Shock and Trauma Resuscitation

Bonjo Batoon, MS, CRNA
Bbatoon@som.umaryland.edu
Disclaimer

• Resuscitation is continuously evolving
• There is no one right way
• “Knowing is half the battle” G.I. Joe
• Having to appropriate resources/information is other half
• How I feel or think now may be very different tomorrow
### Demographics Report

Report generated on 11/15/2013  
Range From 07/01/2012 to 06/30/2013  
Primary Trauma Discharged FY 13  
Number of Records 6555

#### Type of Injury

<table>
<thead>
<tr>
<th>Type of Injury</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt</td>
<td>5618</td>
<td>85.7</td>
</tr>
<tr>
<td>Penetrating</td>
<td>784</td>
<td>12.0</td>
</tr>
<tr>
<td>Burn</td>
<td>8</td>
<td>0.1</td>
</tr>
<tr>
<td>Near drowning</td>
<td>3</td>
<td>0.0</td>
</tr>
<tr>
<td>Hanging</td>
<td>8</td>
<td>0.1</td>
</tr>
<tr>
<td>Inhalation</td>
<td>90</td>
<td>1.4</td>
</tr>
<tr>
<td>Ingestion</td>
<td>4</td>
<td>0.1</td>
</tr>
<tr>
<td>Crush</td>
<td>28</td>
<td>0.4</td>
</tr>
</tbody>
</table>

#### Cause of Injury

<table>
<thead>
<tr>
<th>Cause of Injury</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Vehicle Crash</td>
<td>1834</td>
<td>28.0</td>
</tr>
<tr>
<td>Motorcycle Crash</td>
<td>394</td>
<td>6.0</td>
</tr>
<tr>
<td>Pedestrian Incident</td>
<td>274</td>
<td>4.2</td>
</tr>
<tr>
<td>Falls</td>
<td>2143</td>
<td>32.7</td>
</tr>
<tr>
<td>GSW (Non-Assault)</td>
<td>22</td>
<td>0.3</td>
</tr>
<tr>
<td>GSW (Assault)</td>
<td>353</td>
<td>5.4</td>
</tr>
<tr>
<td>Stabbing (Non-Assault)</td>
<td>56</td>
<td>0.9</td>
</tr>
<tr>
<td>Stabbing (Assault)</td>
<td>336</td>
<td>5.1</td>
</tr>
<tr>
<td>Other Assault</td>
<td>553</td>
<td>8.4</td>
</tr>
<tr>
<td>Other Cause</td>
<td>590</td>
<td>9.0</td>
</tr>
</tbody>
</table>
Shock by definition

- A clinical state of acute circulatory failure with \textit{inadequate oxygen utilization and/or delivery} by the cells resulting in \textit{cellular dysoxia/hypoxia}
  - \textit{Intensive Care Med 2014;40:1795}
- A state of inadequate tissue perfusion
- A cellular and end-organ disorder

\textbf{Not} a disorder of the macro-circulation

- Decreased BP does not equal shock
- BP does not \textit{=} flow
Oxygen Debt

- Energy Metabolism (Oxygen Consumption) (ml/min/m²)
- Time

- Full recovery possible
- Delayed repayment of O₂ debt
- Excessive O₂ deficit produces lethal cell injury with non-recovery
Types of shock

• Hemorrhagic- Most common
• Non-hemorrhagic
  – Cardiogenic
  – Neurogenic
  – Septic
  – Tension pneumothorax
  – Poisoning
Signs & Symptom of Shock

- Tachycardia
- Tachypnea
- Decreased capillary refill
- Hypotension
- Narrow pulse pressure
- Altered mental status
- Elevated lactate
- Coagulopathy

- Cyanosis, pallor, diaphoresis
- Hypothermia
- Decreased urine output
- Absent pulse oximetry signal*
- +FAST/CT*
### Classification of Shock

<table>
<thead>
<tr>
<th>Class of haemorrhagic shock</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (mL)</td>
<td>Up to 750</td>
<td>750–1500</td>
<td>1500–2000</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td>Blood loss (% blood volume)</td>
<td>Up to 15</td>
<td>15–30</td>
<td>30–40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>Pulse rate (per minute)</td>
<td>&lt; 100</td>
<td>100–120</td>
<td>120–140</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Respiratory rate (per minute)</td>
<td>14–20</td>
<td>20–30</td>
<td>30–40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td>Urine output (mL/hour)</td>
<td>&gt; 30</td>
<td>20–30</td>
<td>5–15</td>
<td>Negligible</td>
</tr>
<tr>
<td>Central nervous system/mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious, confused</td>
<td>Confused, lethargic</td>
</tr>
</tbody>
</table>
Lethal Triad

Coagulopathy

Acidosis

Hypothermia
Resuscitation Goals

• Early recognition of the shock state
• Oxygenate and ventilate
• Restore the circulating volume
• Restore organ perfusion
• Restore homeostasis / repay
  “oxygen debt”
• Stop the bleeding- *Surgeon’s job*
• Treat coagulopathy
• Continuous monitoring of the response
Components to Resuscitation

- Airway
- Breathing
- Circulation
- Exposure
Anticipation of Respiratory Consequences of Injuries

- Tension pneumothorax
- Flail chest
- Hemothorax
- Pneumothorax
- Chest wall asymmetry
- Diaphragmatic rupture
- Atelectasis
- Aspiration
- Pulmonary contusion
- Thermal / Smoke
Signs of Airways Problems

- Lacerated pharyngeal tissue
- Cervical hematoma
- Active oral bleeding
- Copious secretions
- Foreign bodies
- Displaced bone
- Dyspnea
- JVD
- Hoarseness
- Stridor
- Dysphonia
- Subcutaneous air
- Hemoptysis
- Cervical deformity
- Edema
- Tracheal deviation
Challenges to Airway Management

- Blood/secretions/emesis
- Airway injuries
- Body habitus
- C-spine issues
- Positioning limitations
Airway

- DL
- Bougie
- Video laryngoscopy
- AFOI
- RSI vs MRSI
- Cricoid pressure- ??
- C-spine issues
- Surgical cricothyrotomy/trach when all else fails
The Effectiveness of Cricoid Pressure for Occluding the Esophageal Entrance in Anesthetized and Paralyzed Patients: An Experimental and Observational Glidescope Study

Ahed M. Zeidan, MD,*† M. Ramez Salem, MD,‡§ Jean-Xavier Mazoit, MD, PhD,¶¶ Mohamad Ali Abdullah, MD,*# Tharwat Ghattas, MD,* and George J. Crystal, PhD†§

Figure 1. Assessment of the position of the esophageal entrance relative to the glottis (Cricoid pressure was not applied.) A vertical line was drawn from the middle of the posterior border of the glottis. If the line crossed the middle third of the esophageal entrance, it was considered in a midline position, whereas if the line crossed to the right or to the left of the middle third of the esophageal entrance, it was considered in a left lateral or right lateral position.
Cricoid Pressure


<table>
<thead>
<tr>
<th>Position of Esophagus</th>
<th>No CP</th>
<th>With CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Directly behind trachea</td>
<td>20%</td>
<td>4%</td>
</tr>
<tr>
<td>Partially behind trachea (left)</td>
<td>60%</td>
<td>26%</td>
</tr>
<tr>
<td>Completely lateral trach (left)</td>
<td>20%</td>
<td>70%</td>
</tr>
</tbody>
</table>
When to Intubate?

- GCS <8
- Combative
- Hypoxia
- Poor / Inadequate Ventilations
- Lack of airway reflexes
- Pain control
- Social issues
- Deteriorating condition
- Manipulation of long bones when muscle relaxation is required
So You Want to Intubate?

• Induction drugs
  – Propofol 2/mg Kg IV
  – Etomidate 0.2-0.3mg/Kg IV
  – Ketamine 2-4mg/Kg IV
  – Ketamine 4-10mg/Kg IM

• Be acutely aware of all of the pt’s issues, allergies, and resuscitation status

• Induction can kill

• Muscle relaxation
  – Succinylcholine 1-1.5 mg/kg
  – 30 second onset
  – Will cause patients to fasciculate (depolarizing block)
  – Approximately 5-12 minute duration of action

• Rocuronium 1.2 mg/kg
  – 60-90 second onset -> may need to gently mask ventilate (“modified RSI”)
  – Patients will not fasciculate (non-depolarizing block)
  – Approximately 60-90 minutes duration of action
Airway Confirmation

- Visualization
- End tidal CO$_2$
- Condensation
- Auscultation
- Chest excursion
- O$_2$ saturation
- Radiography
The Success of Emergency Endotracheal Intubation in Trauma Patients: A 10-Year Experience at a Major Adult Trauma Referral Center

- 6088 pts required intubation within the first 1hr of admission
- 21 (0.3%) received surgical airway
- Unanticipated difficult airway was the leading cause of surgical airway
- 4 pts died but none died as a result of failed intubation
Figure 1. Emergency airway management algorithm at the R Adams Cowley Shock Trauma Center. It is assumed that an airway is absolutely required and that patients cannot be reawakened electively. LMA = laryngeal mask airway.
Breathing

• Secure airway most important
• Adequately oxygenate
• Monitor CO2
• Consider lower Vt in hypotensive pts
• Ventilation strategies??
  – Pressure vs VC
• Massive transfusion- TRALI, TACO, ARDS
Circulation

- Adequate IV access
- Peripheral
  - 16G or greater
  - RIC aka mini cordis
  - Know flow rates for each cathether
- Preferably central access
  - IJ vs SC vs femoral
  - Cordis vs double lumen catheters vs triple lumen
Exposure

- 34°C was the critical point at which enzyme activity slowed significantly, and at which significant alteration in platelet activity was seen. Fibrinolysis was not significantly affected at any of the measured temperatures.

- Keeping pt warm
  - Warm blood products
  - Bair hugger type devices
  - Warm operating room
Hypothermia and Coagulopathy
Effects on Clotting Factor Activity

<table>
<thead>
<tr>
<th>°C</th>
<th>II</th>
<th>V</th>
<th>VII</th>
<th>VIII</th>
<th>IX</th>
<th>X</th>
<th>XI</th>
<th>XII</th>
</tr>
</thead>
<tbody>
<tr>
<td>25°</td>
<td>5</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>27°</td>
<td>7</td>
<td>5</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>29°</td>
<td>10</td>
<td>8</td>
<td>12</td>
<td>3</td>
<td>3</td>
<td>10</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>31°</td>
<td>17</td>
<td>22</td>
<td>34</td>
<td>16</td>
<td>7</td>
<td>20</td>
<td>16</td>
<td>10</td>
</tr>
<tr>
<td>33°</td>
<td>24</td>
<td>50</td>
<td>60</td>
<td>59</td>
<td>32</td>
<td>44</td>
<td>60</td>
<td>17</td>
</tr>
<tr>
<td>35°</td>
<td>82</td>
<td>75</td>
<td>82</td>
<td>79</td>
<td>66</td>
<td>81</td>
<td>85</td>
<td>65</td>
</tr>
<tr>
<td>37°</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Monitoring

- Basic
- Advanced
  - A line
  - CVP?
  - PPV- FloTrac
  - TEE
  - Labs- CBC, coags, lytes, ABGs
  - POC
    - Hemoque- Hgb
    - iStat- lytes/gases
    - ROTEM/TEG- coagulation
Clotting Dynamics

- **Normal**
  - R:K:MA:Angle = Normal

- **Anticoagulants/hemophilia**
  - Factor Deficiency
  - R:K = Prolonged;
  - MA:Angle = Decreased

- **Platelet Blockers**
  - Thrombocytopenia/
    - Thrombocytopathy
  - R = Normal; K = Prolonged;
  - Angle = Normal
  - MA = Very Decreased

- **Fibrinolysis**
  - Presence of t-PA
  - R = Normal;
  - MA = Continuous decrease

- **Hypercoagulability**
  - R:K = Decreased;
  - MA:Angle = Increased

- **D.I.C.**
  - **Stage 1**
    - Hypercoagulable state with secondary fibrinolysis
  - **Stage 2**
    - Hypocoagulable state
Components to Resuscitation

- Crystalloids
- Colloids
- Blood products
Crystalloids

• LR
• NS
• Plasmalyte
• Crystalloids are not and should not be the mainstay of trauma resuscitation!!
IMMEDIATE VERSUS DELAYED FLUID RESUSCITATION FOR HYPOTENSIVE PATIENTS WITH PENETRATING TORSO INJURIES

WILLIAM H. BICKELL, M.D., MATTHEW J. WALL, JR., M.D., PAUL E. PEPPE, M.D.,
R. RUSSELL MARTIN, M.D., VICTORIA F. GINGER, M.S.N., MARY K. ALLEN, B.A.,
AND KENNETH L. MATTOX, M.D.

<table>
<thead>
<tr>
<th></th>
<th>Immediate resus</th>
<th>Delayed resus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>38%</td>
<td>30%</td>
</tr>
<tr>
<td>EBL mL</td>
<td>3127 ± 4937</td>
<td>2555 ± 3546</td>
</tr>
<tr>
<td>Prehospital LR</td>
<td>870 ± 667</td>
<td>92 ± 309</td>
</tr>
<tr>
<td>Trauma center LR</td>
<td>1608 ± 1201</td>
<td>282 ± 722</td>
</tr>
<tr>
<td>OR LR</td>
<td>6772 ± 4688</td>
<td>6529 ± 4863</td>
</tr>
</tbody>
</table>

Fig 2 Clot firmness measured with the ROTEM system in an animal model of controlled and uncontrolled haemorrhage: MCF (in mm) at baseline (1), after removal of 65% of the estimated blood volume (2), after colloid administration (3), after substitution of fibrinogen concentrate (Fib) or placebo (Gel) (4), and after an observation period of 2 h (5).
Table 2. Recommendations for Fluid Resuscitation in Acutely Ill Patients.

**Fluids should be administered with the same caution that is used with any intravenous drug.**
Consider the type, dose, indications, contraindications, potential for toxicity, and cost.

**Fluid resuscitation is a component of a complex physiological process.**
Identify the fluid that is most likely to be lost and replace the fluid lost in equivalent volumes.
Consider serum sodium, osmolality, and acid–base status when selecting a resuscitation fluid.
Consider cumulative fluid balance and actual body weight when selecting the dose of resuscitation fluid.
Consider the early use of catecholamines as concomitant treatment of shock.

**Fluid requirements change over time in critically ill patients.**
The cumulative dose of resuscitation and maintenance fluids is associated with interstitial edema.
Pathological edema is associated with an adverse outcome.
Oliguria is a normal response to hypovolemia and should not be used solely as a trigger or end point for fluid resuscitation, particularly in the post-resuscitation period.
The use of a fluid challenge in the post-resuscitation period (≥24 hours) is questionable.
The use of hypotonic maintenance fluids is questionable once dehydration has been corrected.

**Specific considerations apply to different categories of patients.**
Bleeding patients require control of hemorrhage and transfusion with red cells and blood components as indicated.
Isotonic, balanced salt solutions are a pragmatic initial resuscitation fluid for the majority of acutely ill patients.
Consider saline in patients with hypovolemia and alkalosis.
Consider albumin during the early resuscitation of patients with severe sepsis.
Saline or isotonic crystalloids are indicated in patients with traumatic brain injury.
Albumin is not indicated in patients with traumatic brain injury.
Hydroxyethyl starch is not indicated in patients with sepsis or those at risk for acute kidney injury.
The safety of other semisynthetic colloids has not been established, so the use of these solutions is not recommended.
The safety of hypertonic saline has not been established.
The appropriate type and dose of resuscitation fluid in patients with burns has not been determined.

DOI: 10.1056/NEJMra1208627
Resuscitation Fluids

John A. Myburgh, M.B., B.Ch., Ph.D., and Michael G. Mythen, M.D., M.B., B.S.

- No ideal resuscitation fluid
- Clinician dependent
- No clear benefit colloid over crystalloid
- Albumin safe
  - Increased cost
  - Increased mortality TBI
  - Safe in most other uses
- HES
  - Increased renal replacement therapy
  - Increased adverse effects critically ill
  - Dose dependent decrease in fibrin polymerization
    - coagulopathies

DOI: 10.1056/NEJMra1208627
Colloids

• Starches
  – Coagulopathy r/t decreased fibrin polymerization
  – Hespan max dose 20ml/kg

• Albumin
  – Allergic rxs
Blood Products

• RBCs- uncrossmatched vs crossmatched
• FFP
• Plts
• Cryoprecipitate- high in fibrinogen
• Other hemostatic agents
  – TXA, PCCs, fibrinogen concentrate, fVIIa
Component Therapy

Whole blood 500 mL
(Hct 38%–50%; Plts 150 K–400 K; Plasma coagulation activity 100%)

150 mL anticoagulant added; centrifuged

1 Unit PRBC
(335 mL, Hct 55%)

1 Unit Plasma
(275 mL, coagulation activity 80%)

1 Unit Platelets
(50 mL, 5.5 x 1010 plts)

Patient Receives 650 mL fluid:
Hct 29%, Plts 88 K, 65% coagulation activity

doi: 10.1002/bjs.7731
Resuscitation Strategies

- Permissive hypotension

- Ratio based resuscitation
  - RBC:FFP; RBC:FFP:PLTs

- Point of Care
  - ROTEM/TEG
  - Coagulation concentrates

- Laboratory based resuscitation
  - Lab delays
  - Lost samples
Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial

Findings 10,096 patients were allocated to tranexamic acid and 10,115 to placebo, of whom 10,060 and 10,067, respectively, were analysed. All-cause mortality was significantly reduced with tranexamic acid (1463 [14.5%] tranexamic acid group vs 1613 [16.0%] placebo group; relative risk 0.91, 95% CI 0.85–0.97; p=0.0035). The risk of death due to bleeding was significantly reduced (489 [4.9%] vs 574 [5.7%]; relative risk 0.85, 95% CI 0.76–0.96; p=0.0077).

www.thelancet.com Published online June 15, 2010 DOI:10.1016/S0140-6736(10)60835-5

• Mortality TXA 14.5% vs placebo 16%
• TXA given 1 gram/10 minute
• Followed by 1 gram infusion over 8 hrs
• Design: Selectively randomized?!
### Military Application of Tranexamic Acid in Trauma Emergency Resuscitation (MATTERs) Study

Jonathan J. Morrison, MB ChB, MRCS; Joseph J. Dubose, MD; Todd E. Rasmussen, MD; Mark J. Midwinter, BMedSci, MD, FRCS

*Arch Surg. Published online October 17, 2011.*

<table>
<thead>
<tr>
<th></th>
<th>TXA</th>
<th>No TXA</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISS</td>
<td>25.2</td>
<td>22.5</td>
</tr>
<tr>
<td>Mortality</td>
<td>17.4%</td>
<td>23.9%</td>
</tr>
<tr>
<td>Mortality MT</td>
<td>14.4%</td>
<td>28.1%</td>
</tr>
</tbody>
</table>

- TXA group less over all coagulopathy
Association of Cryoprecipitate and Tranexamic Acid With Improved Survival Following Wartime Injury

Findings From the MATTERs II Study

Jonathan J. Morrison, MB, ChB, MRCS; James D. Ross, PhD; Joseph J. Dubose, MD; Jan O. Jansen, FRCS, FFICM; Mark J. Midwinter, BMedSci, MD, FRCS; Todd E. Rasmussen, MD

**Figure.** Survival plot of patients who received tranexamic acid and cryoprecipitate (TXA/CRYO), tranexamic acid alone (TXA), cryoprecipitate alone (CRYO), or neither product (no TXA/CRYO) as part of a component-based hemostatic resuscitation following combat injury.

*Arch Surg. Published online November 19, 2012.*
**Association of Cryoprecipitate and Tranexamic Acid With Improved Survival Following Wartime Injury**

Findings From the MATTERs II Study

Jonathan J. Morrison, MB, ChB, MRCS; James D. Ross, PhD; Joseph J. Dubose, MD; Jan O. Jansen, FRCS, FFICM; Mark J. Midwinter, BMedSci, MD, FRCS; Todd E. Rasmussen, MD

<table>
<thead>
<tr>
<th></th>
<th>TXA/cryo</th>
<th>Cryo</th>
<th>TXA</th>
<th>TXA/Cryo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>23.6%</td>
<td>21.4%</td>
<td>18.2%</td>
<td>11.6%</td>
</tr>
<tr>
<td>ISS</td>
<td>21.2</td>
<td>28.3</td>
<td>23</td>
<td>26</td>
</tr>
</tbody>
</table>

- Retrospective analysis
- TXA/cryo implemented last 18 months of study
- Benefit may be additive
- Known anti-inflammatory effects of TXA
  - Unknown mechanism

*Arch Surg. Published online November 19, 2012. doi:10.1001/jamasurg.2013.764*
**Table 1. Goals for Damage Control in the Severely Injured Patient**

- Stable airway and oxygenation
- **Hemostasis**—control of life-threatening hemorrhage
  - Exploratory laparotomy or thoracotomy
  - Rapid, wide exposure
  - Excision over repair of “expendable” organs
  - Focus on hemostatic procedures only
    - Vessel ligation or repair
      (avoid grafting if possible)
    - Packing for diffuse bleeding
  - Temporary closure
  - Angiographic embolization in selected cases
- **Effective analgesia and sedation**
- **Appropriate blood composition:**
  - Oxygen-carrying capacity (red blood cells)
  - Clotting potential (platelets, clotting factors)
  - Chemistry (especially calcium, glucose, potassium, chloride)
- **Stabilization/reversal of tissue acidosis**
- Normothermia

<table>
<thead>
<tr>
<th>Table 2. The Essentials of Damage Control Anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Airway and ventilator management</td>
</tr>
<tr>
<td>• Rapid sequence intubation</td>
</tr>
<tr>
<td>• Titration of ventilation</td>
</tr>
<tr>
<td>• Control of bleeding</td>
</tr>
<tr>
<td>• Deliberate hypotensive resuscitation</td>
</tr>
<tr>
<td>• Maintenance of blood composition</td>
</tr>
<tr>
<td>• Preservation of homeostasis</td>
</tr>
<tr>
<td>• Normothermia</td>
</tr>
<tr>
<td>• Restored and sustained end-organ perfusion</td>
</tr>
<tr>
<td>• Analgesia and sedation</td>
</tr>
</tbody>
</table>

### Table 3. Resuscitation Goals

*During Damage Control Surgery*

- Systolic blood pressure 90 mm Hg
- Heart rate <120 beats per minute
- Pulse oximeter functioning, \( \text{SaO}_2 > 95\% \)
- Urine output present
- \( \text{PaCO}_2 < 50 \) torr
- \( \text{pH} > 7.25 \)
- Hematocrit >25%
- Lactate stable or decreasing
- Ionized calcium >1.0
- International normalized ratio <1.6
- Platelets >50,000
- Normothermia
- Deep anesthesia

*Lower blood pressure may be tolerated as long as acidosis is not worsening.*

APPENDIX A  TRAUMA ANESTHESIA CHECKLIST
(adapted from ref.1 and other CENTCOM CPGs)

BEFORE PATIENT ARRIVAL
☐ Room temperature ≥ 25°C
☐ Warm IV line
☐ Machine check
☐ Airway equipment check
☐ Emergency medication check
☐ Blood Bank notified to have blood available per unit SOP

PATIENT ARRIVAL
☐ Patient identified if possible
☐ Blood Bank notified to deliver blood per unit SOP
☐ Ensure large bore IV or CVC access
☐ Monitors (SaO2, BP, ECG)
☐ Pre-oxygenation

INDUCTION
☐ Sedative hypnotic (ketamine vs. propofol vs. etomidate)
☐ Neuromuscular blockade (succinyl choline vs. rocuronium)

INTUBATION (per Trauma Airway Management CPG)
☐ (+) ETCO2
☐ Place orogastric tube

Guideline Only/Not a Substitute for Clinical Judgment
June 2014
APPENDIX A  TRAUMA ANESTHESIA CHECKLIST  
(adapted from ref.1 and other CENTCOM CPGs)

ANESTHETIC

☐ Consider TIVA
☐ (Volatile anesthetic and/or benzodiazepine) + narcotic
☐ Insert additional IV access and/or arterial line if needed

RESUSCITATION (per DCR CPG)

☐ Send baseline labs, type and cross if not yet done
☐ Follow MAP trends
☐ Goal FFP:PRBC:plt 1:1:1 if Massive Transfusion
☐ Goal urine output 0.5-1.0 mL/kg/hr
☐ Consider TXA if <3 hours from injury and indicators for Massive Transfusion identified
☐ Consider calcium chloride 1 gm
☐ Consider hydrocortisone 100 mg
☐ Consider vasopressin 5-10 IU
☐ Administer appropriate antibiotics
☐ Special considerations for TBI as indicated in Severe Head Injury CPG

CLOSING/POST-OPERATIVE

☐ Low volume ventilation per Acute Respiratory Failure CPG

Guideline Only/Not a Substitute for Clinical Judgment
June 2014
Pitfalls

- Airway issues
- Recall
- Coagulopathy
- Hypothermia
- Pain
- Anticoagulants
- Pneumothorax
- Missed injuries
- Substance abuse
- Under resuscitation
- Over resuscitation
- Aspiration
- The UNKNOWN
Thank you!!