**Criteria:** 1. Patient experiencing clinical failure of airway maintenance or protection (includes patients with GCS ≤ 8) **OR**
   2. Patient experiencing clinical failure of ventilation or oxygenation (inability to maintain O2 saturation >90%) unable to be managed by less invasive means **OR**
   3. Anticipated clinical course is such that there is concern for the near term development of one or both of the above conditions, and/or resources or access limited (e.g., thermal airway injury, Airlift)¹

**NOTE:** This is a guideline for optimal advanced airway management. Clinical and logistical circumstances may influence options. Although listed sequentially many steps can be performed simultaneously.

**PRE-INTUBATION (PREPARATION) PHASE**

1. Initiate support of ABC’s, including initiation of pre-oxygenation.
2. Maintain spinal precautions if clinically indicated.
3. For cardiac arrest, refer to the special circumstances section of this protocol as medication adjuncts are generally unnecessary.
4. Apply cardiac monitor, O2 monitor, obtain vitals, and prepare all necessary equipment including rescue airway device.²
5. Evaluate for rapidly reversible causes.³ (e.g., removable foreign body, tension pneumothorax, opiate overdose, and anaphylaxis responding to medication…)
6. Evaluate airway for anticipated difficulties.⁴
7. Prepare patient.
   A. Pre-oxygenate patient.⁵
      • Maximal flow rates through O2 regulator should be used.⁶ Alternately use Non-rebreather at 15 Lpm AND nasal cannula at 15 Lpm.
   B. De-nitrogenate patient (to maximize oxygenated apnea time).⁷
      • Three minutes of tidal volume breathing (normal respirations) with a high FiO2 source is generally acceptable for most patients.
      • Cooperative patients can be asked to take 8 vital capacity breaths (maximal exhalation followed by maximal inhalation). This method can generally reduce pre-oxygenation times by about 60 seconds.
   C. Optimize initial patient positioning (for pre-oxygenation and de-nitrogenation):  
      • If initially found supine and clinically possible, the (approximately) twenty-thirty degree heads up position is optimal for pre-oxygenation over the supine position.⁸
      • If patient is on a backboard reverse Trendelenberg position can be used.
      • Conscious and alert patients can remain sitting in an upright position.
D. Re-evaluate airway for anticipated difficulties.
   • Look in mouth (e.g., dentures - leave dentures in place for pre-oxygenation phase if BVM or initial NiPPV is required as dentures facilitate proper mask seal. Remove dentures just prior to laryngoscopy.)
E. Apply and prepare monitoring (includes preparing for ETCO2 monitoring).
F. Establish reliable access (IV/IO).  
   • Maximize resuscitation efforts such as fluid administration if patient is hemodynamically unstable as the transition to positive pressure ventilation may initially worsen hemodynamics.
     • Consider push-dose epinephrine 10mcg IV in setting of ongoing hypotension/ inadequate perfusion. (May repeat PRN.)
G. When possible, set up for apneic oxygenation.
   • Apneic oxygenation using a nasal cannula at 15 lpm or greater during apnea phase can prevent desaturation. Apply nasal cannula oxygen during pre-oxygenation phase, if high flow rate not tolerated, increase the flow rate during apnea phase.
H. Finalize all equipment preparation and rescue airway materials.
I. Verbalize plan with partner/team for airway and ventilation management.
J. Verbalize failed airway plan.
K. Establish Cricoid status using Cricoid2 model (+/- Mark or Inject) - See Table 5.

8. If patient is resistant to pre-intubation preparations (i.e., delirium or agitation) initiate delayed sequence intubation (DSI).
   A. Administer dissociative dose of Ketamine 1-2mg/kg IV push11 and repeat as needed in doses of 0.5mg/kg slow IV push as needed to achieve disassociation and permit airway management preparations.

9. Evaluate effectiveness of pre-oxygenation prior to medication administration of intubation phase.12
   A. Goal is O2 sat of at least >93%.
   B. If O2 sat ≤ 93 % after initial attempt at oxygenation with high flow non-rebreather mask then initiate Non-invasive Positive Pressure Ventilation (NiPPV) such as CPAP/BiPAP to maximize oxygenation for at least 3 minutes prior to administration of paralytic.
      Alternately use BVM with PEEP valve AND nasal cannula at 15Lpm.
   C. Be aware: oximetry generally reflects a delayed value of central oxygenation by 30-120 seconds, depending on circulatory status.

**INTUBATION PHASE**

1. Administer induction (sedative) medication.13
   A. Ketamine 1-2mg/kg IV push.
      • If used for DSI and patient is adequately disassociated, a repeat dose does not need to be given for a separate induction.
• Ketamine is not recommended for patients with severe hypertension (e.g., isolated head bleed with severe HTN).
• Ketamine is the preferred agent for the hypotensive patient. Consider a reduced dose in the hypotensive shock state.

B. Alternate: Etomidate 0.3mg/kg IV

2. If no contra-indications are present, administer paralytic (neuromuscular blocking agent).
   A. Succinylcholine (depolarizing agent) 1.5-2mg/kg IV.
      • In the setting of severe hypotension or pediatric patient use 2mg/kg IV.
   B. First-line alternate option (non-depolarizing agent): Rocuronium 1-1.2 mg/kg IV.16
      • NOTE: If using rocuronium or vecuronium as paralytic, the pharmacodynamics are such that administration of the paralytic agent first, followed by the induction agent second.17
   C. Second-line alternate option (non-depolarizing agent): Vecuronium 0.15-0.3mg/kg IV.18

3. Finalize patient position for optimal intubating conditions.19
   • Maximize upper airway dimensions and facilitate direct laryngoscopy by positioning of the patient with their external auditory meatus on the same horizontal plane as their sternal notch.
   • The face plane of the patient should be parallel to the ceiling.
   • Raise the torso and head 20-30 degrees or use reverse Trendelenberg.

4. Assess need for ventilations during medication onset (apnea phase).
   • In patients at low risk for desaturation, manual ventilation during the onset phase of muscle relaxants (paralysis) is not necessary or recommended.
   • Ventilations may be required in the hypoxemic, high risk patient.
   • When required, ventilations should be given slowly (over 1-2 seconds), using a low volume (6-7 mL/kg), and at a low rate (6-8 ventilations per minute).20 A PEEP valve recommended to be used on BVM for hypoxic patients.
   • Whenever possible, perform apneic oxygenation with nasal cannula at 15 lpm or higher to extend apnea time without desaturation.

5. Perform Intubation.
   A. Video Laryngoscopy (VL) is the preferred method of intubation.
      • The intubation should be recorded for dedicated QA/QI purposes.
      • Consider brief period of suctioning prior to insertion of VL to remove secretions and improve VL view.
   B. Direct Laryngoscopy is second-line method of intubation.
   C. Consider external laryngeal manipulation to maximize view.
      • Once optimal positioning identified, can hand off to partner to maintain external laryngeal manipulation.
   D. Apneic oxygenation is recommended when feasible.
   E. Laryngoscopy attempt should be aborted if hypoxia occurs during attempt.
      • BVM with airway adjuncts (oral/nasal airway) and PEEP valve is recommended.
      • A supraglottic airway is also an alternative.
   F. The number of attempts at endotracheal intubation should be no more than 2 for a single provider if an alternate provider is available.
G. An Eschmann stylet (gum elastic bougie) is recommended if difficult airway anticipated or encountered, and is also appropriate for the initial laryngoscopy.

H. If a third laryngoscopy is required, it should be performed by an experienced provider.21

I. After 3 unsuccessful attempts at endotracheal intubation, move rapidly to the placement of a supraglottic rescue airway or quality BVM ventilation.

J. Should rescue techniques be ineffective at providing oxygenation or ventilation move to surgical cricothyrotomy or transtracheal jet ventilation.

6. Confirm tube placement.
   A. Visualization
      • Record ET placement using video laryngoscope for dedicated QI/QA purposes.
   B. Immediately perform ETCO2 monitoring with waveform capnography.
      • Use of the suction esophageal detecting device or esophageal bulb detector is indicated only in the setting of cardiac arrest where the endotracheal tube (ET) is suspected to be in the trachea clinically but there is an absence of an ETCO2 waveform.
   C. Auscultate chest and epigastrium.
      • Evaluate for bilateral and equal breath sounds to confirm depth of placement22 and absence of gastric sounds.
   D. Re-assess oxygenation status.

**It is required that the intubated and supraglottic airway be monitored with continuous waveform capnography and pulse oximetry. (Includes surgical airways)**

**POST-INTUBATION PHASE**

1. Secure Endotracheal Tube.
   A. Monitor (and record) depth of insertion and landmark (e.g. - “22cm at teeth”)
      • Average depth for adult female is 21cm, adult male is 23cm - adjust for patient size.
   B. If the patient is pediatric (age less than 12), even in the absence of trauma, routine use of a C-collar is indicated to reduce movement of the neck and decrease likelihood of accidental extubation.
   C. Consider placing a C-collar for all patients to decrease likelihood of accidental extubation.

2. Reassess Patient.
   A. Continuous oximetry, telemetry, and ETCO2 monitoring are mandatory.
      • Evaluate quality and nature of ETCO2 waveform (See Table 6).
   B. Recheck blood pressure and heart rate immediately following intubation.
   C. Recheck and document vitals at least every 5 minutes on an intubated patient or if clinical change in condition occurs.
**INTUBATION PROTOCOL**

**Page 5 of 28**

- Hypotension can frequently occur post-intubation with the transition to positive pressure ventilation and the resultant potential impairment on cardiac preload.
- Be prepared to bolus patients with normal saline.
- In the meta-stable or critically ill patient who is not yet hypotensive but who is at high risk for developing hypotension and who is not in florid, hypoxic pulmonary edema, consider empiric crystalloid bolus of 500ml normal saline. Note: current trauma recommendations are to minimize crystalloid use in setting of hemorrhage.
- In the patient who is hypotensive pre-intubation, administer 500ml normal saline bolus and immediately re-evaluate need for additional boluses to obtain minimum goal of SBP >90 and/or Mean Arterial Pressure (MAP) of > 65.

D. Carefully re-evaluate post intubation ventilation.  
- Aggressive post-intubation hyperventilation should be avoided. Use an external auditory mechanism to assist calculating ventilation rate (e.g. - “1 Mississippi” or physical metronome).
- General goal is to maintain O2 sat >90% and ETCO2 ~35-45 mmHg
- Rapid hyperventilation is not recommended even in the setting of initial hypercarbia, and can be detrimental.
- In the setting of reactive airways disease (asthma, COPD), air trapping is common as the small airways restrict effective exhalation. This can lead to “breath stacking” (auto-PEEP or intrinsic PEEP) and can result in severe barotrauma, ineffective gas exchange, and/or hypotension due to high intra-thoracic pressures. In the reactive airway disease patient, monitor for decreased BVM compliance, use a slow ventilatory rate, and allow time for exhalation.
- The patient population that requires hyperventilation post-intubation is the patient with a primary metabolic acidosis, for which the severe tachypnea is the result of the patient’s compensatory mechanism to drive off CO2 (induced hypocapnia). These patients can be very difficult to determine accurately pre-hospitally. Think of severe metabolic acidosis in the setting of suspected DKA and or known ethylene glycol ingestions. In this isolated setting of suspected primary metabolic acidosis driving tachypnea the goal is hyperventilation with a goal ETCO is 20. If possible, online medical control consultation is recommended if this condition is suspected.

E. Protect from hypothermia.

F. If the clinical scenario permits, elevate the head of bed to at least 20-30 degrees in order to both improve lung mechanics and reduce the risk aspiration. Current data suggest this positioning reduces the frequency of ventilator associated pneumonia (VAP).

3. Evaluate need for post-intubation analgesia and sedation.
   A. Ketamine 1mg/kg slow IV push every 30 minutes as needed.
   - Appropriate for use in the hemodynamically unstable patient.
   - If time permits and patient hemodynamically stable, consider midazolam 0.05mg/kg up to 5mg IV to reduce probability of an emergence reaction, particularly in teenagers and adults.
   - May repeat up to every 15-30 minutes as needed.
• NOTE: If ketamine is used for induction sedation, a repeat dose immediately following successful intubation is not indicated, but a repeat dose can still be given after 15-30 minutes after induction dose.
• Fentanyl 2mcg/kg IV up to every 5 minutes may also be given in setting of suspected severe pain.

B. **Fentanyl** 1-2 mcg/kg IV up to every 5 minutes is an alternative and/or adjunct to ketamine.
   • Appropriate for use in the hemodynamically unstable patient.
   • Recommended in the patient with an apparent painful condition (e.g., trauma).
   • May repeat the 1-2 mcg/kg doses every 5 minutes as needed.

C. **Midazolam** 0.05mg/kg IV up to 10mg is a first line choice in the setting of seizures.
   • May be repeated in that setting up to every 3-5 minutes as needed.
   • Midazolam is generally suboptimal and dosage should be reduced or avoided for the severely hypotensive, unstable patient unless seizures are present.

4. Evaluate need for post-intubation paralysis.
   A. Administration of a post-intubation paralytic is indicated when needed for patient safety, monitoring, or procedures.
   B. Perform a neurologic exam prior to paralytic administration.
   C. Repeat doses of succinylcholine have been associated with severe bradycardia. Therefore succinylcholine is not recommended for post-intubation paralysis, especially if used for induction for the procedure itself.
   D. Ensure adequate analgesia/sedation prior to post intubation paralysis.
   E. **Rocuronium** 1mg/kg IV.
   F. Alternate: **Vecuronium** 0.1mg/kg up to 10mg IV (Note: lower dose than for intubation)
   G. Elevation in HR and BP following administration of post-intubation paralytics can indicate inadequate sedation. Pay careful attention to heart rate and blood pressure.
1. **Special Circumstances - Cardiac Arrest**
   A. Key Points:
      - The role of airway and ventilatory management during CPR is controversial and not well understood.
      - Current evidence indicates that high-quality CPR is measured by maintaining a high compression fraction, satisfactory compression depth, appropriate compression rate, and the limiting of peri-shock pauses—and is essential to optimizing survival with good neurological outcome.\(^{27}\)
      - Studies are conflicted, but some observational data describe an association, but not proven causation, between advanced airway management and poorer outcomes. The question arises as to whether the choice of the type of airway utilized during resuscitation—i.e., BVM vs. advanced airway—is an independent predictor of survival or whether the airway choice is associated with other factors that may affect the chance for survival.
   B. The current recommended approach to the cardiac arrest patient involves a change in priorities: Circulation, Airway, Breathing (instead of Airway, Breathing, Circulation).
   C. The first priority in the management of the cardiac arrest patient is the initiation of chest compressions and quality CPR.
   D. The first attempt at intubation should be made with CPR ongoing (no cessation of compressions) with goal of minimizing interruptions to CPR.
      - Use of the bougie (Eschmann Stylet) is highly recommended.
   E. Primary use of a supraglottic airway such as the King tube is an acceptable approach to managing the airway and ventilation for the patient in cardiac arrest.
   F. ETCO2 monitoring is still mandatory in the setting of cardiac arrest. If there is an initial absence of detectable ETCO2 and the ET tube is thought to be properly placed in the trachea, esophageal suction device confirmation should be employed and documented.
      - Consider ETCO2 of <10mm Hg a marker of potentially inadequate quality CPR.
2. **Special Circumstances - The Pediatric Airway**

In general the overall procedure and approach to rapid sequence intubation in children is the same as for adults with a few important differences outlined as follows:

A. **Preparation**
   - Use the Broselow tape and/or similar resuscitation aid to help calculate drug dosages and choose equipment sizes.
   - Atropine (0.02 mg/kg IV with a minimum dose of 0.1mg IV) should be prepared.

B. **Positioning**
   - Due to relatively larger head in proportion to body, support underneath shoulders to create proper alignment is often needed.

C. **Pre-oxygenation and de-nitrogenation**
   - Additional emphasis is warranted as children desaturate more rapidly than adults.

D. **Pretreatment**
   - Routine pre-treatment with atropine is **no longer required** but can be administered.
     - **Atropine** (0.02 mg/kg IV with a minimum dose of 0.1mg IV) should be available and administered if bradycardia occurs.

E. **Paralysis with Induction**
   - Choice of medications is same as for adults.
   - Succinylcholine should be dosed at 2 mg/kg IV (not 1.5 mg/kg).\(^{28}\)

F. **Tube placement confirmation**
   - Use pediatric compatible ETCO2 detector circuit for pediatric patients.
   - **NOTE:** Pediatric ETCO2 colorimetric devices are required for patients <15kg (too much dead space in adult circuit for accurate readings), while an adult colorimetric device works for those >15kg.

G. **Secure Endotracheal Tube**
   - Use C-collar to minimize neck movement and reduce probability of tube dislodgment.

**NOTES:**
- Seattle Children’s Hospital recommends using cuffed ETT tubes for all pediatric patients except neonates (this would mean pink (6-7kg) and above on the Broselow chart). They recommend using the same size ETT as the uncuffed tube and inflating the cuff only if there is an air leak, and only with enough air to stop the leak. (Formal manometry of cuff insufflation pressure can be deferred to the hospital/CCU environment.)
- Transport with bag valve mask ventilation is an acceptable alternative to endotracheal intubation particularly in children < 2 years old.
- **Caution:** Intubation is **not recommended** for cases of suspected epiglottitis. Bag valve mask ventilation is recommended for primary management of ventilation.

See chart on next page for a summary of the clinically relevant anatomical differences with the pediatric airway.
**Anatomical Differences between Adults and Children** (Adapted from Walls, p281)

<table>
<thead>
<tr>
<th>Anatomy</th>
<th>Clinical Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large tongue occupies proportionally larger volume of the oral cavity, and epiglottis is proportionally larger</td>
<td>Straight blade often preferred over curved to push distensible anatomy out of the way</td>
</tr>
<tr>
<td>High tracheal opening: C-1 in infancy vs C-3 to C-4 at age ~7, C-5 to C-6 in the adult</td>
<td>High anterior airway position of the glottic opening compared with that in adults</td>
</tr>
<tr>
<td>Large occiput that may cause flexion of the airway, large tongue that easily collapses against the posterior pharynx</td>
<td>Sniffing position is preferred. The large occiput actually elevates the head into the sniffing position in most infants and children. A towel may be required under the shoulders to elevate torso relative to head in small infants/children</td>
</tr>
<tr>
<td>Cricoid ring is the narrowest portion of the trachea as compared with the vocal cords in the adult</td>
<td>Uncuffed tubes can provide adequate seal because they fit snugly at the level of the cricoid ring</td>
</tr>
<tr>
<td>Consistent anatomical variations with age with fewer abnormal variations related to body habitus, arthritis, and/or chronic disease</td>
<td>Younger than 2 years, high anterior; Age 2-9, transition period and variable, Ages &gt; 8, small adult</td>
</tr>
<tr>
<td>Large tonsils and adenoids may bleed; more acute angle between epiglottis and laryngeal opening results in nasotracheal intubation attempt failures</td>
<td>Blind nasotracheal intubation not indicated in children</td>
</tr>
<tr>
<td>Small cricothyroid membrane landmark, surgical cricothyrotomy impossible in infants and small children</td>
<td>Needle cricothyrotomy recommended and the landmark is the anterior surface of the trachea, not the cricoid membrane</td>
</tr>
<tr>
<td>Shorter endotracheal tube can lead to susceptibility for dislodgment from patient movement</td>
<td>Consider use of C-collar to help minimize potential movement of neck with attendant risk of ET dislodgment</td>
</tr>
</tbody>
</table>
3. **Special Circumstances - Trauma**

There are a few important components of managing the airway of a trauma patient:

A. **Preparation**
   - In the setting of patients requiring C-spine immobilization, a dedicated individual should remove the C-collar and maintain manual stabilization while the intubation is being performed. It is not possible or appropriate to attempt intubation while the patient is in a C-collar.
   - Assess for injuries to the airway, neck, or chest that may complicate intubation or patient care.
   - Maximize resuscitation efforts during preparation phase of the hemodynamically unstable trauma patient to minimize risk of decompensation during transition to positive pressure ventilation. Attempt to manage/control hemorrhage (e.g., tourniquet, pelvic binder) prior to intubation attempt where feasible. Current guidelines recommending minimizing crystalloid use in hemorrhagic trauma where possible. See Trauma protocols Consider reducing induction dose of ketamine or etomidate, while maximizing dose of succinylcholine or rocuronium.
   - Careful attention to maximizing pre-oxygenation and attempting to prevent and/or address hypotension are critical, as *a single episode of either hypoxia or hypotension has a significant adverse effect on prognosis* for the head injured patient. In the setting of suspected head injury, crystalloid use is appropriate.

B. **Induction Agent choice**
   - Ketamine is the preferred induction agent in the hemodynamically unstable (hypotensive) trauma patient.

C. **Tube Placement confirmation**
   - Careful attention to tube depth, auscultated breath sounds, bag valve compliance, and patient hemodynamics are indicated in the patient with suspected chest trauma. Monitor carefully for tension pneumothorax.

4. **Special Circumstances - Proximity to Hospital**

In the instance of immediate proximity (<2-3 minutes) to the hospital it is a reasonable option to maximize preparation components but consider performing the intubation itself at in the emergency department. Notify hospital ASAP whenever possible.
5. Special Circumstances - Severe Bronchospasm (e.g. Asthma/Severe COPD)
Severe bronchospastic illnesses such as asthma and COPD are characterized by small airway
disease and are not relieved or improved by the act of intubation itself. While the process of
direct intubation itself is not altered by the presence of asthma and/or COPD, extreme care and
attention are required for the mechanical ventilation of such patients. Ventilated air can enter the
lungs with ventilation, but can not easily or rapidly escape - and this leads to “air-trapping” and
“Auto-PEEP”. Hyperventilation should be carefully avoided due to this risk. Additional time to
permit a slower exhalation is required, otherwise hyperinflation can occur. Hyperinflation can
result in direct barotrauma, or IVC compression leading to hypotension. Monitor BVM
compliance carefully. Additional extrinsic PEEP should be avoided in the severely
bronchospastic patient. In severe cases, briefly disconnecting the BVM and manually
compressing the chest intermittent can improve exhalation and improve lung compliance. In the
severe asthmatic the focus is on maintaining adequate oxygenation first, even at the cost of
hypercapnia (“permissive hypercapnia”). Provide aggressive paralysis, analgesia, and sedation
in this setting.

6. Special Circumstances - Anticipated Difficult Airway with low likelihood of success
It is appropriate to defer the administration of a paralytic for an attempt at intubation where
successful intubation is anticipated to be improbable. In this setting alternate means to maintain
oxygenation and ventilation should be attempted first.
  • The critical decision making components regarding management are dependent on the ability
to maintain oxygenation and ventilation.
  • If initial alternate means (such as BVM, NiPPV, or supraglottic airway) are not adequately
    maintaining oxygenation or ventilation, it is reasonable to use paralytics even in the setting of
    anticipated difficulties. Simultaneously prepare for and anticipate use of a planned failed
    airway technique, most commonly a surgical airway and/or transtracheal jet ventilation.
  • A delayed or rapid sequence airway (attempt at placement of a supraglottic device following
    induction and paralytic administration) is an appropriate rescue technique.
7. Special Circumstances - The contaminated airway
When the hypopharynx and/or airway is contaminated (blood or emesis being the most common), visualization and successful intubation become more difficult. Several techniques can be utilized to help manage the contaminated airway:

• **Technique 1:** The Suction Assisted Laryngoscopy and Airway Decontamination (SALAD) approach can be utilized to improve laryngoscopy. Holding the laryngoscope with the left hand and the suction catheter with an overhand grip in the right hand (as if holding a dagger) you use the suction catheter to open the mouth (no scissor technique with the finger) and suction out the hypopharynx, then still holding suction like a dagger push out and up to open the mouth and be able to insert the laryngoscope. *Leading with the suction as you advance* so as to keep the optics and light of the laryngoscope clear. You will then leave the suction catheter in the tip of the esophagus to continue suctioning as you intubate. However, you will not be able to leave the suction catheter on the right side of the mouth, so you will need to move it to the left side. This can be done either by pushing the laryngoscope forward and passing the suction under the laryngoscope, or by removing suction and replacing on the left side of the laryngoscope and advancing into the tip of the esophagus. Once the tip of the catheter is in the esophagus it can be left in place and not fall out while intubation is being performed. If possible suction the endotracheal tube before ventilation. Remove suction catheter only after intubation complete and balloon inflated (or leave suction in place).

• **Technique 2:** Suction Assisted Airway Catheter insertion (Note: This technique is not possible with a traditional Yankauer suction catheter - it requires either a DuCantor or Hi-D suction catheter, both of which are large enough in diameter to permit a bougie (ETI, Eschmann Stylet) to be placed thru the suction catheter) Start with the SALAD technique described above. If possible place suction catheter into larynx itself, then disconnect the suction catheter and place bougie (ETI) through the suction catheter in and intubate the trachea. Remove the suction catheter over the ETI, reattach the suction catheter and place on left side into tip of esophagus again (same as original SALAD technique) Place endotracheal tube over the ETI into the trachea and inflate cuff. If possible suction the endotracheal tube before ventilation.

• **Technique 3:** Esophageal ET tube placement: For high volume airway contaminant where above techniques are not working. Start with SALAD technique (place tip of suction catheter in to esophagus, keep/place suction catheter on left side of laryngoscope). If suction is overwhelmed by contaminant, or suction stops working, then place the ET tube purposely into the esophagus, remove the stylette, and inflate the cuff - allow the contaminant to come up through the esophageal ET tube and freeing up the airway. Move the esophageal tracheal tube to the left side of the laryngoscope, and now complete the intubation with a second endotracheal tube.

NOTE: For an excellent visual reference to the above techniques, view the video by Dr. Jim DuCanto at: [https://vimeo.com/158978573](https://vimeo.com/158978573)
TABLES

Table 1: Intubation Medications

<table>
<thead>
<tr>
<th>DRUG</th>
<th>Normotensive Dose</th>
<th>Normotensive 70 kg Patient Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine</td>
<td>1-2mg/kg</td>
<td>140mg</td>
</tr>
<tr>
<td>Etomidate</td>
<td>0.3mg/kg</td>
<td>20mg</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>1.5-2mg/kg</td>
<td>140mg</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>1-1.2mg/kg</td>
<td>70mg</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.15-0.3mg/kg</td>
<td>20mg</td>
</tr>
</tbody>
</table>

Table 2: Comparison of Paralytics

<table>
<thead>
<tr>
<th>DRUG</th>
<th>Class</th>
<th>Time to intubation paralysis (sec)</th>
<th>Duration (min)</th>
<th>Pregnancy Class</th>
<th>Drawbacks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>depolarizing</td>
<td>45</td>
<td>6-10</td>
<td>C</td>
<td>multiple contraindications, severe bradycardia if repeated, hyperkalemia, malignant hyperthermia</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>non-depolarizing</td>
<td>60</td>
<td>40-60</td>
<td>B</td>
<td>long duration allergy (rare)</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>non-depolarizing</td>
<td>75-90</td>
<td>60-75</td>
<td>C</td>
<td>slow onset, long duration allergy (rare)</td>
</tr>
</tbody>
</table>
Table 3: Sequence for Pre-oxygenation and Prevention of Desaturation

Sequence of Pre-oxygenation and Prevention of Desaturation
(Assuming 2 oxygen regulators*)

Pre-oxygenation Period
- Position the patient in a semi-recumbent position (~20-30°) or in reverse Trendelenberg. Position the patient’s head in the ear-to-sternal-notch position using padding if necessary
- Place a nasal cannula in the patient’s nares. Do not hook the nasal cannula to oxygen regulator.
- Place the patient on a non-rebreather mask at the maximal flow allowed by the regulator (at least 15 lpm, but many allow a much greater uncalibrated flow).
- If patient is not saturating >90%, remove face mask and switch to non-invasive CPAP by using ventilator, non-invasive ventilation machine, commercial CPAP device, or BVM with PEEP valve attached. Titrate between 5-15cm H₂O of PEEP to achieve an oxygen saturation >98%. Consider this step in patients saturating 91-95%.
- Allow patient to breath at tidal volume for 3 minutes or ask the patient to perform 8 maximal exhalations and inhalations.
- Attach a BVM to oxygen regulator and set it to maximal flow (at least 15 lpm). If the patient required CPAP for pre-oxygenation, attach a PEEP valve to the BVM set at the patient’s current CPAP level.

Apneic Period
- Push sedative and paralytic (preferably rocuronium**, if the patient is at risk for rapid desaturation).
- Detach face mask from the oxygen regulator and attach the nasal cannula. Drop the flow rate to 15 lpm.
- Remove the facemask from the patient.
- Perform a jaw thrust to maintain pharyngeal patency.
- If the patient is high risk (required CPAP for pre-oxygenation), consider leaving on CPAP during the apneic period or providing 4-6 ventilations with the BVM with a PEEP valve attached. Maintain a two hand mask seal during the entire apneic period to maintain the CPAP.

Intubation Period
- Leave the nasal cannula on throughout the airway management period to maintain apneic oxygenation.

*If 3 regulators are available, attach reservoir face mask, BVM, and nasal cannula to them. If only one regulator is available, consider using a stand-alone supplemental oxygen tank to offer a second source of oxygen.
**Rocuronium recommended by article authors, but for Skagit County EMS succinylcholine or rocuronium may be used.
• Evaluate all patients undergoing laryngoscopy for potential surgical airway. Estimate risk. Discuss plan for surgical airway as backup with partners/team, feel the anatomy and landmarks, and ensure surgical airway kit is available for all patients.

• For the anticipated difficult airway, have surgical kit visible and on hand, and consider marking the neck with a pen at site of potential cricothyrotomy, or even injecting local anesthetic if available.

• For highest risk patient, have the neck prepped and surgical airway kit open prior initiation of laryngoscopy attempt.

### Table 4: Risk Categorization of Patients During Pre-Oxygenation

<table>
<thead>
<tr>
<th>Risk Category, Based on Pulse Oximetry While Receiving High-Flow Oxygen</th>
<th>Preoxygenation Period (3 Minutes)</th>
<th>Onset of Muscle Relaxation (≤60 Seconds)</th>
<th>Apneic Period During Tracheal Intubation (Variable Duration, Depending on Airway Difficulty; Ideally &lt;30 Seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk, SpO₂ 96%–100%</td>
<td>Nonrebreather mask with maximal oxygen flow rate</td>
<td>Nonrebreather mask and nasal oxygen at 15 L/min</td>
<td>Nasal oxygen at 15 L/min</td>
</tr>
<tr>
<td>High risk, SpO₂ 91%–95%</td>
<td>Nonrebreather mask or CPAP or bag-valve-mask device with PEEP</td>
<td>Nonrebreather mask, CPAP, or bag-valve-mask device with PEEP and nasal oxygen at 15 L/min</td>
<td>Nasal oxygen at 15 L/min</td>
</tr>
<tr>
<td>Hypoxemic, SpO₂ 90% or less</td>
<td>CPAP or bag-valve-mask device with PEEP</td>
<td>CPAP or bag-valve-mask device with PEEP and nasal oxygen at 15 L/min</td>
<td>Nasal oxygen at 15 L/min</td>
</tr>
</tbody>
</table>

* Risk categories are based on patient’s initial response to high-flow oxygen through a tightly fitting nonrebreather mask. Patients who are already hypoxemic exhibit shunt physiology and are prone to rapid desaturation during the peri-intubation. Patients with saturations of 91% to 95% have values close to the precipice of the steep portion of the oxyhemoglobin dissociation curve and should be considered high risk. Patients with saturations greater than or equal to 96% are at low risk for peri-intubation desaturation. Patients in all risk categories should receive preoxygenation in a head-down position (or reverse-Trendelenburg if there is a risk of spine injury).

### Table 5: Cricotr status (adapted from Weingart)

| Level One: Ready (All Patients) | Discuss/Feel/See Kit |
| Level Two: Set (Difficult Airway patient) | Mark/Kit at bedside |
| Level Three: Go (crashing/hypoxemic patient) | Prep, Open, and Set Kit |
### Table 6: End Tidal CO2 Waveform Analysis

<table>
<thead>
<tr>
<th>Sudden loss of waveform</th>
<th>Bronchospasm (&quot;Shark-fin&quot; appearance)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• ET tube disconnected, dislodged, kinked or obstructed</td>
<td>• Asthma</td>
</tr>
<tr>
<td>• Loss of circulatory function</td>
<td>• COPD</td>
</tr>
<tr>
<td><strong>Decreasing EtCO2</strong></td>
<td><strong>Hypoventilation</strong></td>
</tr>
<tr>
<td>• ET tube cuff leak</td>
<td><strong>Hyperventilation</strong></td>
</tr>
<tr>
<td>• ET tube in hypopharynx</td>
<td><strong>Decreased EtCO2</strong></td>
</tr>
<tr>
<td>• Partial obstruction</td>
<td>• Apnea</td>
</tr>
<tr>
<td><strong>CPR Assessment</strong></td>
<td>• Sedation</td>
</tr>
<tr>
<td>• Attempt to maintain minimum of 10mmHg</td>
<td></td>
</tr>
<tr>
<td><strong>Sudden increase in EtCO2</strong></td>
<td></td>
</tr>
<tr>
<td>• Return of spontaneous circulation (ROSC)</td>
<td></td>
</tr>
</tbody>
</table>
EDUCATIONAL NOTES

Main Sources:


**Which induction agents are the most hemodynamically stable when used for RSI?**

In RSI a predetermined dose of an induction agent is given at the same time as a muscle relaxant. The physician makes his or her best estimation of the dose of induction agent required and the dose is not titrated. The physician aims to give a large enough dose of induction agent to prevent awareness, while minimizing risk of hemodynamic collapse. Although virtually all induction agents could be used for RSI, not all are appropriate. We want to avoid both patient awareness and hemodynamic compromise. The ideal induction agent in RSI will have rapid and reliable onset and few adverse (particularly hemodynamic) effects.

Etomidate results in the least variation in blood pressure and heart rate when compared with the other agents used for induction of anesthesia. This cardiovascular stability is seen in both children and adults, including the elderly. The drug is delivered to the CNS in a timely and dependable manner. It is for these reasons that etomidate remains the standard choice for RSI.

Propofol is a very popular induction agent for elective procedures, when the induction dose is titrated against the patient response. It is a poor choice of induction agent in hemodynamically compromised patients, who run the risk of further hemodynamic deterioration coupled with awareness during intubation.

Benzodiazepines are generally not suitable as induction agent in RSI. Midazolam is 95% protein down. Both midazolam and lorazepam require closure of an imidazole ring to have enough lipid solubility to cross the blood brain barrier, which takes as long as 10 minutes. Some authors have referred to benzodiazepines as being "almost useless" for RSI.

Ketamine offer several advantages as an induction agent in hemodynamically compromised patients. Ketamine is a sympathomimetic medication, increasing heart rate, arterial pressure, and cardiac output in animal models. Data on the use of ketamine as an induction agent in RSI are sparse. Conversely, there is significant clinical experience using ketamine for RSI, although much of it is in the resource-poor developing world or in warfare, neither of which lend themselves to clinical trials. In 2009 Jabre, et. al. published the largest clinical trial date involving ketamine 2 mg per kilogram for RSI in adults, and comparing it to etomidate 0.3 mg per kilogram, both with succinylcholine as the neuromuscular blocking agent. There were no significant hemodynamic differences between the two groups. The study concluded that ketamine is a safe alternative to etomidate for endotracheal intubation in critically ill patients, and should be considered in those with sepsis.
In the hemodynamically unstable patient ketamine or etomidate offer the most reliable method of rapidly achieving unconsciousness while limiting further hemodynamic compromise.

**What is the risk of ketamine in the brain injured patient?**

For many years, the use of ketamine was thought to be contra-indicated in brain injured patients because of the risk of increasing Intracranial pressure (ICP) through increased cerebral blood flow (CBF). Subsequent animal models and later clinical data have refuted this earlier hypothesis.

In an injured brain, \( \text{CPP} = \text{MAP} - \text{ICP} \) where CPP is the cerebral perfusion pressure, MAP is the mean arterial pressure, and ICP is the intracranial pressure. Following brain injury, there is a loss of cerebral auto-regulation and cerebral blood flow is largely dependent on cerebral perfusion pressure, which is in turn largely dependent on mean arterial pressure. Consequently, agents such as etomidate and ketamine that maintain mean arterial pressure will maintain cerebral blood flow. This is particularly true in patients with poly-trauma where traumatic brain injury and shock may coexist.

The dangers of hypotension on the injured brain are well known, and any mechanism by which hypotension can be avoided in traumatic brain injury should be encouraged. In ventilated patients with controlled ventilation, ketamine does not increase ICP. In addition to the neuro-protective effects of maintaining cerebral blood flow through cerebral perfusion pressure, ketamine has also been found to have other neuro-protective properties. A comprehensive review of the available experimental and clinical evidence for the neuro-protective properties of ketamine was recently published. Animal models show that ketamine inhibits the NMDA receptor activation, reduces neuronal apoptosis, and reduces the systemic inflammatory response to tissue injury. In the last few years, increasing clinical evidence of the safety of ketamine in brain injured patients has emerged. It is becoming increasingly clear that ketamine is likely not dangerous in brain injured patients, and instead may confer advantages over other agents. Most clinical data come from neurosurgical units with invasive intracranial pressure monitoring using ketamine as a sedative agent.

Very little of these data have been generated using ketamine as an induction agent in the emergency department setting. They are not yet sufficient data to support ketamine induction for RSI in all brain injured patients. If the brain injured patient is also hypotensive, then ketamine is an excellent choice.

**What is the best induction agent for patients with severe bronchospasm?**

Most of the data on the use of induction agents and asthma comes from the anesthesia literature in elective surgical cases, from animal models, and from experience using ketamine as a sedating agent in intubated asthmatic patients. Although ketamine is widely accepted and recommended as the induction agent of choice for severe asthma, the data on ketamine use for induction in RSI for asthmatic patients in the emergency department are sparse. Etomidate caused a mild increase in airway resistance in a very small study of non-asthmatic intubated patients. Midazolam data are lacking. Ketamine and propofol both cause bronchodilation in
asthmatic patients. In the emergency department, severe bronchospasm raises concerns of significantly decreased venous return and cardiovascular collapse, especially following intubation. While propofol may have some bronchodilatory properties, this possible benefit is outweighed in the unstable asthmatic patient by risks of hemodynamic instability, making ketamine the best choice for induction agent in severe bronchospasm. Etomidate also is a good choice as an induction agent in severe bronchospasm because of its excellent hemodynamic stability. Following intubation, either propofol or ketamine are excellent choices for sedation in the patient with severe bronchospasm.

Is etomidate safe safe for use in septic patients?

Etomidate has become the preferred agent for emergent RSI in North America and in much of the rest of the world because of its simple dosing strategies, reliable onset of action, and cardiovascular stability. The debate about the safety of etomidate in patients with sepsis has been raging for much of the last decade. The debate regarding the safety of etomidate in patients with sepsis has occurred within the larger discussion of critical illness relative corticosteroid insufficiency (CIRCI) and the role of corticosteroids in the management at critically ill patients. CIRCI, however, is more complicated than a simple reduction in circulating cortisol levels, and likely stems from a dysfunction at the level of the hypothalamic-pituitary axis. Many of the features of CIRCI are still being identified, but likely include decreased production of corticotropin-releasing hormone, ACTH, cortisol, and perhaps critically, dysfunction of the glucocorticosteroid receptors.

Confounding in this is the inability to precisely characterize the nature and role of adrenal insufficiency in critical illness and how this may or may not relate to total cortisol levels or response to ACTH.

A single dose of etomidate causes a reversible inhibition of adrenal hormone synthesis. It was for this reason that etomidate infusions ceased to be used for ICU sedation in the early 1980s. Following a single dose of etomidate, there is an immediate inhibition of adrenal hormone synthesis that lasts 12 to 24 hours, and may extend on to 72 hours in some patients. What remains unclear is whether or not there are any significant clinical sequelae from the transient inhibition adrenal hormone synthesis.

For the most part, there is broad agreement that a patient without sepsis or sepsis-like syndrome, the advantages of etomidate significantly outweigh concerns about possible inhibition of adrenal hormone synthesis.

For patients with sepsis or sepsis-like syndrome, there remains much debate as to the potential risk of etomidate. The literature is significantly divided. Much of the data has emerged from observational studies and post hoc analysis. There been many review articles and several meta-analyses. However, very few patients have been enrolled in randomized controlled trials, and several studies used cortisol levels as the primary outcomes, and did not address mortality or length of stay. In 2009 Jabre, et. al. published a RCT comparing 234 patients in the etomidate group and 235 in the ketamine group. Although the percent of the patients with adrenal insufficiency was significantly higher in the etomidate group, they found no serious adverse events with either study drug. The number of patients with sepsis is the final diagnosis was 41 in
the etomidate group and 35 in the ketamine group. In August 2010, a comprehensive meta-analysis concluded that although etomidate suppresses adrenal function transiently, there is no significant mortality affect based on current data.

In November 2010 Tekwani et. al. published a RCT comparing etomidate and midazolam as induction agents of patients with a primary infectious cause the illness, with a primary outcome measure of hospital length of stay and secondary outcomes of ICU length of stay, ventilator days, and mortality. They found no significant differences in their primary or secondary outcomes. To date, no study has adequate power to detect a small difference in mortality or in hospital, ICU, or ventilator length of stay.

The debate over the safety of etomidate in sepsis patients has expanded in recent years. There's recognition at some degree of adrenal insufficiency occurs in many patients, and that measurement of total cortisol levels is likely oversimplifying the problem.

For the emergency physician who relies upon a time date for the simple dosing regimen, rapid onset of action, and lack of cardiovascular compromise, even in patients who are hemodynamically unstable, there are three main choices in a patient with presumptive sepsis:

1. **Avoid etomidate use entirely in patients who are presumed to be septic.** Some advocates of this approach emerged early in the debate, but as further data have emerged, the possible risk of etomidate use in septic patients appears to have been overstated and a clinical equipoise has developed. The risk of using etomidate must be balanced against the risk of an alternative agent. Only ketamine provides the hemodynamic instability comparable with etomidate, and ketamine is not available many settings were merging into patient

2. **Routinely administer glucocorticoids to patients with septic shock who have received etomidate.** The emerging recognition of the relationship between critical illness and adrenal insufficiency (CIRCI) has made this question both simpler and more complex. Studies of supplemental corticosteroids in patients with sepsis have had equivocal results. Although is posited that glucocorticoids should be given immediately after the administration of etomidate when the adrenal suppression is likely be greatest, there is no evidence of this approach improves patient outcome. The current international consensus is that supplemental glucocorticoids should be considered in the management of septic patients whenever they have responded poorly the fluid resuscitation and vasopressor of agents.

3. **Communicating clearly the critical care staff that the patient was given a dose of etomidate induction.** It is almost impossible to argue against this commonsense approach

**Adverse affects of succinylcholine**

The recognized effects of succinylcholine include fasciculations, hyperkalemia, bradycardia, prolonged neuromuscular blockade, malignant hyperthermia, and trismus/masseter muscle spasm. Each is discussed separately.

1. **Fasciculations**

   Fasciculations are believed to be produced by stimulation of the nicotinic acetyl choline (ACH) receptors. Fasciculations occur simultaneously with increases in intracranial pressure, intraocular pressure, and intragastric pressure, but these are not the result of concerted muscle activity. Of these, only the increase in ICP is potentially clinically important.

   The exact mechanisms by which these effects occur are not well elucidated. In the past, it was recommended that non-depolarizing agents be given in advance of succinylcholine to mitigate ICP elevation, but there is insufficient evidence to support this practice.

   The relationship between muscle fasciculation and subsequent post operative muscle pain is controversial. Studies have been variable with respect to prevention of fasciculations and subsequent muscle pain. Although there exists a theoretical concern regarding the extrusion of vitreous in patients with open globe injuries who were given succinylcholine, there are no published reports of this potential complication. Anesthesiologists continue to use succinylcholine as a muscle relaxant in cases of open globe injury, with or without an accompanying defasciculating agent. Similarly, the increase in intragastric pressure that has been measured has never been shown to be of any clinical significance, perhaps because it is offset by the corresponding increase in the lower esophageal sphincter pressure.

2. **Hyperkalemia**

   Under normal circumstances, serum potassium increases minimally (0 to 0.5 mEq perL) when succinylcholine is administered. In certain pathologic conditions, however, a rapid and dramatic increase in serum potassium can occur in response to succinylcholine. These pathologic hyperkalemic responses occur by two distinct mechanisms: receptor up-regulation and rhabdomyolysis. In either situation potassium may increase as much as 5 to 10 mEq per L within a few minutes and result in hyperkalemic dysrhythmias or cardiac arrest.

   Two forms of post junctional receptors exist: mature (junctional)and immature (extra-junctional). Each receptor is composed of five proteins arranged in a circular fashion around a common channel. Both types of receptors contain two Alpha subunits. ACH must attach to both alpha subunits to open the channel and effect depolarization and muscle contraction. When receptor up-regulation occurs, the mature receptors at and around the motor endplate or gradually converted over 4 to 5 day period to immature receptors that propagate throughout the entire muscle membrane. Immature receptors are characterized by low conductance and prolonged channel opening times (four times longer than mature receptors), resulting in an increasing release potassium. Most of the entities associated with hyperkalemia during emergency use of succinylcholine are the result of receptor up-regulation. Interestingly, these same extra junctional nicotinic receptors are relatively refractory to non-depolarizing agents, so larger doses of vecuronium, pancuronium, or rocuronium he may be required to produce paralysis. This is not an
issue in emergency RSI, where full intubating doses several times greater than the ED95 for paralysis are used.

Hyperkalemia also may occur with rhabdomyolysis, most often that associated with myopathies, especially inherited forms of muscular dystrophy. When severe hyperkalemia occurs related to rhabdomyolysis, the mortality approaches 30%, almost 3 times higher than that in cases of receptor up-regulation. This mortality increase may be related to coexisting cardiomyopathy. Succinylcholine is a toxin to unstable membranes in any patient with a myopathy and should be avoided.

Patients with the following conditions are at risk of succinylcholine induced hyperkalemia:

I. Receptor Up-regulation

   a. In burn victims, the extra junctional receptor sensitization becomes clinically significant five days post burn. It lasts an indefinite period of time, at least until there is a complete healing of the burned area. If the burn becomes infected or healing is delayed, the patient remains at risk for hyperkalemia. It is prudent to avoid succinylcholine in burn patients beyond day five post-burn if any questions exist regarding the status of their burn. Percent of body surface area burn does not correlate well with the magnitude of hyperkalemia. Significant hyperkalemia has been reported in patients with as little as 8% total body surface area burn (less in the service of one arm), but this is rare. The majority of emergent intubations for burn patients are performed within the safe five day window. Should a later intubation become necessary, however, rocuronium or vecuronium provide excellent alternatives.

   b. Denervation - The patient who suffers a denervation event, such as a spinal cord injury or stroke, is at risk for hyperkalemia from approximately the fifth day post event, until six months post event. Patients with progressive neuromuscular disorders, such as multiple sclerosis or amyotrophic lateral sclerosis (ALS), are perpetually at risk for hyperkalemia. Likewise, patients with transient neuromuscular disorders, such as Guillane-Barre syndrome or wound botulism can develop hyperkalemia after day five, depending on the severity of their disease. As long as the neuromuscular disease is dynamic, there will be augmentation of the extra junctional receptors, which increases the risk for hyperkalemia. These specific clinical situations should be considered absolute contraindications to succinylcholine during the designated time periods.

   c. Crush injuries - The data regarding crush injuries are scant. The hyperkalemic response begins about five days post injury, similar to denervation, and persists for several months after healing seems complete. The mechanism appears to be receptor up-regulation

   d. Severe infections - This entity seems to relate to established, serious infections, usually in the ICU environment. The mechanism is receptor up-regulation, but the initiating event is not established. Total body muscular disuse atrophy and chemical denervation of the ACH receptors, particularly related to long-term infusions of NMBAs (neuromuscular blocking agents), appear to drive the pathologic
receptor changes. Again, the at-risk time begins five days after initiation of the infection and continues indefinitely as long as the disease process is dynamic. Intra-abdominal sepsis has most prominently been identified as the culprit, but any serious, prolonged, debilitating infection should prompt concern.

II. Myopathy

Succinylcholine is actually contraindicated in patients with inherited myopathies, such as muscular dystrophy. Myopathic hyperkalemia can be devastating because the combined effects of a receptor up regulation and rhabdomyolysis. This is a particularly difficult problem in pediatrics, when a child with occult muscular dystrophy receives succinylcholine. Succinylcholine has a black box warning advising against its use in elective pediatric anesthesia, but it continues to be the muscle relaxant of choice for emergency intubation. Any patients suspected of a myopathy should be intubated with non-depolarizing muscle relaxants rather than succinylcholine.

III. Pre-existing Hyperkalemia

Hyperkalemia, per se, is not an absolute contraindication to succinylcholine. There is no evidence that succinylcholine is harmful in patients with pre-existing hyperkalemia, but who are not otherwise at risk of severe succinylcholine induced hyperkalemia by one of the mechanisms described in the preceding section. There is widespread concern that patients with acute hyperkalemia secondary to acute renal failure or diabetic ketoacidosis are more likely to exhibit cardiac dysrhythmias from succinylcholine administration than in patients with chronic or recurrent hyperkalemia. There is, however, no evidence to support this claim. Patient with pre-existing hyperkalemia are subject to the same potential rise of 0 to 0.5 mEq per L of potassium as for "normal" patients. The only study that examined the use of succinylcholine in patients with chronic renal failure (including documented hyperkalemia before intubation) failed to identify any adverse effects related to succinylcholine. A reasonable approach is to assume that succinylcholine is safe to use in patients with renal failure unless the EKG - either monitor tracing the EKG 12 lead - shows evidence of acute hyperkalemia (peaked T waves or prolongation of QRS).

3. Bradycardia

In both adults and children, repeated doses of succinylcholine may produce bradycardia, and administration of atropine may become necessary.
4. **Prolonged Neuromuscular Blockade**

Prolonged neuromuscular blockade may result from an acquired pseudocholinesterase (PCHE) deficiency, a congenital absence of PCHE, or the presence of an atypical form of PCHE, any of the three which will delay the degradation of succinylcholine and prolong paralysis. Acquired PCHE deficiency may be a result of liver disease, chronic cocaine abuse, pregnancy, burns, medications such as oral contraceptives, metoclopramide (Reglan), bambuterol, or esmolol. A 20% reduction in normal levels will increase apnea time about 3 to 9 minutes. The most severe variant (0.04% of the population) will result in prolonged paralysis for 4-8 hours.

5. **Malignant Hyperthermia**

A personal or family history of malignant hyperthermia (MH) is an absolute contraindication to the use of succinylcholine. MH is a myopathy characterized by a genetic skeletal muscle membrane abnormality of the Ry reyanodine receptor. It can be triggered by halogenated anesthetics, succinylcholine, vigorous exercise, and even emotional stress. Following the initiating event, its onset can be acute and progressive, or delayed for hours. General awareness of MH, earlier diagnosis, and the availability of dantrolene (Dantrium) have decreased the mortality from as high as 70% to less than 5%. Acute loss of intracellular calcium control results in a cascade of rapidly progressive events manifested primarily by increased metabolism, muscular rigidity, autonomic instability, hypoxia, hypertension, severe lactic acid doses, hyperkalemia, myoglobinemia, and disseminated intravascular coagulation. Temperature elevation is a late manifestation. The presence of more than one of these clinical signs is suggestive of malignant hyperthermia.

Masseter spasm, once claimed to be the hallmark of MH, is not pathognomonic. Succinylcholine can promote isolated master spasm as an exaggerated response at the neuromuscular junction, especially in children.

The treatment for MH consists of discontinuing the known or suspected precipitant and the immediate administration of dantrolene sodium (Dantrium). Dantrolene is essential to successful resuscitation and must be given as soon as the diagnosis is seriously entertained. Dantrolene is a hydantoin derivative that acts directly on skeletal muscle to prevent calcium release from the sarcoplasmic reticulum without affecting calcium reuptake. The initial dose is 2.5 mg per kilogram IV, repeated every five minutes until muscle relaxation occurs or the maximum dose of 10 mg per kilogram is administered. Dantrolene is free of any serious side effects. In addition, measures to control body temperature, acid-base balance, and renal function must be used. All cases of MH require constant monitoring of pH, arterial blood gases, and serum potassium. Immediate and aggressive management of hyperkalemia with administration of calcium gluconate, glucose, insulin, and sodium bicarb may be necessary. Interestingly, full paralysis with non-depolarizing NMBAs will prevent succinylcholine triggered MH. MH has never been reported related to the use of succinylcholine in the emergency department. The MH emergency hotline number is 1-800 MH-HYPER or 1-800-644-9737 24 hours a day, seven days a week. Ask for “index zero”. The email address for the malignant hyperthermia association United States is mhaus@Norwich.net and the website is www.mhaus.org.
6. Trismus/Masseter muscle spasm
On occasion, succinylcholine may cause transient trismus/masseter muscle spasm, especially in children. This manifests as jaw muscle rigidity associated with limb muscle flaccidity. Pretreatment with defasciculating doses of non-depolarizing NMBAs will not prevent masseter spasm. If masseter spasm interferes with intubation, an intubating dose of a competitive non-depolarizing agent (e.g., rocuronium) should be administered and will relax the involved muscles. The patient may require bag mask ventilation until relaxation is complete and intubation as possible. Masseter spasm should prompt serious consideration of the diagnosis of malignant hyperthermia (see previous discussion).

The following photograph is for Endnote 4 - While the LEMON mnemonic is not perfect, assessment of the patient for potential barriers to intubation - such as having the jaw wired shut - is strongly recommended.
ENDNOTES

1 If there is anticipated deterioration, impending airway compromise, or if there is a possibility of either during transport or a period of limited resources. The threshold for securing the airway for concern about anticipated clinical course is much lower for high risk transports with limited access and/or resources (e.g., Airlift).

2 If resources permit, 2 lines of O2 are recommended, one for BVM/Mask, one for supplementary NC.

3 If anaphylaxis is present, administer epinephrine as early as possible, but proceed with airway securement as clinically indicated.

4 No technique for predicting difficult airways has been demonstrated to accurately predict encountered difficulties with reasonable sensitivity or specificity. The LEMON mnemonic is the most commonly used: L- Look Externally, E - Evaluate 3-3-2, M - Mallampati score, O - Obstruction/Obesity, and N - Neck Mobility. While no technique has been demonstrated to have perfect prediction, a rapid assessment for barriers to success is recommended. See picture on previous page of an example where an examination would identify a difficult airway.

5 See Table 3: Sequence for Pre-oxygenation and Prevention of Desaturation

6 Weingart and Levitan, p166 Standard non-rebreather masks with O2 Flow rates of 15 pm deliver only 60-70% FiO2 and do not provide complete de-nitrogenation. Standard non-breather masks can deliver FiO2 approaching 90% by increasing the flow rate to 30 - 60 lpm. NOTE: A Bag Valve Mask hovering over a patients face provides only ambient O2.

7 Weingart and Levitan, p166

8 Weingart and Levitan, p168 Studies (Lane et al, and Rumkumar et al) have demonstrated in randomized controlled studies that the the 20 degree heads up position improved the time to desaturation by ~100 seconds over the supine position following 3 minutes of pre oxygenation.

9 In setting of no IV/IO access, medications ketamine 4mg/kg and succinylcholine 4mg/kg can be given intramuscularly for sedation and paralysis.

10 Adapted from Scott Weingart, EMCRIT podcast. See Table 5: Cricoid2 status

11 Apnea may result from rapid pushes of ketamine, so push slowly or expect a brief period of apnea to result.

12 See Table 4: Risk Categorization of Patients During Pre-Oxygenation from Weingart and Levitan article.

13 All induction and paralytic agents can also be given intrasosseous (IO).

14 Example: Orotracheal intubation success is anticipated to be improbable/impossible (e.g., jaws wired shut with no ability to remove).

15 Intubating conditions are directly related to dose. Doses less than 1.5mg/kg are suboptimal. Increasing the dose from 0.5 to 2mg/kg increased duration of action minimally from 5.2 to 7.5min (see p262 Ron Walls).
Ron Wall 1mg/kg “is optimal”, Weingart “1.2mg/kg). NOTE from Weingart and Levitan, The choice of paralytic agent may influence the time to desaturation during airway management. In a study of operative patients, the time to desaturation to 95% was 242 seconds in patients receiving succinylcholine versus 378 seconds in a group given rocuronium. Similarly, in obese patients undergoing surgery, the succinylcholine group desaturated to 92% in 283 seconds versus 329 seconds in the rocuronium group. When used at a dose of greater than or equal to 1.2 mg/kg, rocuronium provides intubating conditions identical to those of succinylcholine.

It is hypothesized that the fasciculations induced by succinylcholine may cause increased oxygen use. Pretreatment medications to prevent fasciculations minimize the difference in desaturation times. Recommendation: In patients at high risk of desaturation, rocuronium may (or may not) provide a longer duration of safe apnea than succinylcholine.

Administration of paralytic first when using rocuronium or vecuronium and induction sedative ketamine/etomidate second, is to reduce the sedation lag time. (The interval between onset of sedation and when complete paralysis occurs. During this lag time respiratory efforts drop, alveoli start to de-recruit, PaO2 falls, and PaCO2 rises.) See PulmCrit/EMCrit reference by Weingart 4/24/17.

Weingart and Levitan, p171 Apneic oxygenation requires a patent airway for oxygen to reach the hypopharynx and be entrained into the trachea; once the patient is sedated and paralyzed, it is imperative to keep the posterior pharyngeal structures and tongue from occluding the passage of gas. Head elevation, chin lift, and jaw thrust will accomplish this in most patients; a jaw thrust alone should be used if there is risk for cervical spine injury. In some patients, a nasal trumpet or oral airway may also be required. Patients with sleep apnea or obesity often need a combination of jaw distraction, lifting of submandibular soft tissue, and nasal or oral airways.

Positioning the patient with their external auditory meatus on the same horizontal plane as their sternal notch maximizes upper airway dimensions and facilitates direct laryngoscopy. Head elevation relative to the thorax also permits optimal jaw distraction, and conversely atlanto-occipital extension pivots the base of tongue and epiglottis against the posterior pharynx and promotes obstruction. In all but the thinnest patients, a head-elevated position requires lifting of the head of the bed somewhat, plus padding under the head and upper shoulders. The face plane of the patient should be parallel to the ceiling. For the super-obese, this positioning requires a very large ramp. In cervical spine precautions, elevating the head relative to the neck is not possible, but as previously noted the foot of the stretcher should be tilted downward to improve pulmonary function.

Cricoid pressure, considered an essential aspect of rapid sequence tracheal intubation when it was first conceived, has come under increasing scrutiny within anesthesia and emergency medicine. Theoretically, the application of firm pressure to the cricoid cartilage compresses the esophagus while keeping the trachea patent, but in practice this is not always the case. Computed tomography and magnetic resonance imaging scanning have shown that cricoid pressure causes lateral displacement of the esophagus in more than 90% of patients and laryngeal/tracheal compression in 80%. Numerous ventilation studies have found that cricoid pressure hinders bag-valve-mask device ventilation, increases peak inspiratory pressure, and reduces tidal volumes. For the same reasons that the airway obstruction induced by cricoid pressure may preclude effective manual ventilation, it may limit the effectiveness of apneic oxygenation as well.

Recommendation: Patients should be positioned to maximize upper airway patency before and during the apneic period, using ear-to–sternal notch positioning. Nasal airways may be needed to create a patent upper airway. Once the apneic period begins, the posterior pharyngeal structures should be kept from collapsing backwards by using a jaw thrust. Cricoid pressure may negatively affect apneic oxygenation, but studies examining this question in the setting of modern emergency airway management do not exist to our knowledge.

Weingart and Levitan, p 170

In general, the 3rd attempt at laryngoscopy is not appropriate for a student or inexperienced provider.
22 Evaluate for possible intubation of right mainstem.

23 Over-ventilation (hyperventilation) has been documented frequently in the prehospital setting.

24 Paralyzed patients lose their ability to generate heat.

25 From a Cochrane Analysis published January 8, 2016: Moderate quality evidence from eight studies involving 759 participants demonstrated that a semi-recumbent (30° to 60°) position reduced clinically suspected VAP by 25.7% when compared to a 0° to 10° supine position. Based on this result, we would expect that out of 1000 critically ill adult patients who are nursed in the semi-recumbent position (30° to 60°) for more than 48 hours, 145 patients would experience clinically suspected VAP compared to 402 patients nursed in the 0° to 10° supine position. No adequate evidence is available to draw any definitive conclusion on other outcomes and the comparison of alternative semi-recumbent positions.

26 Ketamine can cause mild increase in HR and BP as well

27 The BVM Effect: An Overview of Studies Assessing Airway Management in Out-of-Hospital Cardiac Arrest by Fowler, et. al, JEMS 9/28/15

28 Succinylcholine is rapidly metabolized by plasma esterase and distributed to extracellular water. Children have a larger reservoir of extracellular water relative to adults. The recommended dose of succinylcholine, therefore, is higher in children. Walls, et. al, p281

29 Scott Weingart


31 Weingart and Levitan

32 Weingart and Levitan


34 Table adapted from Capnography as a Clinical Tool by David Wampler, EMSWorld August 2011