



14th National Emergency Medicine Congress 19-22April 2018 in Turkey Damage Control Resuscitation

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Royal Medical Services





K.H.M.C

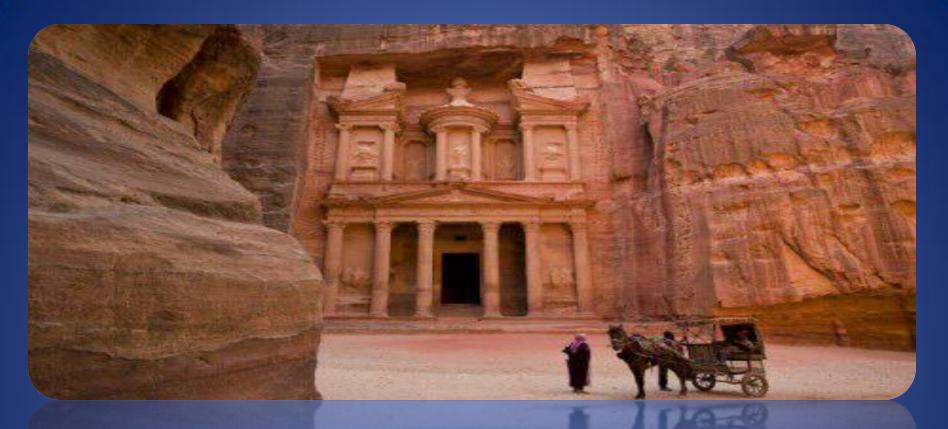




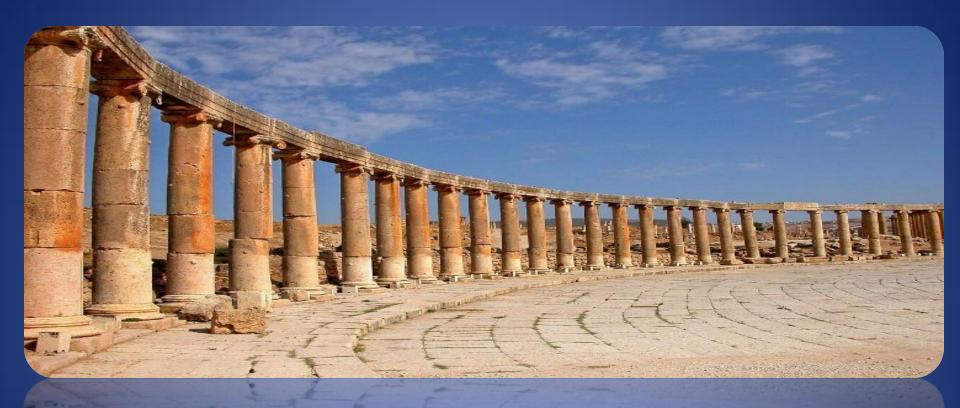
Emergency Department







The city of Petra, capital of the Nabataean Arabs, it is also known as the rose-red city and One of the famous sits in the world, on 7 July 2007 Petra announced as on of the new seven wonders.



Jerash is most Beautiful city in Jordan from Roman empire, The colonnaded street is one of interested place which visited there, Jerash festival of culture and arts is annually held event in Jerash.



Dead Sea ... The Earth's lowest elevation on land

Outline

- Definition
- What is damage Control Resuscitation
- Concept of Damage control
- Criteria (Indications) of Damage Control
- Principles/Lethal Triad
- Haemostatic Resuscitation
- Summary

Definition

Damage control resuscitation (DCR) is a newer development within the damage control paradigm, and describes novel resuscitation strategies aimed to limit the physiological derangement of trauma patients.

What is damage Control Resuscitation

- A hemorrhage in trauma patients
- 1. Find the bleeding, Stop the bleeding
- 2. Recognize the risk of an **uncontrolled** haemorrhage +/-activation of Massive Transfusion Protocols
- 3. Restore blood volume rapidly and effectively (Permissive Hypotension) maximise tissue perfusion whilst minimising clot rupture and excessive blood loss.
- 4. Maintain bloods function by maintaining the blood composition (Haemostatic Resus).

Concept of Damage control

"...keeping a float a badly damaged ship by procedures to limit flooding, stabilize the vessel, isolate fires and explosions and avoid their spreading"

Surface ship survivability, Naval war publication 3-20.31, Washington, DC. Department of defense; 1996

Criteria (Indications):

- Hypothermia
 - <35 degree C</p>
- Massive Blood Transfusion
 - >10 unit PRBC
- Severe Metabolic Acidosis
 - pH< 7.30
- Operative time
 - >90 mins
- Coagulopathy
 - Either on lab results or 'non surgical' bleeding
- Lactate
 - >5 mmol/L

IS IT IMPORTANT TO OUR PRACTICE?

- Traumatic injury is the leading cause of death in under 45s
- Most victims are young males
- Most preventable deaths are due to haemorrhage leading preventable cause of death in trauma



Case Data

25 year old Soldier involved in blast Tourniquet applied at scene one hour ago Systolic BP 90
Mental Status: Awake, confused Blood oozing from superficial wounds Temp 35.5 c

Patient Assessment

Sick or Not Sick

• What are reliable criteria for initial assessment?

• Which criteria can predict outcomes and direct therapy?



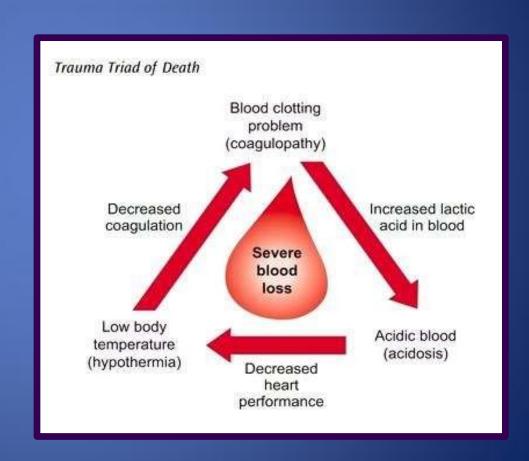


Within the first five minutes in the ED

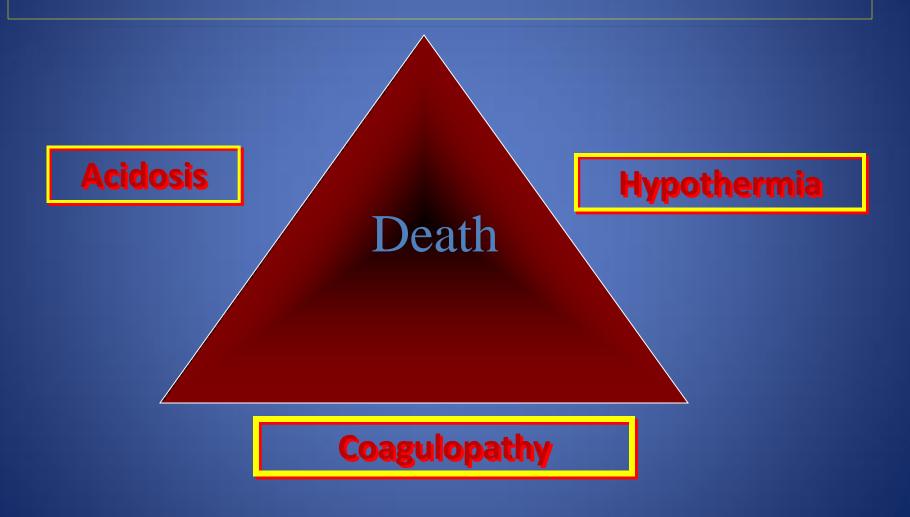
- Identify patients in trouble
- Identify patients with increased mortality
- Identify patients with increased probability of massive transfusion

TRAUMA TRIAD OF DEATH

- Hypothermia
 - Decreased cardiac output
 - Arrhythmias
 - Decreased function of coagulation factors and platelets
- Acidosis
 - reduced contractility, bradycardia, dysrhythmias, vasodilation, hypotension
 - Decreased function of coagulation factors and platelets



The Lethal Triad



Acidosis

Base deficit (BD) \geq 6 identifies patients that

- > require early transfusion,
- > increased ICU days and
- > risk for ARDS and MOF
- O Patients have an elevated BD before their blood pressure drops to classic "hypotension" levels.
- Acidosis contributes more to coagulopathy more than hypothermia (not reversible)

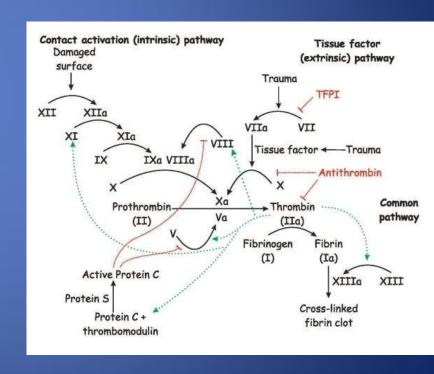
ACIDOSIS AND BASE EXCESS

- Both are independent predictive factors of mortality and identify anaerobic metabolism (tissue hypoperfusion)
- pH strongly affects activity of Factors V, VIIa and X
- Acidosis inhibits Thrombin generation
- pH<7.2 decreased contractility and CO, vasodilation, hypotension, bradycardia, dysrhythmias
- Lactate is demonstrated to have the best association with hypovolaemic shock and death - useful marker as an endpoint of resuscitation

TRAUMA-INDUCED

COAGULOPATHY

- Trauma-induced coagulapathy (TIC) and Acute Traumatic Coagulopathy (ATC)
 - "not simply a dilution coagulopathy or consumptive coagulopathy"
 - Characterized by
 - Isolated factor V inhibition functionally decreases thrombin
 - Dysfibrinogenaemia
 - Systemic Anticoagulation
 - Impaired platelet function
 - Hyperfibrinolysis
- Exacerbated by
 - hypothermia
 - acidosis
 - resuscitation by hypocoagulable fluid



Coagulopathy on Presentation

- Severity of injury and mortality is linearly associated with the degree of the initial coagulopathy.
- O An initial INR ≥ 1.5 reliably predicts those casualties who will require MT.

Hypotension

A systolic blood pressure of 90 mm Hg or less is indicative casualties that have lost over 40% of their blood volume

- \sim (~2000 ml in an adult)
- > They have impending cardiovascular collapse and have significantly increased mortality.

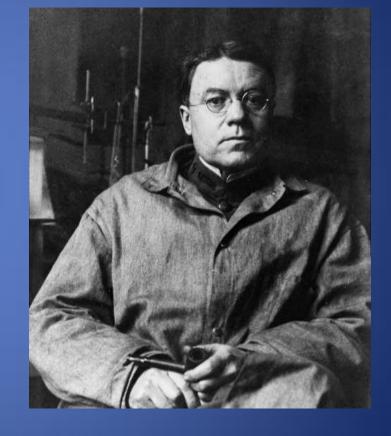
PERMISSIVE HYPOTENSION

 Keep the blood pressure low enough to avoid exsanguination whilst maintaining end-organ perfusion

- Prevent "clot busting"/dislodgement
- Avoid excessive fluid administration

THE PERMISSIVE HYPOTENSION

• will increase blood pressure has dangers in itself. ... If the pressure is raised before the surgeon is ready to check any bleeding that might take place, blood that is sorely needed may be lost."



 Walter Cannon, 1918

Walter Bradford Cannon

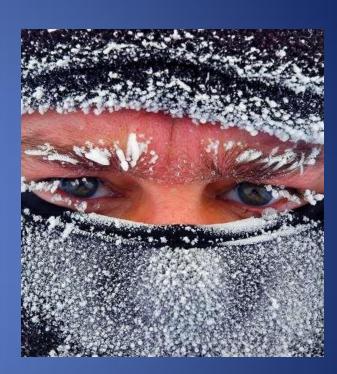
Temperature

- O A temperature < 96°F or 35°C is associated with an increase in mortality.</p>
- Trauma patients that are hypothermic are not per fusing their tissue
- O The coagulation cascade is an enzymatic pathway that degrades with temperature and ceases at 33.5 c

HYPOTHERMIA

- Severe hypothermia is associated with a high mortality
- <35 deg platelet dysfunction
- <33 deg clotting enzyme synthesis/kinetics, plasminogen activator
- Progressive delay in the initiation of thrombus formation
- Causes
 - Pre-hospital
 - ED resuscitation period
 - Theatre exposure of peritoneum, resus fluids
- Treat
 - Warmed fluids
 - Bair Hugger/warm blankets

 - Minimise exposure
 Increase ambient temp.
 Continuous Temp. Monitoring



HAEMORRHAGIC SHOCK

 Hemorrhagic shock is a condition of reduced tissue perfusion, resulting in the inadequate delivery of oxygen and nutrients that are necessary for cellular function. Whenever cellular oxygen demand outweighs supply, both the cell and the organism are in a state of shock.

CAUSES OF SHOCK IN TRAUMA

- Hypovolaemic Shock haemorrhage (accounts for ~40% of all trauma-associated deaths)
- Obstructive Shock Cardiac Tamponade,
 Tension Pneumothorax

- Distributive shock Neurogenic/Spinal
- Cardiogenic direct cardiac contusion

WHAT CAUSES HAEMORRHAGIC SHOCK?

> Direct Tissue Damage

Disruption of blood vessels with associated blood loss can be enough to cause tissue hypoperfusion.

Coagulopathy

- A. Intrinsic Trauma-Induced Coagulopathy (TIC)/Acute
- B. Coagulopathy of Trauma (ACT)
- > Extrinsic
- > Hypothermia
- Dilatational (administration of crystalloids/blood products)
- ➤ Acidosis/Tissue Hypo-perfusion

Diagnosis Done

Diagnosis Done

- > How to resuscitate these casualties?
- Damage Control Resuscitation
 - 1. Hypotensive resuscitation
 - 2. Hemostatic resuscitation

Haemostatic Resuscitation

- O Traditional resuscitation strategies dilute the already deficient coagulation factors and increase multiple organ failure
- O Damage control philosophy can be extended to haemostatic resuscitation
 - restoring normal coagulation
 - · minimizing crystalloid
- O The aggressive haemostatic resuscitation should be combined with equally aggressive control of bleeding

Standard Resuscitation Paradigm

Crystalloid 3:1 Ratio



Transient or no response

Blood

6-10 u PRBC

FFP

Oh- By the way: show me the data



Crystalloid

Haemostatic Resuscitation

- 1. Early Dx in ED
- 2. 1:1 ratio (PRBC to FFP)
- 3. ED use of RFVIIA
- 4. Call for FWB from the ED
- 5. Frequent CRYO and platelets
- 6. Repeated doses of RFVIIA in OR and ICU as required
- 7. Minimal crystalloid

1:1 Ratio of PRBC to Plasma

O Increased FFP transfusions within first 24 hrs of admission were independently associated with increased survival.

O Median ratio of FFP: RBC was 1:1.7 in survivors compared to 1:3 in non-survivors (p<0.001).

Mortality PRBC:FFP

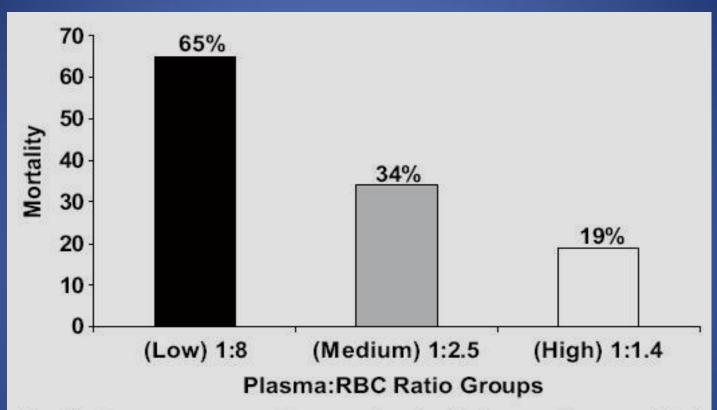


Fig. 1. Percentage mortality associated with low, medium, and high plasma to RBC ratios transfused at admission. Ratios are median ratios per group and include units of fresh whole blood counted both as plasma and RBCs.

Fresh Whole Blood

- Fresh whole blood (FWB) must be called for early after ED arrival, takes 60 minutes
 - Injury Pattern recognition
- FWB is the optimal resuscitation fluid for severely injured casualties.
- FWB is the best fluid for hypotensive resuscitation for hemorrhagic shock.

CRYSTALLOIDS

The Good

- Cheap
- Readily available
- Easily stored
- No risk of transfusion reactions/BBV
- May be appropriate/not harmful in many trauma patients but aggressive use is associated with increased mortality in haemorrhagic shock

The Bad

- Increased haemorrhage increased clot rupture with
 restoration of normal BP
- Dilutional coagulopathy
- Dilutional anaemia impaired oxygen delivery
- Hypothermia
- Metabolic Acidosis (N. Saline)
- Lowers plasma oncotic pressures -

Summary

- Recognize Shock
 - Critical Criterion
 - Identify the critical 10 %
- Resuscitate Immediately
 - Devote attention to Haemostatic resuscitation
- o Provide volume that also restores the haemostatic cascade
- Minimize crystalloid
- Stop the bleeding
 - Fresh Whole Blood

Summary

- O The management of exsanguination requires leadership, prompt thinking and aggressive surgical intervention.
- Delays in the decision to perform DC contribute to a higher morbidity and mortality.
- DC is a vital part of the management of the multiply injured patient and should be performed before metabolic exhaustion.

