RSI: what is it? Quick administration of sedative and paralytic to achieve fast endotracheal intubation. Why do we do it?
-When the patient is unstable and cannot perform one or more of the following:
  1) patient’s inability to ventilate (hypercapnic respiratory failure)
  2) inability to oxygenate (hypoxic respiratory failure)
  3) inability to protect their own airway
  4) for optimal control of the patient (i.e. to deliver care in which the patient would not tolerate or be uncooperative)

Goal: The point is to maximize chances of success on first attempt. In RSI we must assume the stomach has contents and therefore time must be minimized till endotracheal intubation due to risks of respiratory failure and loss of airway protection, as well as aspiration from prolonged BVM. Risks of aspiration, hypotension, esophageal intubation increase with number of attempts (also 15% → 50% increase in adverse events!).

Contraindications: all relative. Most important CI is difficult airway anticipation, of which rescue oxygenation is considered to be impossible or hard to achieve.

If an anatomic difficult airway is anticipated, potential for rapid desaturation, or severe hemodynamic compromise, awake intubation might be needed.

-Pros of awake intubation: avoids hemodynamic collapse, patient maintains spontaneous ventilation
-Cons: takes time, secretion control

When to delay RSI: There are key differences between “emergent” and “urgent” RSI. ABC’s is never 100% the correct order. Sometimes it needs to be BAC or CAB. Some examples:
- Patient is not apneic and unresponsive. This patient needs a definitive airway immediately = ABC
- Patient is in V tach and unresponsive. Still breathing spontaneously = CAB
- Patient is not responding and has no breath sounds on right side and is hypotensive = BAC
- Patient is in shock with BP 90/50 systolic = CAB

Steps to successful RSI: 7P’s of RSI: Prepare, Preoxygenate, Positioning, Paralysis, Paralytic, Proof of placement, Post-intubation management
-Preparation: large bore wall suction, O2, tube, back-up plan x3, 2 large bore IVs
-Preoxygenate: high flow rate for minimum of 3 minutes. Replaces mixed gases in the base of lung with oxygen-rich gas for optimal FRC reservoir. 70 kg adult can maintain O2 saturation ≥90% for 8 minutes during apnea. Children = 4 minutes. Obesity = 3 minutes. Use bag mask for oxygen administration but no need to squeeze bag if patient is cooperative and has adequate spontaneous ventilation. Maintain nasal cannula flow during apneic period. For the very sick:
  -if not able to spontaneously ventilate: positive pressure ventilation with BVM and oxygen flow at max (15 L). Try to synchronize with patient’s respiratory cycle. Do not ventilate >20 cm H2O due to risk of gastric insufflation.
  -if not cooperative, NRB is used with flush oxygen.

Positioning: sniffing position with alignment of vertical axes. Jaw thrust forward and upward, patient’s head more anterior with slight upward extension.

-Pre-intubation optimization: recognize and address obvious or potential pathologies and problems before RSI. Most common issue is hypotension. Causes are multifactorial and include pre-intubation drugs, bleeding, dehydration, patient’s clinical condition (e.g. shock). Must consider giving IV fluids and/or vasopressors if significant concern.

-Paralysis with induction: in the ideal situation, 2 drugs are needed for RSI: a sedative and a paralytic. Both are virtually given within 1 minute apart, with the sedative given first. All the induction agents below work as allosteric agonists of the GABA pathway in the brain. Ketamine is the exception and is primarily an NMDA antagonist.

<table>
<thead>
<tr>
<th>Sedative</th>
<th>Onset</th>
<th>Benefits</th>
<th>Adverse effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate</td>
<td>&lt;30 seconds</td>
<td>Least likely to cause hypotension.</td>
<td>Been documented to suppress adrenal cortisol production.* Can cause myoclonus (not significant but mistaken as seizure).</td>
<td>0.3 mg/kg</td>
</tr>
<tr>
<td>Ketamine</td>
<td>&lt;1 minute; lasts for 20 minutes</td>
<td>Catecholaminergic stimulation leading to increased cerebral blood flow, MAP, cardiac output, and bronchodilation. Analgesic!</td>
<td>Very small, but scary, risk of laryngospasm (&lt;0.4%), can cause paradoxical hypotension in those in shock (‘catechol-depleted’). Avoid in those with high MAP and ICP elevation.</td>
<td>1-2 mg/kg</td>
</tr>
</tbody>
</table>
**Airway Superiority: Principles of RSI**

<table>
<thead>
<tr>
<th>Paralytic</th>
<th>Onset/Offset</th>
<th>Benefit</th>
<th>Contraindications</th>
<th>Dose</th>
<th>Antidote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>&lt;1 minute; offset in 10 minutes</td>
<td>Rapid onset and offset. May be used in Myasthenia Gravis (MG) except an increased dose is needed.</td>
<td>Malignant Hyperthermia*. Hyperkalemia (raising K by about 0.5, so this is only a problem in those already hyperkalemic). Rhabdomyolysis, stroke &gt;72 hours old, burn &gt;72 hours old, muscular dystrophy, ESRD with unknown last dialysis session.**</td>
<td>1.5 mg/kg (better to overestimate!) (2-2.5 mg/kg for MG)</td>
<td>No direct antidote</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>1 min/offset in ~45 min</td>
<td>Less side effects than Succinylcholine. May be used in Myasthenia Gravis but at a lower dose</td>
<td>No absolute contraindications Relative contraindications include possibility of difficult airway</td>
<td>1 mg/kg (0.6 mg/kg for MG)</td>
<td>Sugammadex-binding and reverse agent. Associated with arrhythmias</td>
</tr>
</tbody>
</table>

*This risk of adrenal suppression is due to etomidate inhibition of 11-B-hydroxylation, but this should NOT stop its usage during sepsis if patient has significant hypotension precluding the use of other hypotensive-producing induction agents. If worsening hypotension that is refractory to vasopressors and fluids develops after etomidate usage in the first 24 hours, give glucocorticoids.

**Paralytic- none provide any anesthesia or sedation. Neither of the below medications have any difference in clinical outcomes with regards to successful intubation. The only important differences are related to adverse effects and time of onset/offset.

**-Placement with proof:** There are many steps to ensure adequate tube placement- 1) direct glottis visualization with ETT passing through, 2) ETCO2 via monitor and color change on color-change capnograph. The CXR and listening for breath sounds as well as gastric insufflation is NOT reliable alone but should be done after color change capnography. BELIEVE the End-Tidal! It is more reliable and faster to respond to changes than Pulse Ox (this lags behind by several seconds up to 1 minute in some cases).

**-Postintubation management:** 1) secure the tube, 2) CXR to confirm tube positioning, 3) address any lingering preintubation problems (hypotension, shock, etc), 4) optimize the patient’s respiratory status via ventilator settings, 5) address sedation/analgesia as needed. What if hypotension does not improve after initial recovery from apexa? Think about pneumothorax, tracheal tube cuff rupture, mucus plugging, esophageal intubation.

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**References**