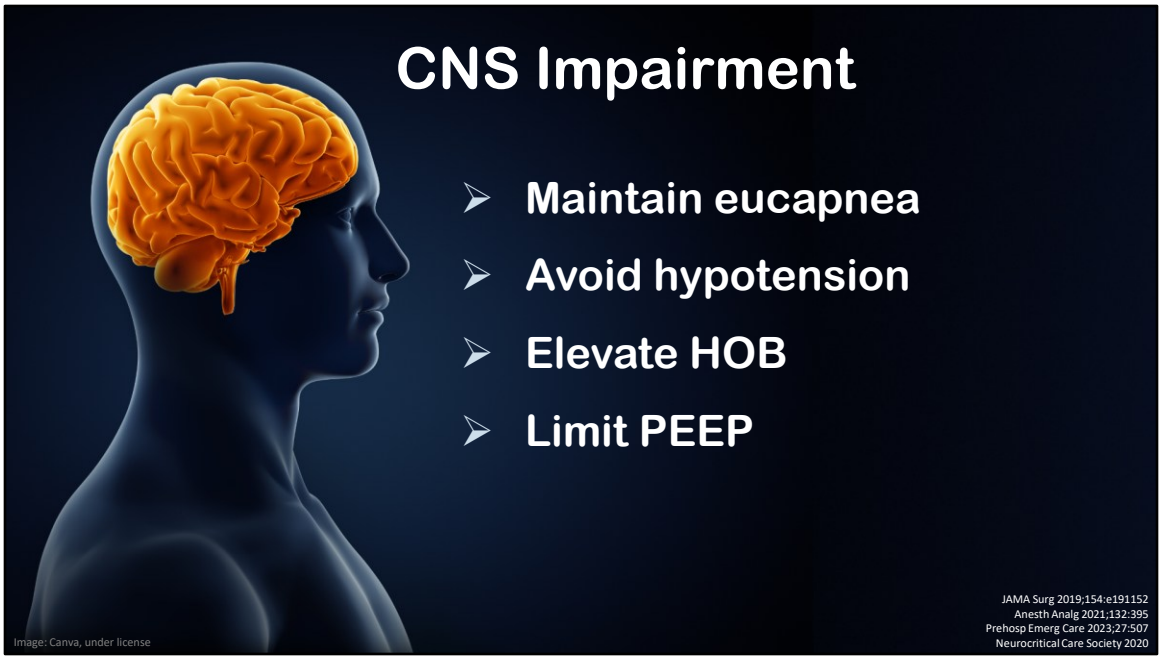
CNS impairment can lead to impaired autoregulation. This can lead to a closer dependence of ICP and cerebral perfusion on the mean arterial pressure.
HypERtension can increase ICP and worsen brain injury, whereas HypOtension can lead to a low CPP and cerebral ischemia.

Recall that cerebral vascular resistance is affected by $PaCO_2$ concentrations.

- Hypercapnea will dilate cerebral blood vessels and may, too, contribute to increased ICP. Prolonged apneic time (i.e., during intubation attempt) will necessarily lead to a rise in CO_2, as can underventilation after the airway is secured. Following $EtCO_2$ targets alone may not fully reflect the true $PaCO_2$ if there is any pathology (acute or comorbid) widening the normal PCO_2:$EtCO_2$ gradient, so an ABG or VBG should be evaluated soon after intubation to ascertain true CO_2 concentrations and whether or not noninvasive $EtCO_2$ readings are correlating.
- Hypocapnea—the result of hyperventilation (often iatrogenic, whether deliberate or accidental)—leads to vasoconstriction, and may worsen cerebral perfusion and cerebral ischemia. **Routine hyperventilation in the TBI patient is no longer recommended.** Even in the setting of suspected herniation, only **MILD** hyperventilation (targeting a PCO_2 of 30-35 mmHg) is recommended.

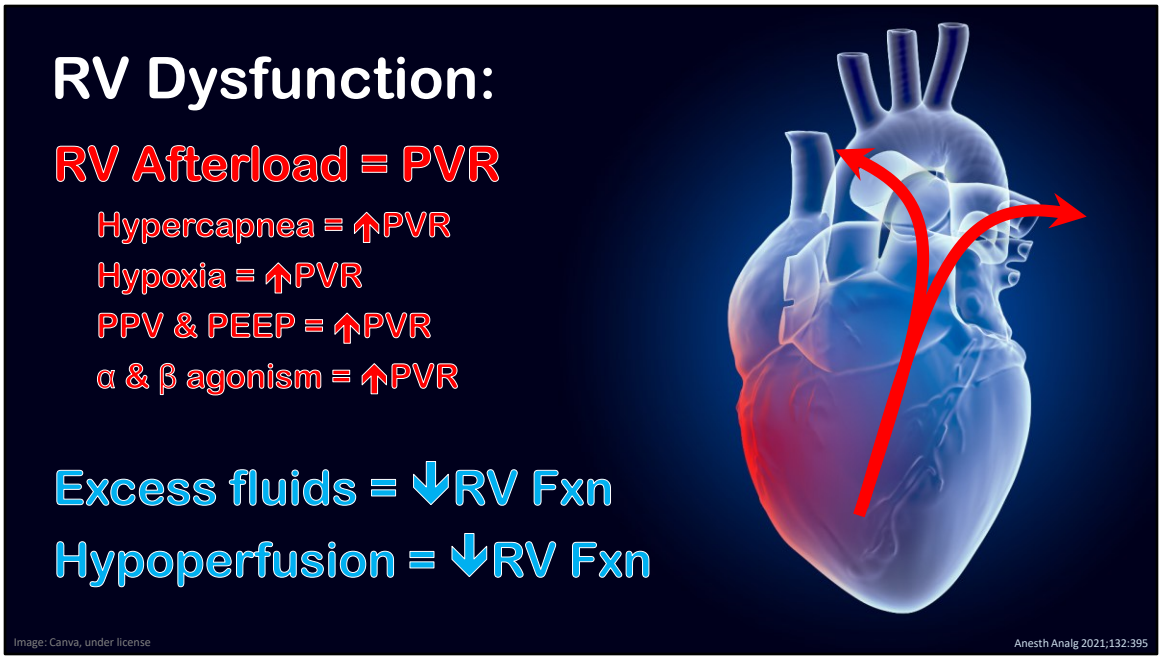
Now, let's look at the other side of ICP: getting blood *out* of the brain. Recall the Monro-Kellie Doctrine and the importance of maintaining balance of blood, brain, and CSF—especially in the setting of any intracranial pathology. The change from normal negative-pressure respiration to positive-pressure ventilation greatly decreases, if not reverses, the pressure gradient between the CNS compartment and the thoracic compartment, making it more difficult for venous drainage to occur. Increasing levels of PEEP can worsen this effect.

Lastly, the brain is dependent on oxygenation (we've seen in the EPIC-TBI registry that a single episode of hypoxia can triple the odds of death in brain-injured patients), so care must be taken to avoid hypoxemia before, during, and after intubation attempts.



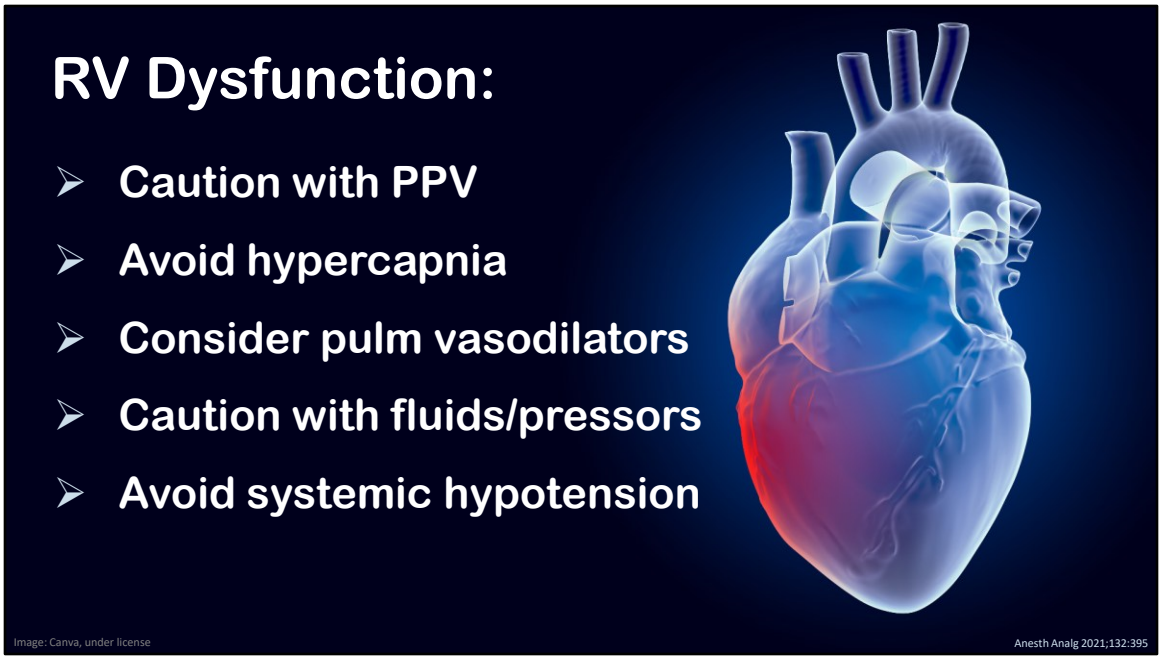
The SAM guidelines recommend these strategies when performing airway management in patients with CNS impairment:

1. Eucapnia should be maintained before, during, and after intubation.
2. Hemodynamically neutral induction agents should be used.
3. Patients should be positioned 30 degrees upright, when possible (to promote venous drainage and prevent increased bloodflow and ICP).
4. Post intubation management should include limiting PEEP to promote cerebral venous drainage.



Right ventricular dysfunction can be challenging to manage in patients requiring airway support.

Recall that the of the right ventricle pumps blood into the pulmonary arteries and then to the pulmonary vascular bed and, therefore, the afterload of the RV is the pulmonary vascular resistance (PVR).



From SAM Guidelines:

- Patients should be **screened for significant RV dysfunction prior to intubation** given the risk of decompensation with the transition to positive pressure ventilation.

- When RV dysfunction is present, patients should be **evaluated for RV systolic function**, and fluid and vasopressor tolerance.

    - Empiric fluid resuscitation without this evaluation can further reduce RV function!

    - Fluid and vasopressor **tolerant patients should be resuscitated accordingly**.

    - **Diuretics should be considered** in patients with RV volume overload.

- Fluid-**intolerant patients** should have **RV afterload reduced** with inhaled or intravenous **pulmonary vasodilators**.

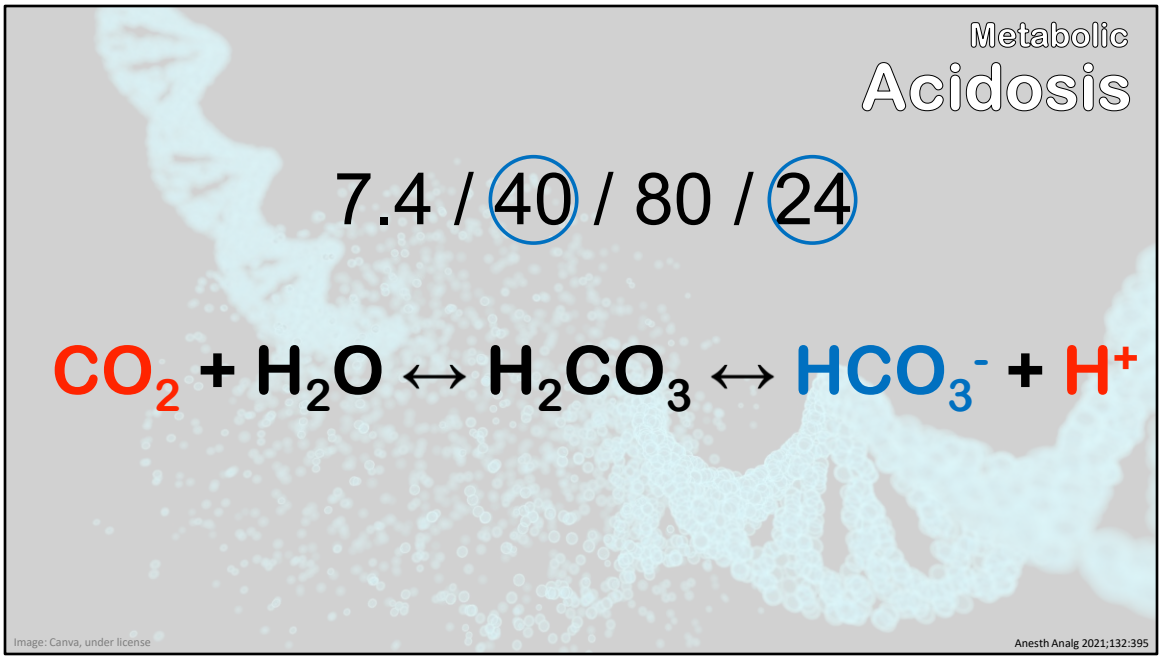
- Hypercapnia should be avoided.

    - **Hypercapnia can increase pulmonary vascular resistance** and worsen RV afterload

- Mean arterial pressure should be augmented to **maintain coronary perfusion pressure**. In patients with **chronic pulmonary arterial hypertension, a higher mean arterial pressure should be targeted** to keep mean arterial pressure > mean pulmonary artery pressure.

Other recommendations:

- **ECMO cannulation should be considered** if available in patients with **RV-failure-induced shock**.



Just a quick review of the physiology of the severely acidotic patient

Recall the basic homeostatic equation.

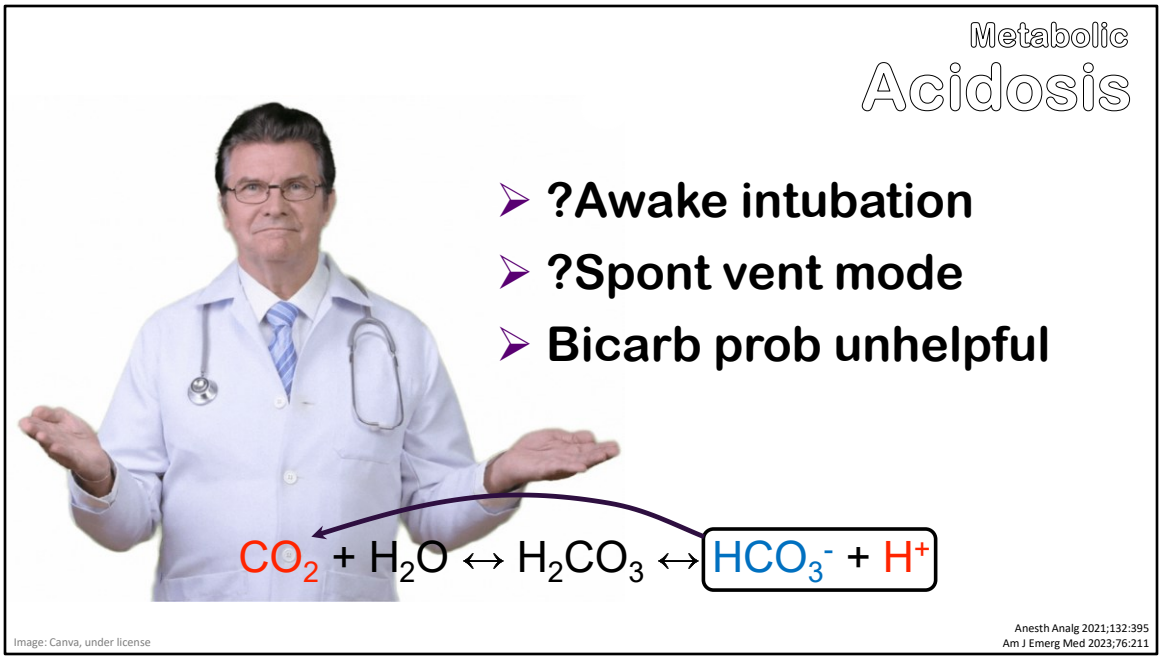
With metabolic acidosis, extra H+ is introduced

Initially, bicarb is able to handle this

As metabolic acids continue to rise, remember your ABG values – there's only a finite amount of bicarb, you can't go negative.

To further compensate, excess CO_2 (a functional acid) begins to get blown off to keep pH normal – but again, this is also a finite resource!

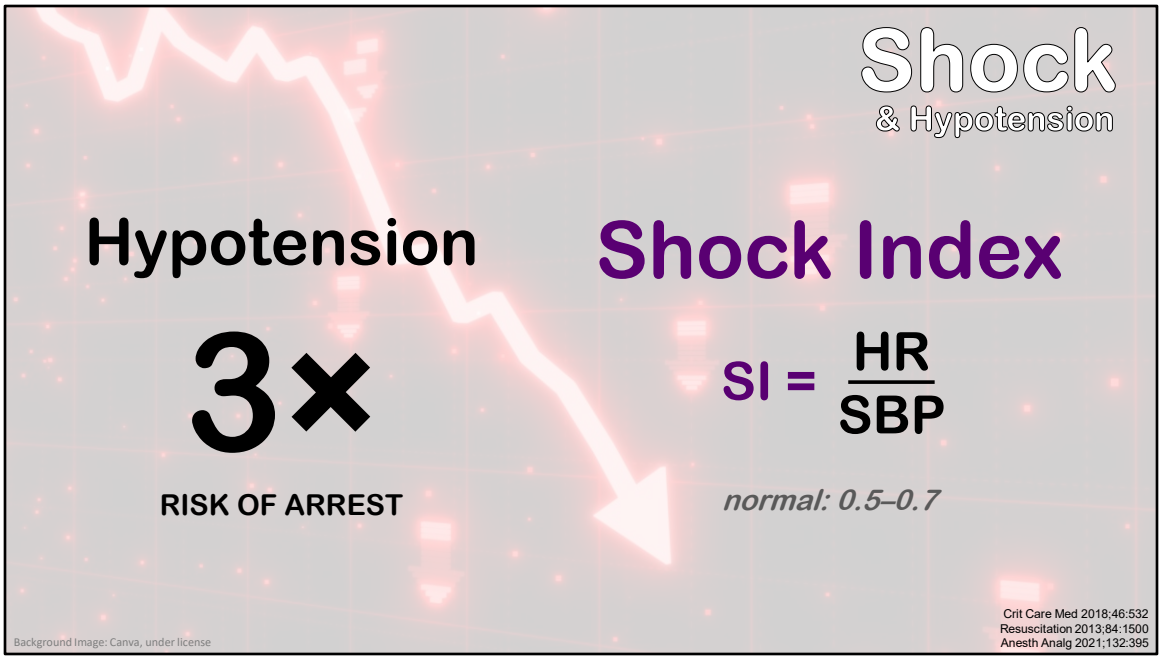
And here's where the physiologic difficulty arises: Patients that have exhausted their bicarbonate buffer system are reliant on CO_2 removal to maintain as close to normal pH as possible; often, this is accomplished through profound increases in minute ventilation (think of Kussmaul's respiratory pattern in the patient with DKA – respirations are both rapid and deep, increasing both rate and tidal volume to maximize minute ventilation and remove CO_2). Any period of apnea will lead to a rise in CO_2 (and subsequent fall in pH), and failure to maintain the same minute ventilation after intubation will also result in accumulation of CO_2 and worsening acidosis. Depending on the patient's pH when RSI is commenced, even a small increase in CO_2 may be fatal.



This is a SUPER tough patient to manage.

The only two recommendations from SAM are:
- Consider awake intubation (i.e., don't make the patient apneic)
- Consider spontaneous vent mode after intubation (maybe AC/PC?) to allow the patient to draw as much minute ventilation as they need

Scott Weingart adds that bicarb is probably not useful because it's mechanism of action is dependent on generating, and clearing, CO2 – if the patient is maximally compensating and cannot increase their minute ventilation any further, giving bicarb might actually *worsen* acidosis when the resulting CO2 can't be cleared!



There is a definite correlation between hypotension and peri-RSI arrest.

However, it's important to remember that not all shock is necessarily "hypotensive"—patients may initially compensate enough to support a normal (or non-hypotensive) blood pressure in early shock states, despite having an underlying shock physiology. Taking away those compensatory mechanisms (through, for example, RSI pharmacology) may lead to cardiovascular collapse.

For that reason, the shock index should also be used to evaluate for occult risk.

The shock index is calculated simply by dividing the patient's heart rate by their systolic blood pressure. A normal shock index should be between 0.5 and 0.7.
(e.g., a normal resting HR of 60 divided by a normal SBP of 120 = 0.5)

Simply put, a shock index of 0.9 or greater is independently associated with increased likelihood of peri-RSI cardiac arrest, and the risk increases with higher shock indices.

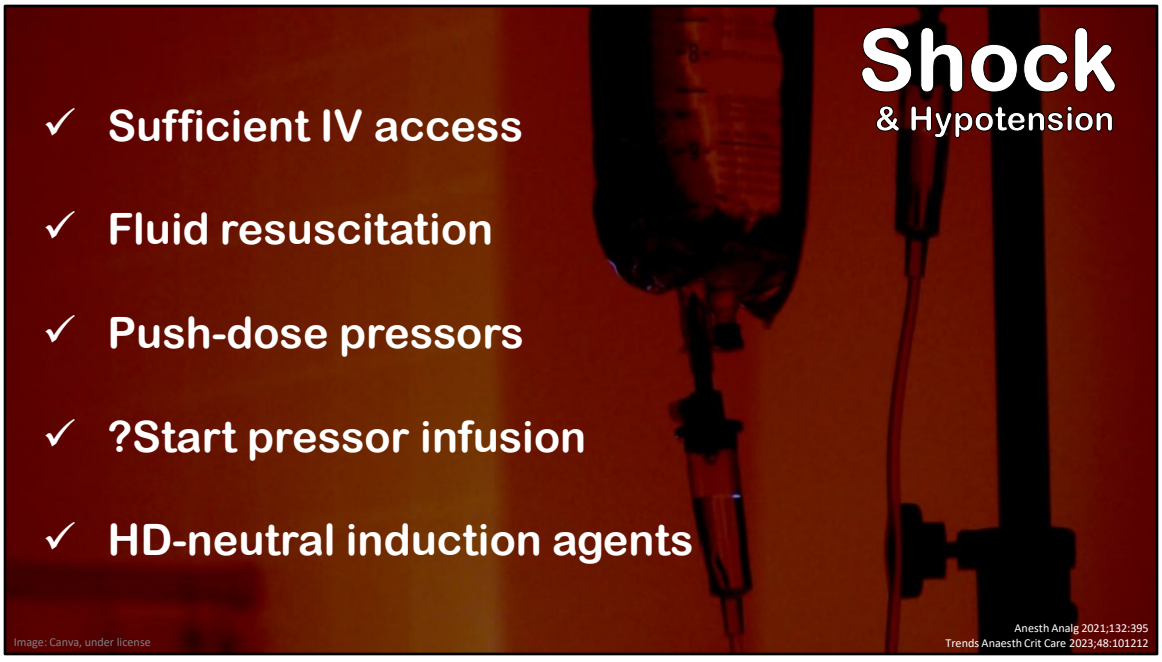
Take that same patient with a SBP of 120 – not "hypotensive" by most definitions, but coupled with a HR of 110, that patient now has a shock index of 0.92 and extreme caution should be used before administering an induction agent for RSI.

But, let's be pragmatic about this: Nobody likes doing mental math, let alone mental division. So, let's consider a few mathematical truths:

- 0.9 is pretty close to 1.0
- Any fraction equaling 1.0 has the same number in the numerator and denominator
- As the numerator gets larger, so does the quotient of that fractional expression

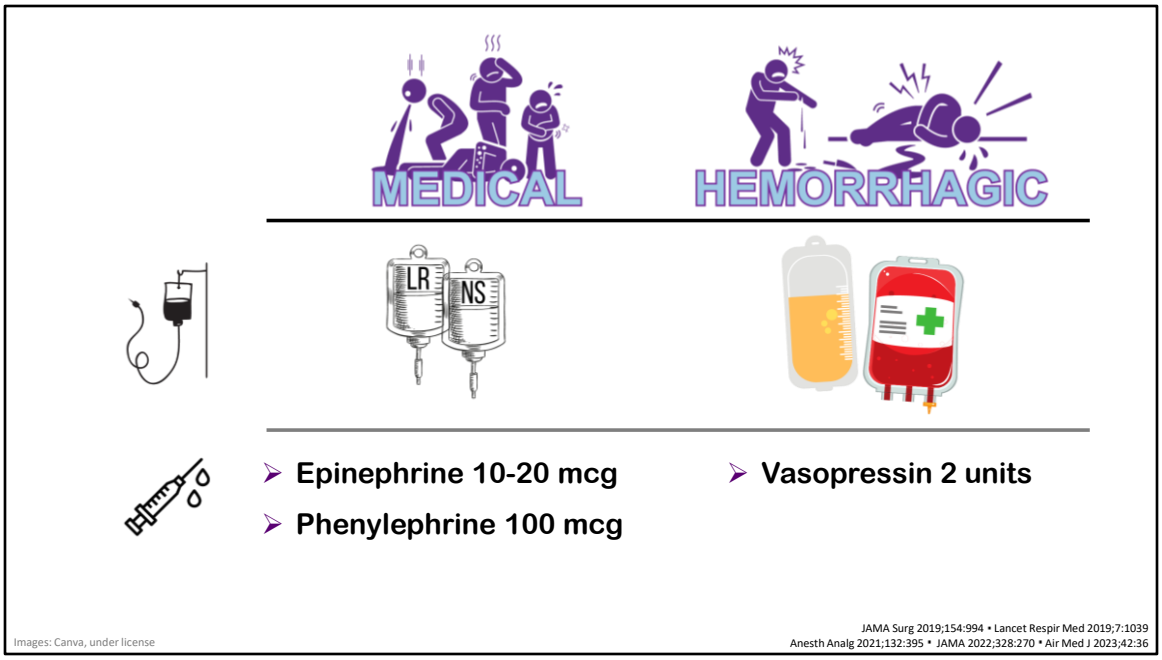
Therefore, here's my 3AM-math-shock-index:

If the patient's heart rate is greater than or equal to their systolic blood pressure, assume they're going to die if you RSI them without aggressive up-front resuscitation!



For patients with pre-induction shock or hypotension:

- Ensure sufficient vascular access
- Initiate fluid resuscitation and have push-dose pressors available (more on those in the next slide)
- Consider starting a preemptive vasopressor infusion – I like this idea, especially those with pre-existing hypotension or borderline ("soft") blood pressures, because if nothing else, it gets the pressors in the room and has them primed, on a pump, and ready to infuse – one less thing to worry about if the patient's condition begins to deteriorate. You can always start at a very low dose, then turn them off if they're ultimately not needed.
- The concept of hemodynamically neutral agents will also be discussed in a couple slides.



Medical resuscitation is bit more common practice so probably don't deserve too much of an in-depth discussion.

For *trauma* (hemorrhage), remember the best "fluids" is going to be blood product resuscitation.

But what about pressors?

Most clinicians are facile with the use of push-dose epinephrine and/or push-dose phenylephrine for medical shock.
If you're not, you can easily make push-dose epi!

- *FIRST: Either squirt out 1 mL of a 10 mL saline flush, or draw 9 mL of saline into a 10 mL syringe.*
- *THEN: Draw up **1 mL** of **code-dose** epinephrine (1:10000, or 1 mg / 10 mL concentration) into that syringe with the 9 mL saline.*
- *You should now have 0.1 mg (100 mcg) in a 10 mL syringe, for a **final concentration of 10 mcg/mL**! Push 1-2 mL (10-20 mcg) at a time.*

More recently, the use of vasopressin (aVP) has been studied for a potential benefit in **hemorrhagic** shock.

While not specific to intubation, the AVERT Shock trial demonstrated safety of aVP use in patients with hemorrhagic shock; aVP use was associated with fewer blood product transfusions and no change in baseline mortality or overall complications.

More recently, the VIPER study demonstrated successful correction of hypotension with aVP in traumatic (hemorrhagic) shock (and use push-dose phenylephrine in medical shock) amongst aeromedical RSI patients – HOWEVER, many patients (84% in the medical cohort and 67% in the trauma cohort) required subsequent doses of push-dose vasopressors for recurrent hypotension.

And, HALF OF ALL REPEAT EPISODES OF HYPOTENSION IN BOTH COHORTS OCCURRED WITHIN 15 MINUTES!
(Remember before about arrest most likely in first 10-15 minutes??)

Lastly – despite push-dose vasopressors and other resuscitative measures, 21% of medical patients and 14% of trauma patients still experienced peri-RSI cardiac arrest in this study!

It's also probably reasonable to use epi or phenylephrine in hemorrhagic shock patients for a limited course to maintain hemodynamics during RSI while actively resuscitating per standard trauma resuscitation guidelines.

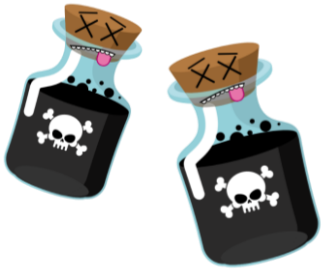
Now let's talk about "hemodynamically neutral" induction agents.

Clearly, some induction agents (looking at you, propofol) are a poor choice

But which is better – etomidate or ketamine?

The bottom line: BOTH can lead to peri-intubation hypotension and arrest!

Ketamine has sympathomimetic effects, but if a patient is maximally stressed and catecholamine depleted, unchecked negative inotropic effects can dominate and cause worsening hypotension



"All things are poison and nothing is without poison. Solely the dose determines that a thing is not a poison"

~Paracelsus

SI≥0.9: 50%

INDUCTION DOSE

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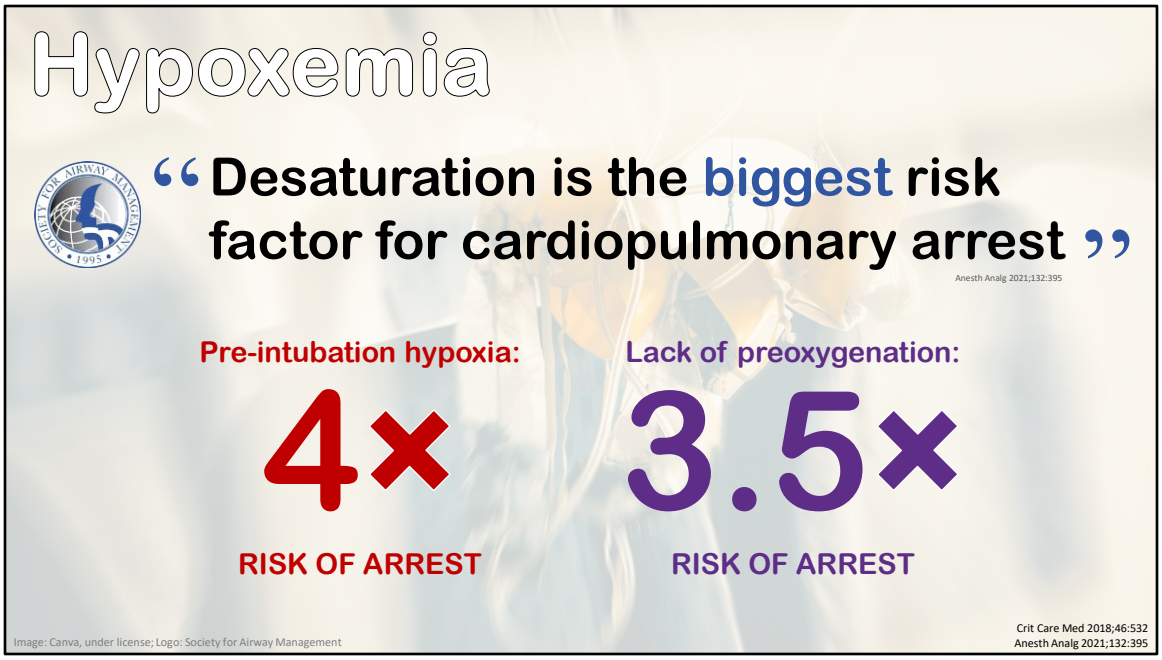
Emerg Med Clin N Am 2018;36:61
Acad Emerg Med 2020;27:1106
Ann Emerg Med 2023;82:471

The current best-practice recommendations are to reduce the dose of the sedative/induction agent – regardless of the drug used – by *at least* 50% in patients with hypotension or elevated Shock Index (consider starting even lower).

Remember: You can always give more of a medication, but once given you can't take it away.

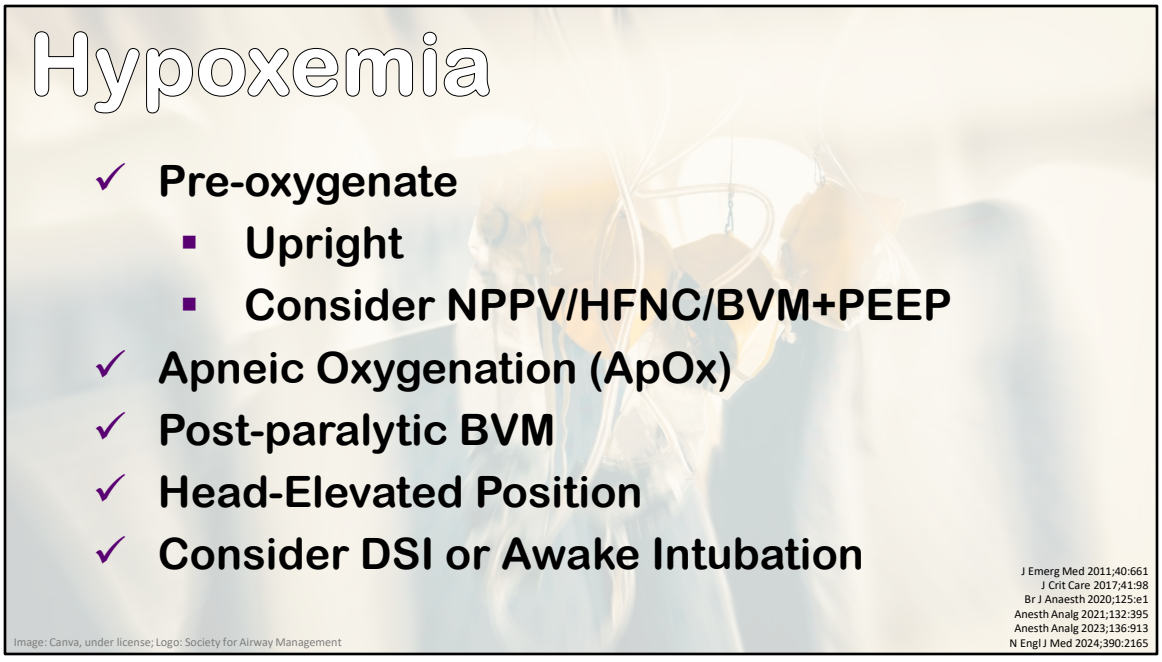
There was a paper published in late 2023 (Ann Emerg Med 2023;82:471) in which a retrospective analysis of the NEAR database demonstrated no difference in peri-intubation hypotension regardless of dose of medication used. However, this was naturally limited by the retrospective nature of the study, and I maintain that the safest course of action is to start with a lower dose and titrate upward to effect.



And this brings us to the last of our “CRASH” killers – hypoxemia.

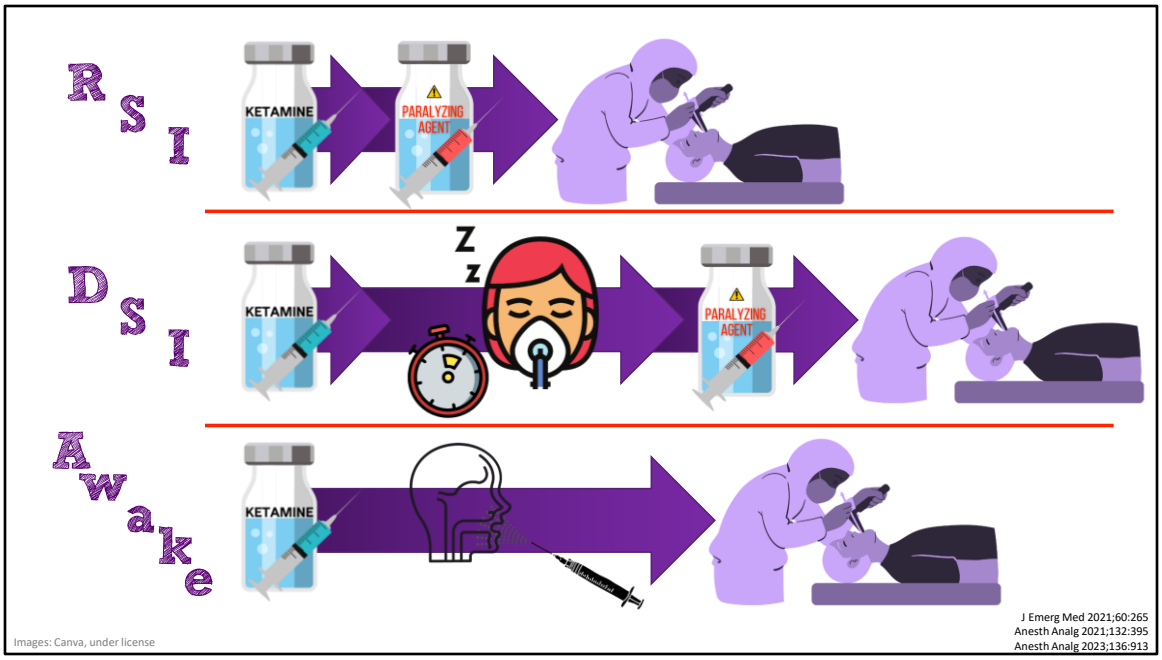
The Society for Airway Management identifies desaturation as the greatest risk factor for hemodynamic collapse and cardiopulmonary arrest in patients undergoing airway management.

Pre-intubation hypoxia increases risk of arrest by four-fold, and failure to adequately preoxygenate can more than triple odds of arrest.



From SAM Guidelines:
- Preoxygenation should be performed using high-flow oxygen for **at least 3 min**, or **8 vital capacity breaths**.
- Patients should be preoxygenated in the upright position when possible.
- If the patient has significant **shunt physiology** or reduced functional residual capacity (eg, pregnancy, obesity, ARDS), **preoxygenation** should be **performed with PEEP using NIPPV**.
- Consider SGA if high PEEP is required for pre-ox
- Delayed sequence intubation is an option for patients who cannot tolerate preoxygenation with NIPPV or HFNO.
- Consider awake intubation to maintain spontaneous respiration should be strongly considered for patients with refractory hypoxemia.

Weingart has long advocated for NIV (CPAP or BiPAP) as preox, and using a *ventilator* as an even better option to NIV (basically put them on AC-PC or AC-VC via BVM mask). The 2024 PREOXI trial demonstrated significant reduction in the rates of hypoxemia during intubation in patients pre-oxygenated with NIV than those preoxygenated with a standard oxygen mask.

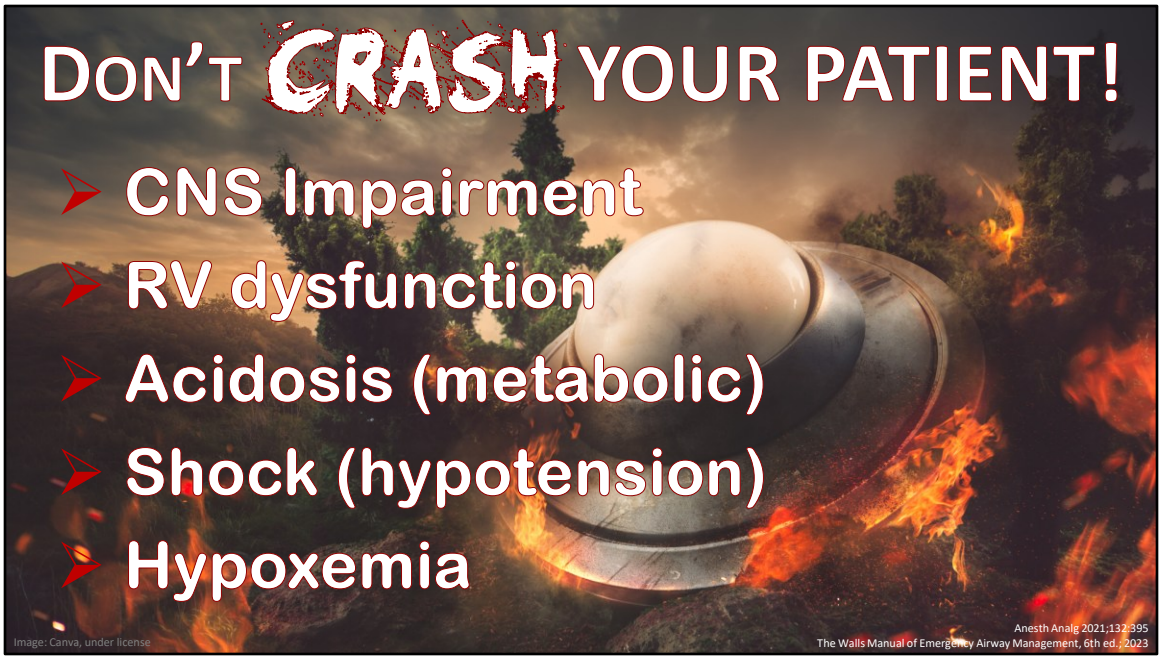


A quick comparison between traditional “RSI” and the concepts of “DSI” and “Awake intubation”

While there are differing practices on how to best perform awake intubation, adding a topical anesthetic to the sedative can provide more favorable conditions that sedation alone. Importantly, a recent study looking at this practice found that *both* ketamine-only intubation and ketamine-plus-topical had lower first-pass success rates and higher complication rates than did RSI. However, ketamine-only fared the worst in both metrics.

DSI Tips:

- Always be ready for emergent intubation with ketamine
- DO NOT SLAM ketamine, can induce apnea
- Consider small aliquots of ketamine (10-20 mg at a time) until the patient is adequately dissociated; this may transiently result in dysphoria (i.e., “the K-hole”), but patients with shock physiology will often require far lower doses of ketamine to fully dissociate than one might think



In summary –Next time you have to RSI a patient –make sure you don't CRASH them!

Be mindful of CNS and RV pathology
Be extremely cautious with severe metabolic acidosis (can they be managed noninvasively?)
Look for, and correct, *shock*—not just hypotension
And, lastly, aggressively correct and prevent hypoxemia

Jacob Miller, ACNP, ENP-C, CNS, NRP, FAEN, FAEMS

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