EMST Immediate Management

Priority Plan

- 1. primary survey
- 2. resuscitation phase
- 3. secondary survey
- 4. definitive treatment & re-evaluation
- 5. transfer if appropriate

Primary Survey

- 1. Airway and Cervical spine
 - assess for patency \pm removal of foreign material, chin lift, jaw thrust
 - assume Cx spine injury in all patients with multisystem trauma
 - lateral Cx spine XRay *does not* exclue all cervical injuries

2. Breathing

- exposure of the chest
- high $F_1O_2 > 0.85$
- three commonest traumatic causes of embarrassed ventilation,
- i. tension pneumothorax
- ii. open pneumothorax
- iii. flail chest with pulmonary contusion

3. Circulation

- i. blood-volume & CO
 - conscious state
 - skin colour & perfusion
 - pulse rate & character
- ii. bleeding
 - esanguinating external haemorrhage should be immediately controlled
 - occult internal haemorrhage may be difficult to detect / control
 - MAST suit may be appropriate for abdominal/lower limb bleeding
- 4. Disability overt neurological status
 - i. level of consciousness
 - A alert
 - V responds to verbal stimuli
 - P responds to painful stimuli
 - U unresponsive
 - ii. pupillary size & response to light
- 5. *Exposure* complete exposure of the patient

• Resuscitation

- 1. supplemental O_2
- 2. IV access
 - i. minimum of 2x 16G cannulae
 - ii. blood drawn for X-match, FBE, Coags, MBA
 - iii. commence fluid replacement
 - initially with either balanced salt solution or synthetic colloid
 - use blood if > 2-3 litres in adult with no improvement
 - *type-specific* unmatched, or *O-negative* blood acceptable
 - adequacy of resuscitation judged by physiological parameters
- 3. ECG monitoring
 - HR, dysrhythmias
 - EMD → hypovolaemia, tamponade, tension pneumothorax, massive embolism: air, fat, AFE, PTE, massive MI, profound metabolic disturbance
- 4. urinary catheter
 - CUD insertion generally contraindicated when,
 - i. blood at the external meatus
 - ii. blood in the scrotum
 - iii. impalpable, or high riding prostate
- 5. naso/oro-gastric tube

Secondary Survey

Def'n: "head-to-toe & front-to-back" examination of patient

1. *Head*

i.	eyes	 pupillary size, response, EOM's, conjunctival haemorrhage visual acuity, lens dislocation, fundi/optic disc changes
ii.	ears	- TM patency, haemorrhage, CSF leak
iii.	scalp	- lacerations, haematoma

2. Maxillo-facial trauma

- when *not* associated with airway compromise, shold be deferred to definitive management
- mid-face fractures may have fractures of the *cribriform plate*
- 3. Cervical Spine / Neck
 - presume in all patients with blunt maxillo-facial trauma & multisystem trauma
 - absence of neurological deficit, pain or tenderness *does not* exclude significant injury
 - helmets should be removed with manual in-line stabilisation
 - penetrating wounds through the platysma require surgical exploration in theatre

4. Chest

- anterior, posterior, rib-cage, thoracic spine
- bone injury, soft-tissue injury, penetrating injury
- diminished apical breath sounds may be only sign of pneumothorax
- soft heart sounds / narrow pulse pressure in tamponade

5. Abdomen

- anterior, posterior, lumbar spine
- obvious swelling, penetrating injury
- consider peritoneal lavage vs CT scan
- many centres now trialling emergency ultrasound in assessment

6. *Rectum*

- anal sphincter tone
- integrity of rectal wall, presence of fractures
- position of prostate
- presence of blood

7. Fractures

- long bones, joints, digits
- pelvis, thoracic cage
- spine, cranium

8. Neurological

- CNS status AVPU
- GCS, pupillary responses
- motor & sensory evaluation of extremities
- vasomotor stability, HR, sphincter tone

9. Investigations

- i. blood X match, FBE, MBA, Coags
- ii. XRays CXR, AXR, Cx spine
 - pelvis, Tx/Lm spine, long bones
 - \pm contrast studies
- iii. DPL
- iv. U/Sound
- v. CT scan
- vi. laparotomy

Haemorrhage

- *class 1* < 15% blood volume
 minimal tachycardia, no measurable changes in BP, pulse pressure, RR, perfusion
- 2. *class 2* ~ 15-30% ~ 800-1500 ml in 70kg adult
 - \uparrow HR > 100 bpm, \downarrow pulse pressure and peripheral perfusion
 - 1 RR
 - SAP changes little, DAP increases due to \uparrow SNS tone
 - \downarrow urine output
 - many in this group will eventually require blood replacement, however this can follow colloid/crystalloid resuscitation
- 3. *class 3* ~ 30-40% ~ 2000 ml
 clinically shocked, ↑ RR, ↑ HR, ↓ BP, CNS changes
- 4. *class 4* > 40%
 - life-threatening shock & CVS collapse
 - require immediate volume resuscitation & blood ASAP
- *NB*: compensation generally prevents a fall in systolic BP until > 30% BV loss

haematocrit/Hb is an unreliable guide to volume loss

Exceptions

- 1. elderly
- 2. athletes
- 3. medications $-\beta$ -blockers
- 4. hypothermia
- 5. pacemakers

• Emergency Transfusion

1. full crossmatch

- preferrable where possible
- takes ~ 45-60/60 in most laboratories
- compatability ~ **99.95%**
- 2. *type specific* "saline crossmatched" blood
 - ABO-Rh typing plus immediate phase X-match ~ 5-10 minutes
 - compatability ~ 99.8%
 - only **1:1000** patients has an unexpected Ab found in full X-match
 - greater risk in previously transfused patients ~ 1:100 unexpected Ab
 - first choice for life-threatening shock states

3. type O Rh-negative

- universal donor, uncrossmatched blood
- some type O donors produce high titres of anti-A,B immunoglobulins

 \rightarrow *packed cells* better than whole blood

- transfusion of > 2 units of whole type O requires continued use until the blood bank determines levels of anti-A/B have declined (theoretically !)
- · continued use of type O results in minor haemolysis & hyperbilirubinaemia

4. *fluid warmers*

- iatrogenic hypothermia associated with,
- i. shivering & $\uparrow VO_2$
- ii. platelet dysfunction and coagulopathy

5. coagulopathy

- rare problem in the first hour & with less than 10 units Tx
- clotting factor deficiency less comon than,
- i. surgical bleeding
- ii. hypothermia
- iii. thrombocytopaenia
- administer FFP according to APTT/INR
- 6. *MAST trousers* see over

MAST Suit

NB: improves early haemodynamics *no* evidence of improved survival

Indications

- 1. splinting and control of pelvic fractures with continued haemorrhage / hypotension
- 2. intra-abdominal trauma with severe hypovolaemia in patients being transported to definitive care

• Contraindications

- 1. absolute
 - i. pulmonary oedema
 - ii. myocardial dysfunction
 - iii. ruptured diaphragm
- 2. relative uncontrolled haemorrhage outside of confines of suit
 - i. intrathoracic haemorrhage
 - ii. severe head injury / raised ICP

• Complications

- a. lower limb ischaemia
- b. compartment syndrome
- c. pulmonary oedema
- d. increased haemorrhage from thoracic injuries
- e. raised ICP
- f. post-deflation
 - i. hypotension
 - ii. acidaemia
 - iii. reperfusion injury

CHEST TRAUMA

• Acute Life Threatening Injuries

- a. airway obstruction
- b. tension pneumothorax
- c. open pneumothorax
- d. haemothorax ≥ 1500 ml
- e. flail chest
- f. cardiac tamponade
- g. aortic rupture
- h. air embolism broncho-pulmonary venous fistula
- *NB*: chest injuries result in ~ 1/4 trauma deaths, only ~ 15% of such injuries require operative intervention

Indications for Intercosal Tube Drainage

- 1. pneumothorax
- 2. haemothorax
- 3. empyema
- 4. bronchial rupture
- 5. oesophageal rupture
- 6. prior to transport in high risk patients

Tension Pneumothorax - Causes

- *NB*: results from "one-way-valve" air leak, either from the lung or through the chest wall
- a. IPPV & barotrauma
- b. spontaneous pneumothorax / ruptured bullae
- c. blunt chest trauma rib fractures
- d. penetrating chest trauma less often
- e. bronchial rupture
- f. iatrogenic
 - i. CVC cannulation
 - ii. pleural aspirate/biopsy
 - iii. non-functioning chest tube
- *clinical diagnosis* for which radiology is used for confirmation,
 - a. respiratory distress
 - b. tracheal deviation
 - c. unilateral diminshed breath sounds
 - d. tympanic percussion note
 - e. pulse paradoxus \pm hypotension
 - f. distended neck veins
 - g. cyanosis
- management,
 - 1. 14G IV cannula insertion 2^{nd} interspace MCL
 - 2. intercostal catheter

Open Pneumothoax

- if the opening defect is > 2/3 the tracheal diameter, effective ventilation is impaired
- management options,
 - 1. apply sterile occlusive dressing with 3/4 sides taped forming valve
 - 2. use totally occlusive dressing & insert remote intercostal catheter

Massive Haemothorax

- usually the result of > 1500 ml blood in the thoracic cavity

- 1. shock
- 2. stony dull percussion note
- 3. neck veins \rightarrow *flat*, due to severe hypovolaemia, or *distended*, due to impaired venous return
- 4. confirmed by CXR

• management,

- 1. simultaneous drainage & volume replacement
- 2. *autotransfusion* if device available
- 3. thoracotomy
 - i. initial drainage > 1500 ml
 - ii. continued loss > 200 ml/hr
- NB: thoracotomy more often required for penetrating wounds medial to the nipple line

Flail Chest

• usually 2° multiple rib fractures as a result of blunt trauma

• if large enough may result in lung pump failure,

- 1. *dominant lesion* is injury to underlying lung
- 2. rarely does flail alone result in respiratory failure
- 3. hypoventilation 2° to pain
- CXR may show multiple fractured ribs, but may miss costo-chondral separation
- paradoxical movement may be hidden by splinting in acute setting
- · AGA's required for assessment
- management,
 - 1. supplemental O₂ humidified
 - 2. volume resuscitation
 - · contused lung is sensitive to both under/over resuscitation
 - 3. pain relief
 - i. oral narcotics, mild analgesics
 - ii. systemic opioids
 - iii. interpleural catheter local anaesthetics \pm opioids
 - iv. thoracic epidural / spinal opioids
 - 4. intubation / IPPV

Cardiac Tamponade

- Beck's triad,
 - 1. hypotension
 - 2. elevated JVP
 - 3. silent heart
- elevated CVP may be absent with hypovolaemia
- distended neck veins may be obscured by a cervical collar
- initial IV resuscitation will elevate CVP and improve CO initially
- *Kussmaul's sign*, a rise in CVP with inspiration, is a true paradoxical venous pressure abnormality associated with tamponade
- · performance of pericardiocentesis pre/post-echocardiography depends upon,
 - 1. the level of suspicion that tamponade exists
 - 2. the degree of decompensation of the patient
 - NB: all patients having a positive pericardiocentesis require a thoracotomy

Chest Trauma - Delayed Major Injuries

- 1. pulmonary contusion without flail chest
- 2. myocardial contusion arrhythmias
- 3. aortic rupture / haemorrhage
- 4. ruptured diaphragm respiratory failure
- 5. ruptured bronchus bronchopleural fistula
 - pneumothorax
- 6. ruptured oesophagus mediastinitis

Pulmonary Contusion

• most common potentially lethal chest injury seen

• may be managed with or without intubation, but require observation due to progression over the first 24-48 hrs

- factors indicating a need for *early intubation*,
 - 1. impaired level of consciousness
 - 2. pre-existing cardiopulmonary disease
 - 3. any injury dictating anaesthesia/operation
 - i. associated abdominal injury requiring laparotomy
 - ii. skeletal injuries requiring immobilization
 - iii. craniotomy ICH, raised ICP and monitor insertion
 - 4. renal failure

Myocardial Contusion

- incidence ~ 65% of major blunt chest trauma
- site affected,
 - a. RV ~ 65%
 - b. LV ~ 15%
 - c. both ~ 20%
- clinical presentation,
 - a. unexplained hypotension
 - b. unexplained elevation of CVP/JVP
 - c. acute cardiac failure
 - d. arrhythmias
 - e. new murmur VSD, MV rupture
 - f. tamponade

investigations,

a.	ECG changes	- ischaemic changes in RV leads
		- multiple ectopics, sinus tachycardia, AF
		- RBBB, ST/T wave changes
		± myocardial infarction
b.	high CK/CK _{MB}	* poor correlation
c.	troponin I	? sensitivity / specificity
d.	gated nucleotide scans	= "gold standard"

Traumatic Aortic Rupture

- most common major vessel injured following blunt chest trauma
- common cause of immediate death in MVA, ~ 90% fatal at the scene of accident
- 50% of surviving patients die per left untreated
 - a. 60% rupture just distal to the origin of the left subclavian arteryat the level of the *ligamentum arteriosum*
 - b. 25% rupture at the ascending aorta
 - just proximal to the origin of the brachiocephalic artery
 - c. characteristic pathophysiology of *contained haematoma*
 - i. initial BP fall associated with loss of 1000-1500 ml
 - ii. hypotension responds to IV volume replacement
 - *NB*: ∴require high index of suspicion & radiological exclusion
- Diagnosis
 - 1. CXR * may be essentially *normal*
 - * no single finding reliably predicts or excludes significant injury
 - i. widening of the superior mediastinum
 - ii. fractures of the 1^{st} & 2^{nd} ribs
 - iii. blurring of the left margin of the aortic knuckle
 - iv. tracheal shift \rightarrow right
 - v. left pleural cap | effusion
 - vi. elevation & right-shift of the RMB
 - vii. depression of the LMB
 - viii. obliteration of the space between PA and aorta
 - ix. oesophageal deviation (NGT)
 - 2. angiography
 - "gold-standard" for diagnosis
 - high index of suspicion, ∴high false negative rate
 - LIGW, "if negative angiography rate < 50%, then not doing enough angiograms"
 - 3. CT scan
 - 4. TEE
 - NEJM 1995, completed studies in 93/101 within 29 ± 12 min
 - 11 positives \rightarrow sensitivity = 100% specificity ~ 98%
 - additional information LV function, valvular competence - tamponade
 - but operator dependent & blind spots in ascending aorta & other arteries
 - transthoracic echo of *no use* in diagnosis

■ <u>Management</u>

- 1. surgical repair
- 2. post-operative paraplegia ~ 5%

Diaphragmatic Rupture

- *NB*: high index of suspicion in the patient with major abdominal injuries, modes of *presentation*,
 - i. acute respiratory failure
 - ii. bowel sounds in left hemithorax
 - iii. peritoneal lavage fluid "disappears", or drains from chest tube
 - iv. bowel / NGT in hemithorax on CXR
 - v. failure to wean from mechanical ventilation

• CXR

- a. loops of bowel in *left hemithorax*
- b. "pseudo-haemopneomothorax" = air/fluid level of stomach
- c. persistent elevated left hemidiaphragm
- d. NG tube in left hemithorax
- *NB:* may be *mimmicked* by (misinterpreted as),
 - i. elevated left hemidiaphragm
 - ii. acute gastric dilatation
 - iii. loculated pneumothorax
 - iv. subpulmonic haematoma

Tracheobronchial Tree Injuries

Laryngeal Injury

- a. history of injury
- b. may be minimal external tissue damage
- c. hoarseness
- d. subcutaneous emphysema
- e. crepitus
- *NB:* in the presence of *acute obstruction* oral intubation should be attenpted, failing this *surgical tracheostomy* is the procedure of choice, *not* cricothyroidotomy

Bronchial Rupture

- a. sites
 - i. lower trachea ~ 80% transverse
 - ii. major bronchus spiral
 - iii. post. tracheal wall vertical
- b. presentation
 - may be insidious
 - high suspicion in major chest trauma
 - i. haemoptysis major or minor
 - ii. subcutaneous emphysema
 - iii. tension pneumothorax
 - iv. acute bronchopleural fistula
 - v. persistent lobal collapse
- *NB*: majority of patients with this injury die at the scene, of those who survive to hospital, *mortality* ~ 30%, usually due to associated injuries
- diagnosis may be confirmed by bronchoscopy
- · large leaks may require insertion of a second intercostal catheter
- · management may be conservative or by operative repair

Oesophageal Rupture

• clinical presentation,

- a. penetrating, most common, or severe closed chest trauma
- b. retrosternal pain
- c. dysphagia
- d. haematemesis
- e. cervical emphysema

f.	CXR:	 left pleural effusion, haemothorax widened mediastinum mediastinal emphysema, air/fluid level cervical emphysema hydropneumothorax / pneumothorax
g.	pleural aspirate	- pH < 6.0 - bloody fluid, high WCC - high amylase
h.	late events	- fever, shock, septicaemia

• confirmatory diagnosis is by gastrograffin swallow or endoscopy

• there is a high associated mortality $\sim 2\%$ / hr left untreated

• aetiology,

a.	traumatic	- external
		- internal / vomiting

- b. iatrogenic
- c. FB
- d. carcinoma

Coma: Causes of Respiratory Failure

- a. airway obstruction
- b. aspiration
- c. acute neurogenic pulmonary oedema
- d. post-obstructive pulmonary oedema
- e. thoracic injuries
 - i. tracheobronchial disruption
 - ii. pulmonary contusion
 - iii. haemothorax / pneumothorax
 - iv. flail chest
 - v. diaphragmatic disruption
- f. acute gastric dilatation
- g. cervical cord trauma
- h. central hypoventilation

ABDOMINAL TRAUMA

Peritoneal Lavage

- a. sensitivity ~ 95-98%
- b. specificity ~ 85%
- c. positive result
 - i. *aspiration*
 - fresh blood ≥ 10 ml prior to lavage
 - faecal soiling or vegetable material
 - ii. *lavage*
 - fluid exits via intercostal or urinary catheter
 - iii. analysis
 - RBC count
- $> 100,000/\mu l$

 $> 500/\mu l$

- $> 50,000/\mu l \equiv$ eqivocal
- > 5,000/µl for penetrating injuries

- WCC
- causes of *false positives* ~ 15%
 - a. traumatic lavage
 - b. retroperitoneal haemorrhage
 - c. pelvic haematoma -2° fractures

• causes of *false negatives* ~ 2-5%

- a. incorrectly performed
- b. diaphragmatic rupture
- c. retroperitoneal injuries haemorrhage
 - duodenum
 - pancreas
 - renal injury
- d. isolated hollow viscus perforation small bowel - bladder

Indications

- a. multiple trauma patient in whom abdominal examination is,
 - i. equivocal
 - unreliable CHI, intoxication, cord injury
 - iii. impractical prolonged XRays, angiography
 - requiring GA
- b. unexplained fluid requirements in resuscitation
- c. penetrating injuries including lower thoracic
- d. gunshot wounds

• Contraindications

ii.

- a. full bladder
- b. pregnancy
- c. recent abdominal surgery
- d. obvious signs of intraperitoneal haemorrhage/infection

NB: the only *absolute contraindication* is an existing indication for laparotomy

• Compications

- a. haemorrhage
- b. intestinal perforation
- c. bladder perforation
- d. infection

Peritoneal Lavage - Technique

- a. empty bladder, sterile technique, IV access
- b. dialysis catheter introduced into pelvis via sub-umbilical incision
- c. aspiration for frank blood
- d. 1000 ml of normal saline introduced over 5 minutes + ballotment
- e. fluid drained sent for,
 - i. red & white blood cell counts
 - ii. urgent gram stain & culture
 - iii. amylase
 - iv. ? cytology

Abdominal CT Scanning

- suggested as a substitute for DPL in haemodynamically stable patients
- · criticism of DPL is indication for laparotomy in cases of minor liver laceration

• several clinical studies suggest double contrast CT may miss life-threatening intra-abdominal injuries

- a. diaphragmatic tears
 - most commonly 5-10 cm length, involving posterolateral left hemidiaphragm
 - initial CXR is usually non-specific
 - high association with wedge #L1, (b) and (c)
- b. duodenal rupture
- c. pancreatic injury
 - normal serum amylase *does not* exclude pancreatic fracture
 - conversely, may be elevated from non-pancreatic sources

Penetrating Trauma

- *NB*: an aggressive policy for exploratory laparotomy is justified, due to the high incidence of hollow visceral injury & vascular involvement
- 1. gunshot wounds
 - entry of the peritoneum mandates laparotomy
 - in the absence of an exit wound, AXR required to establish trajectory
 - broard spectrum antibiotic therapy early
- 2. stab wounds
 - selective laparotomy procedure wound exploration

- DPL

- < 50% of such injuries will require urgent laparotomy
- 3. lower thoracic wounds
 - defined as
 line from 4th ICS anteriorly to 7th ICS posteriorly
 nipple line to inferior border of scapula
 - incidence of significant abdominal injury with penetrating injury,
 - i. stab wound $\sim 15-25\%$
 - ii. gunshot wound $\sim 45-60\%$
 - : laparotomy for all gunshots & sleective for stab wounds
- 4. flank & back wounds
 - risk of significant visceral injury following panetrating,
 - i. back wounds $\sim 5-15\%$
 - ii. flank wounds $\sim 20-30\%$
 - routine laparotomy is the safest policy, as there are no reliable tests

Genitourinary Tract

Blunt Trauma

- increased incidence of injury with,
 - 1. renal
 - i. back / flank haematomas, ecchymoses
 - ii. fractures of lower ribs
 - iii. fractures of spinal transverse processes
 - b. bladder/urethra
 - i. perineal haematomas
 - ii. anterior pelvic fractures
 - c. overt signs of lower tract injury
 - i. blood at the urethral meatus
 - ii. inability to void

• urethral disruptions are divided into,

a.	posterior	 above the urogenital diaphragm usually multisystem trauma. pelvic fractures
b.	anterior	below the urogenital diaphragmusually starddle injuries & isolated

• imaging techniques,

1. IVP

•	unilateral non-function	- congenital absence, previous nephrectomy
		maggiva paranahymal shattaring

- massive parenchymal shattering
- vascular pedical disruption
- 2. urethrography
 - should be performed prior to CUD in all suspected urethral tears
- 3. cystography
- 4. CT scan

Pelvic Fractures

- open pelvic fractures \rightarrow *mortality* > 50%
- rectal & genital injuries should be suspected in all major fractures
- DPL should be performed, preferrably from *above* the umbilicus, due to extension of haematoma
 - a. negative DPL reliably excludes major intraperitoneal bleeding
 - b. positive DPL $\sim 15\%$ FP, due to leaking into the peritoneum

• a MAST suit may be used if there is haemodynamic instability

- continued bleeding then become a therapeutic dilemma,
 - a. if DPL is grossly positive, then laparotomy indicted to exclude co-existent abdominal pathology
 - b. arteriography / embolization may be life-saving for uncontrolled haemorrhage

HEAD TRAUMA

• head injury is associated with,

- a. $\sim 50\%$ of all trauma deaths
- b. $\sim 60\%$ of MVA deaths
- Def'n: coma is defined as,
 - 1. no eye opening = 1
 - 2. not obeying command = 1-5
 - 3. no word verbalisation = 1-2

virtually all patients with GCS < 8 and most with GCS = 8 are comatose

Def'n: head injury is arbitrarily divided according to GCS as,

1.	severe HI	GCS < 9
2.	moderate HI	GCS = 9-12
3.	minor HI	GCS > 12

• other factors considered as severe HI, despite GCS,

- a. unequal pupils > 1 mm difference
- b. unequal motor response
- c. open HI CSF leak, exposed brain tissue
- d. neurological deterioration
- e. depressed skull fracture

• a change in GCS \ge 2 represents clear deterioration, \ge 3 major deterioration requiring immediate assessment & therapy

other factors of concern,

- a. increased severity, or unusually severe headache
- b. unilateral increase in pupil size
- c. unilateral onset of weakness

Imaging Techniques

- a. CT scan
 - examination of first choice in all but trivial injuries
- b. SXR
 - · limited value in early management, except in penetrating injuries
- c. examination with limited/no role in acute head injury
 - i. LP
 - ii. EEG
 - iii. isotope scanning

<u>Skull Fractures</u>

- a. linear, non-depressed
 - across vascular arterial grooves or suture lines increases the risk of *extradural haematoma*
- b. depressed
 - increased risk of sequelae (eg seizures) in depressed > thickness of skull
- c. open

ii.

• early operative intervention, elevation & removal of fragments & closure of the dura

d. basal skull fractures

- internally compound, and loss of CSF may be occult into sinuses
- factors suggestive of diagnosis,
- i. Battle's sign mastoid ecchymoses
 - raccoon eyes bilateral periorbital ecchymoses
 - associated with cribiriform plate fracture
- iii. CSF leak rhinorrhoea, otorrhoea
- iv. haemotympanum

Diffuse Brain Injury

a. concussion

- is a brain injury accompanied by brief loss of neurological function
- various neurological abnormalities may be described, however, these have usually resolve by the time a tertiary institution is reached
- .:. *any* neurological abnormality observed in a patient *should not* be attributed to concussion
- rule of thumb is that if the patient has been unconscious for > 5 minutes then they should be observed for 24 hrs

b. *diffuse axonal injury*

- characterised by prolonged coma, lasting days to weeks
- overall mortality ~ 30%
- autonomic dysfunction, fever, hypertension, sweating etc is common

Focal Injuries

- a. contusions
- b. haemorrhages
- c. haematomas
- **NB:** operative intervention usually only required for mass effect

• Meningeal Haemorrhage

•

1. extradural haemorrhage

relatively rare

- most commonly middle meningeal artery, rarely dural sinus
- usually 2° linear fracture of parietal/temporal bones
 - $\sim 0.5\%$ of unselected HI
 - $\sim 0.9\%$ of HI resulting in coma
- classical description of progress,
- i. LOC followed by lucid interval
- ii. secondary depression of conscious state
- iii. development of contralateral hemiparesis
- iv. ipsilateral dilated pupil
- outcome is directly related to the condition of the patient prior to surgery
- i. light coma \rightarrow ~ 9% mortality
- ii. deep coma \rightarrow ~ 20% mortality
- 2. subdural haemorrhage
 - much more common than extradural $\sim 30\%$ of severe HI
 - most commonly rupture of *bridging veins*, less often cortical arteries or brain
 - underlying primary brain injury is often severe
 - poor prognosis \rightarrow mortality ~ 60%
 - recent studies suggest some improvement of outcome with early evacuation
- 3. subarachnoid haemorrhage
 - blood in CSF \rightarrow meningeal irritation, headache, photophobia, etc.
 - LP not required \rightarrow CT scan

• Parenchymal Haemorrhage

- 1. intracerebral haematomas
- 2. impalement injuries
- 3. bullet wounds

SPINAL TRAUMA

Demographics

a.	age	~ 70-80% are between 11-30 yrs
b.	sex	~ 2/3 are <i>males</i>
c.	mortality	 ~ 30% die before reaching hospital ~ 10% during the first year ~ normal for age thereafter

• although semi-rigid cervical collars are useful, securing the ehad to a spianl board is equally, or more effective

• a conscious patient with paralysis is usually able to identify pain at the site of injury due to sensory loss below the level

NB: paralysis/sensory loss may mask abdominal or lower extremity injury

- in unconscious patients 2° MVA or a fall, chance of Cx spine injury ~ **5-10%**
- risk of Cx spine injury in unconscious patients increases with,
 - 1. flaccid areflexia
 - 2. flaccid rectal sphincter
 - 3. ability to flex, but *not extend* the elbow
 - 4. grimaces to pain above, but not below the clavicle
 - 5. hypotension with bradycardia & dilated veins
 - 6. priapism

Vertebral Assessment

- a. usually associated with pain & tenderness
- b. less often palpable step-deformity
- c. oedema / ecchymoses
- d. tracheal tenderness / deviation retropharyngeal haematoma
- e. muscle spasm \pm head tilt

Neurological Assessment

- a. motor power
- b. tone
- c. reflexes
- d. sensory deficit
 - light touch is conveyed in both lateral and posterior columns & may be the only modality preserved in incomplete injuries
 - sparing of sensation in the *sacral dermatomes* may be the only sign of incomplete injury
 - evaluation of sacral sparing should include sensory perception and voluntary contraction of the anus
- e. autonomic dysfunction bladder / rectal control, priapism

• Neurogenic & Spinal Shock

- 1. neurogenic shock
 - hypotension associated with high thoracic & cervical injuries
 - hypotension, bradycardia & dilated veins \rightarrow "relative hypovolaemia"
 - atropine may be used to Rx bradycardia

2. spinal shock

- refers to neurological function of the spinal cord following injury
- "shock" may result in almost total non-function despite viability of the cord
- produces flaccid paralysis, cf. normal spasticity, brisk reflexes & \uparrow plantars

Fractures / Dislocations

- 1. *C1 Atlas*
 - usually involves a blow-out of the ring \rightarrow *Jefferson* fracture
 - asociated with axial load
 - 30% have associated C2 fracture
 - usually *not* associated with cord injury
 - they are unstable & require immediate immobilization
- 2. *C2 Axis*
 - i. dislocation
 - odontoid may be displaced posteriorly into the spinal canal
 - injury to the transverse ligament, between odontoid & anterior arch of C1
 - consider whenever C1-arch to odontoid distance > 5 mm
 - displacement can occur without injury \rightarrow Steel's rule:

"1/3 of the area in the atlas is occupied by odontoid, 1/3 by spinal cord"

- ii. odontoid fractures
 - *type I*: above the base & stable
 - *type II*: through the base & usually unstable
 - *type III*: extends into the vertebral body
 - NB: in children under 6 yrs the epiphysis may appear as a fracture line cf. type II fractures
- iii. posterior element fractures \rightarrow "hangman's fracture"
 - posterior elements damaged by flexion & distraction
 - unstable

- 3. *C*3-*C*7
 - assess distance from anterior aspect of C3 to pharyngeal shadow
 - \rightarrow prevertebral thickness < 5 mm
 - increased thickness "without" fracture classically seen with minimally displaced C2 fracture
 - "rule-of-thumb" for prevertebral haematoma is the distance to the air-shadow should be $< \frac{1}{2}$ the vertebral body thickness
 - radiological evidence identifying an unstable fracture,
 - i. *disruption* of all of either anterior or posterior elements
 - ii. *over-riding* of a superior vertebral body > 3.5 mm
 - iii. *angulation* between vertebral bodies $> 11^{\circ}$

4. facet dislocations

- unilateral facet injury \rightarrow vertebral displacement ~ 25% of body width
- bilateral facet injury \rightarrow vertebral displacement > 50% of body width
- malalignment of spinous processes on AP film
- bilateral dislocations frequently unstable

Pathophysiology

- · injury results from both primary and secondary injury
- the anatomic and histological findings associated with *primary injury*,
 - a. direct neurilemmal & neuronal disruption \pm destruction
 - b. petechial haemorrhages
 - c. gross haematomyelia
 - d. total cord transection * a rare event
- subsequent secondary injury involves,
 - a. progressive haemorrhagic necrosis
 - b. oedema
 - c. inflammatory response
 - *NB*: \rightarrow proportional to the extent of the 1° injury
- the proposed mechanism of the 2° injury includes,
 - a. activation of *phospholipase* A_2 , due to release of
 - i. Ca++
 - ii. bradykinin
 - iii. thrombin
 - b. formation of arachidonic acid & other FFA's from cell membrane
 - c. metabolism of arachidonic acid to,
 - i. prostaglandins * mainly *thromboxane*
 - ii. leukotrienes → microcirculatory thrombosis & stasis vasogenic oedema tissue ischaemia chemotaxis of inflammatory cells
 - d. free radical formation & hydrolysis of membrane lipid fragments

\rightarrow lipid fragment peroxides

e. lipid hydrolysis and peroxidation of fragment membrane phospholipids

 \rightarrow further release of Ca⁺⁺ & positive feedback

- f. increased $PGF_{2\alpha}$ and thrombin augment phospholipase activity
- g. raised intracellular Ca⁺⁺ leads to disordered energy metabolism and maintenance of cell integrity (Na⁺/K⁺-ATP'ase)
- h. increased endogenous kappa opioid agonist *dynorphin*, plus an increase in receptor binding capacity following experimental SCI in rats

• Effects on Spinal Cord Blood Flow

• immediately following SCI there is a marked *reduction* in SCBF, resulting in ischaemia and biochemical changes as above

- these changes may not commence for up to 1-4 hrs post SCI
- therefore postulated that interruption of the above cascade may protect against ischaemia
- the normal mean *SCBF* ~ 40-50 ml/100g/min
- this is partitioned between grey & white matter ~ 3:1
- SCBF normally *autoregulates* between ~ 60-150 mmHg MAP in rats
- SCBF has been shown to vary with $P_{aCO2} \sim 1:1$ ratio (1 ml/mmHg)
- most of the decrease in SCBF following SCI is in the central cord region
- work with cats has shown that autoregulation is *abolished* following SCI

Management of Acute SCI

- a. pharmacological
 - i. steroids
 - given before, or shortly after decrease 2° injury in animals
 - Braken (1990) showed high dose methylprednisolone improved motor and sensory function at 6 weeks & 6 months
 - benefit is statistically significant only when administered $\pounds 8 hrs$ of SCI
 - there was no increased incidence of septic complications
 - subsequent RCT's have not suported this finding and use currently controversial
 - ii. mannitol
 - effective in reducing parenchymal volume
 - also causes a vigorous osmotic diuresis
 - intravascular volume must be maintained to ensure SCB
- b. spinal cord perfusion
 - following experimental SCI *autoregulation* is lost \rightarrow *pressure passive*
 - hypotension leads to cord hypoperfusion & ischaemia
 - · hypertension leads to increased oedema and haemorrhage
 - therefore the aim is to maintain *MAP* ~ *normal*
- c. experimental*
 - hypothermia
 - hyperbaric oxygen
 - catecholamine antagonists
 - dimethyl sulphoxide
 - naloxone (opioid antagonism)
- **NB:** *none of these has consistently demonstrated a benefit in human clinical trials

Associated Problems

Airway Management

NB: any patient with a significant *closed head injury* potentially has a fractured *cervical spine*

• *neutral position* must be maintained during intubation

• non-incremental traction without radiological control does not protect against further injury

• blind nasal & fibreoptic intubation may be attempted only if base of skull fracture can be excluded, however both tend to produce coughing & bucking which may be deleterious

- NB: RSI & oral intubation are indicated in the presence of,
 - i. complete apnoea
 - ii. associated head injury with GCS < 9
 - iii. an uncontrollable patient

• <u>Respiratory Complications</u>

- 1. *anoxia/hypoxia* is the most common cause of death in acute SCI
- 2. *pneumonia* is the 2nd most common cause of death
- the degree of respiratory embarrassment depends upon SCI level
- *phrenic paralysis* ($C_{3,4,5}$) arises with *lesions* ³ C_4 , leaving only the accessory muscles
 - \rightarrow severe hypoventilation
- intercostal & abdominal paralysis results in significant reduction in pulmonary function ${}^{\mathbf{s}}\mathbf{T}_7$
- pulmonary oedema, *DVT & PTE*, also contribute significantly to early mortality

• *pulmonary oedema* has been seen in up to 44% of patients following resuscitation from spinal shock

• this most likely results from over-enthusiastic volume resuscitation, and attempts to maintain a "normal" arterial BP

• Cardiovascular Complications

1. acute changes

- in experimental SCI there is an abrupt, brief (2-3 min) increase in MAP, ? due to sympathoadrenal outflow
- this is associated with significant increases in CBF/ICP, BBB permeability, extravascular lung water, CVP, PAP, PAOP, and CO
- this supports the tendency for these patients to develop *cerebral & pulmonary oedema* early in resuscitation
- rarely seen by the time of admission to a 3° centre

- 2. *hypotension* \equiv^{t} "neurogenic shock"
 - varying degrees of hypotension, bradycardia, decreased TPR, low-normal CVP and a normal or slightly elevated CO
 - decreased myocardial function, with \downarrow LVSWI (~ 26%) and CI (~ 18%) in response to volume loading in patients for spinal stabilisation surgery
 - loss of the cardioaccelerator fibres (T₁₋₄) produces *bradycardia*
 - ? the Bainbridge reflex (decreased RAP) may contribute as bradycardia is seen in below T_4 SCI
 - lesions ^a T_1 leave only the Frank-Starling mechanism to increase contractility, and may produce a MAP < 40 mmHg
 - the **b**-endorphin surge with SCI may also depress contractility by either a direct action on the heart, or by centrally mediated increases in parasympathetic tone
 - *orthostatic reflexes* are absent & positioning important
 - severe hypotension is observed above a *critical level* ~ T_{6-7}
 - this phase may last days to weeks but is usually *less than* the period of flaccid muscle paralysis
 - cautious addition of fluid is recommended in view of the decreased CVS reserve and tendency to *oedema formation*
 - monitoring by PAOP is frequently indicated as the venous compliance curve is abrupt in the absence of resting tone

3. *autonomic hyperreflexia*

- this follows the phase of hypotension/flaccid paralysis in patients with *lesions* 3 T_{6.7}, usually at 1-3 weeks
- MAP returns to ~ normal or below, with episodes of severe hypertension in
 ~ 85% of patients
- triggered by common noxious stimuli, bladder or rectal distension, labour or surgical pain
- this generalised response begins *below* the level of the lesion, due to the loss of control from the higher centres
- it may spread above the lesion due to *sympathetic divergence*
- symptoms include nasal congestion, severe headache, dyspnoea and nausea
- signs include pallor, sweating, intense somatic & visceral muscle contraction, & piloerection below the lesion
- above the lesion there is flushing & severe hypertension with reflex bradycardia
- SAH & retinal haemorrhages have been observed, with syncope, convulsions and death if unabated
- management has included ganglionic blockers, catecholamine storage depletion, α -adrenergic blockade, and direct vasodilators
- however the studies have been small & lacked controls
- the main aim is to *avoid* known stimuli

4. arrhythmias & ECG abnormalities

- mid thoracic SCI results in sinus or nodal bradycardia ± PAC's, PVC's, AV dissociation, or ventricular tachyarrhythmias
- *atropine* is usually effective for bradyarrhythmias, which are frequently seen with airway manipulations
- **b**-*blockers* may be useful for ventricular tachyarrhythmias
- the ECG frequently shows LV strain ± subendocardial ischaemia
- similar arrhythmias are seen in ~ 75% of autonomic hyperreflexic episodes

• Other Systems

1. genitourinary

- ARF may occur 2° to hypotension, dehydration, sepsis, nephrotoxic drugs, acute obstruction, associated renal trauma, or other factors
- in the chronic phase of SCI, renal failure accounts for ~ 20-75% of mortality

2. *disordered thermoregulation*

- afferent information to the hypothalamus may be interrupted
- sympathetic denervation causes heat loss
- inability to shiver reduces heat production
- general tendency to become *poikilothermic*

3. fluid & electrolytes

- chronic SCI patients tend to be *hypovolaemic & anaemic*
- hypercalcaemia and hypercalcuria follow immobilisation, especially in young male patients (peak ~ 10/52 post-SCI)

4. gastrointestinal complications

- ~ 20% of SCI patients develop *GIT bleeding* acutely
- nonspecific liver dysfunction with a normal bilirubin occurs commonly
- gastric distension & ileus are common
- increased risk of regurgitation / aspiration

5. suxamethonium hyperkalaemia

- may be seen as early as **3 days**
- the magnitude of the rise is more a function of the muscle mass affected than the amount of drug given
- the underlying overgrowth of receptors may occur well *before* spasticity replaces flaccid paralysis
- pretreatment with a nondepolarising agent *does not* reliably prevent the occurrence of significant hyperkalaemia

Management

• between 25-65% of SCI patients have associated problems, most commonly,

- 1. head injury
- 2. thoracic trauma
- 3. abdominal trauma
- 4. major skeletal trauma

• these may compromise respiratory or circulatory function coincident with spinal shock and require a high index of suspicion

• during the acute phase, maintenance of "normal" acid-base & blood gas parameters and adequate cord perfusion are paramount

• experimental animal work has shown *no advantage* in either hypercapnia or hypocapnia in neurological recovery or histological tissue damage

• although not statistically significant, there is some date to suggest hypercapnia is more harmful than hypocapnia

• therefore, should aim for a $P_{aCO2} \sim 35-40$ mmHg and *hypoxaemia* should be avoided at all costs

- contributing factors such must be suspected and managed accordingly,
 - 1. pulmonary contusion
 - 2. pneumothorax, haemothorax
 - 3. pulmonary embolism (fat or thrombus)
 - 4. foreign body
 - 5. gastric aspiration
 - 6. non-cardiogenic pulmonary oedema

• similar to the findings for CNS ischaemia, an elevated *plasma glucose* has been shown to be deleterious upon neurological outcome

• mild to moderate increases of BSL ≤ 2.5 mmol/l, tripled the incidence of paraplegia in rabbits following aortic occlusion

• notably there was a *lack of correlation* between the degree of BSL rise and the extent of neurological injury

• therefore, as for head injury, the administration of dextrose containing fluids should be restricted to proven hypoglycaemia

• the present data are insufficient to recommend active reduction of an elevated plasma glucose

• Cole (1989) looked at various anaesthetic techniques following SCI in the rat

• of the techniques studied, halothane, fentanyl, N_2O , and SA lignocaine, *all* increased the duration of ischaemia required to produce SCI

• no one technique was superior in terms of final *neurological outcome*

THERMAL INJURIES

Management

- 1. airway compromise
 - clinical indications of inhalation injury,
 - i. facial & neck burns
 - ii. singeing of the eyebrows, eyelashes or nasal hair
 - iii. carbon deposits /inflammatory changes in oropharynx
 - iv. carbonaceous sputum
 - v. history of impaired mentation
 - vi. fire in an enclosed environment
 - vii. arterial COHb level
 - any suggestion of significant airway injury supports early intubation & ventilation
- 2. ongoing burning
 - remove all clothing
 - cool burnt area with body temperature saline
- 3. estimate surface area & depth of burn
- 4. IV access
 - upper limbs preferrable due to high incidence of saphenous phlebitis
 - Brook \rightarrow Hartmann's @ 4 ml/kg/%burn in first 24 hrs give half in first 8 hrs, remainder over 16 hrs *in addition* to usual fluid requirements *starting from the time of injury, not assessment
 - all patients with > 20% burn require immediate IV access & IVT replacement
 - formulas are *guides*, ∴ regular assessment of patient essential
 - hourly urine output probably best guide in otherwise healthy patients
- 5. baseline investigation
 - i. blood FBE, XMatch, AGA's/COHb, EC&U
 - ii. CXR
- 6. analgesia
- 7. wound care
 - do not open blisters to apply antiseptic
 - avoid extensive use of cold soaks
 - apply clean dressings to painful second degree burns
- 8. antibiotics
 - *not indicated* in the early post-burn period
- 9. tetanus prophylaxis
- 10. oral fluids
 - no contraindication with small burns < 10%

• Circumferential Burns

- the salient point in maintenance of the *peripheral circulation*
 - 1. remove rings & bracelets
 - 2. assess status of peripheral circulation
 - cyanosis, impeired capillary refill
 - progressive neurological signs
 - doppler ultrasound
 - 3. escharotomy
 - theoretically can be done in ED without anaesthesia, but there would seem little justification for this
 - incision must be entire length of eschar
 - consider bilateral midaxillary escharotomy for circumferential thoracic burns
 - fasciotomy is seldom required unless complicated injury or electrical burn

• Chemical Burns

- alkali burns are generally more serious as alkalis penetrate tissue more deeply
- factors determining severity include,
 - 1. duration of contact
 - 2. concentration of agent
 - 3. amount of agent / area of contact
- main principal of management is copious irrigation
- Electrical Burns
 - 1. high tension / lightning injuries
 - tissue thermal injury/necrosis \propto Joule's Law: Heat \propto I² x W
 - electrical flash burns and flame burns 2° to clothing ignition
 - 2. electrocution
 - disruption of normal physiological function
 - tissue thermal injury
- frequently more serious than appear externally
- muscle, nerve & blood vessels may be destroyed with sparing of the skin due to its high resistance
- *rhabdomyolysis* may be severe enough to result in acute renal failure, : require aggressive fluid resuscitation of evidence of pigmenturia
- fasciotomy may be required for compartmental syndrome

Burns Unit Transfer Criteria

- 1. partial thickness burns > 20%
- 2. partial thickness burns > 10%

+ ages > 50 yrs or < 10 yrs

- 3. full thickness burns >5%
- 4. partial / full thickness burns involving,
 - i. face, eyes, ears
 - ii. hands, feet, major joint
 - iii. genitalia, perineum
- 5. electrical burns / lightening burns
- 6. chemical burns
- 7. complicated injuries, ie. fractures, where the major risk of morbidity is from the burn
- 8. inhalational burns
- 9. lesser burns in patients with significant pre-existing disease

Cold Injury

• Classification

1. frostbite i. first

ii.

iii.

iv.

- first degree hyperaemia & skin oedema without necrosis
 - second degree partial thickness necrosis with vesicle formation
 - third degree full thickness skin, plus some underlying tissue necrosis
 - fourth degree full thickness skin, muscle & bone with gangrene
- 2. nonfreezing injury
 - due to microvascular endothelial injury, with stasis & vascular occlusion eg., "trench foot", "immersion foot"
- 3. hypothermia
 - states with a core temperature $< 35^{\circ}C$
 - see notes on hypothermia

HYPOTHERMIA

Def'n:	core temperature $< 35^{\circ}C$					
	hom	eotherms	s and regulate core temperature	~ 36-37.5°C	(T.Oh)	
				$\sim 37 \pm 0.4^{\circ}C$	(RDM)	
	1.	mild	> 33°C			
	2.	severe	< 33°C			

NB: demarcation is arbitrary, but effects more pronounced & loss of compensation

Aetiology

a. extremes of *age*

b.	debilitating <i>illness</i>
----	-----------------------------

	i.	CNS	CVA, head injury, neoplasmprogressive mental deterioration
	ii.	CVS	- CCF, MI, PVD, PTE
	iii.	infections	- septicaemia, pneumonia
	iv.	renal	- uraemia
c.	expos	sure	 environment IV fluids irrigating fluids
d.	drug	S	 alcohol, GA, vasodilators antipyretics chlorpromazine
e.	endo	crine	 hypothyroidism panhypopituitarism Addisonian crisis, hypoglycaemia diabetes, hyperosmolar coma, ketoacidosis (~ 20%) protein / calorie malnutrition
f.	spina	l cord traun	na
g.	skin (diseases	- psoriasis, icthyosis, erythroderma

h. *iatrogenic* - induced hypothermia & inadequate rewarming

Clinical Effects

Cardiovascular

1.	↑ syı	mpathetic to	ne	- ↑ p	lasma NA/AD and FFA's		
2.	initia	lly \rightarrow	vasoo	vasoconstriction, tachycardia & increased CO			
	<i>later</i> \rightarrow bradycardia		cardi	a, hypotension & decreased CO			
3.	- mainly 2		nly 2°	30-40% at 30°C $\propto \downarrow VO_2$ 2° to <i>bradycardia</i> , SV well preserved y perfusion well maintained			
4.	ECG	changes	- exa	cerbat	ted by <i>acidosis & hyperkalaemia</i>		
	i.	bradycardi	a				
	ii. prolonged		PR, QRS, QT duration		T duration		
	iii.	J point elev	vation		~ 33°C		
	iv.	AF			~ 25-34°C (commonest arrhythmia)		
	v.	AV block		1° 3°	~ 30°C ~ 25°		
	vi.	VF			~ 28°C		
	vii.	asystole			~ 20°C		

- 5. CPK & LDH levels are elevated
 - ? leakage from cells or microinfarction

<u>Central Nervous System</u>

• reasonably well preserved to 33°C, below this function deteriorates progressively,

- 1. initial confusion \rightarrow coma \leq 30°C with pupillary *dilatation*
- 2. \downarrow CBF $\propto \downarrow$ CMRO₂ ~ **6-7%** / °C ~ similar change cf. whole body VO₂
- 3. progressive brainstem depression $\rightarrow \downarrow$ HR & \downarrow RR
- 4. \downarrow temperature regulation \rightarrow
 - \downarrow shivering $\leq 33^{\circ}C$
 - loss of T control $\leq 28^{\circ}$ C
- 5. cerebral protection
 - over and above metabolic depression
 - deep circulatory arrest
 - recovery from near drowning

Pulmonary Changes

1.	central depression $\rightarrow \downarrow RR \le 33^{\circ}C$					
	~ 4 bpr	$m \pm respiratory arrest at 25^{\circ}C$				
2.	impaired cough & gag reflexes –	→ aspiration risk				
3.	reduced CO ₂ drive					
4.	no change in hypoxic drive					
5.	impaired hypoxic pulmonary vasocon	impaired hypoxic pulmonary vasoconstriction				
6.	\downarrow FRC, increased atelectasis					
7.	\downarrow gaseous diffusion capacity					
8.	\uparrow VO ₂ with <i>shivering</i> $\rightarrow \downarrow$	$VO_2 \leq 33^{\circ}C$				
9.	$\downarrow O_2$ availability $\propto \uparrow$	HbO_2 affinity				
10.	increased gas solubility					
	i. $\uparrow \alpha CO_2 / \downarrow P_{aCO2} \rightarrow \uparrow$	pH				
	-	rate of rise of F_A/F_I & elimination halothane MAC _{27°C} ~ 50% MAC _{37°C}				

Metabolic

- 1. $\downarrow VO_2$ ~ 6-7% / °C
- 2. severe *acidosis* \rightarrow HbO₂ curve shifts to the *right*
 - i. respiratory $\downarrow CO_2$ elimination due to hypoventilation
 - ii. metabolic \downarrow tissue perfusion
 - \downarrow hepatic lactate clearance
 - \downarrow renal tubular H⁺ excretion
 - iii. temperature correction of blood gas values offer no advantage in management

 \rightarrow $\delta pH \sim -0.0147/^{\circ}C$

3. hyperkalaemia / hypokalaemia

- causes for expected rise in K^+
- i. decreased activity Na⁺/K⁺-ATPase $\rightarrow \quad \downarrow Na^+ / \uparrow K^+$
- ii. cellular hypoxia, membrane damage & acidosis
- however, hypokalaemia more commonly observed
- i. $? 2^{\circ}$ diuresis
- ii. ICF shift
- 4. *hyperglycaemia* ↓ insulin secretion & ↓ peripheral glucose utilisation ? mild pancreatitis
 - hypoglycaemia may ensue in longstanding hypothermia
- 5. \uparrow drug t_{1/2} \propto \downarrow hepatic blood flow & enzyme reaction rates

 \rightarrow heparin, citrate & lactate

Renal

- 1. \downarrow GFR $\propto \downarrow$ renal blood flow $\sim 50\%$ at 30°C $\rightarrow \downarrow$ drug clearance
- 2. \downarrow tubular function
 - i. cold diuresis volume of urine initially increased or the same
 - ii. hypoosmolar urine
 - iii. glycosuria, kaluria \rightarrow additional diuresis

<u>Neuromuscular Junction</u>

- 1. shivering occurs $\sim 33-36^{\circ}C$
- 2. increased muscle tone \rightarrow *myoclonus* ~ 26°C
- 3. increased sensitivity to *both* depolarising & nondepolarising with mild hypothermia

Haematological

1.	coagulopathy	
	i. \downarrow coagulation	\downarrow enzyme activity
	ii. thrombocytopaenia	↑ portal platelet sequestration↑ bleeding time
2.	increased blood <i>viscosity</i>	- haemoconcentration - \downarrow microcirculatory blood flow
3.	immunoparesis	- \downarrow WCC & function
4.	marrow hypoplasia	

Immunological

- 1. \downarrow neutrophils, phagocytes, migration, bactericidal activity
- 2. organ hypoperfusion & increased infection risk
- 3. diminished gag/cough reflexes
- 4. atelectasis

Regulation of Body Temperature

NB: balance between heat production and heat loss

- a. heat production / gain
 - i. basal VO_2
 - ii. SDA of food
 - iii. muscular activity
 - iv. non-shivering thermogenesis
 - v. gain from the environment

b. heat loss

i.	radiation	~ 40%
ii.	convection	~ 30%
iii.	evaporation	~ 29%
iv.	conduction,	
	feces/urine	~ 1%
respiratory losses		~ 10%

i.	humidification	~ 8%
1.	numumeation	0/0

ii. convection $\sim 2\%$

Sensory Systems

NB:

- a. cutaneous thermoreceptors $\sim 15\%$ of input
 - i. cold receptors $< 24^{\circ}C$
 - ii. heat receptors $> 44^{\circ}C$
- b. deep/core thermore ceptors $\sim 85\%$ of input
 - i. anterior hypothalamus
 - ii. spinal cord
 - iii. hollow viscera

• Central Integration

• some processing in the spinal cord, majority in the *posterior hypothalamus*

- "central thermostat" regulated by,
 - 1. diurnal rhythm, age, sex, hormones
 - 2. endogenous pyrogens
 - 3. drugs
 - 4. neurotransmitters (? 5HT)
 - 5. exercise

Effector Systems

- 1. higher control centres
 - i. posture, avoidance behaviour
 - ii. apetite/hunger
 - iii. clothing
 - iv. level of activity \rightarrow voluntary muscle metabolism $\uparrow VO_2 \le 10x$ with exercise

2. *cutaneous blood flow*

- first line of defence activated against heat loss
- especially the extremities, cf. normal may decrease
- i. skin blood flow $\sim 5\%$
- ii. heat loss to $\sim 12\%$

3. shivering thermogenesis

- involuntary incoordinate muscular activity ~ 50 Hz
- may \uparrow VO₂ ~ 2-5x
- may \uparrow core temperature ~ 2-3°C/hr
- requires $\uparrow VO_2 \sim 100\% / \uparrow 1^{\circ}C$

4. nonshivering thermogenesis

- increased combustion of FFA's and glucose, regulated by,
- i. sympathoadrenal outflow \rightarrow fast response noradrenaline
- ii. thyroid function \rightarrow slow response adrenaline & T₄
- liver and skeletal muscles in adults $\sim 25\% \uparrow VO_2$
- *brown fat* in neonates $\sim 100\% \uparrow VO_2$ ~ 25% of total CO
- 5. *sweating*
 - direct or reflex stimulation of the spinal cord, medulla, hypothalamus or cortex
 - provides only coarse control of temperature
- 6. horripilation / piloerection minimal effects in man cf. animals
- *NB*: usually order of activation,
 - i. behavioural modification
 - ii. vasoconstriction
 - iii. nonshivering thermogenesis
 - iv. shivering thermogenesis

Effects of Anaesthesia

• Unintentional Hypothermia

1.	↓ he i. ii.	eat <i>production</i> $\downarrow VO_2 \sim 25-30\%$ $\sim 1 \text{ kcal/kg/hr}$ $\downarrow \text{ muscular activity & shiveri}$	Ωœ
•		•	18
2.	he	eat <i>losses</i>	
	i.	\uparrow radiation / convection	undressed in cold theatrelarge surgical incisions
	ii.	↑ evaporation	 cold preparation solutions from the wound cold/dry anaesthetic gases bypassing of upper airway
	iii.	\uparrow conduction	
		• cold IV solutions	- 1 kcal/°C/1 \rightarrow ~ 17 kcal / 1000ml / 20-37°C ~ 1% of BMR
		• cold table (minimal) & we	drapes

3. inhibition of *thermoregulation*

- i. \downarrow hypothalamic set point
- ii. inhibition of effector responses
 - vasoconstriction & NST only means of heat gain available
 - GA \rightarrow vasodilatation & redistribution of heat decreased core-shell gradient responsible for the initial rapid fall ~ 0.5-1.5°C
 - RA → similar initial loss of core-peripheral gradient central regulation preserved this *is* the origin of *shivering* with epidural blockade (?? not spinal)

4. *at risk* groups

- i. neonates high SA:V ratio, immature thermoregulation, no shivering
- ii. elderly $-\log BMR$, $\downarrow body mass$
- iii. prolonged procedures
- iv. large central incisions
- v. burns
- vi. trauma patients, large volume transfusions/blood loss
- vii. \downarrow metabolism adrenal insufficiency, hypothyroidism, hypopituitarism

Perioperative Effects

- 1. protection against CNS ischaemia, even with mild hypothermia (Sano et al. 1992)
- 2. metabolic acidosis, hyperkalaemia
 - decreased drug metabolism
- 3. haematological $-\uparrow$ viscosity, $\downarrow O_2$ delivery & tissue hypoxaemia - impaired coagulation
- 4. CVS $-\downarrow$ CO & arrhythmias (*AF)

5. postoperative problems

i. ii.	shivering marked vasoconstriction	 ↑ VO₂ & hypoxia if borderline lung function decreased microvascular flow ? graft survival & wound infection haemodynamic instability on rewarming
iii. iv.	impaired drug clearance impaired immune function	- naemodynamic instability on rewarning - predisposes to <i>wound infection</i>

v. impaired conscious level

Intraoperative Management

↑ am	bient temperature			
i.	adults under cove	$\sim 21^{\circ}C$		
ii.	neonates	≤26°C		
radia	nt warmers	mainly useful in children (higher SA:V ratio)limited by accesspotential for burns		
drape	es / coverings	 ↓ radiant & convective losses <i>area</i> more important than type, but must remain <i>dry</i> losses from the head important in neonates/bald adults * forced air convective warmers most effective means 		
warn	ning blankets	 most effective <i>above</i> patient, minimal losses to table useful when patient < 10 kg 		
respi	ratory losses	< 10% losses through the respiratory tract		
i. heat & moisture		exchangers prevent most of this loss		
ii. heater humidifier		rs will prevent all of this loss		
		lults are <i>unable</i> to significantly raise body heat content ng otherwise actually looking at oesophageal probe changes		
iii.	heater humidifiers	s rarely, if ever, indicated in adults		
	i. ii. radia drape warn respi i. ii.	 ii. neonates radiant warmers drapes / coverings warming blankets respiratory losses i. heat & moisture e ii. heater humidifiers however, in ac studies showing 		

6. blood / IV warmers - especially large volumes given rapidly

<u>Monitoring During Anaesthesia</u>

a.	central	B	\rightarrow \rightarrow	heart brain
b.	rectal	intermediatechanges lag behind core/shell	ll duri	ing cooling & warming
c.	shell	skin/peripheralmay estimate vasoconstricto	r/vaso	odilator responses
NB:	useful to measure both core & shell,			

core-shell gradient	\rightarrow better assessment of overall body temperature
	\rightarrow adequacy of rewarming & predicts "afterdrop"

Deliberate Hypothermia

• Surface Cooling

• principally historical interest, main use currently is in the management of *malignant hyperthermia*, or severe hyperthermia in septic ICU patients

- cold environment, ice bathing, especially groins & axillae
- problems of slow & uneven effects both during cooling and rewarming,
 - a. 2-6°C *afterdrop* when cooling / rewarming
 - b. *uneven* effects mean some tissues are still "at risk" for ischaemia

• Cardiopulmonary Bypass

- a. more rapid & even cooling / rewarming
- b. more precise temperature regulation
- c. maintenance of *tissue perfusion* despite \downarrow CO / arrest
- d. combined with *haemodilution*
 - i. offsets the effects on viscosity
 - ii. "optimal Hct." ~ 18-22%

• Deep Hypothermia & Total Circulatory Arrest

- a. allows operation on still & bloodless heart
- b. principally for correction of complex CHD
- c. current operative times ~ 50-60 minutes at 18-20°C
- d. need for more thorough longterm outcome studies on CNS effects

PAEDIATRIC TRAUMA

- ratio of blunt:penetrating trauma highest for paediatric group
- MVA's and falls account for ~ 80% of trauma
- *multisystem* injury is the rule, rather than the exception

• Unique Characteristics

- 1. smaller size greater force per unit area
- 2. skeletal immaturity
 - soft bones with active growth centres can absorb large amount of energy without fracture
 - · higher incidence of internal organ damage without overlying fracture
 - $\ \ \, \text{ includes spinal column } \ \ \, \rightarrow \quad \text{SCIWORA}$
 - open sutures < 18 months
- 3. surface area
 - thermal energy loss higher
 - absorption of toxins higher
 - systemic effects of burns greater
- 4. GCS modified for age
- 5. higher incidence of
 - i. seizure activity
 - ii. mass lesions
 - iii. white matter tears frontal and temporal lobes
 - especially infants < 6 months
 - iv. subdural haematomas especially NAI
- 6. major blood loss with *hypotension* may be *concealed*
- 7. IV access often more difficult
- 8. acute gastric distension \rightarrow NG tube
- 9. psychological immatuirty
- 10. long-term effects
 - growth & deformity
- 11. equipment
 - specific equipment required \rightarrow size for age
 - *not* small adults

Shock

- a. normal blood volume ~ 80 ml/kg
- b. estimated weight
 - i.< 9 years $\sim (2 \text{ x age}) + 9$ ii.> 9 years $\sim 3 \text{ x age}$
- c. systolic arterial pressure $\sim 80 + (2 \text{ x age})$
 - cuff width 2/3 upper arm

Vital Signs			
	HR	SAP	RR
Infant	160	80	40
Preschool	140	90	30
Adolescent	120	100	20

Response to Blood-Loss			
	BL < 25%	BL 25-40%	BL > 40%
CVS	 ↑ HR weak pulse 	 ↑ HR • BP drop with tilting 	 frank hypotension ↑ or ↓ HR
CNS	lethargic, irritableconfused, combative	 ↓ LOC diminshed response to pain 	• comatose
Skin	 cold, clammy mottled	 cyanotic ↓ capillary refill 	• pale, cold
Renal	 ↓ urine output high SG 	• minimal urine output	• no urine

• initial fluid challenge ~ 20 ml/kg, or 25% blood volume, for colloid, 3x for crystalloid

- if remain unstable, then give 10 ml/kg P-RBCs
- most common acid-base abnormality is *respiratory acidosis*, correctable by adequate ventilation
- sites for emergency IV acces,
 - 1. intra-osseus tibial needle
 - 2. median antecubital vein
 - 3. long saphenous at ankle
 - 4. CVC IJV or subclavian

• Chest Trauma

- a. majority blunt
- b. underlying *pulmonary contusion* / haemorrhage most common significant injury
- c. tension pneumothorax / haemothorax are less common cf adults, but may be rapidly lethal if unrecognised
- d. injuries relatively rare in paaediatric group,
 - i. fractures ribs
 - ii. diaphragmatic rupture
 - iii. injury to great vessels

• Abdominal Trauma

NB: all should have stomach decompressed by oro/nasograstric tube

- a. high incidence of visceral injury
- b. more difficult to assess cf adults
- c. limited role for DPL \rightarrow *CT* scan
 - CT must be immediately available
 - not delay diagnostic algorithm
 - suit must have resuscitation facitlity
- d. majority of solid visceral injuries are managed *conservatively*
 - but require adequate observation
- e. indications for operating on spleen/liver trauma,
 - i. failure to respond to resuscitation
 - ii. continued major haemorrhage > 40 ml/kg/24 hrs
 - iii. suspicion of associated hollow visceral injury
 - iv. severe concomitant HI, where haemodynamic instability is deleterious
- f. many liver/spleen injuries can be repaired, rarely perform *splenectomy*

• Extremity Trauma

- a. presence of growth plates makes assessment difficult
 - XRay of the opposite limb often useful
- b. blood-loss associated with long-bone & pelvic fractures is proportionately more significant
- c. *physeal* fractures classified by Salter-Harris
 - i. type I linear through growth plate
 - ii. type II cf. type I, plus small chip of metaphysis
 - iii. type III through growth plate & epiphysis
 - iv. type IV through both metaphysis & epiphysis
 - v. type V compression fractures
 - types I & II have best prognosis for normal growth
 - type V has worst prognosis & difficult to spot on XRay
- d. greenstick fracture fracture of cortex only
 - most require reduction
- e. buckle fracture angulation without cortical fracture
- f. supracondylar fracture
 - high propensity for neurovascular injury
 - high incidence of growth deformity

• <u>Head Trauma</u>

- a. children < 3 years have worse outcomes following severe HI
 - cf. older children who generally recover better than adults
- b. small children may develop hypovolaemic shock 2° head injury alone, or associated scalp laceration
- c. children < 18 months with open sutures have increased tolerance of expanding intracranial masses
 - check for fontanelle bulging and suture diastasis
- d. vomiting is a common response to injury and may, or may not, equal raised ICP
- e. seizures occurring shortly after injury are usually self-limiting
 - recurrent seizures require Ix & Rx
- f. focal lesions are less common
- g. generalised oedema and raised ICP is more common

h. GCS, verbal score modified for age,

i.	appropriate words social smile		
	fixes and follows	=	5
ii.	cries but consolable	=	4
iii.	persistently irritable	=	3
iv.	restless, agitated	=	2
v.	none	=	1

- i. indications for ICP monitoring in children
 - i. GCS < 5, or motor scores < 3
 - ii. where raised ICP is known, or likely to develop, and signs are masked by neuromuscular paralysis
 - iii. multiple associated injuries, where CT scanning delayed
 - ICP monitoring/management does not improve outcome in global ischaemic events
 - near-drowning victims with \uparrow ICP / \downarrow CPP have poor prognosis, and maintenance of 'normal' ICP does not correlate with outcome
- j. child drug doses

i.	diazepam	~ 0.25 mk/kg

- ii. phenytoin ~ 15-20 mg/kg
- iii. mannitol $\sim 0.5-1.0 \text{ g/kg}$

Spinal Trauma

- paediatric spinal trauma is relatively rare $\rightarrow ~ \sim 5\%$ of all spinal injuries
- of children with severe trauma ~ 5% will have a cervical spine injury
- injuries will occur at more than one spinal level in ~ 16% of cases
- the commonest causes are,
 - a. road trauma MVA, pedestrian, cyclist
 - b. falls especially diving
- anatomical differences include,
 - a. interspinous ligaments & joint capsules are more flexible
 - b. uncinate articulations are poorly developed & slide forward
 - c. the facet joints are flat
 - d. the vertebral bodies are wedged anteriorly & slide forward with flexion
 - e. the head is relatively large
 - \rightarrow greater angular momentum can be generated with flexion / extension

- normal radiological variations include,
 - a. anterior displacement of C_2 on $C_3 \rightarrow$
 - i. ~ 40% of children < 7 yrs
 - ii. ~ 20% of children \leq 16 yrs
 - iii. $\pm \ge 3$ mm movement on flexion/extension
 - b. increased distance between the dens and anterior arch of $C_1 \sim 20\%$ of children
 - c. skeletal growth centres may resemble fractures
 - d. basilar *odontoid synchondrosis* appears as a radiolucent line at the base of the dens (especially \leq 5 years)

• spinal cord injury without radiographic abnormality, *SCIWORA* is almost unique to the paediatric age group

~ 20-60% of all SCI~ 30-50% of these the lesion is complete

• SCI in the first decade of life is,

- a. almost exclusively at $C_{1/2}$
- b. either subluxation or SCIWORA and severe cord injury
- c. rarely associated with fractures

• a high proportion of children who die in MVA's, or suffer cardiorespiratory arrest prior to reaching hospital have cord trauma above C_3 , particularly at the *cervico-medullary junction* • this is difficult to diagnose in the unconscious patient, signs including,

- a. flaccid immobility & areflexia
- b. hypoventilation with paradoxical chest movement
- c. apnoea and rhythmic flaring of the alae nasi (above C_3)
- d. hypotension with inappropriate bradycardia - peripheral vasodilatation ± priapism

Spinal Shock

• the syndrome of spinal shock occurs more commonly in children,

- a. SCI lesion resolves after 2-3 days
- b. progressive return of reflexes bulbocavernous & anal first
- c. incomplete lesions may then become apparent
 - i. Brown-Sequard hemisection
 - ii. anterior cord lesion
 - iii. central cord lesion

Non-Accidental Injury

- a. physical
- b. sexual and emotional abuse
- c. deprivation of medical care and nutrition

· children are also intentionally poisoned, and endure the consequences of inadequate supervision

• diagnosis of children who suffer from abuse or neglect is difficult

• NAI should be suspected where,

- a. an injury is unexplained
- b. the history is not consistent with the type of injury
- c. it is alleged that the injury was self-inflicted
- d. relatives delay in seeking medical aid
- e. there are repeated suspicious injuries

• the history is rarely volunteered by the child

- the pattern of physical findings can be helpful,
 - a. head injury skull fractures - subdural haematomas
 - b. retinal haemorrhages occur with head shaking, but also have other causes
 - c. bruises and scars on the back and buttocks in different stages of development and of unusual shapes
 - d. burns from cigarettes or forced immersion in hot water
 - e. overt bone fractures or *healing fractures*
 - f. long-bone fractures in children < 3 years
 - g. injury to genital or perianal areas

• when non-accidental injury is suspected, referral to a specialised child protection unit to enable appropriate counselling and intervention is helpful

· safety of siblings must be considered