PRINCIPLES OF DAMAGE CONTROL IN

TRAUMA:

18th Annual Controversies and Problems in Surgery

DEPARTMENT OF SURGERY SBAH/UP

TORSO TRAUMA

L.M. NTLHE
Introduction

Damage control surgery (DCS) has been the standard of care for the last 20 years in multiple trauma patients (all cutting disciplines). Necessitated by excessive haemorrhage and high mortality from “total care.” Damage Control Resuscitation (DCR) includes DCS and intensivists occupy a pivotal role in the last 10 years.

(Futile surgery & DC resurrection: best avoided)
DEFINITION
• INJURIES TO MORE THAN ONE ANATOMICAL AREA
• INCIDENCE – 10-15% OF TRAUMA PATIENTS
BAT
BAT
The concept of *damage control* originates from the USA Navy (*early 1900’s*)

Damaged ships and personnel underwent quick **temporary** repairs for mission integrity and to be stable enough to reach port for definitive repair.
• Damage: Not only to anatomical relations but also to the physiological/metabolic ambience and internal homeostasis.

• Traditionally, the total care ab initio concentrated mainly on the former hence the very high mortality and morbidity associated with the classic lethal triangle of death in severely injured patients (Dangerous triplets)
DEFINITIONS :-

**DCS**: abbreviated surgical intervention to stop *haemorrhage*, limit *contamination* then ICU

**DCR**: includes DCS “A systematic approach to major trauma combining the <C>ABC paradigm with a series of techniques from the point of wounding to definitive treatment in order to *minimize blood loss, maximize tissue oxygenation and optimize outcome*“ (6). Hodgetts et.al

**TORSO TRAUMA**: MOI
Penetrating, blunt, blast and any combination
PATHOPHYSIOLOGY

LETHAL TRIAD:

Hypothermia/Acidosis/Coagulation
Coagulopathy in trauma has a complex etiology. (DO THEY BLEED !!!)

It is Multifactorial & involves all components of the coagulation system. (Hess JR, Brohi K et.al)

*Six key factors interplay in Coagulopathy:*
- Tissue trauma
- Shock
- Hypothermia
- Haemodilution
- Acidosis
- INFLAMMATION

37.2 Trillion cells!! (cross-talk). TSHAHI
Coagulopathy continued

**Tissue Trauma** (Brohi et al)

*Endothelium* injury → exposes sub-endothelial collagen type III & tissue factor V which binds to platelets, von Willebrand factor, aFactor VII → Coagulation cascading *at site* (minor event)

**Amplification** via Factor IX → Hyperfibrinolysis

Exacerbated by the Thrombin/Thrombomodulin /APC pathway
Coagulopathy
continued

Platelet function: Aggregation, Degranulation and Shape change.

There is Early/admission time platelet dysfunction.

ADP and activated Collagen Surface Receptors:

ADP induction of platelet aggregation relies on a Subgroup of nucleotide-activated platelet receptors P2T: (a) P2X...Ca^{++} influx channel (b)P2Tac...Adenyl Cyclase inhibitors (c) P2Tplc ...mobilizes Ca^{++} stores via inositol phosphate production
Coagulopathy continued

Collagen surface receptors *activation* $\rightarrow$ *reversal* of the Na$^+$/Ca$^{++}$ *exchanger.*

Thus platelet dysfunction may be due to

1) inhibition of the above multiple receptors
2) with alteration in Ca$^{++}$ influx
3) thrombocyte Energy deficit
Coagulopathy continued

**SHOCK:**

Hypoperfusion from shock is associated with thrombin-thrombomodulin complexing which activates the *Protein C pathway* → inhibits the Plasminogen Activator Inhibitor (PAI-1) → **Hyperfibrinolysis**

Inactivation of factors Va, VIIIa and Xa due to lactic acidosis

Worsened by undetected continued bleeding.
Coagulopathy continued

**Haemodilution**: Anormal admission INR, PT, APTT and Base deficits is reported. Haemorrhage results in *Fluid shifts* with low factor levels (from intracellular, interstitial) Made worse by crystalloid & prbc, colloid *therapy*

*Hence* 1: 1: 1 ratios of prbc / **plasma** / platelets early therapy and permissive hypotension ....65mmHg until haemostasis is secured
HYPOTHERMIA

Mortality increase from 40% at 34°C to 100% when body core temperature drops to 32°C
HYPOTHERMIA (continued)

Haemorrhage leads to sympatho-adrenal overstimulation, vasoconstriction, decreased tissue perfusion and hypothermia. Heat generation by skeletal muscles is decreased.

Dysrhythmias, impaired myocardial function and reduced Cardiac Output.

Hb Oxygen dissociation curve is left shifted with oxygen retention at tissue level, worsens anaerobism & lactic acidosis. Clotting cascade impairment affecting temperature sensitive serine esterases.

Endothelial damage and reduced thromboxane B2 production leads to inhibition of platelet aggregation (includes transfused platelets!).

Hyperfrinolysis due to alteration in the fibrinolytic system.
Coagulopathy continued

**INFLAMMATION:**
Severe trauma (ISS>20 surrogate for trauma volume..37.2Trillion cells!) induces SIRS early (Systemic Inflammatory Response Syndrome)

Endothelial damage promotes early Cellular & Humoral elements of the immune system

**Cross-Talk** between the 2 systems.(all systems)

Coagulation proteases activate Complement(C4b)

Degranulating platelets release mediators which activate neutrophils & monocytes that elaborate cytokines

**NB:** patients may switch from Coagulation to a Hyperfibrinolytic state during the course of the disease

Hence goal directed therapy with Rotational ThromboElastoGraphy ROTEM
ACIDOSIS

• Shock causes **anaerobic** metabolism and lactic acidosis
• Admission Base Deficit > 6
• Lactate clearance is a useful marker of successful resuscitation
• Abramson et al observed a **100%** survival with 24 hour lactate clearance compared to only **14%** in the 24hr non-lactate clearance group
• Acidosis usually corrects itself *when* resuscitation is successful
• Buffers are rarely needed eg. bicarbonate & sometimes THAM (tris-hydroxy-methyl aminomethane) (only with **pH** < 7.2)
Damage Control Sequence

Part I – C ABCDEs (Pre-hospital or Emergency dept)
  - THEATER , HAEMOSTATIC Agents
  - or in Interventional Radiology Suite

Part II – ICU: Hypothermia, Acidosis, Coagulopathy
Hypotensive Resusc, 1:1:1 Ratio and Component Px &
Haemostatic resusc ROTEM guided
Monitor all systems CLOSELY

Part III – definitive surgery
  definitive closure
  Hernia repairs later
Patient selection guidelines

Table 1. Damage Control: Key Factors in Patient Selection

<table>
<thead>
<tr>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-energy blunt torso trauma</td>
</tr>
<tr>
<td>Multiple torso penetrations</td>
</tr>
<tr>
<td>Hemodynamic instability</td>
</tr>
<tr>
<td>Presenting coagulopathy and/or hypothermia</td>
</tr>
<tr>
<td>Complexes</td>
</tr>
<tr>
<td>Major abdominal vascular injury with multiple visceral injuries</td>
</tr>
<tr>
<td>Multicavitary exsanguination with concomitant visceral injuries</td>
</tr>
<tr>
<td>Multiregional injury with competing priorities</td>
</tr>
<tr>
<td>Critical factors</td>
</tr>
<tr>
<td>Severe metabolic acidosis (pH &lt;7.3)</td>
</tr>
<tr>
<td>Hypothermia (temperature &lt;35°C)</td>
</tr>
<tr>
<td>Resuscitation and operative time &gt;90 minutes</td>
</tr>
<tr>
<td>Coagulopathy as evidenced by development of nonmechanical bleeding</td>
</tr>
<tr>
<td>Massive transfusion (&gt;10 units packed red blood cells)</td>
</tr>
</tbody>
</table>

Torso trauma: operative techniques

ATLS principles are observed at first contact with all patients BUT now with emphasis on C THORACIC:

INCISION - Sternotomy or clam shell

Majority of the patients end up receiving definitive operations because of exsanguination
Lung:

Mode of Haemorrhage control

• 1) clamping of hilar vessels with De Bakey vascular clamps
• 2) twist the hilum to kink the major vessels
• Tractotomy or pulmonotomy using a linear stapling and cutting device to expose and ligate bleeders
• **Non-anatomic** approach recommended
• Suture the tract with non-absorbable sutures
• Packing if coagulopathy is evident
Heart:

- Finger occlusion and suture of perforations over teflon pledgets
- May temporize with skin staplers in the emergency room before theater
- Significant lacerations – may clamp the SVC and IVC and quickly apply the sutures when the hearts slows down (to avoid putting the patient on bypass machine). Remember to **vent the air** before removing the clamps from the vena cavae
ABDOMEN:

Incision - Midline.... EXTENDABLE chest & neck
Evacuate blood & clots manually
Pack all quadrants & remove packs Sequentially
   start with the most likely site or organ
Central haematoma : Aorta or IVC or Renal
Get haemostasis then attend to spielage
LIVER:
Early decision making is essential and experience is key. Adequate mobilization of the liver is important.
Pringle manoeuvre precedes - finger fracture
1) Hepatotomy and selective vascular ligation or
2) Resectional debridement and vascular ligation
3) Packing is highly recommended in many cases to avoid conglobopathy, or if it is already present
Packing technique is important
Spleen:

Splenectomy for AAST Grade III, IV and V Grades I and II may be sutured or controlled with an absorbable vicryl mesh
Pancreatico - duodenal:

Major injuries are best treated with **drainage** once **haemorrhage is controlled or by packing**

Severe duodenal injuries may be treated with duodenal stapling, ligation and exclusion

There is **no place for elaborate repairs/Whipple**

Major ductal injury are treated with drainage

Distal injuries – distal pancreatectomy and splenectomy
Small and Large intestines:

- Stapling devices, ligation and **no anastomoses** are performed (only simple one layer sutures)
- Avoid stomas and complex repairs to save time

Abdominal wall closure: vac dressings/bogota bag OR

Skin running suture
URINARY BLADDER/ URETER

Bladder: simple suture & urethral foley catheter

Severe injuries.. Drainage & Packing

Urologist will repair at next op

Ureter: repair over a stent if simple

Proximal diversion through abdominal wall for severe injuries or ligate & nephrostomy later
DEFINITIVE SURGERY

1) Careful pack removal: these should be wet irrigated first, to allow ease of removal if not covered with opsite

NB: May have to repack if haemotasis fails again

2) Repair all injuries & newly discovered ones

3) Abdominal wall closure.

If peak pressures increase by > 10 cmH2O

Resort to temporary devices again
Complication of DCS and DCR

1) Compartment Hypertension and Syndromes
   • abdomen
   • extremeties
   • intracranial
   • chest
2) Sepsis – local and systemic with ARDS FMOF
3) Enteric fistulae
4) Wound dehiscence
5) Difficult- to- treat hernias
6) ICU related complications
Damage Control Resuscitation

HYPOTHERMIA:
Rewarming in ICU and continued in theater
Warm – IV Fluids, Ventilatory circuits and adjusted accordingly to encourage pulmonary recruitment, remove wet clothes and cover with warm blankets
- **aim** for a temperature of **37° in 12-36hrs**, adequate haemodynamics, O₂ delivery, coagulation.
- *active* rewarming, e.g. with warm saline instillation into the pleural cavity
Damage Control Resuscitation continued

COAGULOPATHY:
Stop any surgically correctable haemorrhage, this may entail an unscheduled theatre trip.
Blood component therapy of 1:1:1 Rbc/Plasma/Platelets ratio or validated massive blood transfusion protocols rFVII (Boffard et al).
Tranexamic acid 1g IV bolus and 1g IV over 8 hrs
Topical haemostatic agents
New strategies: avoid coagulopathy
Avoid large ivf volume infusion & clot dislodgement & further shock

Permissive Hypotension- prehospital mental status, present pulse ; if absent... IVF to regain these.

Haemostatic Resuscitation-
1unit prbc:1plasma:1plates:1 cryoprecipitate→
Hct29%;coagulant activity=65%,plates=150-400
PROCOAGULANTS:
Procoagulants therapy –
1) \textbf{rFvii} reduced the need for blood, ARDS, MBT.
2) ProthrombinComplexConcentration (PCC)
   Has Factors II, VII, IX and X (3000 units $\rightarrow$ 40-80% normal factor activity
3) Fibrinogen Concentrate (FC) as lyophilized powder
   [concentrate] = to cryoprecipitate (1g/50 ml)
# Topical Haemostatic Agents

<table>
<thead>
<tr>
<th>Mode of delivery (commercial examples)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factor concentrators</strong>^*^</td>
</tr>
<tr>
<td>Mineral zeolite</td>
</tr>
<tr>
<td>Granules (QuikClot); mesh bags (QuikClot Sport Advanced Clotting Sponge); gauze (QuikClot Combat Gauze)</td>
</tr>
<tr>
<td>Biological polymers</td>
</tr>
<tr>
<td>Powder (TraumaDex); nylon bags (self-expanding haemostatic polymer)</td>
</tr>
<tr>
<td><strong>Mucoadhesives</strong>†</td>
</tr>
<tr>
<td>Chitosan</td>
</tr>
<tr>
<td>Granules (TraumaStat); gauze (Chitogauze PRO, Celox, Hemogrip)</td>
</tr>
<tr>
<td>Chitin</td>
</tr>
<tr>
<td>Gauze (Modified Rapid Deployment Hemostat)</td>
</tr>
<tr>
<td>Mineral-based</td>
</tr>
<tr>
<td>Granules (WoundStat, WoundSeal Powder)</td>
</tr>
<tr>
<td>Synthetic peptides</td>
</tr>
<tr>
<td>Powder (InstaClot)</td>
</tr>
<tr>
<td>Polyethylene glycol</td>
</tr>
<tr>
<td>Gel (Coseal)</td>
</tr>
<tr>
<td>Oxidised cellulose</td>
</tr>
<tr>
<td>Gauze (BloodSTOP, Surgicel Fibrillar, Surgicel Nu-Knit)</td>
</tr>
<tr>
<td>Gelatin</td>
</tr>
<tr>
<td>Foam (Sugifoam, Gelfoam, Gelfilm)</td>
</tr>
<tr>
<td>Microfibrillar collagen</td>
</tr>
<tr>
<td>Powder (Avitene Flour, Helitene, Instat); rolled sheet (Avitene Sheets, EndoAvitene); sponge (Avitene Ultrafoam, Avitene Ultrawrap, Helisstat); gel (Vitatel)</td>
</tr>
<tr>
<td><strong>Procoagulant supplementors</strong>‡</td>
</tr>
<tr>
<td>Human-derived factors§</td>
</tr>
<tr>
<td>Dry; oxidised cellulose and polyglactin matrix with thrombin and fibrinogen coating (Fibrin Pad); gauze imbedded with lyophilised fibrinogen and thrombin (Dry Fibrin Sealant Dressings); equine collagen patch with fibrinogen and thrombin (TachoSil); liquid or aerosol: fibrin sealants (Tisseel, Evicel, Crosseal); gelatin-thrombin suspension (Floseal)</td>
</tr>
<tr>
<td>Bovine-derived factors¶</td>
</tr>
<tr>
<td>Gauze (FastAct); glue (BioGlue); sponge (TachoComb)</td>
</tr>
<tr>
<td>Plant-derived factors</td>
</tr>
<tr>
<td>Powder (HemoStase MPH, Arista)</td>
</tr>
<tr>
<td>Synthetic factors δ</td>
</tr>
<tr>
<td>Solution (Recothrom)</td>
</tr>
</tbody>
</table>

The appendix lists the manufacturers of all products. ^Rapidly absorb water from blood to concentrate factors that promote clot formation. †Adhere to tissues and form a physical barrier to seal bleeding wounds. ‡Deliver procoagulant factors to bleeding wounds to promote clot formation. §Examples include fibrinogen, thrombin, calcium, and coagulation factor XIII. ¶Examples include thrombin.
MONITORING

- Close monitoring of vitals, Urine output and other organ functions
- Tertiary Survey for missed injuries
- ROTEM for coagulopathy is highly recommended
- Invasive monitoring – Swan-Ganz, Oesophageal Temperature probes and echocardiography, arterial lines
- Pharmacotherapy – inotropes
- Constant search for complications & attend to them
The beginning of the end for DCSurgery?
Successful DCResuscitation with novel strategies may in future allow another paradigm shift “TOTAL CARE again”
But...more research work needs to be done
(M.A. Schreiber)
REFERENCES:

1) Scott G. Sagraves, Eric A. Toschlog and Michael F. Rotondo. Damage Control Surgery – The Intensivist’s Role; J Intensive Care Med 2006; Vol 21(5)


4) Kaafarani HM, Velmahos GC. Damage Control Resuscitation In Trauma; Scandinavian Journal of Surgery. APRIL 28, 2014: 1457496914524388


7) Timothy E Miller. New evidence in trauma resuscitation – is 1:1:1 the answer? Perioperative medicine 2013, 2: 13


12) Spahn DR, Ganter MT. Towards early individual goal-directed coagulation management in trauma patients; British Journal of Anaesthesia 2010. 105(2) 103-105

14) Brohi K; Cohen MJ; Ganter MT; et al. Aute coagulopathy of trauma: hypoprefusion induces systemic anticoagulation and hyperfibrinolysis. Journal of Trauma, 2008, 64(5) 1211-1217
