**Resuscitation Prior to Definitive Airway Management in the Operational Environment.**

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Prioritizing compressible hemorrhage prior to airway management was a great leap forward in modern pre-hospital trauma management. With adoption of the MARCH algorithm, our order of priorities became more in line with the prevalence of potentially preventable causes of battlefield death.1  Yet, recent work from some of the great minds in Emergency Medicine has shown us that immediate airway management can be refined further still and for good reason.2,3,11,15 Aside from potentially distracting us from more likely life threats in the polytrauma patient, securing a definitive airway, either cricothyroidotomy or endotracheal intubation , is both technically and physiologically challenging. The failure rate of surgical airways performed in the field is upwards of 33%, resulting in further airway compromise through creation of false passages, subcutaneous emphysema and iatrogenic bleeding.4 Reports from recently deployed combat physicians, supported by this author's anecdotal experience in a Role I facility in Afghanistan in the summer of 2019, suggest that many surgical airways performed in the field are not required, making these complications even more tragic. Even with successful pre-hospital intubation there is no demonstrable mortality benefit in trauma, in fact evidence points towards worsening outcomes.18, 19 The analgesic and sedative medications we give for these painful procedures reduce the circulating catecholamines, risking circulatory collapse in hypovolemic patients that are dependent on that sympathomimetic compensation.5,11 Even our darling, ketamine, has been shown to cause sympathetic relaxation and worsening hypotension in the highly adrenergic state of traumatic hypoperfusion.2,5,6  Should the patient subsequently require ventilatory support with positive pressure secondary to now having to breathe through a large straw (especially if respiratory drive is already maxed out from metabolic demands of hemorrhagic shock or compromised from any analgesics, sedatives or paralytics that were required for the procedure), the hemodynamic effects of increasing intrathoracic pressure on venous return can be disastrous when filling pressures are already tenuous to start. All these risks can be mitigated by correctly separating the very rare subset of trauma patients that demand immediate intubation/surgical airway from the overwhelming majority of those where basic maneuvers and adjuncts will suffice until after initial resuscitation and optimization of hemodynamics. The patients that can be placed in the later cohort, particularly with profound AMS and unconsciousness (either from cerebral hypoperfusion or severe TBI), represent a significant deviation from traditional trauma airway management.

With the initial assessment of *any* airway two separate questions should be asked: 1) Is this patient maintaining a patent airway? 2) Is this patient protecting that airway?7 For many years the standard practice in trauma has been that the lack of either spontaneous patency or protection demanded an immediate tube in the trachea. In actuality, all we really need from the patient in the immediate management is air moving freely through the upper airway without making abnormal noises (gurgling, stridor, stertor, etc.).  Rephrased another way, even the most profoundly altered or unconscious trauma patients who are lacking either airway maintenance, airway protection or both can often be managed by basic airway interventions, allowing the provider to focus on more likely causes of preventable death while deferring the risks of a definitive airway until later in the resuscitation.

**A deeper dive into assessment of airway maintenance in the polytrauma patient:**

In absence of trauma to the airway itself, adequate maintenance is present when there exists a level of consciousness to maintain muscular tone that prevents posterior displacement of the tongue or collapse of the hypopharynx. This is assessed best in the pre-hospital environment by placing one's head over the casualty's mouth and nose; listening for adventitial airway noises, feeling for lack of exhalatory air movement while simultaneously looking at the bare chest for retractions and paradoxical thoracoabdominal movements suggesting diffuse airflow limitation. When the chest wall attempts to expand against a collapsed upper airway, the deflated lungs cannot expand, pulling the intercostals inward from the ribs. The diaphragm which has slightly more freedom to travel, flattens pushing the abdominal cavity upwards, paradoxical to the relatively downward motion of the chest on inspiration.16 Resolution of these findings following a correctly performed jaw thrust identifies obstruction as the underlying problem; at which point nasopharyngeal airways, repositioning and recovery position are indicated to maintain the maneuver. For a jaw-thrust to be effective, it is critical to first open the mouth fully, allowing the mandible to subluxate out of the temporal mandibular joint. This allows for full translation of the mandible anteriorly when force is applied to the mandibular rami. Without first subluxating the jaw, anterior movement is severely restricted and thus connected soft tissue cannot be effectively lifted off the posterior wall. It would be extraordinarily rare to encounter an *atraumatic* airway that cannot be opened by a jaw thrust in conjunction with airway adjuncts and re-positioning; especially in the healthy population of military and police that we most often encounter in TEMS. Fiberoptic studies show that more than enough airway clearance can be achieved, even in obstructive sleep apnea patients with adipose laden necks, when the jaw thrust is done correctly.17 A definitive airway is only immediately necessary from a maintenance perspective when the same signs of complete or partial airway obstruction are present in the setting of *direct* airway trauma. Destruction of the bony and cartilaginous architecture and distortion of soft tissues may create occlusion that is refractory to manual maneuvers and airway adjuncts are unlikely to have a lasting effect in these dynamic airways. Noisy airways (stridor, stertor) indicating partial obstruction, typically more prominent on inspiration, similarly mandates expediency towards a definitive airway when in the setting of *direct* airway trauma. Thankfully, these injuries are present only in a minority of the polytrauma patient population.

The exam findings specific to airway obstruction (above) must be discerned from non-specific respiratory signs seen in a multitude of traumatic injuries; tachypnea, air hunger, dyspnea, increased work of breathing, diaphoresis, accessory muscle use or the patient simply stating "I can't breathe!"  These findings are more likely secondary to hemorrhagic shock (hyperadrenergic state coupled with volatile acid compensation), hemothorax, pneumothorax, blast injury, anxiety or pain rather than airway pathology. Too often these findings, at times dramatic, lead to knee-jerk intubation or cricothyroidotomy. This not only fails to treat the underlying problem, but instead likely exacerbates the true pathology. If air is moving freely with basic techniques, inserting a piece of plastic into the trachea prior to addressing hypovolemic or obstructive forms of shock offers no additional benefit and has the potential for disastrous consequences.

**A closer look into the initial assessment and management of airway protection:**

The second part of the airway assessment, protection, is surrounded by even more confusion and misinformation. To start, GCS and gag reflex have absolutely no role in the assessment of airway protection.8,9,10 Not only is the gag reflex misleading in that its presence does not confer protection to an airway, a significant part of the population lacks the reflex (~ 30%, that's a lot of unnecessary intubations if you subscribe to the false teaching that an absent gag demands a definitive airway).12, 13, 14 More importantly, performing a maneuver that has a strong chance of inducing vomiting in a patient with a questionable ability to protect their airway is foolhardy at best. Instead, the presence of the highly complex efferent and afferent neuronal pathways of spontaneous swallowing reflex, evidenced by absence of pooling secretions, is a far better indicator of airway protection.7 For the purposes of the primary trauma survey, absence of pooling secretions (initially or after suctioning and repositioning) in the atraumatic airway is the green light to move on. It is only with pooling secretions that quickly reaccumulate after suctioning or not managed with repositioning that we must move to definitive airway for a protection standpoint. Similarly, in the setting of direct airway trauma causing loss of protection, either from rapid blood accumulation or soft tissue distortion, delaying definitive airway is not an option.

For many years profoundly unconscious or altered trauma patients not responding appropriately to noxious stimuli would warrant immediate intubation (the false dogma of "GCS<8, intubate"), even without pooling secretions.  The concern being they would not defend their airway during a potential vomiting event. However, it has been shown that these airways can be observed safely, particularly during initial resuscitation with the provider immediately available to intervene.7,10 It is important to note that an inflated tube below the vocal cords does not definitively protect one from aspiration either. Certainly, these profoundly altered and unconscious patients should not be left alone and have frequent re-evaluation. The appropriateness of securing a more definitive airway should be readdressed prior to patient transport (ground or air med-evac) or if circumstances in the field no longer allow for the provider's undivided attention.

It is critical that we strive for mastery of basic airway assessment and skills. We must foster our pre-hospital providers to respect the dangers of securing a definitive airway in the critically injured patient. Progression to surgical airway is rarely needed and we must make sure the risks associated with it are truly warranted. In the recent past we had little to offer our patients in the operational environment that could offset the hemodynamic consequences of intubation, but thankfully that is changing. Ketamine is widely available with a more favorable hemodynamic profile when given at low doses and titrated to effect. Whole blood is being pushed further towards the point of injury. Resuscitation with increasingly available freeze dried plasma will hopefully end the practice of crystalloid induced coagulopathy when blood products are not available. Limiting immediate intubation or surgical airways to only those that have direct airway trauma that is compromising maintenance or protection allows us to address the more common life threats in the polytrauma patient and increase the likelihood of them surviving the definitive airway when appropriate.

1. Eastridge, Brian J. et al. *Death on the Battlefield (2001-2011): Implications of the future of combat casualty care.* Journal of Trauma and Acute Care Surgery. 2012;73(6)
2. Scott Weingart. EMCrit RACC Podcast 216 – *The Hemodynamically Neutral Intubation*. *EMCRIT Blog*. Published on January 22,2018.
3. Petrosoniak, Andrew and Hicks, Christopher*. Resuscitation Resequenced A Rational Approach to Patients with Trauma in Shock*. Emergency Medicine Clinics. 2018;36(1):41-60
4. Prehospital Trauma Life Support Committee of the NAEMT. Prehospital Trauma Life Support. Military 8th edition. 2016
5. Morris, C et al. *Anesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent.* Anesthesia 2009; 101:567
6. Waxman K et al. *Cardiovascular Effects of anesthetic induction with ketamine.* Anesth Analg 1980;59(5): 355-358
7. Walls Ron M., and Michael F. Murphy. *Manual of Emergency Airway Management.* Philadelphia: Wolters Kluwer/ Lippincott Williams & Wilkins Health, 2012.