Controversies in Postinjury Hemostasis

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Controversies in Postinjury Hemostasis
The ponderous literature on the subject of hemostasis could perhaps be considered a classical example of the infinite ability of the human mind for abstract speculation. For several years, the number of working theories of the hemostatic mechanism greatly exceeded and not always respected the confirmed experimental facts. In recent years, however, the revived interest in this field has led to an accumulation of new findings which has been almost too rapid for their orderly incorporation into a logical working pattern. As a result, we have rapidly gone from a state of “orderly ignorance” to one of “confused enlightenment.”
Postinjury Hemostasis: Controversies

1. Acute Coagulopathy of Trauma
   Endothelial TM + Thrombin = Activated PC
   Tissue Factor / Thrombin = DIC + Fibrinolysis

2. Pre-emptive Blood Components
   ? PLT : FFP : RBC

3. Goal Directed Therapy
   ? Coagulation Assessment
<table>
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<th>Authors</th>
<th>Journal</th>
<th>Year</th>
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Fatal Hepatic Hemorrhage After Trauma

Steven C. Elerding MD, Ernest E. Moore, MD and G. E. Aragon MD

The characteristic picture was uncontrollable hepatic hemorrhage and diffuse bleeding from all exposed surfaces. Only 11 patients (39%) received fresh frozen plasma. In only eight patients (29%) were arterial blood gases measured; the average pH was 7.11. Core temperature was obtained in only six patients (21 percent); the average temperature was 32.2°C.


ACIDOSIS-INDUCED COAGULOPATHY

Ernest L. Dunn, MD, Ernest E. Moore, MD, Diane J. Breslich, MD, and William B. Galloway, MD

Thirteen adult mongrel dogs (15-20 kg) were anesthetized with pentobarbital (25 mg/kg) and placed in a volume respirator. Thermodilution cardiac outputs, pulmonary artery pressures, and systemic arterial pressures were recorded hourly. Metabolic acidosis was induced by slowly infusing sterile 0.15N hydrochloric acid into the inferior vena cava over 4 hours.


Hypothermia-Induced Coagulopathy

David Bar-Or, MD, Ernest E Moore, MD, John A Marx, MD, and Jim T Good, MD

At lower body temperatures a bleeding diathesis is observed. This phenomenon was studied in 8 adult mongrel dog who were anesthetized and mechanically ventilated. Hypothermia was induced by surface cooling (submersion in ice water) and coagulation studies were performed at 37, 34, 32, 30 and 28°C.
"THE BLOODY VICIOUS CYCLE"

Major Abdominal Vascular Trauma

Hemorrhage

Coagulopathy

Hypothermia

Acidosis

Cellular Shock

AAST 1981
J. Trauma 1982
Major Abdominal Vascular Trauma—A Unified Approach

JEFFRY L. KASHUK, M.D., ERNEST E. MOORE, M.D., J. SCOTT MILLIKAN, M.D., AND JOHN B. MOORE, M.D.

Although coagulation studies were often poorly documented, indirect evidence of inadequate factor replacement was obtained by calculating the ratio of bank blood to unit of fresh frozen plasma (FFP) given. A consistent deviation from the commonly accepted ratio of 4–5:1 was evident, increasing to 8:1 in nonsurvivors and 9:1 in those where an overt coagulopathy was documented.

Factor replacement is certainly involved. We believe fresh frozen plasma should be administered with the first four units of bank blood in the hypotensive patient, as well as...
Staged Laparotomy: Global Objective

“Abort laparotomy ... establish intra-abdominal pack tamponade ... complete the surgical procedure once coagulation has returned to an acceptable level.”

H H Stone et al
Ann Surg 1983
Hypothermia, Acidosis, and Coagulopathy

Staged Laparotomy for the Hypothermia, Acidosis, and Coagulopathy Syndrome

MODEL: Indication for Damage Control

- pH < 7.1
- Temp < 34 °C
- ISS > 25
- SBP < 70 mm Hg

Tissue Injury and Cellular Shock
Coagulation Factor Deficiency: ? FFP Transfusion

- Pre-emptive FFP: RBC = 1:4  
  DGH ...  
  J Trauma 1982

- Canine hemorrhage shock  
  Lucas, et al  
  Ann Surg 1985  
  (no benefit of presumptive FFP)

- Pre-emptive FFP: RBC = 1:5  
  Wilson, et al  
  J Trauma 1987

- FFP after > 10 RBC  
  Lucas, et al  
  J Trauma 1989

- Pre-emptive FFP: RBC = 1:1  
  DGH ...  
  Ann Surg 2001
Acute Coagulopathy of Trauma

San Francisco General
208 Trauma Activation

Sampling < 10 min

BD > 6mEq/L = 27% ACS

Brohi, Cohen, et al
Ann Surg 2007
Life-Threatening Trauma

Blood Loss

Progressive Systemic Coagulopathy

Core Hypothermia

Metabolic Acidosis

Acute Endogenous Coagulopathy

Clotting Factor Deficiencies

Massive RBC Transfusion

Tissue Injury

Cellular Shock

Iatrogenic Factors

Pre-existing Diseases

Postinjury Coagulopathy

Bloody Vicious Cycle
Military Strategy = Replace Lost Blood

Component Therapy:
1U PRBC + 6U PLT + 1U FFP + 10 pk Cryo

- Hct 29%
- Plt 87K
- Coag Factor Activity 65%
- 750 mg Fibrinogen

UNCLASS ALARACT

SUBJECT: OPTIMAL RESUSCITATION OF SEVERELY INJURED SOLDIERS

1. COMBAT RESUSCITATION DATA ANALYZED BY THE US ARMY INSTITUTE OF SURGICAL RESEARCH (USAISR) DEMONSTRATE THAT CASUALTIES WHO RECEIVE MORE THAN 10 UNITS OF PACKED RED BLOOD CELLS (PRBCS) IN A 24-HOUR PERIOD (MASSIVE TRANSFUSION) HAVE A PROFOUND SURVIVAL BENEFIT WHEN THE PLASMA (FFP) TO PRBC TRANSFUSION RATIO IS 1:1. CASUALTIES WHO RECEIVE LESS FFP (1 UNIT FFP TO 4 UNITS PRBCS, OR LESS) HAVE AN OVERALL MORTALITY OF 65%, WHILE THOSE WHO RECEIVE A 1:1 RATIO HAVE AN OVERALL MORTALITY OF 20% (P< 0.001).

2. SEVERELY INJURED CASUALTIES SHOULD HAVE THE 1:1 RATIO INITIATED AS EARLY AFTER INJURY AS POSSIBLE. TRANSFUSIONS MUST BE ACCOMPLISHED ACCORDING TO GUIDELINES ESTABLISHED BY THE CENTCOM BLOOD PROGRAM MANAGER. THE CURRENT APPROVED CENTCOM CLINICAL PRACTICE GUIDELINE FOR DAMAGE CONTROL RESUSCITATION AND TRANSFUSION IS POSTED ON THE JOINT PATIENT TRACKING APPLICATION (JPTA) WEBSITE:
Platelet Transfusion

- **WB Derived Single Unit** = $5.5 \times 10^{10}$
  - (50 ml ; 40 = Plasma)
  - Recipient = > 10,000

- **Apheresis Platelets** = $3.0 \times 10^{11}$ (300 = Plasma)
  - Recipient = > 60,000
  - ( $575 $ )

- $1 : 1 : 1 = 0.2 : 1.1 : 1$
  - ( PLT : FFP : RBC )
FFP : RBC Ratio – Military Experience

Plasma: RBC Ratio Groups

Borgman et al
Brooke Army Medical Center
J Trauma 2007
Postinjury Massive Transfusion: First 6 Hours

Massive Transfusion >10 Units RBC / 6 Hrs

% of Blood Products Transfused

- PRBCs
- FFP
- Platelets

Hours After Injury

AAST / J Trauma 2008
FFP : RBC Ratio – Civilian Experience

Predicted Probability

Upper Quartile

Trendline

Lower Quartile

FFP : RBC Ratio

Civilian Experience

AAST / J Trauma 2008
Civilian Trauma ... Massive Transfusion

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n = 383 (Head Injury Excluded)  

Teixera et al  
J Trauma 2009
US Military – FFP:RBC

Mar 2003 - Feb 2006  Pre 1:1  =  1: 2.0

Mar 2006 - Sept 2008  1:1 Policy  =  1: 1.2

- Similar cohorts
- No difference in Mortality

Simmons et al
J Trauma  2010
Massive Transfusion Analyses: Issues

1) No Coagulation Functional Response

2) Analysis over 24 hr versus 6 hr

3) Selection Bias / Product Availability

4) Variability in Bioactivity of Blood Products

5) Differences in Injury Patterns
Postinjury Coagulopathy: Scientific Basis

↑ PLT: FFP : RBC → → → → ↓↓ Coagulopathy → → → ↓ Mortality

??? ↓ Coagulopathy
Death from Hemorrhage Occurs Within the First 6 Hours

66% within 6 Hr

1st 24 Hours After Admission

J Trauma 2007
### FFP : RBC >>>> Selection Bias

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Snyder et al  
J Trauma  2009

Are they dying because they are bleeding or bleeding because they are dying?  
Ben Galloway, MD 1976
"The Clinical Efficacy of FFP is Largely Unproven"
The Risk of ALI / ARDS is Higher with FFP and Platelets than RBCs

**PubMed**

U.S. National Library of Medicine
National Institutes of Health

Display Settings: ☑ Abstract


**Fresh-frozen plasma and platelet transfusions are associated with development of acute lung injury in critically ill medical patients.**

Khan H, Belsher J, Yilmaz M, Afessa B, Winters JL, Moore SB, Hubmayr RD, Gajic O.

Department of Internal Medicine, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA.

**BACKGROUND:** Transfusion has long been identified as a risk factor for acute lung injury (ALI)/ARDS. No study formally evaluated the transfusion of specific blood products as a risk factor for ALI/ARDS in critically ill medical patients.

**METHOD:** In this single-center retrospective cohort study, 841 consecutive critically ill patients were studied for the development of ALI/ARDS. Patients who received blood product transfusions were compared with those who did not. Univariate and multivariate propensity analyses were conducted. RESULTS: Two hundred ninety-eight patients (35%) received blood transfusions. Transfused patients were older (mean [± SD] age, 67 ± 17 years vs 62 ± 19 years; p < 0.001) and had higher acute physiologic and chronic health evaluation (APACHE) III scores (74 ± 32 vs 58 ± 23; p < 0.001) than those who had not received transfusions. ALI/ARDS developed more commonly (25% vs 18%; p = 0.025) in patients exposed to transfusion. Seventeen patients received massive RBC transfusions (i.e., > 10 U of blood transfused within 24 h) and 13 also received fresh-frozen plasma (FFP) and 11 received platelet transfusions. When adjusted for the probability of transfusion and other ALI/ARDS risk factors, any transfusion was associated with the development of ALI/ARDS (odds ratio [OR], 2.14; 95% confidence interval [CI], 1.24 to 3.75). Among those patients receiving individual blood products, ALI/ARDS was more likely to develop in patients who received FFP transfusions (OR, 2.48; 95% CI, 1.05 to 4.74) and platelet transfusions (OR, 3.89; 95% CI, 1.36 to 11.52) than in those who received only RBC transfusions (OR, 1.39; 95% CI, 0.79 to 2.43). CONCLUSION: Transfusion is associated with an increased risk of the development of ALI/ARDS in critically ill medical patients. The risk is higher with transfusions of plasma-rich blood products, FFP, and platelets, than with RBCs.
Fresh Frozen Plasma: Adverse Effects

Packed Red Blood Cells: MOF
Fresh Frozen Plasma: MOF
Fresh Frozen Plasma: MOF

Arch Surg 1997
Arch Surg 2010
J Trauma 2009
Physiologic Changes with Storage

- RBC
- FFP
- PLT

↓ Coagulation

↑ Inflammation
Unique mechanism of action bypasses the intrinsic pathway to form a complex with tissue factor at the site of injury.

Extrinsic Pathway

NovoSeven

Tissue Factor

Intrinsic Pathway

Thrombin Burst

Hemostasis

HMWK Kallikrein

FXI = FXla

FIX = FXa

FVIII = FVIIa

Fibrinogen = Fibrin

Prothrombin = Thrombin

FX = FXa

Prothrombin = Thrombin

Hemostasis
• No Difference in Mortality

• Decreased PRBC ... Blunt Trauma
• Decreased ARDS ... Blunt Trauma

• Safe ( ~ 3% Complication Rate )
CONTROL Trial: RCT / 150 Hospitals / 26 Countries

No Difference in Mortality

Decreased Blood Products

No Safety Issues

Hauser et al
J Trauma 2010
US Military – Recombinant Factor VIIa

- Combat Casualties (n=2050) 2003 – 2009
- 25% Received FVIIa; Propensity Scoring Match
- No Difference in Mortality
- No Safety Issues

Wade et al
J Trauma 2010
Role of Postinjury Fibrinolytics

- **Inclusion**: Massive Transfusion Protocol

- **Massive - Moderate – Minor / 6 Hr**
  - >10u
  - 5-10u
  - <5u

- **Thrombelastogram**

- **Logistic Regression Models:**
  - Risk Stratification for Fibrinolysis

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Each shipment can include the option of “doubling up” (e.g., 8 pRBC + 4 Plasma) as determined by the MTP ordering M.D. Shipment >4 determined by lab values and TEG results.

Ann Surg 2010
Postinjury Fibrinolysis

61 ED RBC

Transient Fibrinolysis
n = 28 (46%)

Primary Fibrinolysis (PF)
n = 11 (18%)

No Fibrinolysis
n = 22 (36%)

Conventional measures associated with PF:

- Higher ISS (p=0.06)
- Increased RBC’s (p=0.002)
- Depressed Fibrinogen@ 1 hour (p=0.0005)
- Increased Base Deficit/ Lactate (p=0.0001)

r-TEG findings associated with PF

- ACT
- K time
- MA (Maximum Amplitude)
- G value (Clot Strength)
- ALL p<0.0001

Timing of Fibrinolysis

Primary: 58 minutes (IQR 18.2-95.9)
Transient: 104 minutes (IQR 13.0-1200)

Ann Surg 2010
Mortality Associated with Fibrinolysis

- No: 18%
- Transient: 29%
- Primary: 64%

p = 0.02

Ann Surg 2010
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

**Summary**

Background: Tranexamic acid can reduce bleeding in patients undergoing elective surgery. We assessed the effects of early administration of a short course of tranexamic acid on death, vascular occlusive events, and the receipt of blood transfusion in trauma patients.

20,211 Adult: SBP < 90 or HR > 110, within 8 Hr

- Mortality 14.5% vs 16.0% (p<.0035)
- No safety issues

CRASH-2 Trial: RCT / 274 Hospitals / 40 Countries

Lancet 2010
In Search of the Scientific Basis for Postinjury Coagulopathy.

The Hemostasis Process
Thrombelastography: Answers

- Acute Coagulopathy of Trauma
- Pre-emptive Blood Components
- TEG
- Goal Directed Therapy
1) **Initiation**
- Endothelial damage
- Tissue Factor exposed
- TF / VIIa complex
- Small amount of thrombin

2) **Amplification**
- Thrombin activates platelets

3) **Propagation**
- Tenase and prothrombinase complexes
  - = rapid thrombin burst
Rapid Thrombelastography

- Coagulation
- Fibrinolysis

- Enzymatic (R)
  - Clotting time

- Fibrinogen (K, α)
  - Clot kinetics

- Platelets (MA)
  - Platelet function
  - Clot strength (G)

- Thrombolysins (Ly30, EPL)
  - Clot stability
  - Clot breakdown

- Tissue Factor ... Uncitrated Whole Blood

- Time (min)

- Amplilute (mm)
- LY

- SP
Angle (\(\alpha\)) < 54 °

Rate of clot growth … fibrin build-up and cross-linking

10 units pooled cryoprecipitate or 1 unit / 5 kg
~ 30-50 mg/dL increase in fibrinogen
MA < 50 mm

Strength / stiffness of the developed clot contributed mainly by platelets

1 unit apheresed platelets or 1 random donor equivalent /10 kg
~ 30,000-50,000/µL increase in platelet
Pattern Recognition

Thrombelastrograph

Normal
- \( R;K;MA;\text{Angle} = \text{Normal} \)

D.I.C.
- Stage 1
  - Hypercoagulable state with secondary fibrinolysis
- Stage 2
  - Hypocoagulable state

Platelet Dysfunction
- Thrombocytopenia/
  - Thrombocytopathy
- \( R \sim \text{Normal}; K = \text{Prolonged} \)
- \( \text{Angle} = \text{Normal} \)
- \( MA = \text{Very Decreased} \)

Anticoagulants/hemophilia
- Factor Deficiency
- \( R;K = \text{Prolonged}; \)
- \( MA;\text{Angle} = \text{Decreased} \)

Fibrinolysis (UK, SK or t-PA)
- Presence of t-PA
- \( R \sim \text{Normal}; \)
- \( MA = \text{Continuous decrease} \)
- \( LY30 > 7.5\% \)
- \( Ly60 > 15.0\% \)

Hypercoagulability
- \( R;K = \text{Decreased}; \)
- \( MA; \text{Angle} = \text{Increased} \)
Massive Transfusion Protocol

rapid-TEG

ACT >110
FFP

angle <60°
CRYO

MA <50
PLT

EPL >15%
ACA*

Re-assess via rapid-TEG

FFP = fresh frozen plasma; CRYO = cryopercipitate; PLT = apheresis platelets; ACA = aminocaproicacid * 5 grams in 250 ml infused over 1 hr
Thromboelastography: ED Assessment

GSW: Right Middle & Lower Lobes / Grade IV Right Liver

G = 1.8 (> 5.3)
Global Clot

ACT = 205 (< 110)
Coagulation Factors

K = 700 (< 120)
Fibrinogen

MA = 26 (< 72)
Platelets

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<th>SP</th>
<th>R</th>
<th>K</th>
<th>Angle</th>
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Thromboelastography : OR Resuscitation

GSW: Right Middle & Lower Lobes / Grade IV Right Liver

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<td>0 — 8</td>
<td>50 — 80</td>
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Trauma Transfer: 9 RBC / 4 FFP

- **G = 3.2 (>5.3)**
  Clot strength

- **ACT = 113 (>110)**
  Enzymatic

- **K = 335 (<120)**
  Fibrinogen

- **MA = 38 (>54)**
  Platelets
ED Thoracotomy: SW LV ... Prehospital CPR 11 min

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<th>K (min)</th>
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<th>MA (mm)</th>
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<td>-3-3</td>
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Antifibrinolytic Agents

- **Aminocaproic Acid** … lysine binding site plasminogen
- **Tranexamic Acid** … lysine binding site (10X)
- **Aprotinin** … directly inhibits plasmin (thrombotic complications)
Postinjury Fibrinolysis : S/P MVC
1. Pathogenesis of Postinjury Coagulopathy
   ~ 1/3 Requiring MT / 6 hr Arrive with ACT… Activated PC
   Hypothermia, Acidosis, Dilution, Consumption, Fibrinolysis, etc

2. Pre-emptive Therapy
   FFP : RBC Ratio = 1:2
   Antifibrinolytics … Selective
   Platelets, Fibrinogen

3. Goal Directed Management
   Thrombelastography
Postinjury Hemostasis: Our Protocol

- Correct Shock ... ASAP !!!
- Prevent Hypothermia
- Avoid Hypocalcemia
- Pre-emptive FFP: RBC = 1:2
- Pre-emptive Apheresis PLT / Cryo if > 4 RBC 1st 30 min
- Goal - directed via rTEG
Thank you !!!
Qualitative Platelet Dysfunction: ? PLT Transfusion

Korea WB  Scott, et al  Blood  1954


( no evidence for presumptive PLT )

> 12 U / 12 hr RBC  Reed, et al  Ann Surg  1986
( no benefit of presumptive PLT )
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<thead>
<tr>
<th>Coagulation Factor Deficiency:  ? FFP Transfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Pre-emptive FFP: RBC = 1:4</td>
</tr>
<tr>
<td>( no benefit of presumptive FFP )</td>
</tr>
<tr>
<td>➢ Pre-emptive FFP: RBC = 1:5</td>
</tr>
<tr>
<td>➢ FFP after &gt; 10 RBC</td>
</tr>
<tr>
<td>➢ Pre-emptive FFP: RBC = 1:1</td>
</tr>
<tr>
<td>PLT:RBC = 5:5</td>
</tr>
<tr>
<td>➢ Pre-emptive FFP:RBC = 1:1</td>
</tr>
<tr>
<td>( Iraq )</td>
</tr>
<tr>
<td>( Civilian )</td>
</tr>
</tbody>
</table>
Postinjury Hemostasis with Massive Transfusion

**Qualitative Platelet Dysfunction:** ? PLT Transfusion

- Korea WB  Scott, et al  Blood 1954
  (no evidence for presumptive PLT)
- > 12 U / 12 hr RBC  Reed, et al  Ann Surg 1986
  (no benefit of presumptive PLT)

**Coagulation Factor Deficiency:** ? FFP Transfusion

- Pre-emptive FFP: RBC = 1:4  DGH ... J Trauma 1982
  (no benefit of presumptive FFP)
- Pre-emptive FFP: RBC = 1:5  Wilson, et al  J Trauma 1987
- FFP after > 10 RBC  Lucas, et al  J Trauma 1989
- Pre-emptive FFP:RBC = 1:1  DGH ... Ann Surg 2001
  PLT:RBC = 5:5  (pelvic fracture hemorrhage guidelines)
- Pre-emptive FFP:RBC = 1:1  Holcomb et al  J Trauma (Iraq) 2007
**Civilian Trauma – Massive Transfusion**

<table>
<thead>
<tr>
<th></th>
<th>FFP : RBC &gt; 1:2</th>
<th>PLT : RBC &gt; 1:2</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFP</td>
<td>FFP</td>
<td>PLT</td>
<td>= 29%</td>
</tr>
<tr>
<td>FFP</td>
<td>-</td>
<td>-</td>
<td>= 48%</td>
</tr>
<tr>
<td>-</td>
<td>PLT</td>
<td>-</td>
<td>= 34%</td>
</tr>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>= 59%</td>
</tr>
</tbody>
</table>

*n = 466 (10% Head)*

Holcomb et al
Ann Surg 2008
Blood Component Expenses

L – RBC = $ 225

FFP = $ 70

PLT_{aph} = $ 575

Bonfils Blood Center 2010
Recombinant Factor VIIa: Coagulopathy

FDA Approved – Hemophiliac VIII / IX Inhibitors

Multiple Trauma Series: Randomized Trial x 2

Off-label Use:
- ? Indication
- ? Dose
- ? Risk / Benefit
Postinjury Coagulopathy
Pharmacologic Control of Thrombus Formation

Aspirin

Cyclooxygenase

Thromboxane

Persantine
Toradol
Pletal

ATP
CAMP
AMP
PD

GPIb

Reopro
Aggrastat
Integrilin

GPIIb/IIIa

ADP

GPIIb/IIIa

Platelet

Plavix
Ticlid
Postinjury Fibrinolysis

Profound Shock
(Not Brain Tissue)
POC Rapid Thrombelastography

Transfusion Triggers Activated: Conventional Coagulation Tests vs. r-TEG
Non-citrated Whole Blood Specimens (n=27)

Launch Microsoft Office Outlook

P=0.02

INR vs. TEG ACT
Fibrinogen vs. Angle
Platelets vs. MA
Overall

P=0.02
P=0.41
P=0.65

Conventional Coagulation tests
r-TEG
# POC Rapid Thromboelastography

<table>
<thead>
<tr>
<th>Pre TEG</th>
<th>( n = 68 )</th>
<th>Post TEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>-13</td>
<td>ED : BD</td>
<td>-15</td>
</tr>
<tr>
<td>1.6</td>
<td>ED : INR</td>
<td>1.8</td>
</tr>
<tr>
<td>18.0</td>
<td>RBC / 6 hr</td>
<td>17.2</td>
</tr>
<tr>
<td>6.8</td>
<td>FFP / 6 hr</td>
<td>6.5</td>
</tr>
<tr>
<td>65%</td>
<td>Mortality</td>
<td>29%</td>
</tr>
<tr>
<td>21%</td>
<td>Coagulopathy</td>
<td>3%</td>
</tr>
</tbody>
</table>

AAST 2009
Combat Injury Mechanism ... FFP : RBC

Mace et al
JACS 2009
POSTINJURY FIBRINOLYSIS

61 Acute Injury / ED RBC

- Massive >10u /6hrs N=32 (52%)
  - Mortality 53%
- Moderate 5-10u / 6hrs N=15 (25%)
  - Mortality 7%
- Minimal <5u / 6hrs N=14 (23%)
  - Mortality 7%

Ann Surg 2010
Estimated Probability of Primary Fibrinolysis and Death by G value at 1 hour Postinjury

For every one unit drop in G value (clot strength) by one hour, risk of PF increases by 30% and death by >10%