Polytrauma and Critical Care

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Northwick Park and St Mark’s Hospital
Harrow
England
My Hospital
My Football Team
My Favourite Place
Trauma - Introduction
Introduction

- Trauma is the leading cause of death in the first 4 decades of life in most European countries.
- Third cause of death in all age groups (in 2020 – may be second cause of death)
- 3.8 million deaths / year worldwide
- 312 million injured / year worldwide
- 3 patients permanently disabled / death
Lost life years

Claire Merrick et. al. Prehospital Trauma Life Support, Mosby, 2003
Introduction

- 1 in 3 deaths occurred in hospital as a result of an injury which could have been prevented

(Royal College of Surgeons of England, 1988)
# Motor vehicle accidents prevention

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| Vehicle |       |           |

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<td>Impact-absorbing barriers</td>
<td>Emergency medical service (access to trauma system)</td>
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Road deaths fall to record lows

Road deaths have fallen to their lowest level since records began in 1928, according to figures published by the Department for Transport.

Last year a total of 2,946 people died - a 7% reduction on the previous year when 3,172 died.
Introduction

- 1 in 3 deaths occurred in hospital as a result of an injury which could have been prevented.
- Deaths occur due to failure of simple management in the early stages ("GOLDEN HOUR"), rather than a failure of complex definitive treatment.

(Royal College of surgeons of England, 1988)
Trimodal Death Distribution

- Immediate Deaths
- Early Deaths
- Late Deaths

Number of Deaths vs. Time After Injury (Hours and Weeks)
Trimodal Death Distribution

THE GOLDEN HOUR

Number of Deaths

Time After Injury

Early Deaths
Plane crash in Nebraska, 1976
1 killed, 4 injured
“When I can provide better care in the field with limited resources than what my children and I received at the primary care facility — there is something wrong with the system and the system has to be changed.”

~ James Styner, MD, FACS ~

1977
ATLS provides a common language
These guidelines seek to set achievable standards for trauma treatment services which could realistically be made available to almost every injured person in the world.

World Health Organization 2004
ATLS Concept

- ABCDE-approach to evaluation / treatment
ATLS Concept

A Airway
B Breathing
C Circulation
D Disability
E Expose
ATLS Concept

- ABCDE-approach to evaluation / treatment
- Treat greatest threat to life first
- Definitive diagnosis not immediately important
- Time is of the essence
- Do no further harm
ATLS® - Initial Assessment / Management

- Primary Survey
- Resuscitation
- Re-evaluation
- Detailed Secondary Survey
- Definitive Care
- Re-evaluation
Assessing the patient in 10 seconds

- Identify yourself
- Ask the patient his / her name
- Ask the patient what happened
Appropriate response confirms

**A** Patent airway

**B** Breathing

**C** **D** Clear thinking
Beware

- Continue to observe and have a “high index of suspicion”

- Especially if patient has
  - Maxillofacial injury
  - Soft-tissue injury of neck
  - Facial or neck burns
  - Hoarse voice (Laryngeal injury / tracheal burn)
If no response, proceed with rapid primary survey
Primary Survey

A. Airway: C-spine protection
B. Breathing: Oxygenation
C. Circulation: Stop the Bleeding!
D. Disability: Neuro Status
E. Expose: Environment & Body Temp
Airway

- Simple management manoeuvres
  - Suction
  - Chin lift
  - Jaw thrust
- Use simple equipment to help
Airway
Airway
Airway

- Simple management manoeuvres
  - Suction
  - Chin lift
  - Jaw thrust

- “Definitive airway:” Cuffed tube in trachea
ALWAYS THINK ABOUT THE CERVICAL SPINE
The reported incidence of cervical spine injury in the setting of major trauma is 1.5% - 4%.

Protecting the spine

- Immobilize entire patient on long spine board
Protecting the spine

- Apply semirigid cervical collar/head block
Airway management in trauma
Preoxygenate and BLS manoeuvres
Airway management in trauma

Preoxygenate and BLS manoeuvres

RSI, cricoid pressure, MILS
Manual in-line stabilisation (MILS)
Airway management in trauma

- Preoxygenate and BLS manoeuvres
- RSI, cricoid pressure, MILS
- Laryngoscopy
Airway management in trauma

- Preoxygenate and BLS manoeuvres
- RSI, cricoid pressure, MILS
- Laryngoscopy
- Oral intubation with GEB/introducer
Airway management in trauma

Preoxygenate and BLS manoeuvres

RSI, cricoid pressure, MILS

Laryngoscopy

Oral intubation with GEB/introducer

Success  
Failure  
Reoxygenate, retry
Airway management in trauma

Preoxygenate and BLS manoeuvres

RSI, cricoid pressure, MILS

Laryngoscopy

Oral intubation with GEB/introducer

Success

Failure

Reoxygenate, retry

Cricothyroidotomy
Airway management in trauma

Preoxygenate and BLS manoeuvres

RSI, cricoid pressure, MILS

Laryngoscopy

Oral intubation with GEB/introducer

Success

Failure

Reoxygenate, retry

ILMA
Intubating Laryngeal Mask Airway (ILMA)
Combitube

- Twin Lumen
- Distal cuff
- Proximal cuff
Combitube

98%

2%
Combitube

- Inflation of the large pharyngeal cuff of the Combitube exerts pressure against the vertebral bodies of C2, C3 and/or C4, depending on placement.
“Gold Standard” airway management for patient with potential C-spine injury requiring immediate and/or urgent airway control

- rapid sequence induction of anaesthesia
- orotracheal intubation with
  - cricoid pressure
  - manual in-line immobilization of the head and neck
- Precise cervical spine in-line immobilization should be maintained throughout the intubation manoeuvres.

Breathing

- Confirm ETT in the right place
  - Look at chest movements
    - Paradoxical
    - Asymmetric
  - Listen in the axillae
Breathing

Right main bronchus intubation
Breathing

- Confirm ETT in the right place
  - Look at chest movements
    - Paradoxical
    - Asymmetric
  - Listen in the axillae
  - End tidal CO₂
- Assess oxygenation
  - Pulse oximeter
End Tidal CO₂ Monitors/Pulse Oximeters

The StatCO2™

The Capnocheck II Hand-Held O₂/SpO₂ Monitor
Breathing

Think about

- Pneumothorax (tension)
- Haemothorax
- ETT in the wrong place (if in doubt....take it out!!)
Breathing

GIVE 100% O₂
Breathing
Oxygen delivery (VO$_2$)

- VO$_2$
Oxygen delivery (VO$_2$)

- VO$_2$ = [Hb] x 1.34
Oxygen delivery (VO$_2$)

- VO$_2$ = [Hb] x 1.34 x SaO$_2$
Oxygen delivery (VO₂)

- \( \text{VO}_2 = [\text{Hb}] \times 1.34 \times \text{SaO}_2 \times \text{CO} \)
Oxygen delivery ($VO_2$)

$VO_2 = [Hb] \times 1.34 \times SaO_2 \times CO$

$= 150 \text{ (g/dl)} \times 1.34 \times 100\% \times 5 \text{ (l/min)}$
Oxygen delivery \( (\text{VO}_2) \)

\[ \text{VO}_2 = [\text{Hb}] \times 1.34 \times \text{SaO}_2 \times \text{CO} \]

\[ = 150 \text{ (g/dl)} \times 1.34 \times 100\% \times 5 \text{ (l/min)} \]

\[ = 1000 \text{ ml/min} \]
Shock is a generalized state of hypoperfusion causing:
- Anaerobic metabolism
- Lactic acid production
- Cellular dysfunction
- Cell death
- MOF
Causes of shock

Haemorrhagic vs Nonhaemorrhagic

- Blood loss
- Tension pneumothorax
- Cardiac tamponade
- Cardiogenic
- Neurogenic
- Septic
Treatment of shock

Direct pressure

STOP the bleeding!

Operation

Splint fractures
Treatment of shock

- **Restore volume!**

\[
CO = \text{Stroke volume (SV)} \times \text{Heart rate (HR)}
\]
Treatment of shock

Starlings Law of the Heart

Stroke volume (BP, CO, UO)

Initial fibre length (CVP, PCWP, LVEDP)
Oxygen delivery (VO$_2$)

- $\text{VO}_2 = [\text{Hb}] \times 1.34 \times \text{SaO}_2 \times \text{CO}$

  = $150 \text{ (g/dl)} \times 1.34 \times 100\% \times 5 \text{ (l/min)}$

  = $1000 \text{ ml/min}$
The Rule of 2’s and 5’s

- Give 2 mls/kg fluid stat
  - If CVP goes up by 5 stop giving fluid
  - If CVP goes up by less than 5, give 2 mls/kg fluid again
  - Keep giving 2 mls/kg fluid until CVP goes up by 5 and stays up
Monitoring the Response

Identify Improved Organ Function

- CNS: Improved level of consciousness
- Renal: ↑ urinary output
- Skin: Warm, capillary refill
- Respirations: Improved rate and depth
- Vital signs: Return to normal
Treatment of shock

- **Restore volume!**
  - Vascular access

(1) The femoral vein (FV) and artery (FA) run side by side.
Treatment of shock

- Restore volume!
  - Warmed fluid
Which fluid???????

Crystalloid  v  Colloid

DOESN'T MATTER!!
If patient is losing blood....

GIVE BLOOD !!
Oxygen delivery ($\text{VO}_2$)

- $\text{VO}_2 = [\text{Hb}] \times 1.34 \times \text{SaO}_2 \times \text{CO}$

  - $= 150 \text{ (g/dl)} \times 1.34 \times 100\% \times 5 \text{ (l/min)}$

  - $= 1000 \text{ ml/min}$
Treatment of shock

Prevent hypothermia!
Disability

- Baseline neurological examination
  - GCS score
  - Pupillary response

- OBSERVE FOR DETERIORATION !!!!
E Exposure / Environment

- Completely undress the patient
- PREVENT HYPOTHERMIA !!
Multiple organ failure after trauma affects even long-term survival and functional status

*Critical Care* 2007, **11**:R95 doi:10.1186/cc6111
Multiple organ failure after trauma affects even long-term survival and functional status


- MOF increased the overall risk of death 6.0 times.
"The Golden Hour"

- Multiple organ failure after trauma affects even long-term survival and functional status. 
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- MOF increased the overall risk of death 6.0 times.

- Patients with MOF were 3.9 times more likely to require personal assistance in activities of daily living.
Multiple organ failure after trauma affects even long-term survival and functional status. 

*Critical Care* 2007, **11**:R95 doi:10.1186/cc6111

- MOF increased the overall risk of death 6.0 times.
- Patients with MOF were 3.9 times more likely to require personal assistance in activities of daily living.

**Conclusion** - the presence of MOF greatly increased mortality and the risk of impaired functional status.
Secondary Survey

- Start **only when**
  - ABCDE has been done and reassessed
  - Vital functions are returning to normal
Secondary Survey - Components

- History
Secondary Survey - Components

- History …… AMPLE

A  Allergies
M  Medications
P  Past illnesses
L  Last meal
E  Events / Environment
Secondary Survey - HEAD

- GCS Score (again)

- Full neurological exam
  - Motor and sensory and reflexes

- Full eye and ear exam
Intracranial Pressure (ICP)

- 10 mm Hg = Normal
- > 20 mm Hg = Abnormal
- > 40 mm Hg = Severe

Sustained ↑ ICP leads to ↓ brain function and outcome
Monro-Kellie Doctrine

ICP (mm Hg)

Volume of Mass

Compensation

Herniation
Cerebral Perfusion Pressure

MBP - ICP = CPP
Cerebral Perfusion Pressure

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# Cerebral Perfusion Pressure

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure (MBP) and intracranial pressure (ICP). It is a critical parameter in the management of patients with neurological conditions, as it affects cerebral blood flow and oxygen delivery.

<table>
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<tr>
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Minimize secondary brain injury

- ABCDE

- Maintain CPP
  - Maintain blood pressure (systolic > 90 mm Hg)
  - Reduce ICP
Treatment

- Maintain MAP
  - Euvolaemic fluid resuscitation
  - Isotonic fluids
  - Inotropes
Treatment

- Reduce ICP
  - Controlled ventilation
    - Goal: PaCO$_2$ at 35 mm Hg
  - Head up tilt
  - Paralysis
  - Mannitol
  - Surgery
Treatment

- Mannitol
  - Use with signs of tentorial herniation
  - Dose: 1.0 g / kg IV bolus
  - Consult with neurosurgeon first
Secondary Survey – Cervical Spine

- Tenderness
- Complete motor and sensory exam
- Reflexes
- X-ray
Normal C-Spine Xray
Secondary Survey – Cervical Spine

Only 6 vertebrae seen
Secondary Survey – Cervical Spine

Atlas fracture
Secondary Survey – Cervical Spine

C5/6 step fracture
Secondary Survey – Cervical Spine

Er !!!!!!!!
Secondary Survey – Cervical Spine

MRI of cervical fracture-dislocation with cord injury
Neurogenic shock

- Associated with cervical / high thoracic spine injury
- Neurological (not a hemodynamic) phenomenon
- Occurs shortly after cord injury
- Variable duration
- Flaccidity and loss of reflexes
- Hypotension and slow heart rate
Treat / prevent secondary injury

- Ensure adequate ventilation and oxygenation
- Properly immobilize entire patient
- Maintain blood pressure with fluid and inotropes
- Atropine as needed for bradycardia
- Avoid transfer delay!
- Methylprednisolone
**IV Methylprednisolone**

- **Blunt injury only**

- **Start within 8 hours of injury**
  - 30 mg/kg over 15 minutes
  - 5.4 mg/kg over next
  - 23 hours if started within 3 hours of injury
  - 48 hours if started within 3 to 8 hours after injury
Secondary Survey - Neck

- Type of injury Blunt v Penetrating
Secondary Survey - Neck
Secondary Survey - Neck

- Type of injury Blunt v Penetrating

- Look for increasing airway obstruction, stridor or hoarseness

- KEEP LOOKING AND LISTENING !!
Secondary Survey - Chest

- Inspect
- Palpate
- Percuss
- Auscultate
- X-rays
Secondary Survey - Chest

Haemothorax and flail
Secondary Survey - Chest

Pneumothorax
Secondary Survey - Chest

Pneumothorax
- deep sulcus sign
Chest Drain Insertion
Chest Drain Insertion
Chest Drain Insertion
Chest Drain Insertion
Chest Drain Insertion
Secondary Survey - Chest

Tension pneumothorax

This is a post mortem X-ray!!
Tension Pneumothorax

- Respiratory distress
- Distended neck veins
- Non-central trachea
- Hypotension and tachycardia
- Hyperresonance
- Absent breath sounds
Tension Pneumothorax - Treatment

- Immediate decompression
Secondary Survey - Chest

Haemothorax
Haemothorax

- At least 1500 ml blood
- Flat (not distended) neck veins
- Hypotension and tachycardia
- Absent breath sounds
- Dullness to percussion (not hyperresonance)
Haemothorax - Treatment

- Rapid volume resuscitation
- Operation
Haemothorax

Multiple stab wounds

Don’t forget the back
Secondary Survey – Perineum

- Urethral blood
- Rectal tone, integrity, blood
- Vaginal blood, lacerations
Secondary Survey - Pelvis

- Pain on palpation
- Unequal leg length
- Instability
- SIGNIFICANT BLOOD LOSS!!
Secondary Survey
Musculoskeletal: Extremities

- Contusion, deformity
- Pain
- Perfusion
- Peripheral nerve status
- Potential blood loss
- Compartment syndrome
- Fat embolus
Fat Embolism Syndrome (FES)

- Fat emboli were first noted by F.A. Zenker in 1861 in a railroad worker with a thoraco-lumbar crush injury.

- Fat embolism develops in nearly all patients with fractured bones and is asymptomatic.

- 1-3% chance of developing FES with single long bone fracture (increases with number of fractures).

- Mortality rate 10-20%.

Chest 2003;123:4
FES - pathophysiology

- 2 theories:

  - Mechanical theory:
    - Large fat droplets deposited in pulmonary capillary beds, and via a-v shunts to the brain. Causes local ischaemia and inflammation.

  - Biochemical theory:
    - Degradation of fat to free fatty acids cause vasculitis/ARDS in animals. Probably phospholipase A2 and CRP mediated.
Pathogenesis of ARDS in FES

- Fat emboli obstructs lung vessel (20 microns) platelets and fibrin adhere
- Lipase creates FFA
- Inflammatory changes --> endothelial damage --> ARDS
FES - clinical signs

- Cardiopulmonary (12-72 hrs after insult)
  - Early persistent tachycardia
  - Tachypnoea, dyspnoea, and hypoxia due to V-Q mismatch
  - ½ of pts with FES require mechanical ventilation
    (Bulger, Archives of Surgery 1997; 132: 435-9)
  - High temperature spikes
FES – clinical signs

- Dermatological (24-36 hrs after insult)
  - Reddish-brown nonpalpable petechiae over upper body, especially axillae
  - Occur in 20-50% of patients and resolve quickly
FES – clinical signs

- Subconjunctival & oral hemorrhages/petechiae
FES – clinical signs

- Neurologic (80% of patients with FES)
  - Agitated delirium
  - Stupor, seizures, or coma
  - Retinal haemorrhages with intra-arterial fat globules visible on fundoscopy
FES - investigations

- Blood (non-specific)
  - Thrombocytopenia
  - Anaemia
  - Hypofibrinogenaemia
  - hypocalcaemia

- Fat globules seen in urine, blood & sputum stained with Sudan or oil red
  - N.B. fat in urine common in all trauma patients

- Imaging
  - CXR-diffuse bilateral pulmonary infiltrates
FES - investigations

- Imaging
  - Head CT-nil or diffuse white matter petechial haemorrhages

- BAL-staining of alveolar macrophages for fat (controversial benefit)
FES - treatment

- Medical care
  - Supportive in nature
  - Maintain oxygenation and ventilation
  - Stabilize haemodynamics
  - Blood products as needed

- Surgical care
  - Early stabilization of fractures to minimize bone marrow embolization

Arch Surg 1997; 132:435-439
FES - steroids

- Steroid prophylaxis is controversial to prevent FES
- Theorized blunting of inflammatory response and complement activation
- Prospective studies suggests prophylactic steroids benefit high risk patients
- Few studies and small study size, so remains controversial
- Once FES established, steroids have not shown improved outcomes
FES – alcohol !!!!!!!

ABSTRACT:

In an analysis of the cases of 100 consecutive patients with diaphyseal fractures in the major bones of the lower limb, the incidence of fat embolism syndrome was 17%. The blood alcohol level was determined at the time of admission.

A raised level of alcohol in the blood was associated with a lower incidence of fat embolism.

R Myers and JJ Taljaard
Marmara earthquake – Turkey 1999
Crush Injury

- “Crush syndrome” first recorded in bombing of London during WWII: 5 people who were crushed presented in shock with swollen extremities, dark urine.
- Later died from renal failure.
Rhabdomyolysis - incidence

- Occurs in up to 85% of patients with traumatic injuries.

- Those with severe injury who develop rhabdomyolysis-induced renal failure have a 20% mortality rate.

- Crush injury to any part of the body (e.g. hand)

- Laying on limb for long period of time
Rhabdomyolysis - pathophysiology

- Not usually directly due to ischaemia
- Muscle sarcolemma stretch and permeability increases
- Influx of sodium, water, & extracellular calcium into the sarcoplasm
- Cells swell → reduced function → myocytic death
- Leak of intracellular metabolites into circulation
- Free radicals then cause further membrane injury
Rhabdomyolysis – symptoms and signs

- **Clinical:**
  - Muscle pain & weakness
  - dark urine (myoglobin)
  - Hypovolaemic shock (fluid moves into damaged tissue)

- **Electrolyte abnormalities:**
  - $\uparrow K^+$ (leakage)
  - $\downarrow Ca^{2+}$ (sequestered in injured tissues)
  - Metabolic acidosis
  - $\downarrow$ phosphate
  - Myoglobinemia/uria
Rhabdomyolysis - ARF

- Myoglobin precipitates (particularly in hypovolemia) and obstructs renal tubules
Rhabdomyolysis - ARF

- Myoglobin directly toxic to the renal tubular cells especially in acid urine

- At pH < 5.6, myoglobin dissociates into its 2 components
  - Globin (nontoxic)
  - Ferrihaemate (probably toxic)
Lab tests

- **Serum Creatinine Kinase**
  - Correlates with severity of rhabdomyolysis
  - Levels peak within 24h
  - Normally 145-260 U/L
  - >5000 U/L - high correlation with renal failure

- **Serum myoglobin**
  - t(1/2) 2-3 h
  - Excreted in bile

- **Myoglobinuria**
  - Dipstick will be (+) for haemoglobin, RBC’s and myoglobin
  - Microscopy: no RBC’s, brown casts, uric acid crystals
Early Treatment

- Begin early (on the field)
  - Ideally ½ NS with 100mmol/L bicarb
    - corrects acidosis
    - prevents tubular precipitation of myoglobin
    - reduces risk of hyperkalaemia
  - 10ml/h 15% mannitol
    - renal vasodilator
    - free radical scavenger
- Forced diuresis w/in 6 hrs of admission
Late Treatment
In Summary
Successful management of the trauma patient requires …

- Teamwork
- Speed
- Efficiency
- Keeping it simple
- Using your skills
- Effective
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Thank You

Any Questions?