

DAMAGE CONTROL RESUSCITATION

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DAMAGE CONTROL RESUSCITATION

- Damage control is a Naval term
- SAVE THE SHIP
- Limit damage
- Emergency repair
- Finish mission



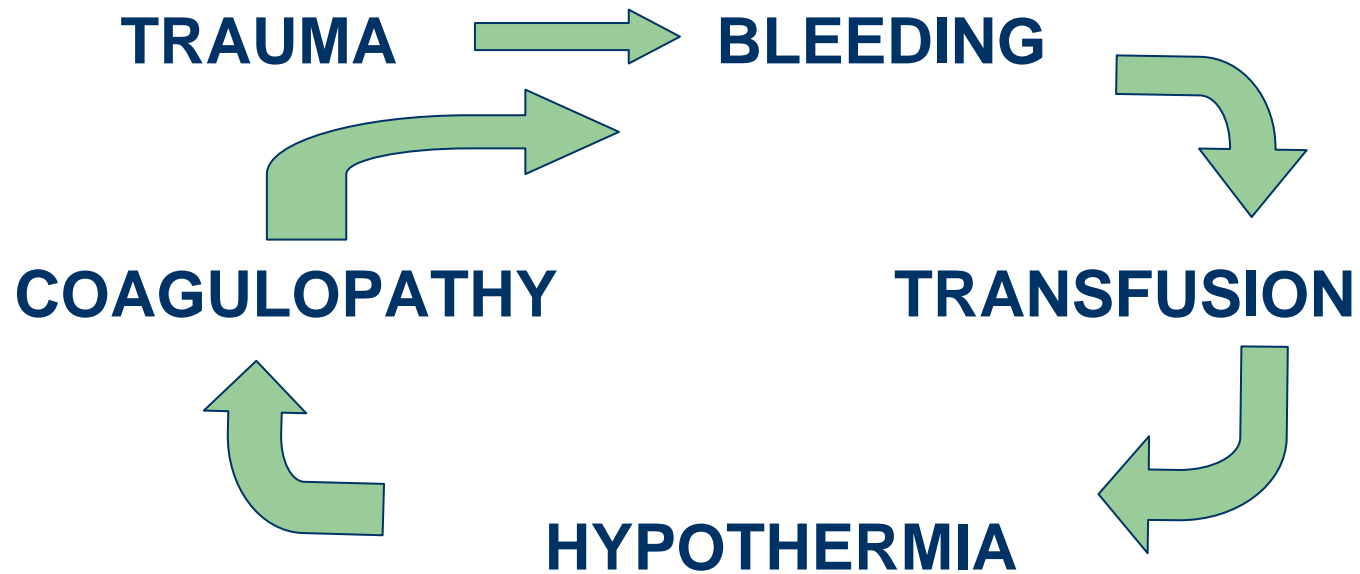
DAMAGE CONTROL

- Apply to surgery:
- Trauma patient with abdominal injury
- Control bleeding
- Stop spillage and contamination
- Get out !
- Resuscitation in ICU, optimisation
- Elective repair when optimised

DAMAGE CONTROL

- Apply to surgery:
- All trauma patients
- Initial procedure to control damage:
stop bleeding, stop contamination
- Operating time max 1 hour
- Prevent the Bloody Vicious Cycle
- Resuscitate in ICU
- Return electively for definitive repair of injuries

DAMAGE CONTROL



THE BLOODY VICIOUS CYCLE

DAMAGE CONTROL

- TRAUMA CAUSES OF DEATH:
- Hypoxia
- Hypovolemia
- Hypothermia
- METABOLIC MAYHEM

DAMAGE CONTROL

- Wider application: Other disciplines
- Orthopaedics: Debride, stabilise with ExFix, leave wounds open
- Return electively for wound inspection and definitive treatment

DAMAGE CONTROL

- Neurosurgery:
- Craniotomy, decompressive craniectomy, leave cranium open for oedema
- Partial resection of brain tissue
- Reconstruction later

DAMAGE CONTROL

- DAMAGE CONTROL is
- DAMAGE CONTROL RESUSCITATION
(give components the patient needs)
and
- DAMAGE CONTROL SURGERY
(staged surgical procedures)

DAMAGE CONTROL RESUSCITATION

- Military deployment: US OIF/OEF (IRAQ)
- Resuscitation with crystalloids, and PRBC
- Bleeding tendency: less / no clotting factors
- Increased the use of FFP
- Use PRBC : FFP : platelets 1 : 1 : 1
- Improved outcomes

DAMAGE CONTROL RESUSCITATION

- Rationale:
- Patients lose whole blood
- Blood banks supply components
- Patient develops bleeding tendency, with loss of clotting factors +/- consumption
- Needs replacement of all components, ie **WHOLE BLOOD**

HEMOSTATIC RESUSCITATION

- Can hemostatic resuscitation (ie blood components) reverse coagulopathy of trauma?
- Polytrauma pig model (40 Kg)
- Pre-hosp phase: femur #, hemorrhage (60% blood volume), 30 min shock, saline (3x shed blood), hypothermia (33 C)
- Early hosp phase: Gr 5 liver injury,
- Operative phase: Liver packing

Alam H B. et al. Hemostatic Resuscitation Research Group, Mass Gen Hosp.
Presented at AAST Hawaii, Sep 2008

HEMOSTATIC RESUSCITATION

- 75 pigs: randomised to 6 groups:
- 1. Sham
- 2. Control (no treatment)
- 3. Fresh whole blood
- 4. FFP / PRBC in ratio 1 : 1
- 5. FFP alone
- 6. 6% Hetastarch (synthetic colloid) equal to shed blood

Alam H B. et al. Hemostatic Resuscitation Research Group, Mass Gen Hosp.
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HEMOSTATIC RESUSCITATION

- Acidosis (lactate > 6 mmol/l, and coagulopathy (30-80% increase in PT)

- 30% animals died

- Mortality (%)

- Control FWB FFP/PRBC FFP Hetastarch
75 <10 <10 <10 80

anemia

Alam H B. et al. Hemostatic Resuscitation Research Group, Mass Gen Hosp. Presented at AAST Hawaii, Sep 2008

HEMOSTATIC RESUSCITATION

- Cause of death is metabolic, not hemorrhage
- All stopped bleeding after packing
- Acidosis and hypothermia are the “bad guys” in trauma.

Alam H B. et al. Hemostatic Resuscitation Research Group, Mass Gen Hosp. Presented at AAST Hawaii, Sep 2008

DAMAGE CONTROL RESUSCITATION

- Massive transfusion protocol:
- Trauma unit must have a protocol for the exsanguinated patient
- Discuss with blood bank beforehand
- When called, bloodbank will supply packed cells, FFP and platelets in correct ratio of 1 to 1 to 1

DAMAGE CONTROL RESUSCITATION

- Vascular surgery in Combat support hospital
- 16 pts with vascular injuries, hypotensive (105/60), acidotic (pH 7.27, BE -7.5), coagulopathic (INR 1.3)
- Resuscitated with FWB, plasma, platelets, cryoprecipitate and rF VIIa
- Allowed prolonged complex surgery (vein grafts, 4.5 h) with a good outcome

Fox CJ (Holcomb) J Trauma 2008;65:1-9

HEMOSTATIC RESUSCITATION

- Massive Transfusion Protocol reduces organ failure and post injury complications
- Vanderbilt University TEP (Trauma Exsanguination Protocol) supplies 3:2 RBC: FFP, and 5:1 RBC: platelets.
- 2 years (04-05, preTEP) and 2 years (06-07, TEP)
- Cotton B A, et al. Vanderbilt University, as presented at AAST, Hawaii, Sep 2008, J Trauma 2009; 66:41-49

HEMOSTATIC RESUSCITATION

- Results

	preTEP (140)	TEP (124)	P value
Sepsis	28(20%)	11(9%)	0.011
Vent free days	16.1 d	19.9 d	0.001
Open abdomen	42(30%)	9(7%)	<0.001
MOF	52(37%)	20(16%)	<0.001

- Cotton B A, et al. Vanderbilt University, as presented at AAST, Hawaii, Sep 2008, J Trauma 2009;66:41-49

HEMOSTATIC RESUSCITATION

- Question: Is this result better because of More Plasma, or less crystalloid?
- Shires (1976): gave blood, and a little crystalloid
- Fred Moore uses more plasma, less crystalloids, reducing the resus time, and patient earlier in the ICU

- Cotton B A, et al. Vanderbilt University, as presented at AAST, Hawaii, Sep 2008, J Trauma 2009;66:41-49

HEMOSTATIC RESUSCITATION

- FFP: PRBC ratio $> 1:1.5$ is associated with lower risk of mortality after massive transfusion
- Pts needing massive transfusion ($>8u$) in 12 H (415), high ratio (102) vs Low ratio (313)
- High: lower risk of mortality, especially in first 48 H, but higher risk of ARDS

Sperry J L, et al. (group of Pittsburg PA, Dallas Tx, Seattle Wa, Denver Co).
J Trauma 2008; 65: 986-993

HEMOSTATIC RESUSCITATION

- Civilian Trauma Unit, retrospective analysis
- Trauma induced coagulopathy (TIC):
PT > 16 sec, PTT > 50 sec
- 435 pts (7 years) received FFP with > 10 u blood in OR.
- 31% had TIC with 53 (40%) mortality.
- More FFP, lower mortality. (1:1 has 26%, 1:2 has 36%, 1:3 has 40%, and 1:4 has 52% mortality)

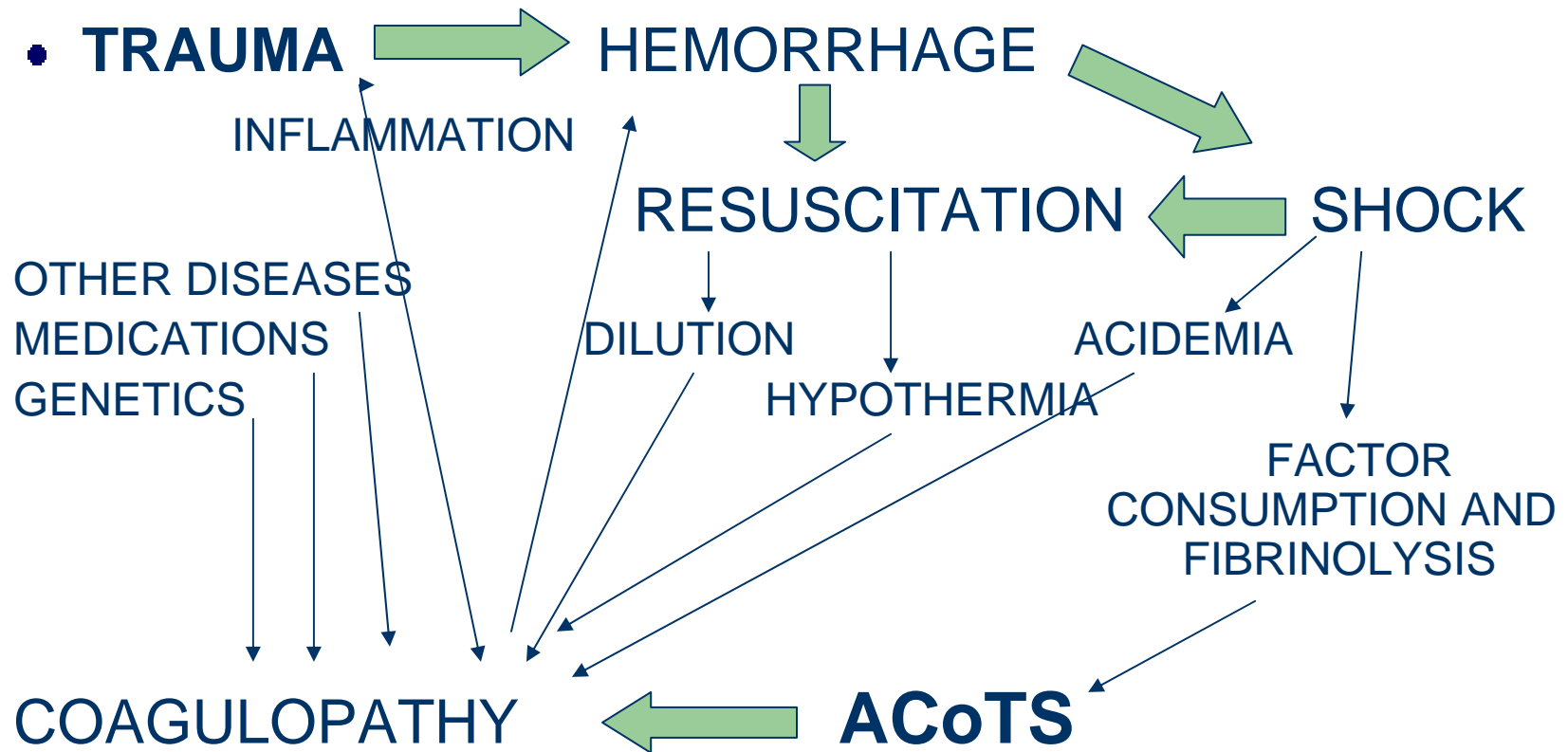
Duchesne J C et al. Tulane University (Norm McSwain) N O, as presented at AAST Hawaii. Sep 2008

HEMOSTATIC RESUSCITATION

- The Coagulopathy of Trauma: A Review of Mechanisms: Interactions of multiple factors:
- Tissue Trauma
- Shock
- Hemodilution
- Hypothermia
- Acidemia
- Inflammation

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

COAGULOPATHY AFTER TRAUMA



HEMOSTATIC RESUSCITATION

Tissue Trauma

- Injury severity closely associated with degree of coagulopathy
- Tissue damage – endothelial damage – coagulation – F VIIa – thrombin + fibrin
- Fibrinolysis – tPA – increased by presence of thrombin
- Specific organs: Brain (release brain-specific thromboplastins), long bone fractures, F E S.

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

HEMOSTATIC RESUSCITATION

Shock

- Prime driver of early coagulopathy
- Association between severity of tissue hypoperfusion and degree of admission coagulopathy (PT and PTT)
- B E >-6 associated with coagulopathy in 25% of patients, with 4-fold increase in mortality
- Mechanism unclear. Possible association with aProt C

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

HEMOSTATIC RESUSCITATION

Hemodilution

- Dilution of clotting factors is a major cause
- Fluid shifts from interstitium and fluid resuscitation both contribute
- Colloids may interfere with clot formation and stability
- PRBC also dilute clotting factors, and reduce clot stability

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

HEMOSTATIC RESUSCITATION

Hypothermia

- Inhibits coagulation protease activity and platelet function
- F VIIa activity decreases with lowered temperature (50% activity at 28 C)
- Environmental exposure, reduced production from muscle, increased loss during surgery, cold fluids.
- Clinical effects seen at < 34 C, mortality $\wedge < 32$ C

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

HEMOSTATIC RESUSCITATION

Acidemia

- Low flow shock states, excess ionic Chloride
- Impairs function of plasma proteases
- Activity of F Xa/Va complex reduced by 50% at pH 7.2, 70% at pH 7.0, and 90% at pH 6.8
- Prolong clotting times and reduce clot strength, and increase degradation of fibrinogen
- Buffer solutions correct pH but not the coagulopathy

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

HEMOSTATIC RESUSCITATION

Inflammation (1)

- Strong inducer of inflammation, and SIRS is a common consequence of severe injury
- Cellular and humoral elements of immune system activated by trans membrane protease receptors on cells, as well as activation of complement
- Platelet degranulation activate neutrophils, endothelium, and monocytes that adhere to platelets at the site of injury

HEMOSTATIC RESUSCITATION

Inflammation (2)

- Trauma patients switch to hypercoagulable state with increased risk of thrombotic events
- Similar to the coagulopathy of severe sepsis and the depletion of protein C
- Higher incidence of sepsis, and development of MOF

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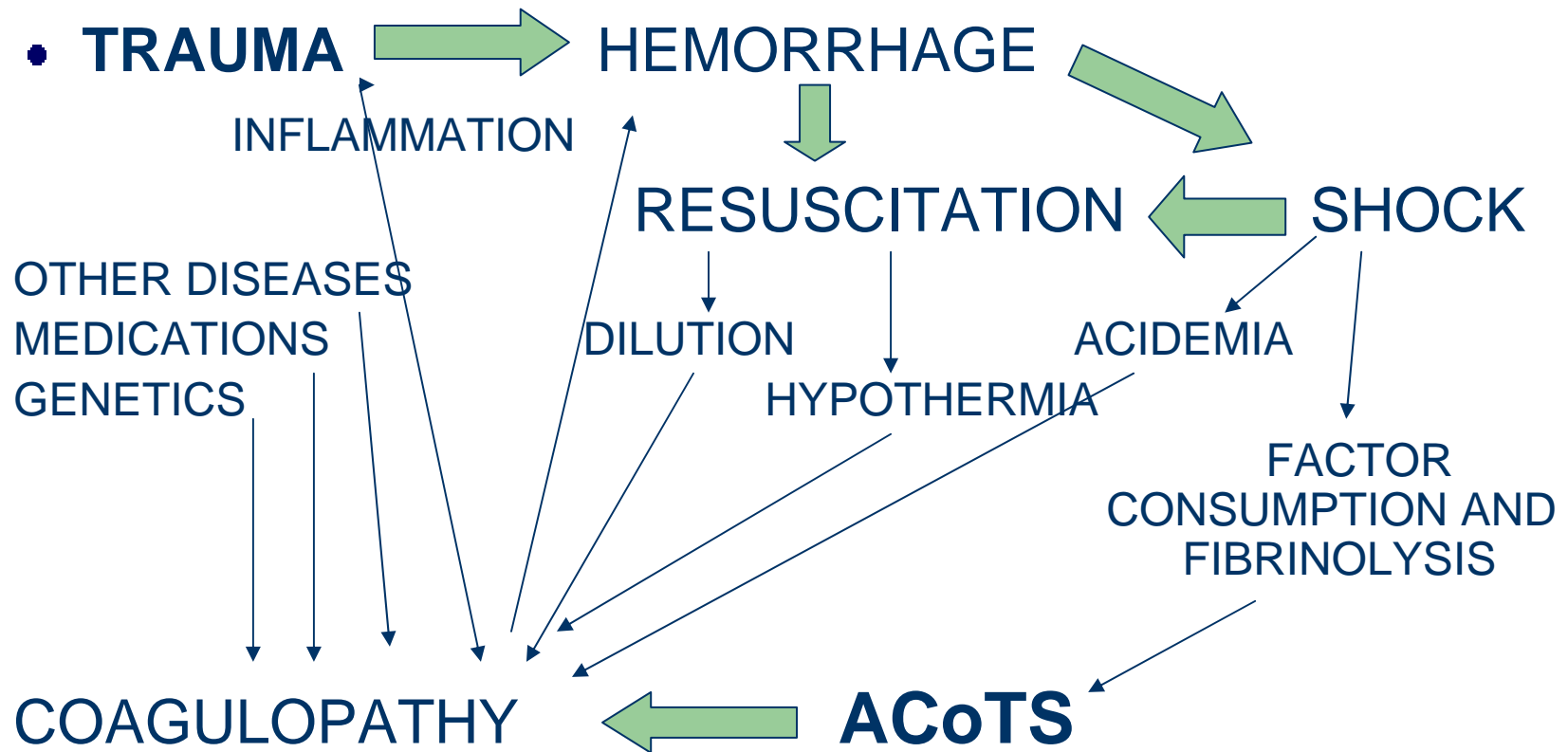
HEMOSTATIC RESUSCITATION

Different names:

- Acute traumatic Coagulopathy
- Early Coagulopathy of Trauma
- Trauma-Induced Coagulopathy
- Propose a new term:
- **ACoTS: Acute Coagulopathy of Trauma-Shock**

Hess J R et al. (EICBT group) J Trauma 2008; 65: 748-754

COAGULOPATHY AFTER TRAUMA



HEMOSTATIC RESUSCITATION

- HEMOSTATIC RESUSCITATION
- DAMAGE CONTROL RESUSCITATION
- BACK TO BASICS: SUPPLY WHAT THE PATIENT NEEDS
- WHOLE BLOOD , OR ALL COMPONENTS

HEMOSTATIC RESUSCITATION



COAGULOPATHY AFTER TRAUMA

