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Rural Airway Manual

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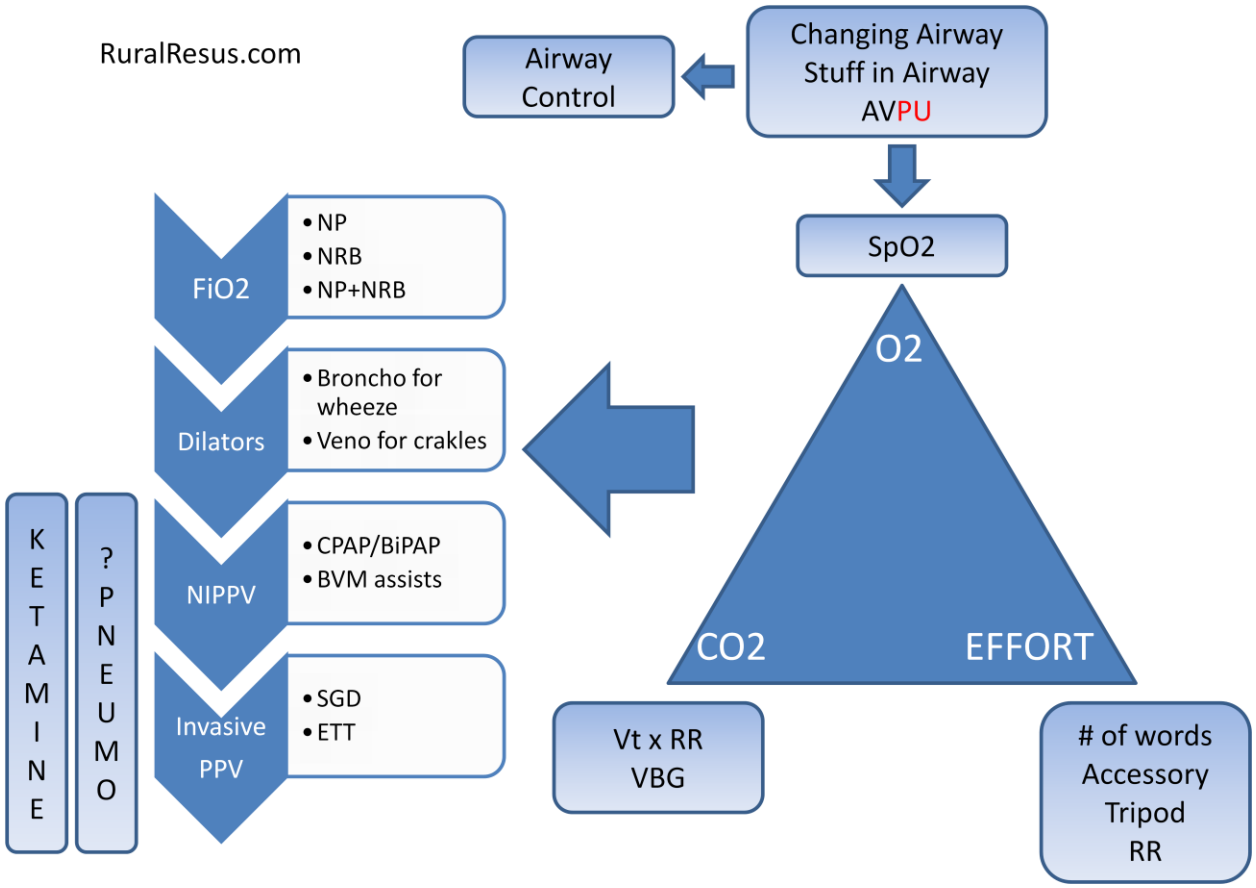
Intubation is very resource intensive. It takes time, manpower, equipment, cognitive load and medication. It carries the potential for catastrophic failure. Deciding to intubate in a rural or community hospital will typically put all other resuscitative efforts on hold for at least 20 to 30 minutes. Therefore, the first step is to decide whether intubation actually solves the problem you have, and whether it is the only means of solving it. Rather than going into a resuscitation with the mindset of "I must secure the airway at all costs", a more graduated response is beneficial.

Intubation can only accomplish 4 things:

1. **CONDITION 1:** Protect airway access if the airway is altered, or is changing for the worse (e.g. angiodema, burns, trauma).
2. **CONDITION 2:** Protect the airway against aspiration where there is "crap" in the mouth (e.g. blood, vomit, pus) and/or the patient is too obtunded to mount effective aspiration protection.
3. **CONDITION 3:** Ventilate passively when all other methods (including patient's respiratory muscular efforts) have failed.
4. **CONDITION 4:** Reduce the oxygen demand of respiratory muscle use in a patient who can't afford such demand (e.g. septic shock with poor tissue perfusion).

Keep these ***4 conditions*** in mind as we look at a global approach to a patient with shortness of breath/respiratory difficulty. Please use the SOB cognitive aid as you go through the next section.



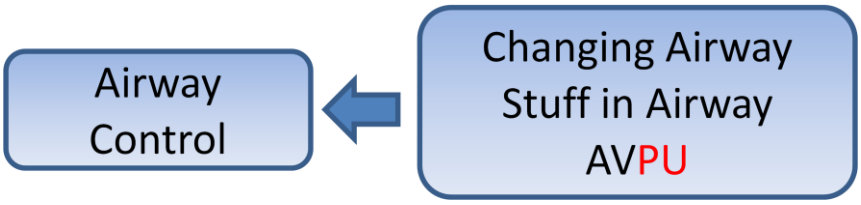
Shortness of Breath Cognitive Aid

The first step is to see if **CONDITION 1 OR 2** apply, as they genuinely can only be solved by an endotracheal tube (ETT). It is a very brief step with 3 questions

1. **CONDITION 1:** Is the airway altered, or changing for the worse (e.g. angioedema, burns, trauma)?
2. **CONDITION 2:** Is there "crap" in the mouth (e.g. blood, vomit, pus)?
3. **CONDITION 2:** Is the patient P or U on the AVPU scale (Awake, responds to Verbal, responds to Physical, Unresponsive).

If the answer to any of the questions is ***YES***, then start mobilizing the resources for an intubation in parallel to other resuscitative efforts. Just how urgently I will focus on the ETT versus other efforts will depend on which of the questions is positive, in a descending order of urgency (i.e. a changing airway is more urgent then an obtunded patient).

You probably noticed we are using AVPU instead of GCS to determine the level of obtundation. GCS is too cumbersome, takes too long, is unreliable and does not accurately predict the airway protection ability of comatose patients. However, the general concept of decreasing ability to protect the airway with increasing levels of obtundation is a valid one, and one captured with the AVPU score much faster and simpler then the GCS. Score of P reliably corresponds to a GCS of 8, and U to a GCS of 6. If we take the traditional cut-off of 8 for intubation (taken from ATLS), P or U are markers for deep obtundation, that will probably need airway protection in the near future.



If the 3 questions above are negative, we get to assess the patient's respiratory function, in order to judge the potential for **CONDITION 3** to be present or impending.

Functionally, lungs are a simple organ that accomplish two things: 1) transport O2 to tissues, and 2) remove CO2. All of this is accomplished using muscular effort. This gives us the 3 apices of the respiratory failure triangle: you can fail oxygenation, CO2 removal or you can tire out due to increased effort.

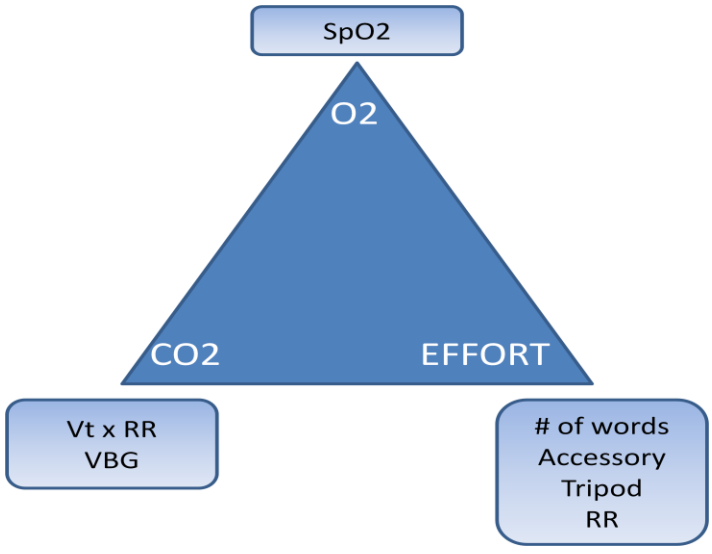
Oxygenation is simply measured using an SpO2 monitor as about 98% of O2 is carried by hemoglobin and only about 2% is carried dissolved in the blood (this is the PaO2 that is measured by ABG and is functionally irrelevant).

CO2 removal depends on only two things: 1) metabolic output, and 2) minute ventilation (i.e. the amount of air we move in and out of the lungs each minute). Metabolic output tends to be stable or decreasing once we begin our medical management of a patient. So in essence, CO2 removal only depends on the minute ventilation, which is measured by the simple formula of RR X Vt (ventilatory volumes). RR can be counted and Vt can be estimated by looking and listening with a stethoscope. The normal tidal volume is 500cc and we all have a lifetime of experience of looking at people moving 500cc per breath. We are not looking for precision here, just a gestalt assessment of *adequate* versus *decreased* versus *increased* ventilation. Once things are more stable, you can confirm your gestalt by getting a VBG and interpreting the pCO2 from this VBG.

Effort is also a clinical assessment. My best predictor is the number of words a person can string together. It takes a lot of respiratory reserve and coordination to speak full sentences (just try holding a conversation while doing wind sprints). If the patient can only say a couple of words at a time, their effort is very large. Other useful parameters are the RR (increased with effort and decreased if tiring out), accessory muscle use and tripod positioning or pursed lips.

The advantage of using the respiratory triangle is that it allows us to estimate a patient's respiratory status within seconds, and with very limited tools (SpO2 monitor, eyes and a stethoscope).

If the patient is failing on any of the respiratory triangle apices (or multiple ones), we get to apply the **Respiratory Treatment Progression** which is, luckily, identical no matter what the underlying condition is.



The Respiratory Assessment Triangle with the failure apices

We start by increasing FiO2. Nasal prongs (NP) give us about 3% bonus per L of flow and if cranked way past the 15L/min can actually provide close to 100%, assuming the patient keeps their mouth closed. It is quite uncomfortable, so we reserve this approach for people who have deep failure on an apex or two.

Non-rebreather (NRB) gives us about 70% FiO2 and its valve should be cranked past 15L as well, to ensure that most of the inspired gas will be pure O2 instead of air admixed from patient's surroundings.

NRB+ NPs, both cranked past 15L/min gives us 100% FiO2 reliably.

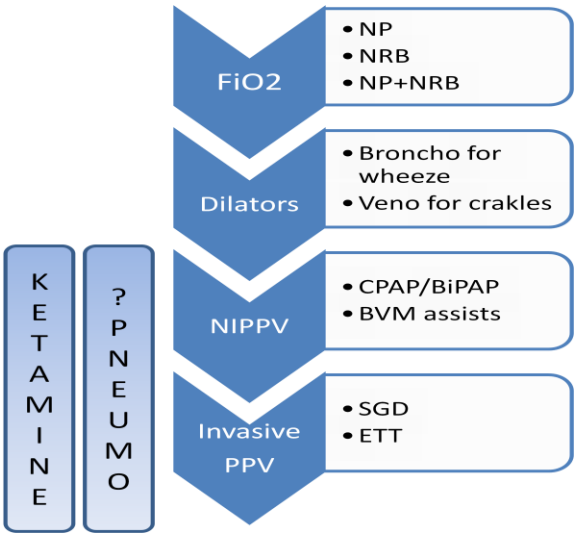
Warmed, humidified nasal high-flow oxygen device (commercial name Optiflow) can also give 100% FiO2 comfortably for days on end

If added O2 is not fixing the failure of one or more of the apices, we do a quick assessment to see if the dilators could be of use: bronchodilators (salbutamol) for wheezy patients and venodilators (nitroglycerin, furosemide) for bilaterally crackly patients.

If neither of the above treatment have restored all 3 apices to satisfactory status, it is time to apply positive pressure ventilation (PPV). However, before we do that we need to do an assessment, with stethoscope, CXR or bedside ultrasound, to exclude the presence of a pneumothorax. This is the only cause of respiratory difficulty that would get worse with PPV.

There are 4 ways to deliver PPV. The escalating order to follow is: Non Invasive PPV (NIPPV: CPAP or BiPAP), BVM (assists or full ventilation), Supraglottic device (SGD: LMA or King LT) or an ETT. There is no fundamental difference in ability to deliver PPV through any of these devices. They simply differ in the location in the respiratory tree where the PPV is delivered (mouth for NIPPV and BVM, pharynx for SGDs or trachea for ETT) and how much aspiration protection they offer (none for NIPPV and BVM, decent with SGDs, most with ETT). All of these methods can also provide 100% FiO2.

As we apply these methods of PPV delivery, we might find that a hypoxic, hypoventilated, tiring patient is too agitated to tolerate them comfortably. This is where Ketamine can be of help. I prepare a syringe with 100mg and if the patient is not doing well with PPV, I inject aliquots of 20mg IV q2-3 minutes until the patient is dissociated sufficiently to tolerate the chosen method. If I find that NIPPV or BVM are still not working well, I would inject another 200mg of Ketamine and proceed to insertion of an SGD.

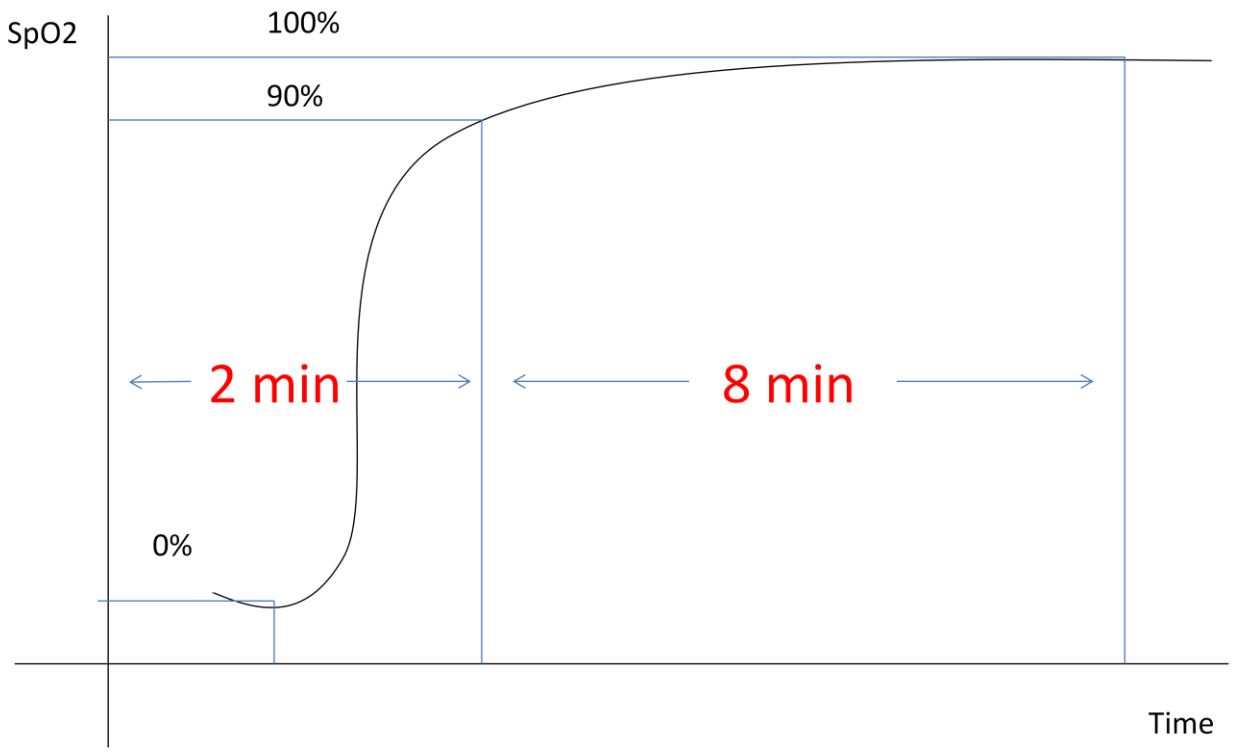


The respiratory treatment progression

But wait, why mess around with all this progressive treatment stuff, when the good old Rapid Sequence intubation (RSI) with an ETT will solve all these problems definitively and quickly? Why not just secure the airway?

The first reason, as mentioned in the beginning, is that an RSI with an ETT is neither quick nor easy. In a single provider situation, it will become your sole focus of attention. It will rob you and your team of situational awareness for quite a while, and grind to a halt all the other (possibly more important) resuscitative efforts.

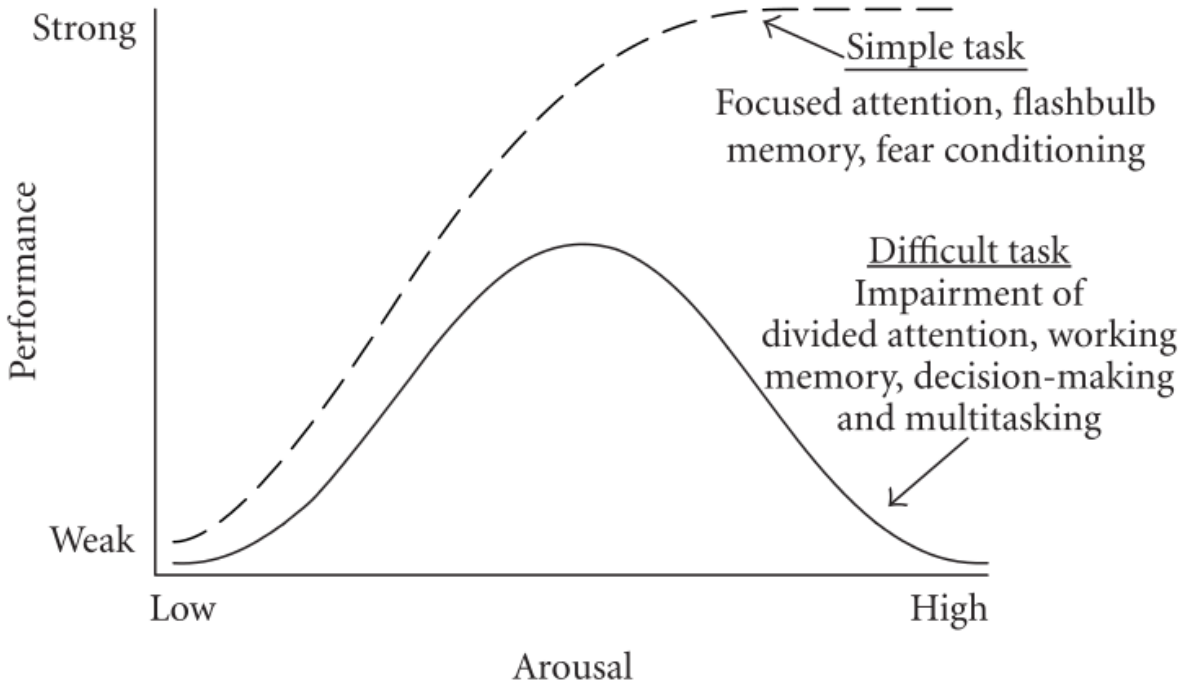
The second reason is the oxygen dissociation curve and its implications for safe apnea. A properly (progressively) hyperoxygenated patient who has all the nitrogen in their lungs replaced with oxygen (typically 3 minutes of tidal volume breathing with 100% FiO2) will tolerate an average of 8 minutes of apnea before their O2 saturation will drop from 100% to 90%. If you start with the patient who is at 90% (and has not been hyperoxygenated), they will only tolerate about 2 minutes of apnea before their O2 saturation will go down to ZERO! The drugs take about 1 minute to fully take effect (during which time the patient is getting progressively more apneic), larynx visualization can take about 30 seconds and ETT placement 15-20 seconds more (assuming everything is going fine with visualization and tube placement). Thus, it is easy to see how a “crash tube” can result in a deeply hypoxic patient, and why it is generally a safer approach to use the progressive methods to achieve proper hyperoxygenation before one begins the process of placing an ETT.



Desaturation under apnea

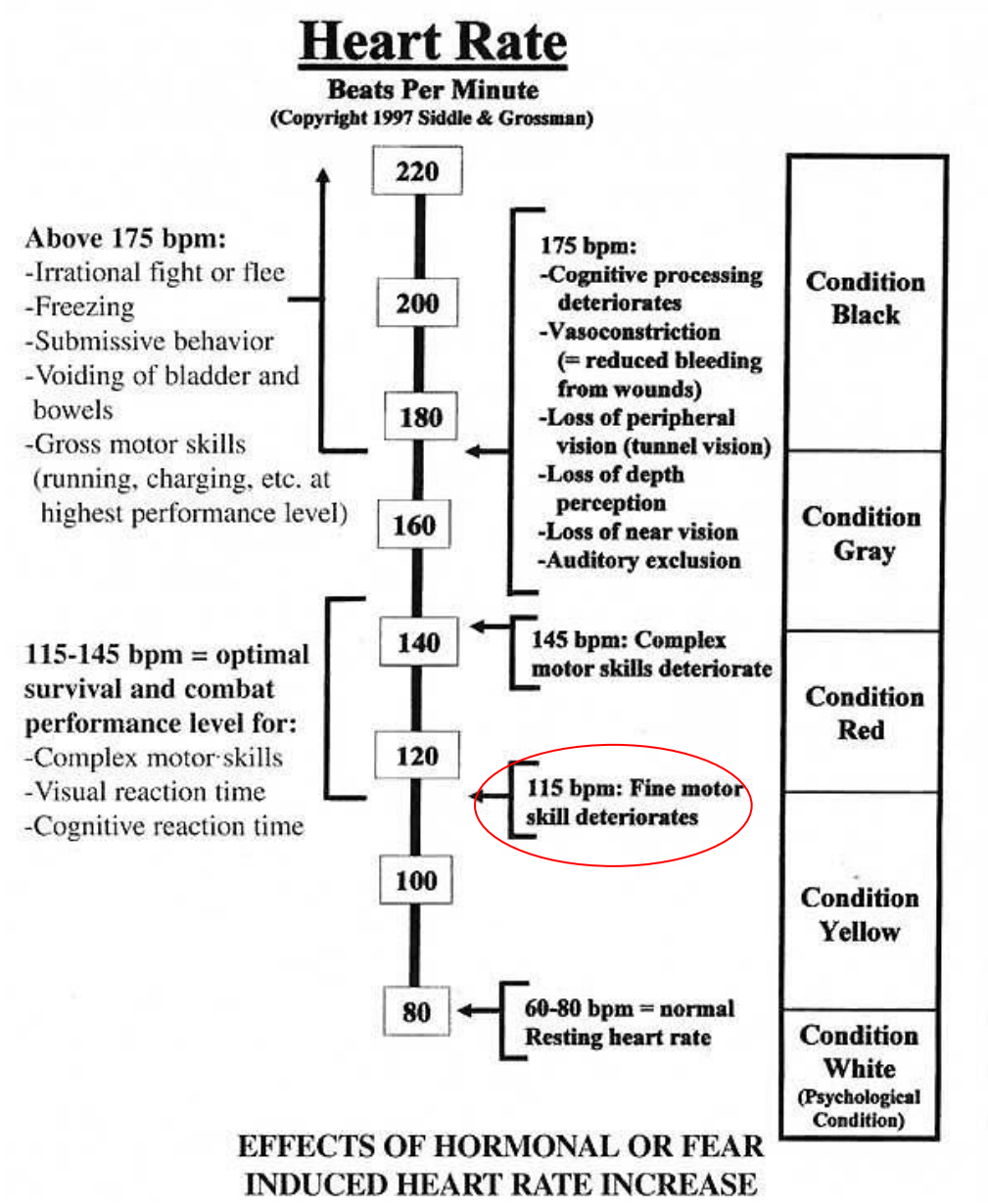
(adapted from Manual of Emergency Airway Management, 3rd ed)

The final reason is that a hasty RSI will likely be done in a state where both the intubator and the other staff are highly activated and even agitated. Such a state of agitation can lead to poor teamwork, medication errors, inadequate preparation, poor positioning and faulty laryngoscopy technique. Laryngoscopy is an especially complex task requiring fine motor skills. Thus, it is heavily subjected to Yerkes-Dodson law which simply states that for complex tasks, a little bit of arousal is beneficial, but hyperarousal quickly diminishes performance. Fine motor skills especially degrade quickly as heart rate and anxiety response mounts.



Yerkes-Dodson Law.

Laryngoscopy and RSI are highly complex tasks.



Degradation of fine motor skills with operator heart rate and anxiety increases

Adapted from Grossman, 1996

All right, we have finally decided to intubate the patient. Let's proceed through the mental and physical process of doing so. Everyone worries about the patient with the difficult anatomy. But that is something that we can't change or influence. It is not under our control, nor is it is not the cause of most of failed intubations. Most intubations fail because of operator hyperarousal, inadequate oxygenation or poor preparation and positioning of the patient. Luckily, all those things ARE under our control.

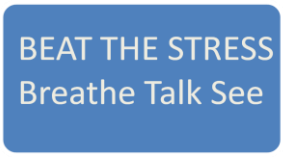
The first step is getting yourself to a proper physiological and psychological state, so you are on the good part of the Yerkes-Dodson arousal curve. Don't skip this step, even if it looks a bit silly at first. A keyed up intubator is not a good intubator.

A useful tool developed by an Australian critical care paramedic is called **Beat The Stress**, or Breathe Talk See.

Breathe: Take two slow, controlled deep breaths.

Talk: Positive self-talk (e.g. "I have the skills and knowledge to do this properly and I will see it through").

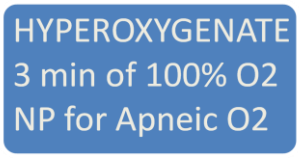
See: Visualize your next two actions to get your brain on the proper train of thought (e.g. "Now I am going to apply high flow O2 and then position the patient").



You might need to come back to this step during the intubation process if you feel your level of arousal rising to unhelpful levels. This is perfectly ok.

The next step is to get the patient properly hyperoxygenated using the **Respiratory Treatment Progression** so their oxygen saturation is at 100% for 3 minutes, while receiving 100% FiO2 if at all possible. Note that this could involve NRB+NP, Optiflow, NIPPV, BVM assists or even an SGD placement, in addition to Ketamine.

The second component of this step is leaving a set of Nasal Prongs cranked at 15+L underneath whatever method of preoxygenation you are using, in order to engage in **Apneic Oxygenation** during your intubation attempt. This will increase your period of safe apnea by another **2 minutes**.



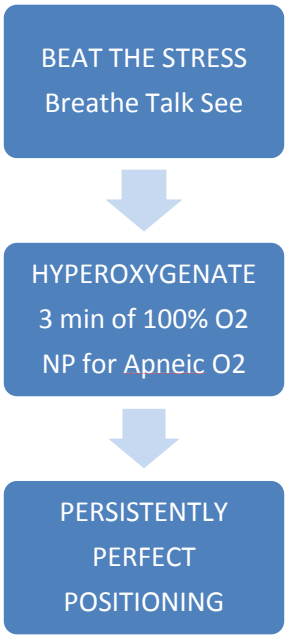
After we have achieved hyperoxygenation, we will put the patient in **Persistently Perfect Positioning**. Positioning has two goals: 1) aligning the visual axis in such a way to convert the 90 degree pharynx angle into a straight line, and 2) giving us maximal mechanical advantage to achieve the 20 Newton force a typical laryngoscopy will need.

This has 4 steps:

1. Patient's head at the very top edge of the bed.
2. Patient's head positioned between your symphysis pubis and your umbilicus.
3. Occipital elevation achieved by use of an occipital pad, pillow, towels, or in the obsese patient, a ramp until the ear lobe is in line with the sternal notch
4. Head of bed elevated to about 30 degrees

Realistically, you will have to wait for your induction drugs to take effect to put the patient in this position and that is fine. You just have to anticipate this step, brief your team ahead of time, and once the patient is fully sedated, perform it in a rapid and organized fashion. Avoid the urge to rush this step. That is why we used hyperoxygenation and are using apneic oxygenation. You will have time to position

the patient correctly. A small misalignment here, can have profound consequences on your laryngoscopy success. We are aiming for perfection every single time.



Okay, we have now optimized all of the factors under our control. Realistically, you will find that doing these 3 steps will turn most of your intubations into a very manageable task with little drama. However, an airway can still surprise you in a negative way, so it is time to focus on the patient characteristics

Airway courses tend to have complicated protocols using many mnemonics (LEMON, TOPS, etc). While it is very important to be aware of those factors (and in fact, we expect you to have the **Strikes** we use memorized by the time you come to the course), when you are staring at a potentially difficult airway as a sole provider with no immediate backup, it is very hard to remember all those details. Cognitive space is at a premium and you will need it for other things. This is why we are going to use a very simplified approach.

We will split the patient into 3 possible groups:

1. **Shit Show Airway**. A "shit show airway" is the one that makes you go "oh, shit"! It could be a burned airway in C spine precautions. It could be a massive hemoptysis. It could be a massively obese patient with a tiny mouth. It is a gestalt assessment that includes the **Strikes** into one global judgement. I am purposefully not giving a formal definition or a number of Strikes or which ones you need. What creates an "oh shit" intubation at the beginning of your carrier might be a mildly interesting airway with a few years of experience under your belt. Bottom line is that the "shit show airway" is the one where you are not at all certain you will be able to visualize the larynx or pass the tube.

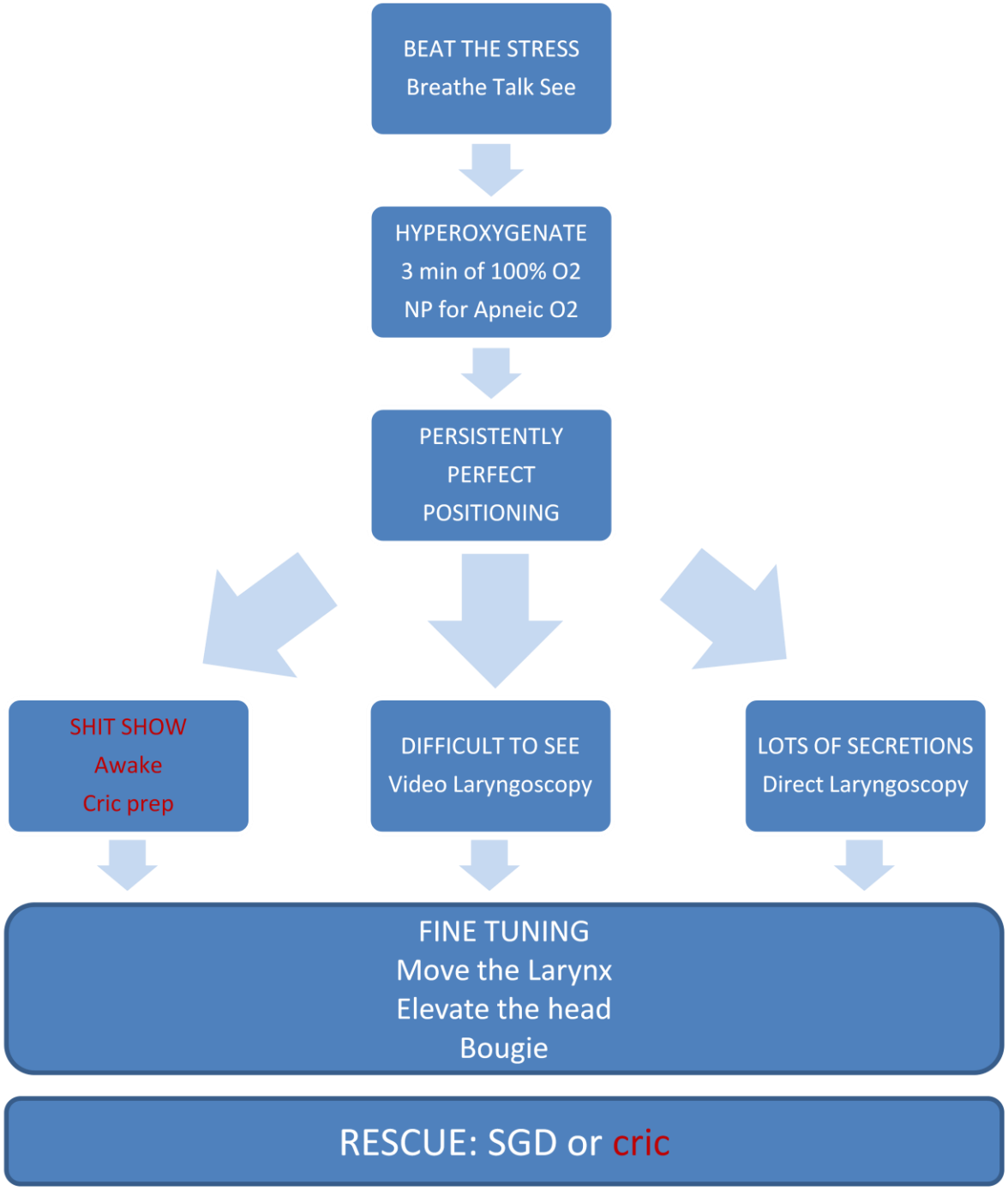
In that case, we will prepare for a surgical airway right away. Have your 3-step surgical airway kit ready and assembled and the patient landmarked, skin marked and cleaned for a cricothyrotomy.
You will also prepare for a potential awake intubation. Topicalize the airway with nebulized 2% lidocaine followed by deep squirts of lidocaine spray. Use the method of laryngoscopy you are most comfortable with (direct or video laryngoscopy). Then give dissociating doses of Ketamine (20-100mg). Have another 100mg of Ketamine ready as well as Rocuronium drawn up. Take a look at the airway. If the view is good AND you can maintain it, give the rest of the Ketamine plus the Rocuronium and intubate once the drugs take full effect.
If you cannot get a great look, try to intubate ONLY once you improve the view using our **Fine Tuning** tools (see below). If that one attempt fails, move on to a surgical airway.

2. If it is not a "shit show airway", you get to decide if the video or direct laryngoscopy (DL) will be a better tool. In general, video laryngoscopy (VL) is helpful when we have trouble converting the 90 degree pharynx angle into a straight line. A classical case is a trauma patient in C-spine precautions that prevents us putting the patient in the perfect sniffing position. Another example is an obese, short necked patient with limited space in the mouth. On the other hand, VL has difficulty with lots of secretions as they tend to get smeared on the tip of the camera and obstruct the view. In those cases, DL is our preferred choice.

In either case, if we are not getting a great view, we will use the same set of sequential **Fine Tuning** tools in order to improve the view.

1. **Laryngeal manipulation with the right hand**. Get an assistant's thumb and forefinger to grasp the larynx **gently** and put your right hand over theirs. **Gently** move the larynx to where you can see it. Most of the time, it will be to the right, posterior and up (BURP). This is not the same as using lots of pressure to occlude the esophagus (Sellick's manouver) which has been shown to be counterproductive.
2. **Head lift**. This helps with an anterior larynx. Put the palm of the right hand under the occiput and lift the patient's head until the larynx drops in to view. Have folded towels ready to put under the occiput by an assistant (on top of your already placed occipital pad as part of Persistently Perfect Positioning).
3. **Bougie.** If you still don't have a great view, try a bougie. Note that the standard bougie is not optimized for hyperangulated VL blades like the GlideScope and thus this step might not be useful with that particular method.

If the intubation fails and it is not a "shit show airway", place a supraglottic device and ventilate through that until more experienced help arrives. If you have trouble placing the SGD or ventilating through it, proceed to surgical airway.


BEAT THE STRESS
Breathe Talk See
HYPEROXYGENATE
3 min of 100% O2
NP for Apneic O2
PERSISTENTLY
PERFECT
POSITIONING
SHIT SHOW
Awake
Cric prep
DIFFICULT TO SEE
Video Laryngoscopy
LOTS OF SECRETIONS
Direct Laryngoscopy
FINE TUNING
Move the Larynx
Elevate the head
Bougie
RESCUE: SGD or cric

STRIKES

Altered anatomy (burns, andiogema, hematoma, prev surgery)
Crap in mouth (blood, puss, vomit)
Obesity
Small mouth or short neck (less then 3-3-2)
Tight space in the mouth (high Malempati score)
Limited neck mobility (C spine, RA, spinal fusion)

A brief note on **CONDITION 4** (eg a septic patient we are intubating to optimize tissue O2 delivery). You would apply the same methods as the other 3 condition. The only difference is that you might have to anticipate post-intubation hypotension and treat it with Phenylephrine (see below).

Meds:

Induction

Ketamine is going to be out mainstay induction drug. First, it can be given in dissociating doses, facilitating our Awake intubation. Second, it does not produce the same central apnea and pharyngeal tone collapse that other induction drugs do. It has a very high LD50, making it virtually impossible to poison the patient with it. Finally, it is not associated with post intubation hypotension, like some of the other agents. Contrary to popular opinion, it is safe to give in head-injured patients.

The only downside is that it results in secondary release of catheholamines, leading to tachycardia and hypertension. Sometimes, that can be a problem if the patient already has those two going on already. However, most of the tachycardia/hypertension in a patient needing an intubation is due to hypoxic and hypercarbic agitation, and the dissociative state that the Ketamine induces will usually have a more

profound relaxing effect then the moderate catecholamine release it induces. If you are really concerned about this, add a bit of Propofol.

Ketamine does have other, rarer, side effects, and some of them are important to know about

1. Hypersalivation. Generally with large doses and delayed, compared to the onset of sedation. If it happens, suction.
2. Post sedation vomiting and agitation. Again, should not be a problem as we are not planning to have the patient awake at the end of all this. Propofol top up at the time of induction reduces the chance of this developing. Alternatively, can use the Propofol top up when the agitation happens.
3. Laryngospasm. Really rare and short acting usually, though terrifying. Start with strong jaw thrust. If it doesn't fix it, add gentle BVM. Next, give Propofol top up. If all this it doesn't resolve it, paralyze the patient and ventilate passively (BVM or SGD or ETT)

Ketamine.

NOTE: dosing is much easier if you dilute it to 10mg/cc rather than 50mg/cc that some vials come with

1. Dissociation: 20-100mg. 20 mg at a time, q2-3 min, until patient is happy
2. Induction 100mg for normal size person, 150mg for big person.
3. Ongoing sedation: 75-100mg/hr drip

Propofol

1. Top up: 20mg at a time q2-3 min

Paralysis

We will always use Rocuronium (Roc). Succynilcholine (Sux) has contraindications. Roc does not. Sux can produce bradycardia. Roc does not. Sux produces fasciculations that can increase ICP. Roc does not. Sux produces hyperkalemia. Roc does not. At doses of 1.2mg/kg, they both produce equally good intubation conditions. Both of them have duration of action that is longer than the typical apneic desaturation time of a patient. Thus, there is no real benefit to Sux, except in the very rare cases of a previously known Roc allergy (in that case, dose of Sux is the same as Roc-100mg)

Rocuronium

1. Induction: 100mg. This should last 30-45 min
2. Maintenance of paralysis: 50mg whenever the patient starts moving

Maintenance of sedation

Of the agents below, Fentanyl is not a true sedative, but rather a pain killer with sedative properties at high doses. However, adequate pain control with it can significantly reduce the sedative doses of other agents needed. Thus, our usual approach is Fentanyl drip+ a sedative top up until the patient does not exhibit signs of agitation (movement if not paralyzed; heart rate and BP elevation +/- tearing if paralyzed)

1. Ketamine 75-100mg/hr
2. Propofol 30-80 mcg/kg/min (if converted to mg/hour, it will come in a 200-300mg/hour range)
3. Fentanyl 100-400mcg/hr

Adjuncts

The only adjunct we will use is Phenyelphrine to deal with a peri or post intubation hypotension (also, give fluid boluses). Lidoicaine and Fentanyl for traumatic brain injury have really weak evidence base and are complicated to remember and use if TBI intubations are not your daily reality. Atropine for drying out secretions takes 15-30 min to work, which is usually not a useful timeframe for a sick patient needing an ETT tube.

1. Phenylephrine. Comes in 10mg vials. Instill into a 100cc NS bag. This gives us a concentration of 100mcg/cc. Give 1-3cc at a time, or drip at 1-3cc/minute.

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