Shock: Concepts in Resuscitation

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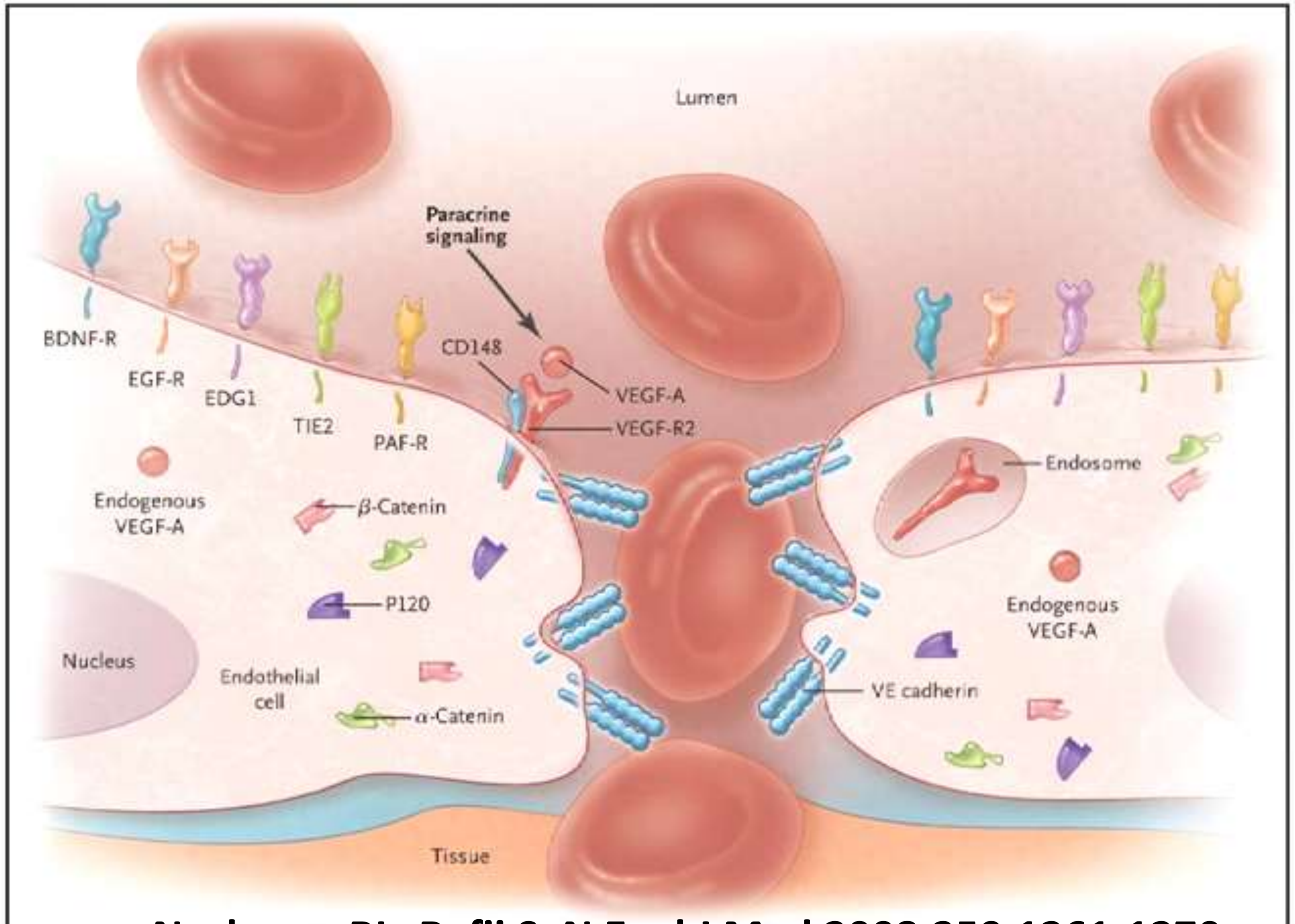
The speaker has no financial or other disclosures

Shock

- Final common pathway
  - Inadequate tissue perfusion
  - Worsening acidosis
  - Tissue ischemia/cell death
  - Cardiovascular collapse>death
- Inflammatory cascade
- Pro/anticoagulant balance lost



Nachman RL, Rafii S. N Engl J Med 2008;359:1261-1270.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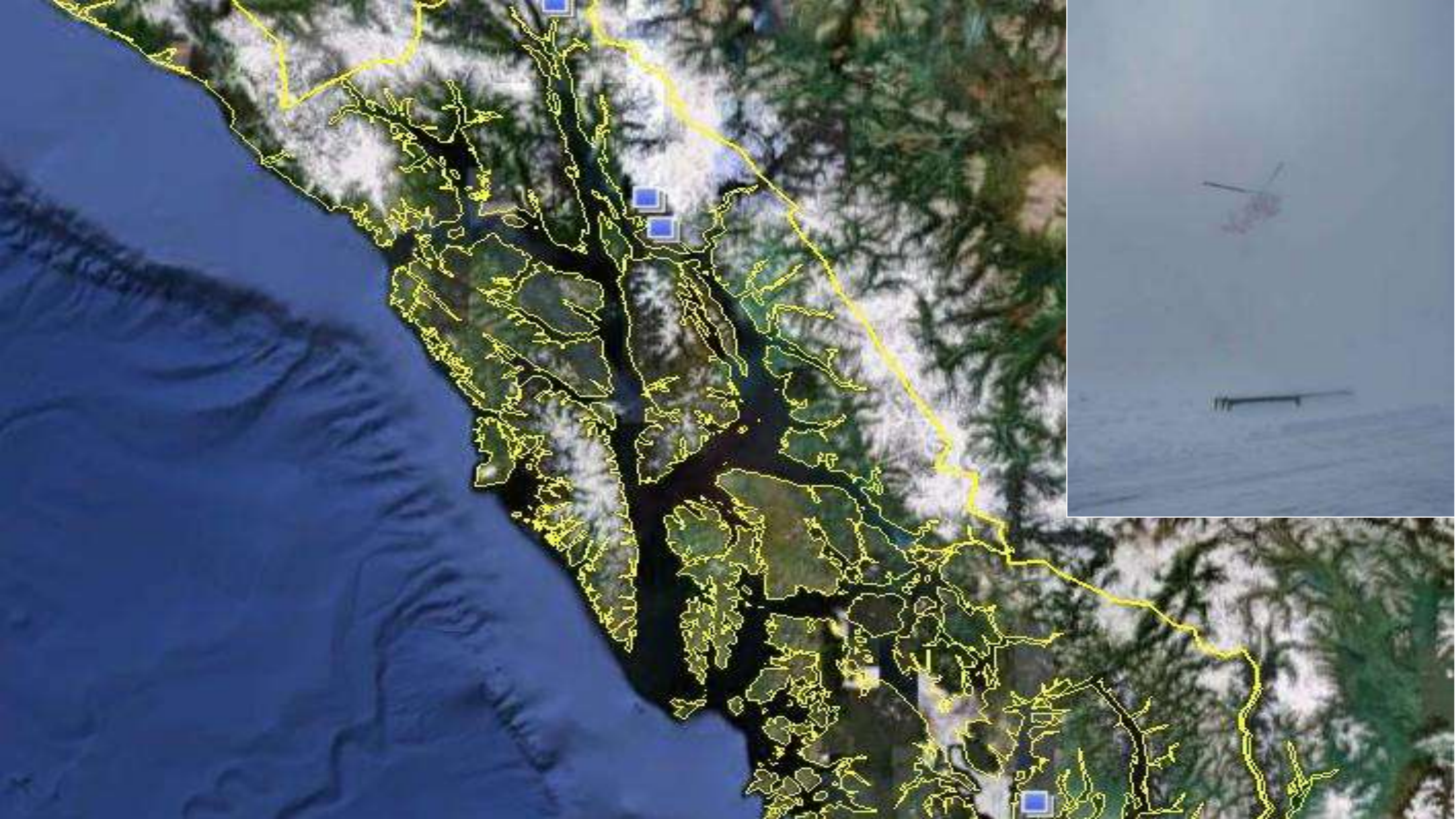
Background

- Trauma ranks fifth as a cause of death in all patients in the US
- Trauma is the leading cause of death in patients 44 years or younger
- Hemorrhage is a leading cause of death in trauma[1]
- Most preventable deaths from trauma are due to uncontrolled hemorrhage and resultant coagulopathy[2]
- Progressive blood loss leads to worsening tissue perfusion (shock), progressive acidosis, cardiovascular collapse and death

It was a dark and stormy night…



Accidental GSW

- Location: A village in SE Alaska, remote from town down a logging road at the town dump
- 17 yo man accidental GSW with .357 Mag to thigh
- Arousable to pain, crying out when moved
- Bleeding uncontrolled

Resources

- BLS EMS Crew with ambulance
- Clinic in town staffed with CHAs and a PA
- Landline phones in town. Radio comms to town via Marine VHF
- Weather below minimums for commercial aeromedevac transport
- USCG helicopter from Sitka willing to try to get in
- Boat transport >24 hours to effect transport

Accidental GSW(continued)

- Vital Signs
  - T 95.9 °F
  - HR 130
  - RR 26
  - BP 80/palp

Accidental GSW(continued)

- Physical Assessment
  - P on AVPU scale, crying out in pain with movement
  - No respiratory distress
  - GSW mid-thigh on right with evidence of arterial bleeding and midshaft femoral bony deformity, (+) pulse in foot
  - No evidence of other injury

Accidental GSW (continued)

- Belt tourniquet dressing placed on scene
- Patient transported to local clinic
- IV resuscitation restored normal BP and HR<100 with crystalloid fluids
- USCG helicopter able to get in to local runway on third attempt. Sager splint placed.
- Patient transported to regional hospital (Level IV), stabilized and transported to Level II trauma center

Concepts in Resuscitation

- Lethal triad
- Control of hemorrhage
- Choice of resuscitation fluid
- Goals of resuscitation
- Analgesia in the field

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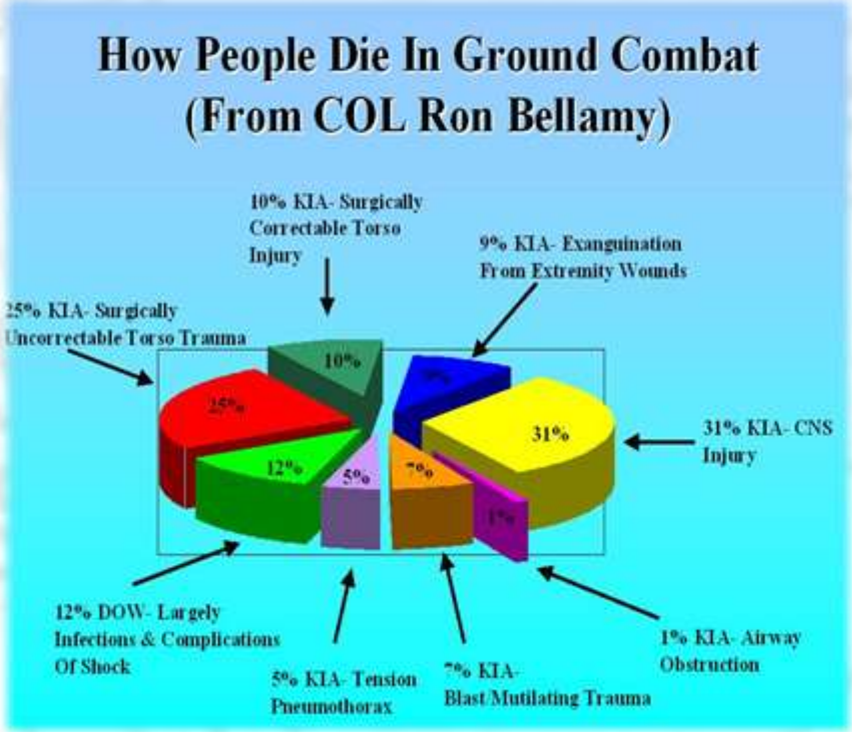
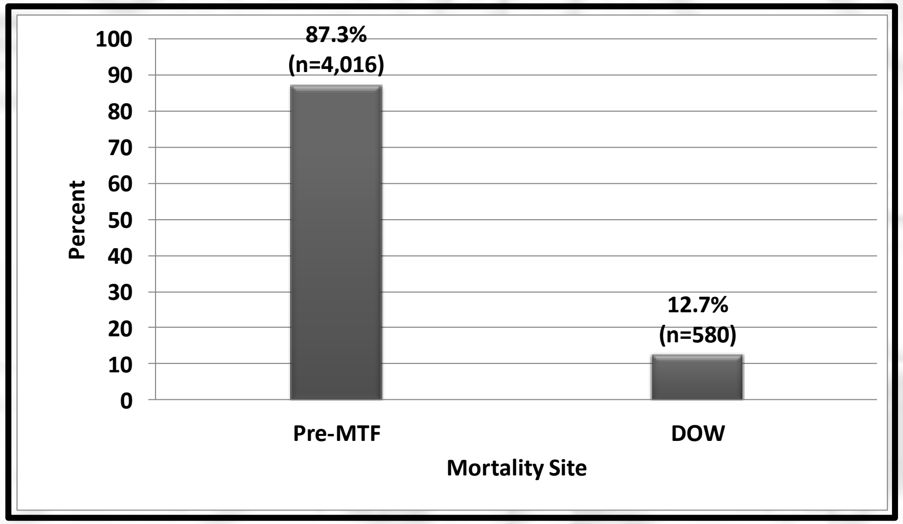
"Damage Control Resuscitation"

Where Do Battlefield Casualties Die?

Past = 88% Prehospital

Present = 87.3% Prehospital
(4016/4596)





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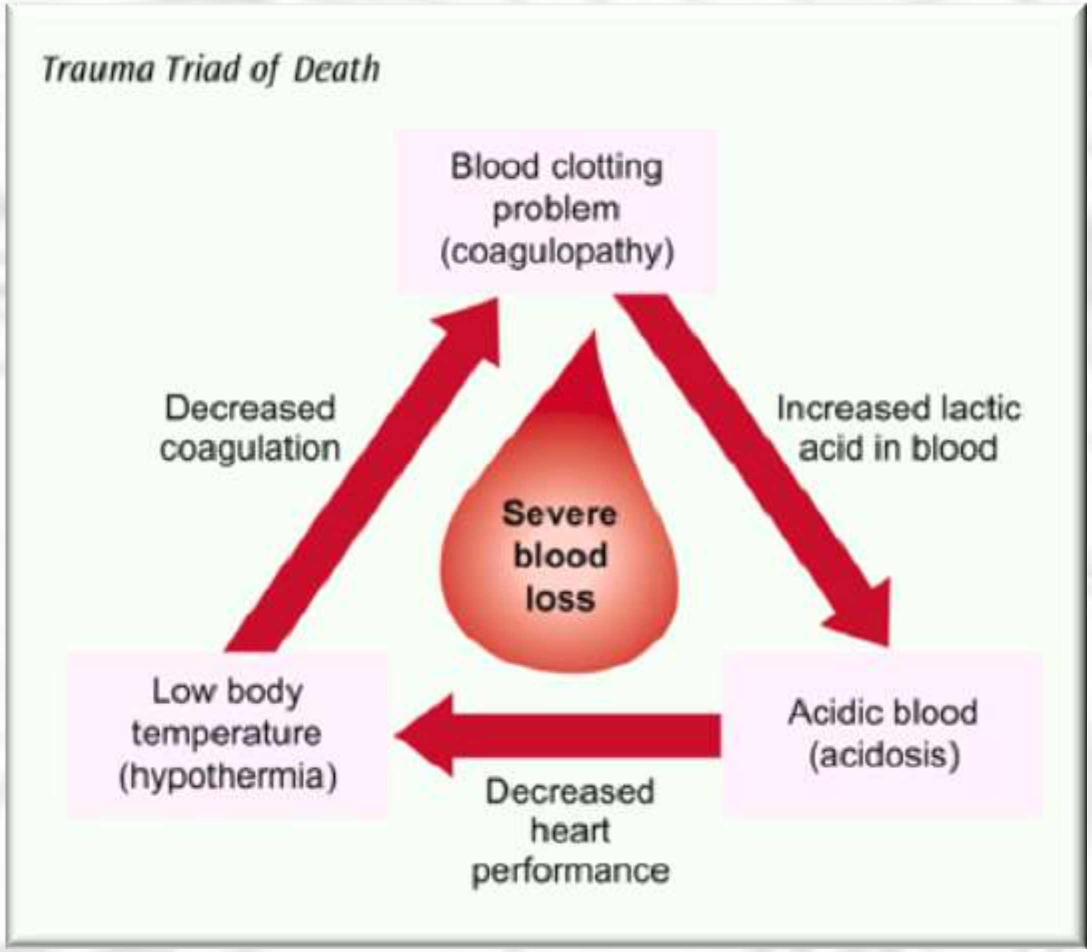
Slide courtesy of COL R. Kotwal

Lethal Triad

- Hypothermia
- Coagulopathy
- Acidosis



Lethal Triad: Hypothermia

- Hypothermia in the trauma patient contributes to worse coagulopathy, worse metabolic acidosis, and cardiac dysrhythmias and serious electrolyte disorders
- Cold exposure and body heat loss
  - Environmental
  - Resuscitation
  - Anaerobic metabolism is less exothermic than aerobic metabolism
- Core temperature <36°C clinically important
- Increases bleeding
  - Impeding platelet adhesion (due to decreased thromboxane production)
  - Dysregulating the coagulation factors and enzymes, and interfering with fibrinolysis
  - Hard to test

Lethal Triad: Coagulopathy

- Dilutional resuscitation- related coagulopathy
  - ≥ 40% of patients who received more than 2 l of intravenous fluids
  - ≥ 50% of those who received more than 3 l of fluids
  - ≥ 70% of patients who received more than 4 l of fluids
  - Massive transfusion protocols with inadequate plasma
- Non-dilutional acute traumatic coagulopathy
  - Early trauma-related coagulopathy that occurs before hemodilution of coagulation factors
  - Affects about one-third of major trauma patients
  - Thought to result mainly from direct activation of the protein c pathway by tissue injury and hypoperfusion

Lethal Triad: Acidosis

- Inadequate tissue perfusion > lactic acidosis through anaerobic metabolism.
- Crystalloid IV fluids – normal saline
- Decreased coagulation factors' activity with decreasing pH;
  - pH decrease from 7.4 to 7.0 reduces the activity of key clotting factors (VIIaXa/Va) by over 70-90%
- The degree of base deficit (i.e. acidosis) and the lactate levels on admission to the trauma may strongly correlate with worse patient mortality

Control of Hemorrhage

- Tourniquets in the field
- Hemostatic adjuncts
- Damage Control Resuscitation (DCR)


CRoC shown with carrier bag

Hemorrhage Control: Tourniquets

Tourniquets

- Get tourniquets on BEFORE onset of shock
  - Mortality is very high if casualties already in shock before tourniquet application
- If bleeding is not controlled and distal pulse not eliminated with first tourniquet – use a second one just proximal to first
  - Increasing the tourniquet WIDTH with a second tourniquet controls bleeding more effectively and reduces complications

Control of Hemorrhage: Hemostatic Adjuncts

- Hemostatic adjuncts
  - Tranexamic acid (tXa), an anti-fibrinolytic agent, was shown in a large international, multicenter, randomized, placebo-controlled trial to slightly decrease the risk of death from bleeding (n = 20,211; relative risk = 0.85; p = 0.0077)
  - “We recommend the use of tXa in massively bleeding trauma patients, and incorporating it in MTPs”
  - Evidence is lacking for the use of recombinant factor VIIa or prothrombin complex concentrates (PCCs) in the massively bleeding trauma patients are still lacking and there does not appear to be a clear benefit from their use


Antifibrinolytic drugs for acute traumatic injury (Review)

Roberts I, Shakur H, Ker K, Coats T, on behalf of the CRASH-2 Trial collaborators

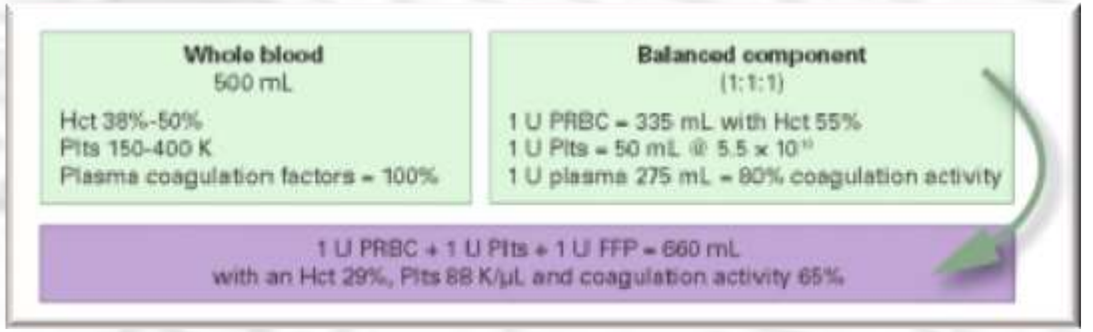
THE COCHRANE COLLABORATION®

WILEY

Control of Hemorrhage: Damage Control Resuscitation

- Concept evolved with the recognition of the nature of trauma related coagulopathy and with the recent combat trauma experiences in Iraq and Afghanistan
- The term "damage control" itself originated from World War II description of the US navy's strategy to salvage sinking ships
  - Avoided immediate definitive repair of the damaged vessel
  - Focused instead on preserving only what was needed to return the ship safely back into the port for eventual definitive repair
- Focus on targeted resuscitation to avoid lethal triad

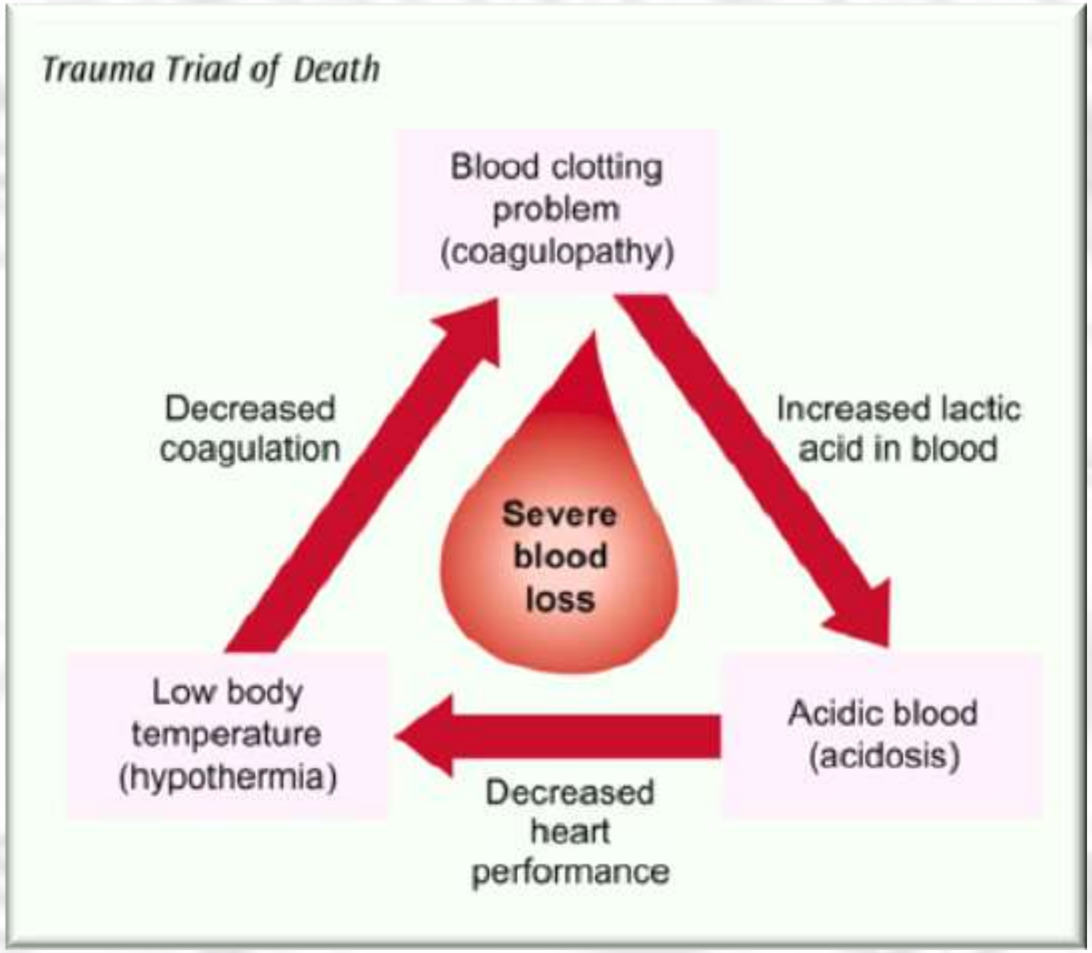
Choice of Resuscitation Fluid



- Crystalloids vs Colloids
  - Avoid aggressive volume resuscitation
  - Albumin vs. Crystalloid both OK
  - No evidence of benefit for hypertonIc salIne
  - Balanced crystalloids (LR, plasmalyte) better than NS
- Balanced resuscitation
  - Early administration of blood products in addition to pRBCs can help prevent trauma-related coagulopathy.
  - Target ratio of pRBCs, FFP, and platelets 1:1:1.
    - Retrospective JTS study, the survival of patients receiving FFPs in a 1:8 ratio compared to PrBcs was dramatically higher than those receiving FFPs in a 1:1.4 ratio (92.5% vs 37%, p < 0.001).

Goals of Resuscitation

- Limit crystalloid use
- Early balanced transfusion
- Permissive hypotension
- Damage control surgery
- Definitive care/surgery



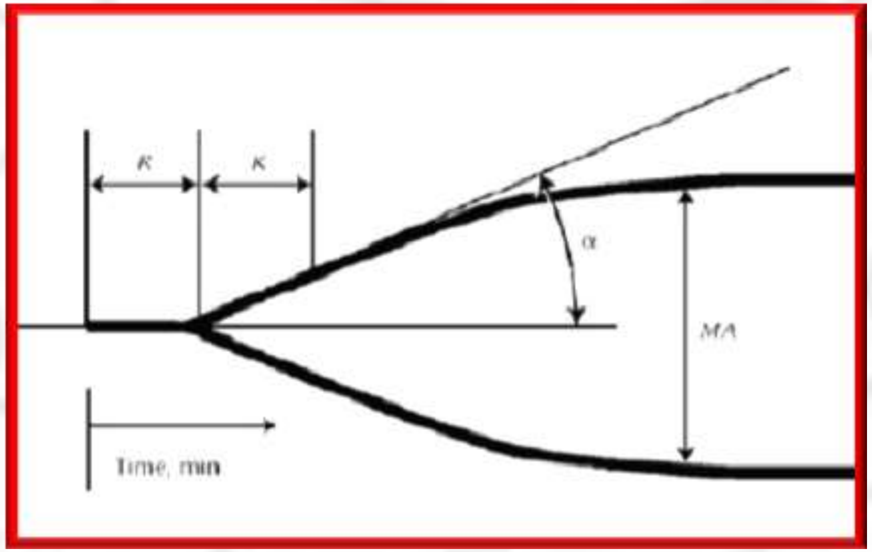
Goals of Resuscitation

- Permissive hypotension
  - Permissive hypotension is the strategic decision to delay the initiation of fluid resuscitation and limit the volume of resuscitation fluids/blood products administered to the bleeding trauma patient by targeting a lower than normal blood pressure, usually a systolic blood pressure of 80–90 mmHg or a mean arterial pressure (MAP) of 50 mmhg.
- Hemostatic endpoints
  - pRBcs should be given to target a hemoglobin >7 g/dl
  - FFPs to target an interna- tional normalized ratio (Inr) <2
  - Platelets to target a count >50,000
  - Cryoprecipitate to target a fibrinogen level >100 mg/dl. the use of thromboelastog- raphy-based protocols that assess the different aspects of clot formation and stability in a real time point-of-care output graph has been incorporated into some MtPs or suggested as an alternative to MtPs. although promising, this methodology is lacking the multi-institutional rigorous data sup- porting its use (68–71).

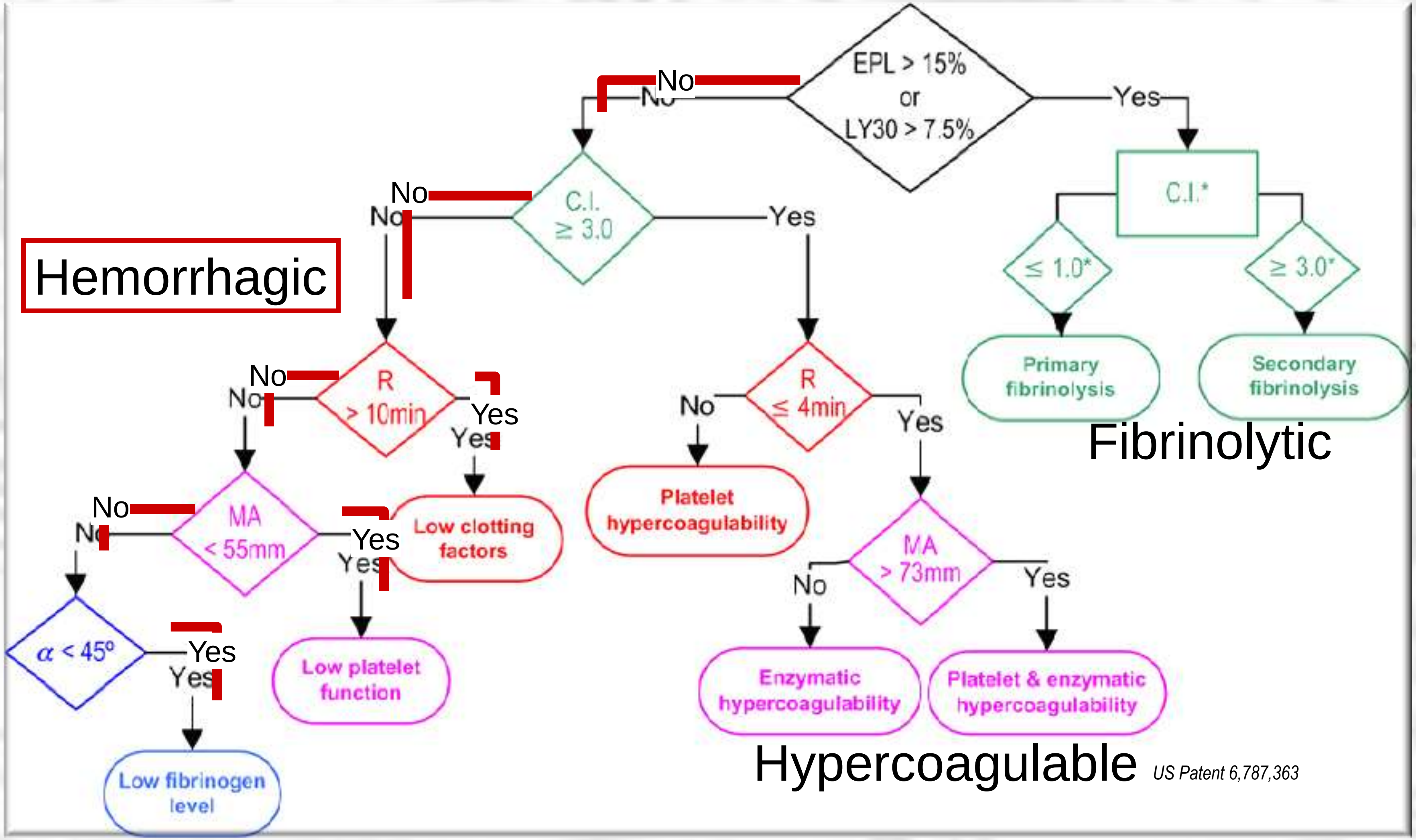
Hemostatic Endpoints: Thromboelastogram

- A whole blood assay that attempts to quantitate the viscoelastic properties of clot formation and breakdown.
- The blood sample is transferred to an oscillating cup of the TEG device. A pin, suspended within the blood sample, monitors the motion of the blood within the cup.
- As the blood sample clots, the torque of the rotating cup is transmitted to the pin and the mechanical change is translated to an electrical signal that is monitored and recorded as TEG tracings by a computer.

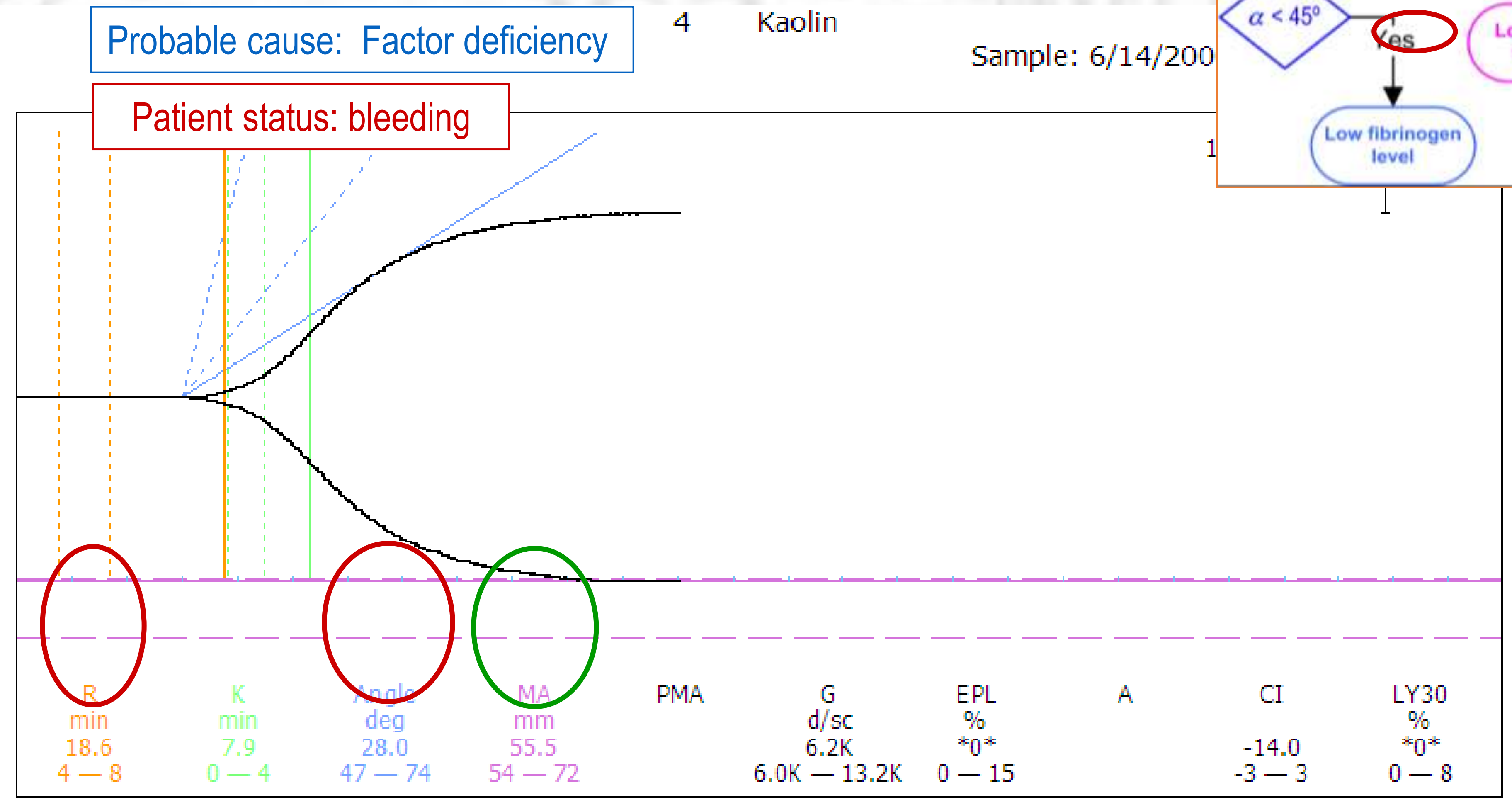
- A <u>plasma transfusion trigger</u> can be built from a critically prolonged R-time.
- A <u>cryoprecipitate transfusion trigger</u> can be built from a critically low angle.
- A <u>platelet transfusion trigger</u> can be built from a critically low maximum amplitude result

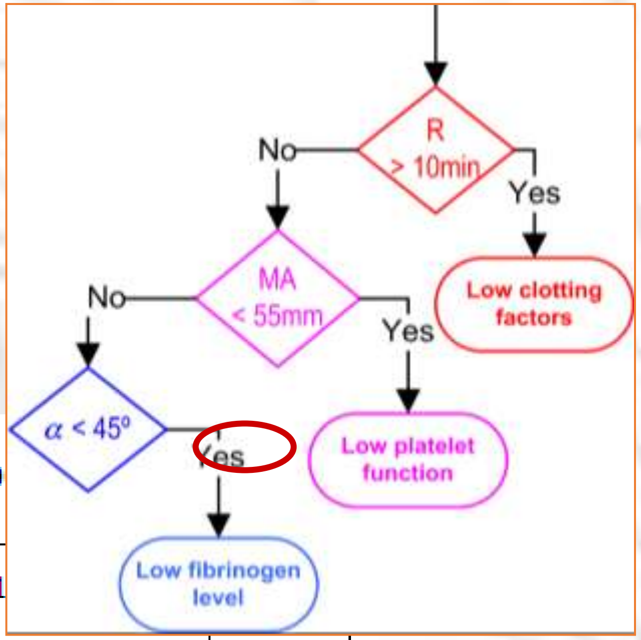


TEG:Decision Tree



Enzymatic Pathway Abnormality: Coagulation Factor Deficiency





Analgesia in the Field

- Narcotics
- Ketamine
- PTSD
- HD effects

ORIGINAL ARTICLE

Morphine Use after Combat Injury in Iraq and Post-Traumatic Stress Disorder

Troy Lisa Holbrook, Ph.D., Michael R. Galarneau, M.S., Judy L. Dye, M.S., R.N., A.N.P., Kimberly Quinn, B.S.N., and Amber L. Dougherty, M.P.H.

ABSTRACT

BACKGROUND

Post-traumatic stress disorder (PTSD) is a common adverse mental health outcome among seriously injured civilians and military personnel who are survivors of trauma. Pharmacotherapy in the aftermath of serious physical injury or exposure to traumatic events may be effective for the secondary prevention of PTSD.

METHODS

We identified 696 injured U.S. military personnel without serious traumatic brain injury from the Navy–Marine Corps Combat Trauma Registry Expeditionary Medical Encounter Database. Complete data on medications administered were available for all personnel selected. The diagnosis of PTSD was obtained from the Career History Archival Medical and Personnel System and verified in a review of medical records.

RESULTS

Among the 696 patients studied, 243 received a diagnosis of PTSD and 453 did not. The use of morphine during early resuscitation and trauma care was significantly associated with a lower risk of PTSD after injury. Among the patients in whom PTSD developed, 61% received morphine; among those in whom PTSD did not develop, 76% received morphine (odds ratio, 0.47; $P<0.001$). This association remained significant after adjustment for injury severity, age, mechanism of injury, status with respect to amputation, and selected injury-related clinical factors.

CONCLUSIONS

Our findings suggest that the use of morphine during trauma care may reduce the risk of subsequent development of PTSD after serious injury.

From the Naval Health Research Center (T.L.H., M.R.G, J.L.D., K.Q., A.L.D.) and EPI-SOAR Consulting (T.L.H.) — both in San Diego, CA. Address reprint requests to Dr. Holbrook at the Naval Health Research Center, 140 Sylvester Rd., San Diego, CA 92106-3521, or at troy@epi-soar.com.

N Engl J Med 2010;362:110-7.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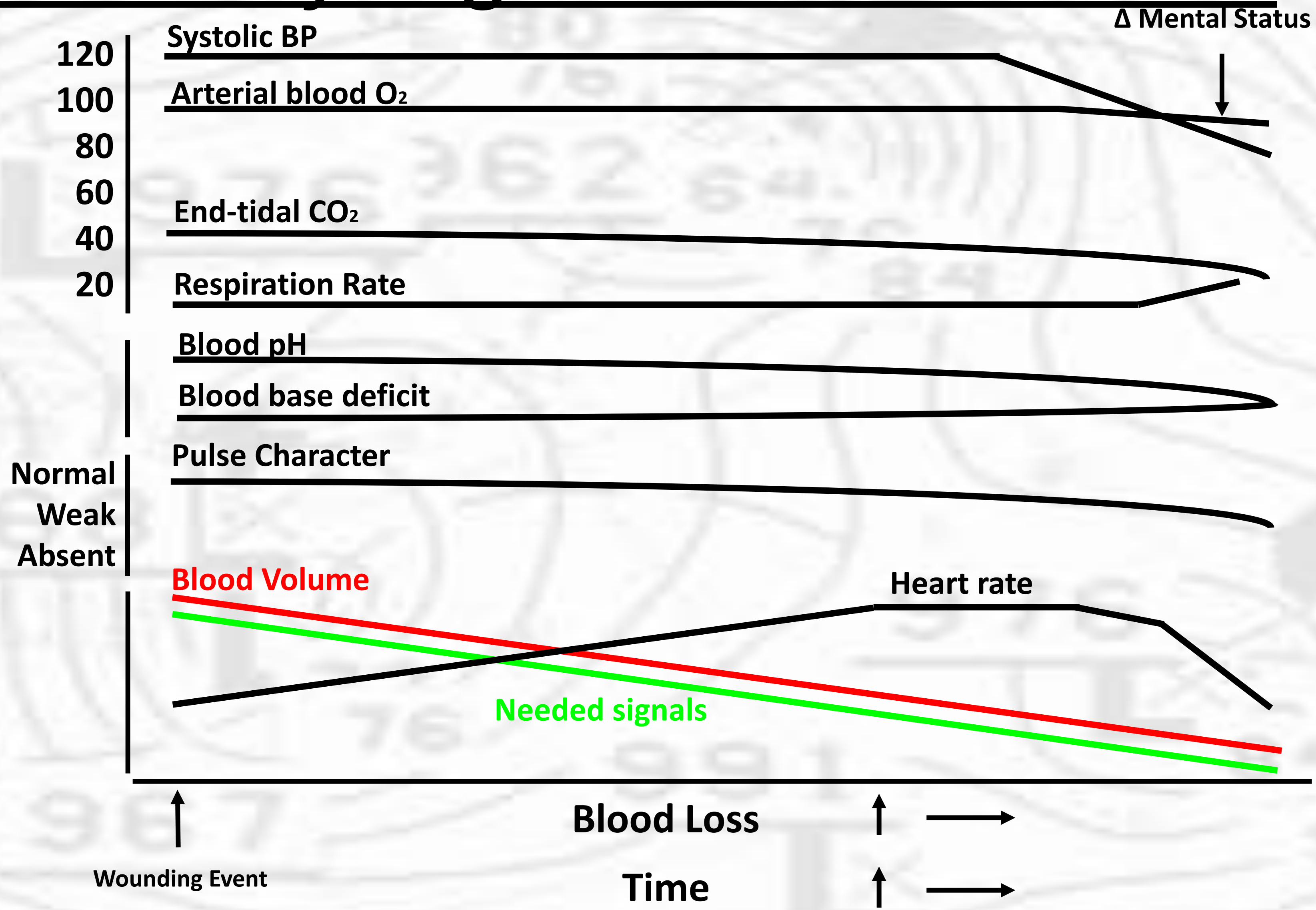
Hemodynamic Signals

- Systolic blood pressure
- Heart rate
- Respiratory rate
- Mental status
- Weak pulse
- Oxygen saturation
- End-tidal carbon dioxide (etCO_2)
- Base excess
- Blood pH

Need for Early Signals of Blood Loss



[4]Convertino et al. *J Trauma* 2011;**71**:S25-S32.

"Shock is defined by inadequate substrate for aerobic cellular respiration. Unfortunately, we can't measure this directly."

Hemodynamic Signals

- PA catheter/thermodilution
- Lactate/lactate clearance
- Ultrasound
- Pulse plethysmography
- Near infra-red reflectance spectroscopy
- Bioreactance/Bioimpedance
- Machine learning algorithms

FloTrac™ Sensor & Vigileo Monitor

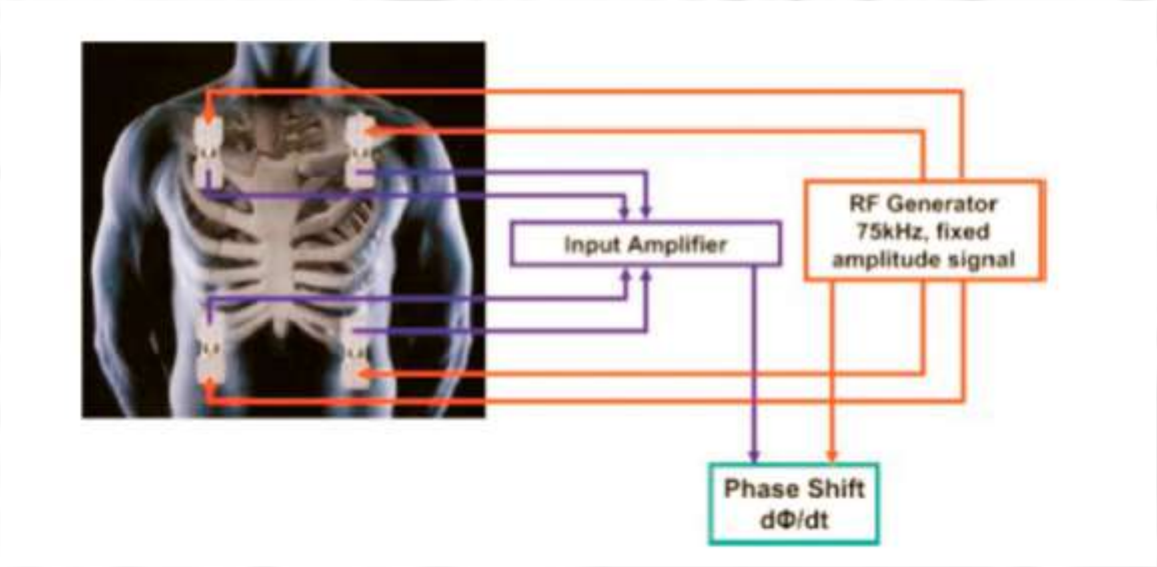
FloTrac™ Sensor

Vigileo™ Sensor

PreSep Oximetry Catheter

- Adapted from: http://ht.edwards.com/scin/edwards/sitecollectionimages/edwards/ar08052.pdf.

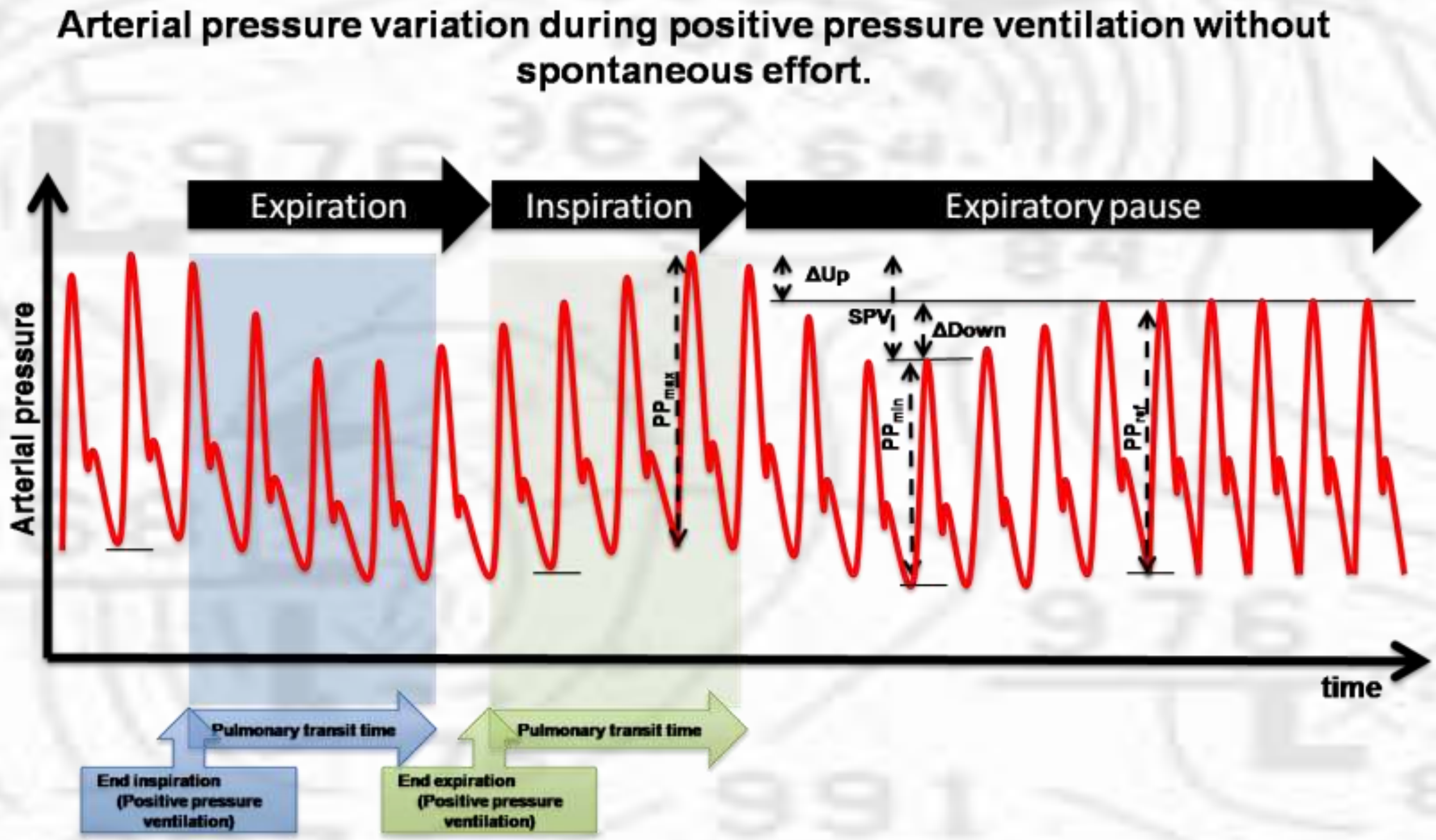
NICOM® System



Validation of the NICOM® System

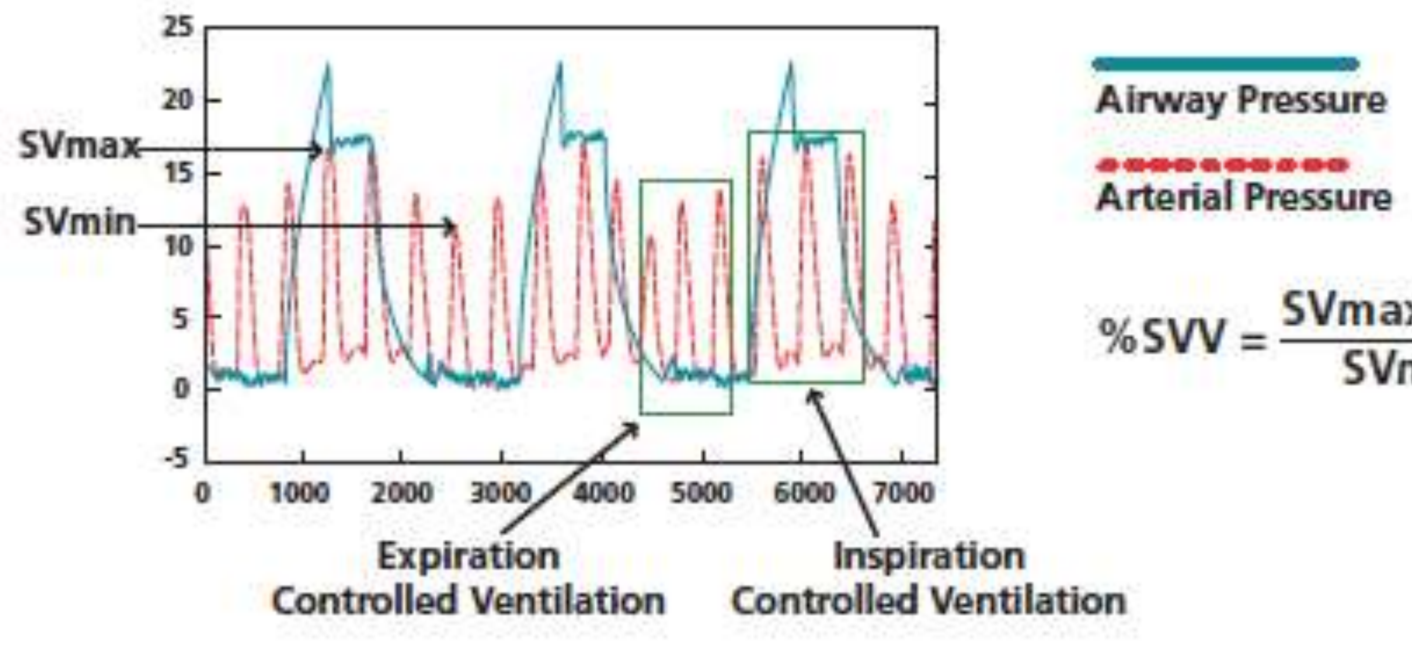
- The CO measured by bioreactance® is highly correlated with that measured by thermodilution and pulse contour analysis
- Rich *et al.* performed right heart caths in 24 patients with pulmonary HTN
  - NICOM® was more precise than thermodilution (3.6% vs 9.9%, $p < 0.001$)
- Benomar *et al*. showed that the NICOM® system could predict fluid responsiveness accurately from changes in CO during PLR
- Marik *et al.* found nearly 100% concordance between fluid responsiveness as determined by carotid flow and the NICOM® system

Pulse Pressure Variation



- Adapted from: http://www.genevahemodynamic.com/formation/pulse-pressure-variation-ppv/.

Stroke Volume Variation



$$\%SVV = \frac{SVmax - SVmin}{SVmean}$$

- Adapted from: http://ht.edwards.com/scin/edwards/sitecollectionimages/edwards/ar08052.pdf.

Interpretation of PPV/SVV

- A PPV/SVV of greater than 12 to 13% has been reported to be predictive of volume responsiveness

Factors that Affect PPV & SVV

- Volume status
  - PPV amplitude increases in hypovolemic states
- Arterial compliance
  - PPV may vary significantly when arterial compliance decreases
- Heart rate
  - PPV may increase with decreasing HR
- Heart rhythm
  - PPV may vary independently with irregular rhythms
- RV failure
  - PPV amplitude may increase due to as RV afterload increases

Factors that Affect PPV & SVV

- Spontaneous respiration
  - PPV is difficult to interpret as pleural & alveolar pressure are influenced by spontaneous effort.
- Tidal volume
  - VT must be large enough (8-10mL/kg) to induce significant cardiovascular perturbation in order to induce a PPV.
- Chest compliance
  - Decreased compliance will decrease the change in transpulmonary pressure, resulting in decreased PPV.

Ultrasonographic Techniques

- Two primary indices:
  - Positive pressure ventilation-induced changes in vena-caval diameter
    - Fluid responsiveness can be predicted by measuring IVC distensibility index or SVC collapsibility.
    - SVC collapsibility may be more reliable than IVC distensibility.[46]
  - Dynamic changes in aortic flow velocity/stroke volume
    - Changes in aortic blood flow reflect changes in LV stroke volume.
    - Feissel *et al.* demonstrated that the respiratory changes in aortic flow velocity predict fluid responsiveness in mechanically ventilated patients.

Inferior Vena-Cava (IVC) Diameter

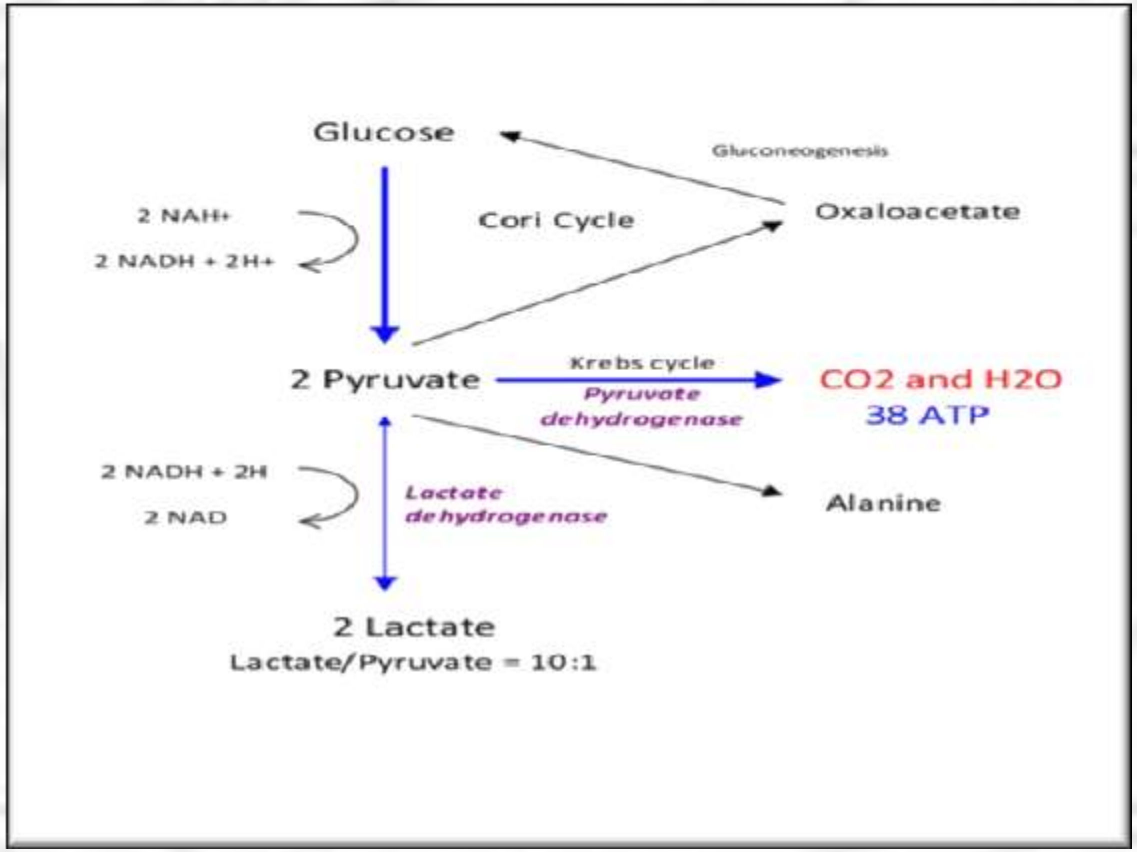
- Transthoracic echocardiography can be used to measure the IVC diameter as it enters the RA.

- A collapsed IVC is assumed to represent hypovolemia.

- A distended IVC is assumed to represent an elevated RAP (i.e. volume overload).

* Adapted from: https://acep.org/Content.aspx?id=80791.

Lactate



Prehospital Serum Lactate as a Predictor of Outcomes in Trauma Patients: A Retrospective Observational Study

Francis Guyette, MD, Brian Suffoletto, MD, Jose-Luis Castillo, MD, Jorge Quintero, MD, Clifton Callaway, MD, PhD, and Juan-Carlos Puyana, MD

Background: Lactate is associated with morbidity and mortality; however, the value of prehospital lactate (pLA) is unknown. Our objective was to determine whether pLA improves identification of mortality and morbidity independent of vital signs.

Methods: We measured pLA in 1,168 patients transported by rotorcraft to a Level I trauma center over 18 months. The primary outcome was in-hospital mortality; secondary outcomes were emergent surgery and multiple organ dysfunction syndrome (MODS). Covariates include age, sex, prehospital vital signs, and mental status. We created multivariable logistic regression models and tested them for interaction terms and goodness of fit. Cutoff values were established for reporting operating characteristics using shock (defined as shock index >0.8, heart rate >110, and systolic blood pressure <100), tachypnea (RR ≥30), and altered sensorium (Glasgow Coma Scale score <15).

Results: In-hospital mortality was 5.6%, 7.4% required surgery and 5.7% developed MODS. Median lactate was 2.4 mmol/L. Lactate was associated with mortality (odds ratio [OR], 1.23; $p < 0.0001$), surgery (OR, 1.13; $p < 0.001$), and MODS (OR, 1.14; $p < 0.0001$). Inclusion of pLA into a logistic model significantly improved the area under the receiver operator curves from 0.85 to 0.89 for death ($p < 0.001$), 0.68 to 0.71 for surgery ($p = 0.02$), and 0.78 to 0.81 for MODS ($p = 0.002$). When a threshold lactate value of >2 mmol/L was added to a predictive model of shock, respiratory distress, or altered sensorium, it improved sensitivity from 88% to 97% for death, 64% to 86% for surgery, and 94% to 99% for MODS.

Conclusion: The pLA measurements improve prediction of mortality, surgery, and MODS. Lactate may improve the identification of patients who require monitoring, resources, and resuscitation.

Key Words: Prehospital, Lactate, Trauma.

(*J Trauma.* 2011;70: 782–786)

Traditional trauma triage uses physiologic variables, including vital signs, which often do not predict need for surgical resources or outcomes.[1,2] Blood pressure and heart rate may change later in shock when compensatory mechanisms fail in hemorrhagic shock. Compensated shock is not easily recognizable by prehospital providers.[2–6] Delayed identification of hypoperfusion may lead to triage of some patients away from specialized trauma centers and to inadequate or delayed resuscitation, which is strongly associated with increase in infection, multiple organ dysfunction (MOD), and mortality.[3,7,8]

Serum lactate is a byproduct of anaerobic metabolism, is a circulating biomarker of organ oxygen supply/demand mismatch, and is directly related to mortality in patients with sepsis, myocardial infarction, and trauma.[9,10] Trends in serum lactate levels can also monitor the effectiveness of resuscitation, even in patients with normal vital signs.[10,11] Currently, technological advances have led to the production of hand-held, point of care (POC) lactate analyzers that produce fast, reliable, and valid measurements.[12] Use of POC lactate is now feasible in the out-of-hospital setting, but the additional value of prehospital lactate (pLA) for predicting in-hospital outcomes is unknown.

Importance

If increased pLA identifies patients who go on to have clinically significant outcomes after trauma, then it may assist prehospital triage by directing transport to regionalized trauma centers for more aggressive early resuscitation. If pLA improves identification of outcomes independent of clinically available prehospital variables, then it could be especially useful to risk stratify patients who do not show any initial signs of physiologic perturbation. Ultimately, protocols designed to intervene with earlier resuscitation based on pLA may have a protective effect in trauma-associated mortality.

Goals of This Investigation

This study sought to determine the additional value of pLA as a biomarker for mortality and morbidity in patients presenting to prehospital providers with acute trauma. We investigated the odds ratios of in-hospital outcomes in patients with increased pLA compared with and adjusting for traditional covariates using multivariable logistic regression models. We then explored the improved sensitivity of identifying outcomes when pLA above a threshold of 2 mmol/L was added to other traditional prehospital markers of shock, respiratory distress, and altered sensorium.

PATIENTS AND METHODS

We conducted an observational cohort study of consecutive trauma patients transported to a single hospital by an air

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Presented as a poster at the 68th Annual Meeting of the American Association for the Surgery of Trauma, October 1–3, 2009, Pittsburgh, Pennsylvania.
Supported, in part, by the Fogarty International Center NIH Grant No. 1 D43 TW007560-01.
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DOI: 10.1097/TA.0b013e318210f5c9

Machine Learning Algorithm: CRI

- Utilizes pulse waveform from oximeter
- Analyzes waveform characteristics to assess features predict shock

Figure 4. CipherOx is a small bluetooth-enabled pulse oximeter with a wrist worn CRI display, mini-USB port for battery charging and data download.

Running on empty? The compensatory reserve index

Steven L. Moulton, MD, Jane Mulligan, PhD, Greg Z. Grudic, PhD,
and **Victor A. Convertino, PhD,** *San Antonio, Texas*

BACKGROUND: Hemorrhage is a leading cause of traumatic death. We hypothesized that state-of-the-art feature extraction and machine learning techniques could be used to discover, detect, and continuously trend beat-to-beat changes in arterial pulse waveforms associated with the progression to hemodynamic decompensation.

METHODS: We exposed 184 healthy humans to progressive central hypovolemia using lower-body negative pressure to the point of hemodynamic decompensation (systolic blood pressure > 80 mm Hg with or without bradycardia). Initial models were developed using continuous noninvasive blood pressure waveform data. The resulting algorithm calculates a compensatory reserve index (CRI), where 1 represents supine normovolemia and 0 represents the circulatory volume at which hemodynamic decompensation occurs (i.e., "running on empty"). Values between 1 and 0 indicate the proportion of reserve remaining before hemodynamic decompensation—much like the fuel gauge of a car indicates the amount of fuel remaining in the tank. A CRI estimate is produced after the first 30 heart beats, followed by a new CRI estimate after each subsequent beat.

RESULTS: The CRI model with a 30-beat window has an absolute difference between actual and expected time to decompensation of 0.1, with a SD of 0.09. The model distinguishes individuals with low tolerance to reduced central blood volume (i.e., those most likely to develop early shock) from those with high tolerance and are able to estimate how near or far an individual may be from hemodynamic decompensation.

CONCLUSION: Machine modeling can quickly and accurately detect and trend central blood volume reduction in real time during the compensatory phase of hemorrhage as well as estimate when an individual is "running on empty" and will decompensate (CRI, 0), well in advance of meaningful changes in traditional vital signs. (*J Trauma Acute Care Surg.* 2013;75: 1053–1059. Copyright © 2013 by Lippincott Williams & Wilkins)

KEY WORDS: Hypotension; lower-body negative pressure; pulse oximetry.

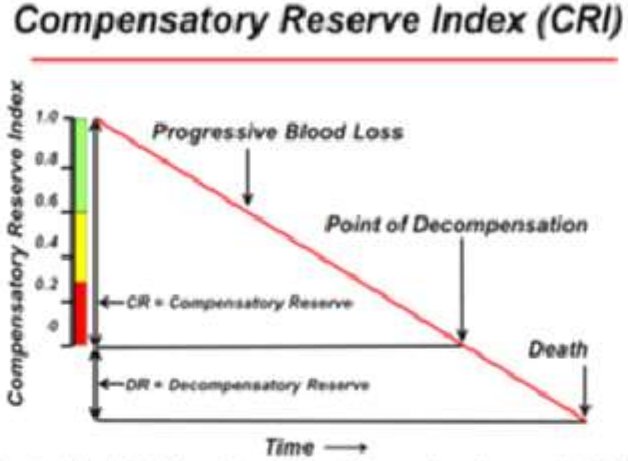


Figure 1. The CRI is indicative of the individual-specific proportion of intravascular volume remaining before the onset of cardiovascular collapse. The *red line* shows a hypothetical decline in CRI over time in the setting of blood loss caused by hemorrhage or plasma leakage. A calculated CRI of 1 represents normovolemia, whereas a calculated CRI of 0 represents the point of hemodynamic decompensation.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision unless so designated by other documentation.

Address for reprints: Steven L. Moulton, MD, Pediatric Surgery, B-323, Children's Hospital Colorado, 13123 E 16th Ave, Aurora, CO 80045; email: steven.moulton@childrenscolorado.org.

DOI: 10.1097/TA.0b013e3182aa811a

respond to real-world situations, where previous knowledge and experience may be unknown or uncertain. A robot uses sensors and interpretive algorithms to explore its environment and make decisions about the actions it should perform to reach its intended goal. Clinicians are responsible for interpreting growing volumes of clinical data to identify underlying

Monitoring in Shock Trial (MIST)

- Do available noninvasive monitoring modalities provide accurate and reliable information in states of altered tissue perfusion to guide resuscitation of patients in shock allowing for a “real time” tool to guide resuscitation?

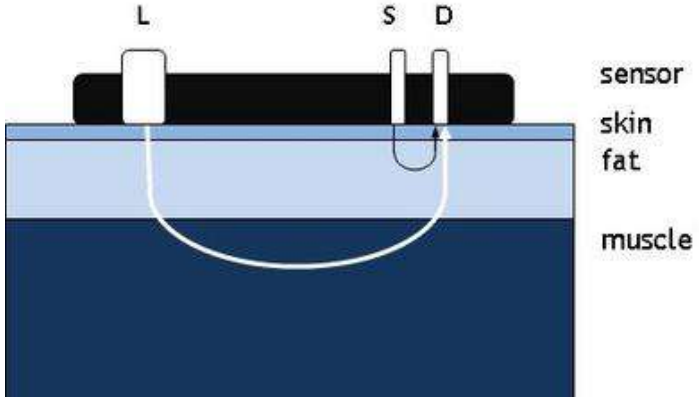
Study Process

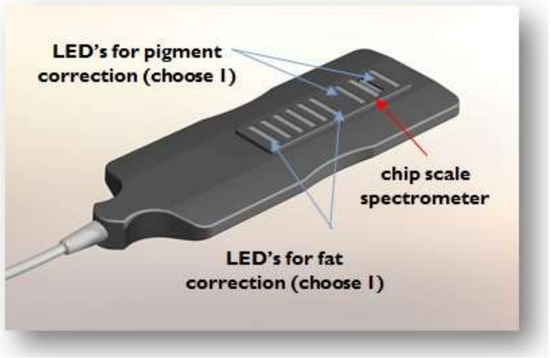
Near Infra-red Reflectance Spectroscopy (NIRS)

- Muscle oxygen saturation (S_mO_2) has been shown to decrease in shock in a human model of hemorrhage
  - Lower body negative pressure[5]
  - Swine hemorrhage study[6]
  - Human study of dengue hemorrhagic fever patients[7]
- Decreased muscle pH (pH_m) has been shown to predict liver injury in swine hemorrhagic shock[8]
- An objective of this study is to determine whether NIRS measurement of deep tissue oxygen saturation (S_mO_2) and deep tissue pH (pH_m) identifies shock in critically ill adults

Near Infra-red Reflectance Spectroscopy (NIRS)

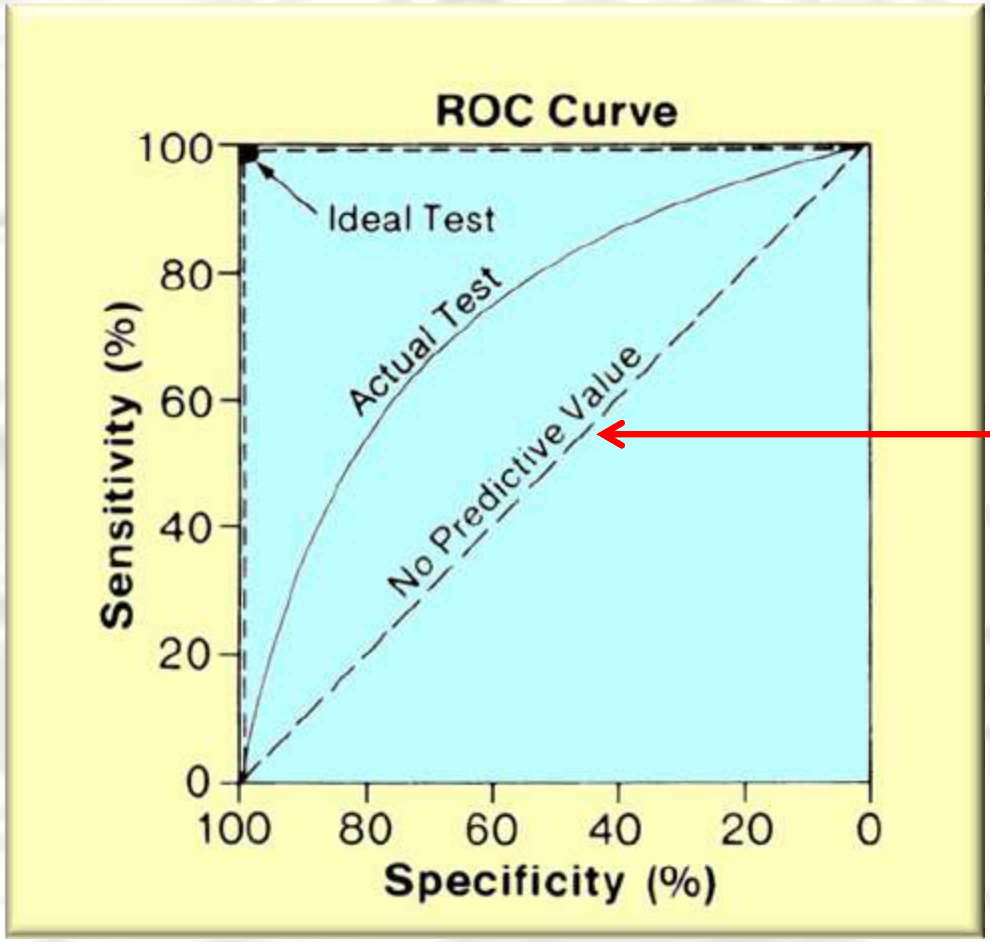
- Careguide™ 1100 NIRS sensor measurement of tissue oximetry and pH





Images from: http://www.reflectancemedical.com/Technology.html

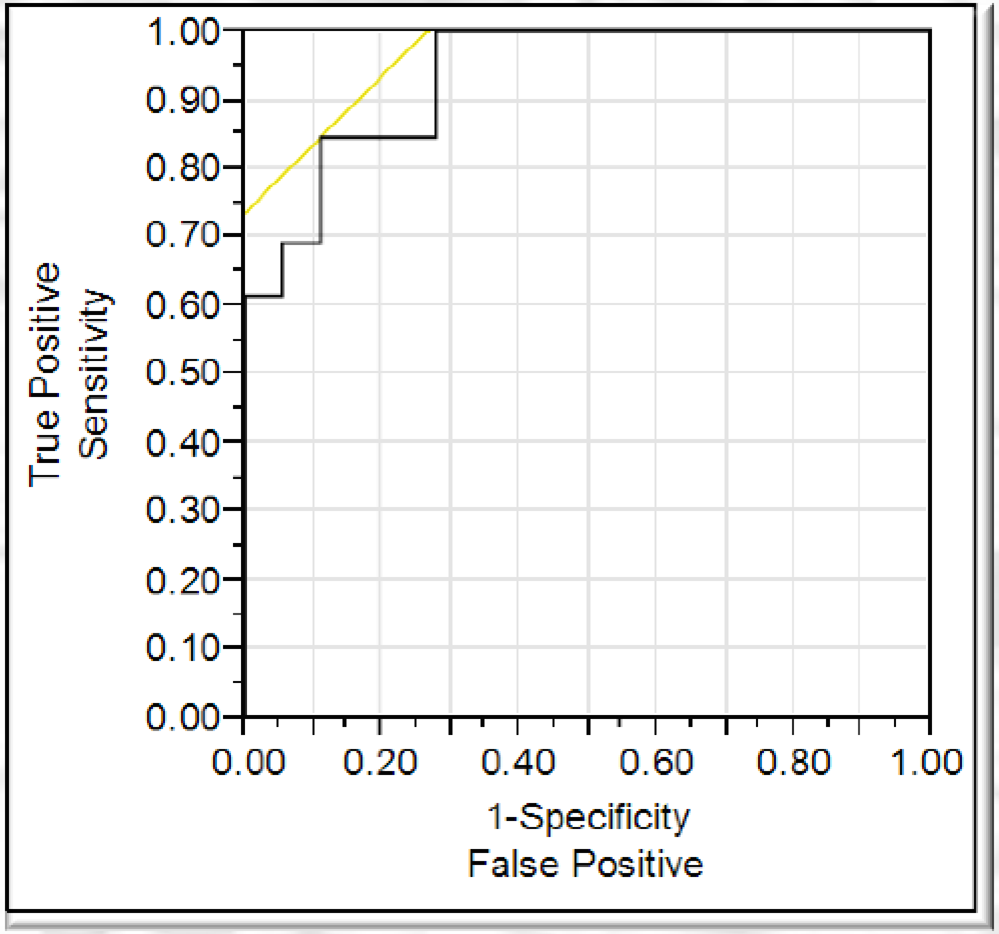
Receiver Operating Characteristic Curve



Coin toss

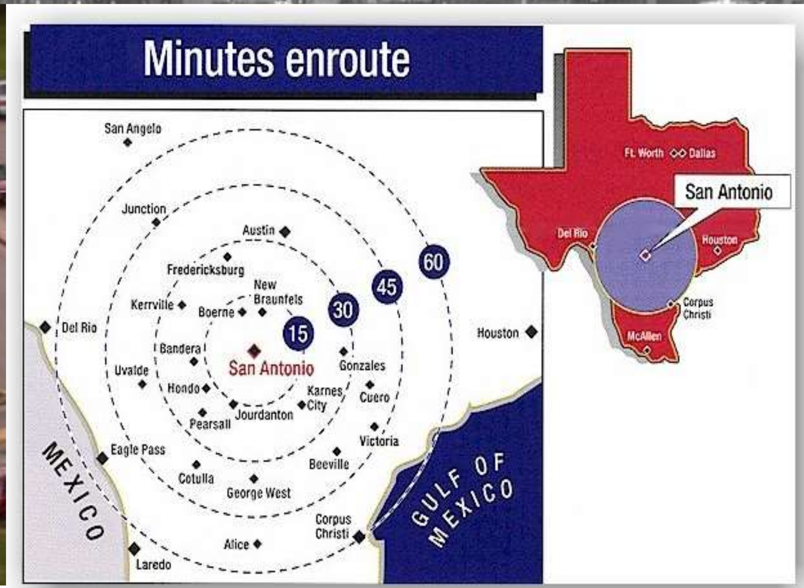
ROC: Lactate + S_mO_2 + pH_m

- Lactate ($p=0.019$)
- S_mO_2 ($p=0.039$)
- pH_m ($p=0.56$)
- AUC = 0.94
- Sensitivity 85%
- Specificity 89%



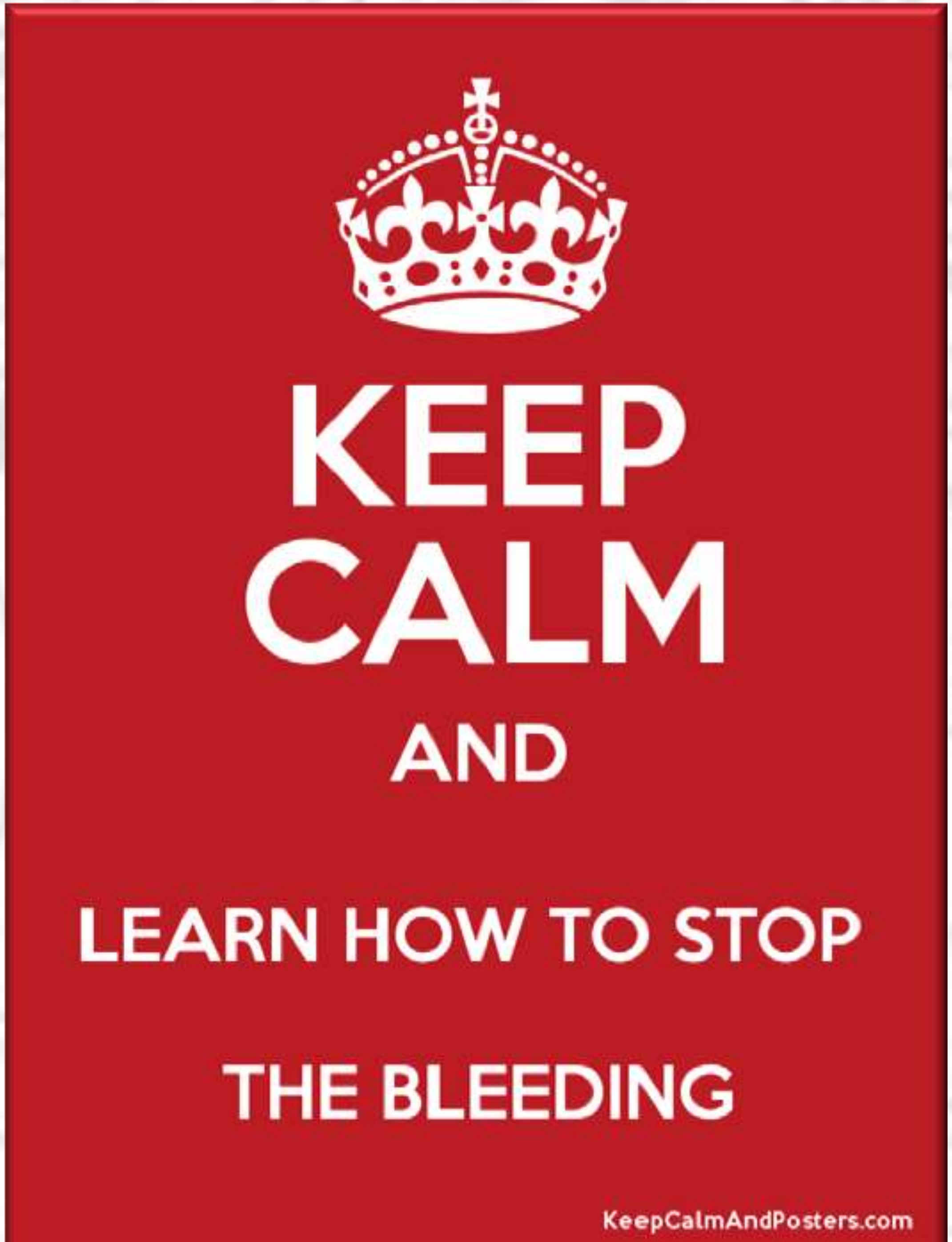
MIST Phase II: DCR Enroute Care Study

- Inbound major trauma to Level I within regional trauma system
- Standard physiologic monitoring
- POC Lactate and ROTEM
- Do monitoring waveforms allow CRI to predict shock in the enroute care environment?



Shock: Take Home Points

- Hemorrhage control is key
- Avoid the Lethal Triad
- Damage control resuscitation
- Identification of compensated shock is important but difficult
- The ideal signal for inadequate tissue perfusion has yet to be identified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