Anaesthesia, Trauma and Critical Care
Course Manual 2014

Frontmatter

Editors

Dr Mark Forrest

BSc Hons MB ChB FRCA FICM
Consultant in Anaesthetics, Critical and Prehospital Care
Warrington & Halton Hospitals NHS Foundation Trust
ATACC Medical Director
Medical Director NW Police Firearms Collaboration
Cheshire Fire & Rescue Service Medical Director
UK

Dr Peter Lax

MB ChB (Hons.), Dip RTM, Dip IMC RCSEd, FRCA, RAF
Squadron Leader
Anaesthetics/Intensive Care Medicine SpR, Royal Air Force Critical Care Air Support Team
Tactical Medical Wing, RAF Brize Norton, Oxfordshire
UK

Dr Jason van der Velde

BAA, MBChB, M.Sc. (DM), MCEM
Prehospital Emergency Medicine and Critical Care Retrieval Physician
Clinical Lead HSE National 24 hour Emergency Telemedical Support Unit
Cork University Hospital
ATACC Assistant Medical Director
West Cork Rapid Response Medical Director
Ireland

Contributors

Dr Carin Dear   Consultant Anaesthesia and Prehospital Care, Southampton MTC, UK

Dr Hugh Doran   GP and Prehospital Emergency Medicine, East Cork, Ireland

Mr Ian Dunbar   International Extrication & Rescue Specialist, Holmatro, Holland

Dr John Glasheen   Critical Care Retrieval Physician, Sydney HEMS, Australia

Ms Jennie Grainger   Surgical SpR, RCS link, Cheshire, UK

Dr Peter Larcombe   Consultant Anaesthetist and Neuro Intensive Care, Brighton MTC, UK

Prof Mike Mcnicholas   Trauma & Orthopaedic Surgeon, Aintree MTC, UK

Dr Jenny Parsons   Anaesthetics and Intensive Care SpR, Dundee, Scotland

Dr Geoff Roberts   Deputy Coroner, Cheshire, UK

Dr Phil Russell   SpR Anaesthetics and Prehospital Emergency Medicine, UK

Mr Noaman Sarfraz   Consultant Surgeon, Pakistan Army (Ret), Cheshire, UK

Paul Savage OBE   Clinical Operations Manager RNLI, Chairman UKSAR Medical Group

Dr Marc Scott   Senior Consultant Anaesthetist, Amsterdam Medical Centre, Holland

Miss Brigid Wall   Advanced Paramedic, Queensland Ambulance Service, Australia

Dr Craig Williams   Consultant Anaesthetics and Intensive Care, Swansea, Wales

Mr Lee Winterbottom   Paramedic, Course Director, Frontier Medex, UK

Ms Dawn Wood   Senior NHS Manager, Trauma & Orthopaedics, Cheshire, UK

Copyright

 @ATACCFaculty

 ataccmanual@gmail.com

 www.atacc.co.uk

Foreword and FOAMed

Welcome to the new ATACC 'Virtual' trauma manual. All six editions of our previous manual have been a printed text and whilst far more current than many traditional texts, we were always left somewhat frustrated that once it had reached print, that elements were already out of date.

15 years ago, ATACC made a commitment to be as up to date as we possibly could be and we constantly update our lectures and educational content. We have also been committed to utilising every new educational modality or tool and this continues with our increasingly immersive simulations and innovative skill stations.

In this manner the world of FOAMed (Free Open Access Meducation) has given us the ideal platform and opportunity for us to similarly update our manual and textbook content and as a non-commercial body, ATACC can launch this into the open forum of the FOAM community, with free access to all.

Whilst there are many outstanding blogs, Twitter feeds and podcasts that we all follow on a regular basis, as far as we are aware, this is the first Virtual Textbook in the FOAM community and we are hugely excited about its potential.

As it stands we know that it is still not perfect and as such this will be an on-going project. We aim to keep the text as current as possible with regular re-writes and updates in response to new research, best practice and feedback from the FOAM community. We hope that you embrace it and support it with your comments.

We also hope that it will encourage many individuals to come and see how on the course we bring together the knowledge with the safe, practical delivery of high quality trauma care. It may also inspire others to develop their own local up to date training courses, moving away from outdated tick box courses.

Enjoy the text and we would be delighted to receive feedback, comment and discussion as this is very much a 'work in progress' and will continue to evolve and improve. We will also hopefully see you on an ATACC course very soon!

Table of Contents

N.B. Due to formatting differences between the iBooks and PDF versions of the manual the page numbers below refer to pages as viewed in your PDF viewer, which are usually one higher than the numbers displayed on the pages themselves.

Table of Contents

1

Introduction to ATACC

Welcome to the Virtual ATACC Manual
8th edition: 2014

Trauma remains one of the major killers in our modern society. Improved medical care enables us to sustain life in the face of many serious conditions but until we reach 40-50 years of age, trauma is the greatest risk to life.

If we total up all of the lost life years, from cancer, heart disease and stroke premature deaths, then it will still not be equivalent to the number lost as a result of trauma. It is a killer of all age groups, but especially the young.

Even for survivors, without delivery of a high standard of care, at the earliest opportunity, they can be left with severe life-changing long term disability. As a result the impact on patients, families, the health service and us all, can be considerable with huge costs to everyone.

In light of these facts there is a constant drive to improve trauma care standards, but for many years there was little real measurable improvement. In the UK, despite several reports in the past and more recently the 2007 NCEPOD 'Trauma Who Cares' document and the subsequent National Audit Office 2011 report, it was clear that despite increasing levels of ATLS training, improvements in paramedic skill sets, greater understanding of the complexities of trauma and recent wartime medical experience, little had actually improved in terms of outcome.

What was required was a national structure for managing trauma and also training that moved rapidly with the times, which could easily be adapted, modified and enhanced as knowledge expanded. From this the UK National Trauma Network was established and modern trauma courses such as ATACC, European Trauma Course and the Emergency Trauma Medicine course became far more popular, offering a far more current and advanced level of training, including newer concepts and methods, such as effective crew resource management.

Since 1997, ATACC has gone to considerable lengths to keep our course as up to date as possible and the educational con-

tent is reviewed and updated between every single course. As such, it remains academically far ahead of many rival courses and the Faculty are always keen to here of anything new that enthusiastic candidates may have also seen or read.

In many areas of trauma, we are still far from having all the answers and no area is a better demonstration than that of trauma induced coagulopathy. We now understand far more about coagulation in trauma, but it just seems to complicate the whole picture still further, without providing all of the necessary answers or solutions. This is typical of many aspects of trauma care and presents courses and instructors with a dilemma, as there is often no clear answer to many questions. Faced with such challenges, some courses just simplify the issue and offer an old or basic solution, whereas ATACC will do its utmost to present all of the current best evidence and then suggest what we consider the best current solution, often just based on the educated opinions of key players in trauma in the UK and around the world.

The ATACC Faculty aim to offer the latest and most advanced trauma methods, taught in a stimulating and innovative manner, using all the modalities that we can to develop team skills, trauma skills and an appreciation of relevant human factors.

History

Back in 1997, Dr Mark Forrest was approached by the Head of the Mersey School of Anaesthesia to address the issue of trauma training for the local anaesthetic & critical care community.

As an ATLS instructor he realised that there were many very good qualities to this course, but it was Americanised, surgically biased and it was proving difficult for it to keep up with the changes and developments that were occurring within trauma practice. To be fair ATLS aimed to do nothing more than provide a robust background of trauma skills for clinicians who may face major trauma on an infrequent basis and have to manage it in a systematic fashion as part of a trauma team.

It was never designed to be the course for leading major trauma centres and as such 'advanced' may not be the best title, but as an initial introduction it was a good start. Sadly, as our approach to trauma has progressed, a number of the methods and principles of ATLS have proved incorrect, but these were slow to be changed, resulting in many disillusioned candidates and Faculty members. As such ATACC offered a fresh alternative, with s similar approach but an ongoing commitment to update the educational material before every course. Over the last 15 years the Faculty

have achieved that and the current best practice and evidence is reflected in this course manual.

In addition to the science, trauma is typically managed by a multi-disciplinary team, many of whom were excluded from the ATLS course. ATACC took a far more inclusive view, as everyone has a part to play in the Trauma Team on the patient journey from 'Roadside to critical care'

The first course ran in late 1997, produced and written by local enthusiastic consultants in Merseyside. Since then, the Faculty has grown to several hundred and includes many of the leading trauma clinicians in UK hospitals and Major Trauma Centres. Thousands of candidates from around the world have completed the course from over 20 different countries.

ATACC has developed still further and we now adopt many unique and ground breaking teaching modalities. In the last 12 months alone, we have introduced Laerdal SimPad as a tool for our rolling, on going assessment process, an additional six major scenarios on day 3 for the trauma teams, a large scale, half day major incident for MERIT training and our new Virtual World immersive simulator.

ATACC remains a non-profit making organisation with all income reinvested in the course and trauma care, in support of the emergency services.

Demand for the course has spilled over into these emergency services and the ATACC course has now become a multi-level integrated package with suitable levels for every provider, who can then progress up to ATACC, which remains the most advanced trauma course that we are aware of in Europe or even worldwide. These levels are indicated by the badges and titles overleaf:

- **FTACC (First Aid)**

- **BTACC (Basic Trauma & Casualty Care)**

- **RTACC (Rescue Trauma & Casualty Care**

ATACC (Anaesthesia Trauma and Critical Care)

The ATACC Course

The course runs over three very long days, but planning for each starts over 3 months before and is almost continuous throughout the year. The Faculty give up their time for free because of their passion for trauma care and as a body they are hugely knowledgeable.

As a candidate the course may seem exhausting with its long and intense timetable, but there is still plenty of time to relax in the evening, meet the Faculty and make new friends from around the world.

After the course candidates often reflect and in their feedback, they will frequently comment on the huge complexity and sheer scale of the course and the major scenarios that we create. This is all down to an incredible amount of hard work by the Faculty and also the tremendous Facilities in our adopted course 'home' at the Cheshire Fire and Rescue Headquarters. A partnership for which we are hugely grateful.

Those of you who are invited to come back and join the Faculty as Instructor Candidates will quickly get some idea of the scale of work and effort put in by the instructors, as this course rarely has many bodies sat around in the Faculty room.

In summary, if you like managing major trauma then you will love ATACC. If you want to know more, or the latest methods, principles and concepts, then you will love ATACC. If you want to challenge your skills and work with a diverse range of trauma care providers from firefighters, firearms officers, trauma surgeons, anaesthetists, nurses and many more, then you will love ATACC.

During our Royal College Accreditation one of the assessors admitted that he was unsure about non-medics being on the course, but when he saw how the teams work in such diverse conditions as prehospital, resus rooms and many other areas and how they gel, share and cross over skills he was truly stunned and give his overwhelming support to the concept.

He went on to say: ' I would recommend this course for anyone who works in a Trauma Unit or Major Trauma Centre, who needs something more than just the basics'

Royal College of Surgeons

The Anaesthesia, Trauma and Critical Care (ATACC) course was initially developed in 1997 by a group of Merseyside anaesthetic consultants as a UK practice-based multidisciplinary advanced trauma care course. Throughout its 15 - year history it has grown in stature to become one of the leading multidisciplinary trauma courses currently available in Europe. It has trained more than 1,000 surgeons, doctors, nurses, paramedics, military medics and other trauma care providers.

Mark Forrest, a consultant in anaesthetics and critical care who founded the course and remains the driving force behind it, explains that the course aims to deliver the highest standards of current evidence-based trauma practice. He says: 'For three long days we teach a philosophy of a therapeutic continuum from 'roadside to critical care', whereby the same principles are applied but with increasingly complex methods.'

In March 2013, the RCS panel paid a visit to observe the nature and conduct of this educational endeavour and to discuss the peculiarities and benefits of the course with its faculty and participants. The course's aim could be defined as an excellent niche trauma course with a strong emphasis on trauma skills from roadside to critical care, providing an extensive hands-on trauma/ disaster management experience in a multidisciplinary team comprising doctors, firefighters, police and ambulance. As ATLS is a well-established trauma course that is part of the surgical and anaesthetic curriculum, it is important to emphasise that the two courses do not contradict but rather supplement each other. ATACC would expand the knowledge gained at ATLS, with particular emphasis on the multidisciplinary pre-hospital management of trauma and disaster situations.

The course has been usually run at the Cheshire Fire and Rescue Service headquarters, utilising all inside and outside space, parking, stations, etc.

The course is constantly updated and has been adapted to meet the needs of anyone working in a trauma unit of a major trauma centre. Faculty are committed to review the course constantly to keep each lecture or skill station as up to date as possible. The course is now on its sixth edition. The faculty instructors are selected for their knowledge, their passion and their enthusiasm for teaching in innovative and realistic ways. ATACC goes to considerable lengths to maximise realism and the immersion of candidates, be it in a complex pre-hospital incident, the resuscitation room, theatre or critical care. Interestingly, even for those with little enthusiasm for pre-hospital care, the faculty have identified that there is huge benefit in developing trauma team skills and human factor training in such situations, which can then be utilised in the more therapeutically advanced in-hospital scenarios.

ATACC should be viewed as a very valuable supplement to anyone dealing with trauma/disaster/emergency management. ATACC would rather concentrate on providing the most recent updates and reinforc-

ing an individual early trauma care experience. One of the main emphases of the course is bringing together a team approach to trauma care, providing an overview and linking all relevant rescue services with the hospital/trauma centre.

Jennie Grainger, ST5 in general surgery and a participant on the 2012 ATACC course, noted: 'As opposed to simulated patient scenarios that you encounter on other courses, ATACC uses real actors in real-life situations. The course is completely "hands-on" and really allowed me to develop prehospital skills, which I felt had never been covered elsewhere in my training. Techniques of extrication, proper spinal immobilisation, triage of multiple traumas, decision making in the field and aggressive haemorrhage control were to name but a few, with plenty of time for practice and on-hand feedback. I actually felt that if I stopped by an accident on the motorway I would be able to offer proper support and medical care, something that before this course I would not have confidently done.'

Noaman Sarfraz, a consultant general surgeon at Warrington General Hospital, emphasised the importance of this course to trainees' experience of trauma: 'From a trainer's perspective I would definitely now

want my trainees to go on this course. Once they get out in the rain on their elbows and knees and get their hands dirty, it would give them a realisation of what is entailed when that trauma bleep goes off at three in the morning. They would empathise with the paramedics and respect the effort that has gone into bringing the casualty to them. These comments emanate from occasional reports of a casual and aloof attitude of some surgical trainees towards A&E and trauma. This course is definitely an awakening for such trainees.'

Paul Sherry, consultant orthopaedic surgeon at Warrington General Hospital, identified the strengths of the course: 'From the perspective of a consultant orthopaedic surgeon with 18 years' experience of hospital medicine, one of the main strengths was to take participants out of their "comfort zone", expose them to situations where they had to think on their feet using the skills that they had been taught, and to value and capitalise on the relevant experience of team members from different backgrounds.'

ATACC will never be a course delivered in numerous locations across the globe or as a basic introduction to trauma care for all, as it demands a huge amount of faculty time and effort to constantly update the lecture material and to maximise the potential of every simulation and scenario. However, this is an ideal course for those involved with the more advanced levels of trauma care; for example, those working in trauma units, major trauma centres and enhanced pre-hospital care. The ATACC course was accredited by The Royal College of Surgeons of England in June 2013 and it is felt that this type of the course is of particular relevance, benefit and use to surgeons, as it addresses current issues related to emergency surgery and development of major trauma centres.

Martin Bircher
RCS Council lead for Trauma
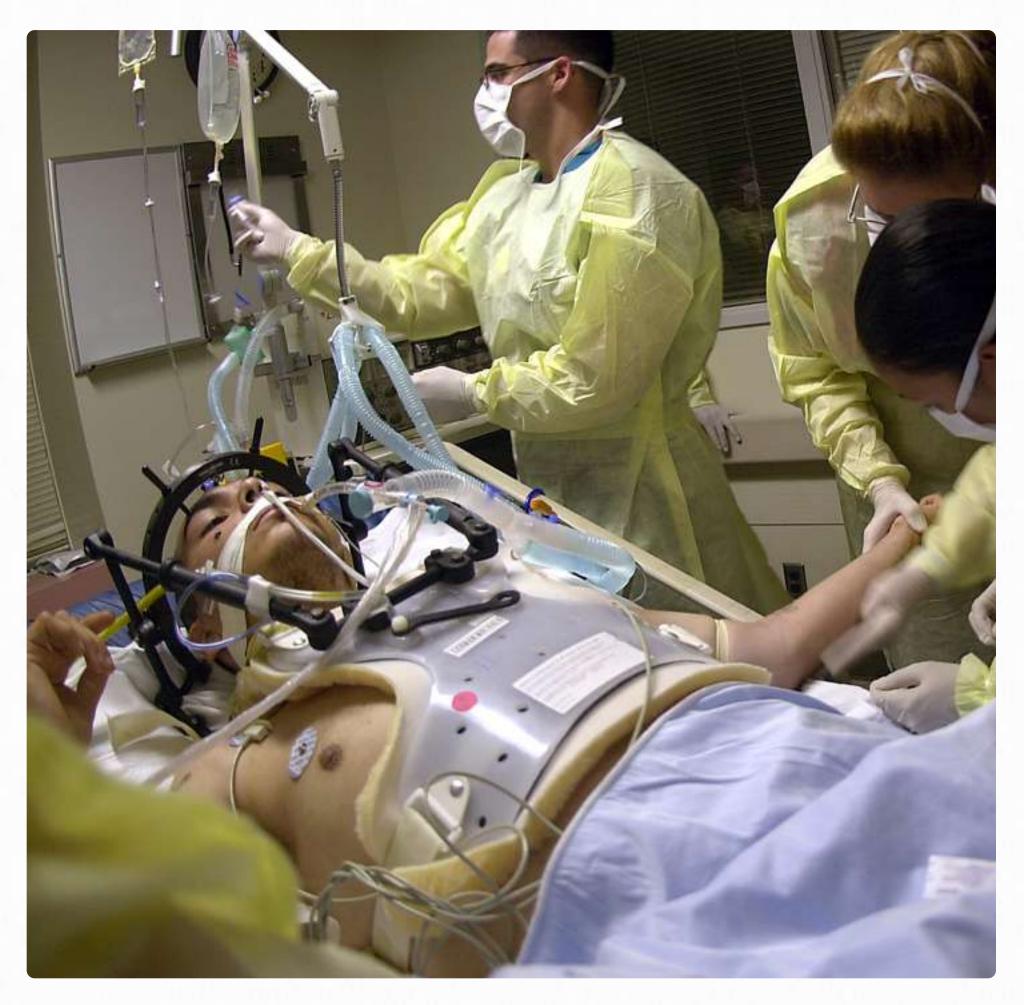
Anna Yerokhina
Head of Quality Assurance and Accreditation

2

Trauma Care Delivery

"Next to Creating Life, The Finest Thing a Man Can Do is Save One"

– Abraham Lincoln

Roadside to Critical Care

More than simply ATACCs ethos, a Roadside to Critical Care approach to trauma "resuscitation" is increasingly being recognised as the key to addressing the burden of trauma.

Trauma consists of a wide spectrum of clinical conditions that are initiated in the immediate aftermath following injury. In England, trauma is the leading cause of death across all age groups, with over 16,000 deaths per year. It is one of the few disease categories in which mortality is increasing. So Who "owns" a trauma patient?

- Is it the Emergency Services Disease?

- Is it a Prehospital or Emergency Medicine disease?

- Perhaps a surgical disease? If so what surgical specialty?

- Is it a medical disease? Renal Physicians and Haematologists are frequently involved in care.

- Is it a radiological disease? Diagnostic and increasingly interventional radiology are involved.

- Is it an Intensivists disease? If so, what brand of intensivist?

- Is it the general public's disease? The key to cardiac arrest survival lies in the community, could the key to massive external haemorrhage control fit a similar "push hard and keep up the pressure" model?

Where the seamless and frequently generalist approach to military medicine has constantly lead the way in trauma innovations, the acute hospital services have rapidly fallen by the wayside as clinicians of all disciplines become increasingly more super-specialised.

The Goal of the Individual Practitioner

Your goal must be to safely recognise and act upon time critical injuries. This is fundamental in providing both immediate life saving care, but also recognising when this definitive management is or is not unachievable given:

- Your level of expertise
- Your current crew and resource configuration
- Your current location
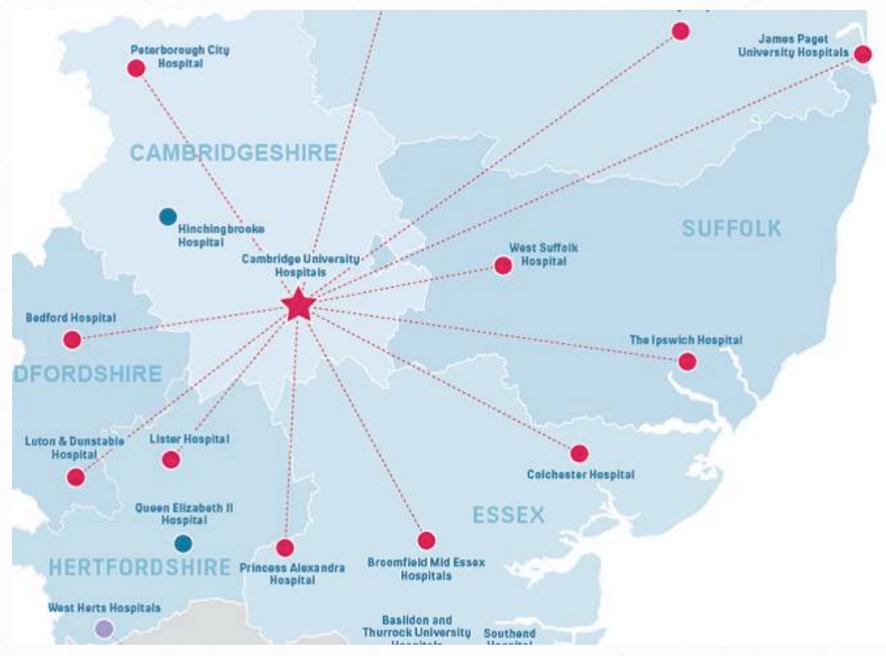
Once mindful of both the needs of your patient(s) and the level of care you can deliver, a decision needs to be quickly taken as to how best to achieve the care required for your patient(s). This is the role of Trauma Networks.

Trauma Networks

Successive reports have documented the current ad hoc unstructured management of trauma patients, consistently highlighting both avoidable mortality and morbidity. The high acuity and need for trauma readiness around the clock, the reality of delayed secondary transfers, and the justified severe criticism of available care and waste of resources all set the scene for an urgently needed reformat of trauma services across the UK.

The crude numbers representing the huge burden of trauma, the increasing public awareness of the issue, and the documented underperformance of the existing UK trauma services, eventually led to the restructuring of the National Health Service (NHS) trauma services. The result being the formation of Regional Trauma Networks.

The 'ideal' Trauma Network, as proposed by the American College of Surgeons Committee on Trauma, is a complex all-inclusive system of trauma-related services, from prehospital care through to acute care and rehabilitation. It affects and is affected by numerous patient and healthcare-related parameters, as well as

An example of a Major Trauma Network

legislation and finances. The goal is to match the utilised resources of the provider to the needs of the injured patients at the appropriate facility in a timely manner, achieving optimal management from the initial recognition of the injury to the return of the patient to the community.

Obviously one size does not fit all, particularly in a European context with multiple borders and multiple models of health care delivery. In the UK the power of local politics has been at increasing odds with the establishment of trauma networks, with each local DGH support group vowing to fight the reassignment of specialties to distant Trauma Units (TU) or regional Major Trauma Centres (MTC).

The key to success is that MTCs must maintain a policy of automatic acceptance for patients requiring MTC care from within the network who have been correctly triaged to a TU, under triaged or self-presented. Equally as important as automatic acceptance to, TU and DGHs in the networks must work together, collaboratively ensuring

patients have seamless access to care and transfer back to their locality hospital or host TU when medically fit. Networks must meet regularly to examine performance through formal governance processes, through regular mortality and morbidity meetings which generate action plans for improvement.

Key Statistics from the RCS

The following Key Statistics regarding Major Trauma where published by the Royal College of Surgeons of England in their 2007 briefing paper on a "Provision of Trauma Care" in support of Major Trauma Networks:

- Regionalisation of care to specialist trauma centres reduces mortality by 25% and length of stay by 4 days

- High volume trauma centres reduce death from major injury by up to 50%

- Time from injury to definitive surgery is the primary determinant of outcome in major trauma. (Not time to arrival in the nearest emergency department)

- Major trauma patients managed initially in local hospitals are 1.5 to 5 times more likely to die than patients transported directly to trauma centres.

- There is an average delay of 6 hours in transferring patients from a local hospital to a specialist centre. Delays of 12 hours or more are not uncommon. Across the UK, almost all ambulance bypasses can be achieved in <30 minutes.

- Long prehospital times have a minimal effect on trauma mortality or morbidity – even in very rural areas such as the west of Scotland.

- Trauma centres have significant improvements in quality and process of care. This effect extends to non-trauma patients managed in these hospitals.

- Costs per life saved and per life-year saved are very low compared with other comparable medical interventions.

- Currently UK mortality for severely injured trauma patients who are alive when they reach a hospital is 40% higher than the US.

- Without regionalisation, trauma mortality and morbidity in the UK will remain unacceptably high. The likelihood of dying from injuries has remained static since 1994, despite improvements in trauma care, education & training.

Trauma Unit
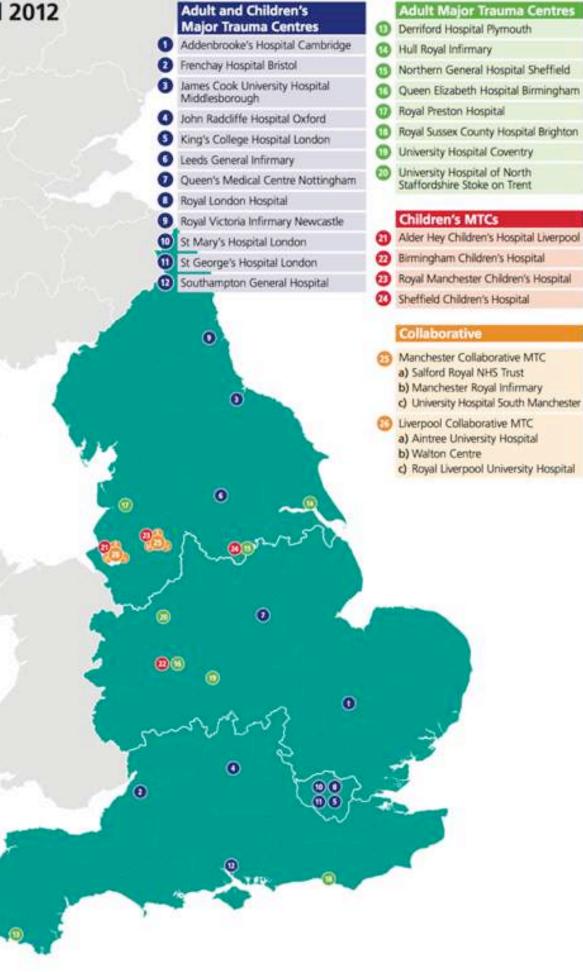
The majority of injured patients (for example, patients with fractures or single system injuries) do not need to access MTCs. To do so would cause inconvenience for patients (due to the potential for longer travel times), and also reduce the quality of care provided in both the specialist unit (which may become overburdened with 'routine' cases) and in surrounding hospitals (where staff would be unable to maintain their skills in treating injured patients).
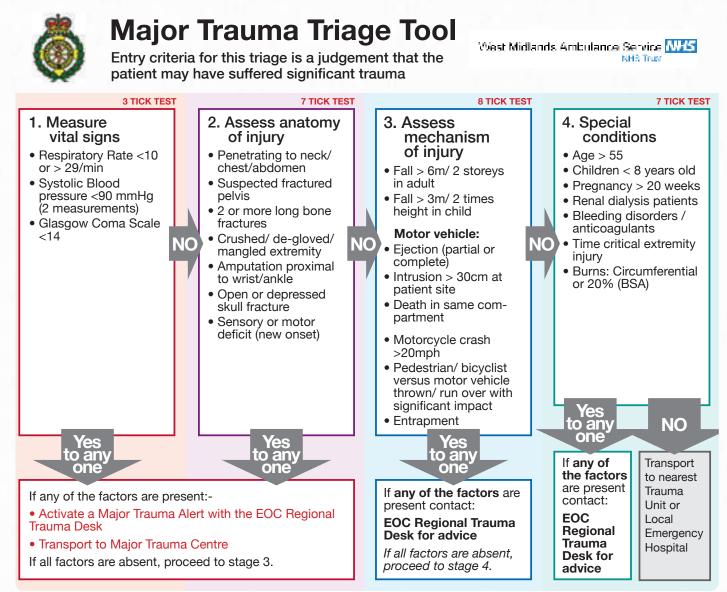
The concept of a Trauma unit maintains regional services (as opposed to a tertiary service) and maintains a highly skilled level of definitive care that is capable of managing the bulk of trauma patient presentations that do not trigger the criteria for MTC transfer.

Major Trauma Centres

NHS

April 2012

Major Trauma Triage Tool

Entry criteria for this triage is a judgement that the patient may have suffered significant trauma

West Midlands Ambulance Service NHS
NHS Trust

| 3 TICK TEST	7 TICK TEST	8 TICK TEST	7 TICK TEST
1. Measure vital signs • Respiratory Rate <10 or > 29/min • Systolic Blood pressure <90 mmHg (2 measurements) • Glasgow Coma Scale <14	**2. Assess anatomy of injury** • Penetrating to neck/chest/abdomen • Suspected fractured pelvis • 2 or more long bone fractures • Crushed/ de-gloved/ mangled extremity • Amputation proximal to wrist/ankle • Open or depressed skull fracture • Sensory or motor deficit (new onset)	**3. Assess mechanism of injury** • Fall > 6m/ 2 storeys in adult • Fall > 3m/ 2 times height in child **Motor vehicle:** • Ejection (partial or complete) • Intrusion > 30cm at patient site • Death in same compartment • Motorcycle crash >20mph • Pedestrian/ bicyclist versus motor vehicle thrown/ run over with significant impact • Entrapment	**4. Special conditions** • Age > 55 • Children < 8 years old • Pregnancy > 20 weeks • Renal dialysis patients • Bleeding disorders / anticoagulants • Time critical extremity injury • Burns: Circumferential or 20% (BSA)

NO → **NO** → **NO** →

Yes to any one ↓

Yes to any one ↓

Yes to any one ↓

Yes to any one ↓   **NO** ↓

If any of the factors are present:-
• **Activate a Major Trauma Alert with the EOC Regional Trauma Desk**
• **Transport to Major Trauma Centre**
If all factors are absent, proceed to stage 3.

If **any of the factors** are present contact:
EOC Regional Trauma Desk for advice
If all factors are absent, proceed to stage 4.

If **any of the factors** are present contact:
EOC Regional Trauma Desk for advice

Transport to nearest Trauma Unit or Local Emergency Hospital

Example of a major Trauma Triage Tool

Major Trauma Centre

Patients are triaged to a MTCs if they trigger predefined Major Trauma and secondary transfer protocols; these may differ across regions and systems. Where an MTC has key services located across more than one site, an operational plan describes how major trauma patients are treated and patient outcomes delivered.

A MTC (adult, child or combined) must have all the facilities and specialties required to be able to treat patients with any type of injury in any combination. Patients who have ISS greater than 8 and are treated in a MTC are covered by this specification. Elements of the service to manage these patients include:

Emergency Care and Surgery

• 24/7 consultant available on site to lead the trauma team

• The trauma team should be appropriately trained

• Trauma team present 24 hours a day for immediate reception of the patient.

• Ability to undertake resuscitative thoracotomy in the emergency department.

- A massive haemorrhage protocol in place for patients with severe blood loss, which includes the administration of tranexamic acid within 3 hours of injury, and transfusion specialist advice should be available 24 hours a day.

- 24/7 immediate availability of fully staffed operating theatres.

- A consultant will be involved in making any surgical decision making.

- Emergency trauma surgery will be undertaken by or under the direct supervision of a consultant surgeon.

- There will be a network protocol in place and operational at the MTC for assessing the whole spine in Major Trauma patients.

- Specialty Consultants available on site within 30 minutes when required:

  - Neurosurgery

  - Spinal and spinal cord surgery

  - Vascular surgery

  - General surgery (adult or child)

  - Trauma and Orthopaedic surgery

  - Cardiothoracic surgery

  - Plastic surgery

  - Maxillofacial surgery

  - Ear nose and throat surgery

- Anaesthetics

- Interventional radiology

- Intensive care

- For standalone children's MTC, local agreement in relation to middle grade attendance and timeframes within which they are required to attend.

Diagnostics and Radiology

- Immediate (defined as within a maximum of 60 minutes, ideally within 30 minutes) access to computerised tomography (CT) scanning and appropriate reporting within 60 minutes of scan;

- Availability of interventional radiology within 60 minutes of referral.

Ongoing care and reconstruction

- Immediate access to critical care or high dependency care (adult or paediatric) when required

- A defined team to manage on-going patient care, including a key worker (also referred to as trauma and rehabilitation co-ordinator) to support patients through the pathway and into rehabilitation. The model and background of the key worker may vary from centre to centre.

- Specialist nursing and allied health professional trauma roles.

- Access to cross speciality supporting services which will include pain management, rehabilitation medicine (which usually includes management of disturbed behaviour) and neuropsychology and neuropsychiatry.

- A defined ward for major trauma patients.

- A ward environment suitable for people with disability to practice and maintain their activities, specifically having enough space for people to get up and dress with some privacy, having toilets and baths/showers safely accessible for assisted or independent use by patients, and having facilities to allow the making of snacks and hot drinks.

- A nursing team in the ward, who are able to facilitate practice of and independence in functional activities by the patient, and undertake activities with the patient as advised, by the rehabilitation team.

Acute/Early Phase Rehabilitation

- A defined service for acute trauma rehabilitation which meets the needs of patients with ISS >8.

- Assessment within 72 hours by a rehabilitation Medicine consultant or alternative consultant with skills and competencies in rehabilitation. allowing up to 96 hours if seriously at risk of dying or assessment prior to 96 hours not clinically possible), with the output being an initial formulation (analysis of relevant factors) and plan to complete and inform the initial rehabilitation prescription.

- The prescription for rehabilitation reflects the assessment of the physical, functional, vocational, educational, cognitive, psychological and social rehabilitation needs of a patient.

- An initial assessment by the relevant members of a specialist rehabilitation team (including nurses) to add to the medical assessment.

- The output of the above two actions will be that all patients covered by this speci-

fication (without exception) have an initial rehabilitation prescription within 48-96 hours of presentation. Note that the prescription may identify no further need for rehabilitation, or may simply recommend monitoring or may require full active engagement of the wider rehabilitation team.

- All patients to receive early phase rehabilitation as indicated by the rehabilitation prescription, and all other actions identified in the rehabilitation prescription to be undertaken; if action or input cannot be delivered, the reason should be recorded and intervening action to be undertaken.

- All patients needing rehabilitation input or monitoring to be under the care of a multidisciplinary team that includes rehabilitation nurses, allied health professionals and a consultant in rehabilitation medicine. This team will meet weekly to discuss all patients within the scope of this specification in the MTC (Including those in Intensive Care Units (ICU) and ward areas); a specialist registrar in rehabilitation may deputise for a consultant on occasion but a consultant should attend over 60% of meetings and continue to provide supervision and support to the team.

Mechanism of injury

Just as the assessment of a medical patient starts with careful history taking, a "Medical History" in trauma is a careful collation of not only the events and risk factors leading up to a particular injury, but the injuring forces involved. Various techniques (by far not an exhaustive list) can aid in this history taking:

• Doing full scene surveys on arrival, not just for safety, but for information gathering.

• Taking video or photographs of the scene as a rapid documentation when handing over care.

• Stopping to imagine the forces involved, and how falling or crashing or being struck in a particular way might translate into pathology

• Designated time-out protocols when receiving care, so vital information is not lost.

Various injury patterns fit in with modes of injury and for example, position in a crashing motor-vehicle. The more thought put into this history taking, the more likely you are to not miss a significant injury. A very detailed chapter on Mechanism of Injury is included later in this manual.

Time Critical Concept

At each stage of the ATACC algorithm, responders should carefully look for signs of any time critical injuries following the sequence:

- Massive Haemorrhage
- Airway
- Respiration
- Circulation
- Head or Spinal Injury

If there is nothing life threatening found in a particular system, follow the small black arrows down the algorithm.

The time critical concept allows responders to not only identify life threatening injuries but to also establish the degree of urgency and, if necessary, indicate the speed of extrication or onward transport required. Once a time critical problem has been identified, solo responders should immediately stop their assessment to manage this problem. Following the red arrow to the right, the ATACC Algorithm provides a very brief aide-memoire of possible solutions/considerations under each heading in the pink box.

Solo responders should not move to the next stage of MARCH until the time critical problem has been managed effectively. Of course, the majority of trauma care is team based and ATACC actively encourage simultaneous activity, provided it is well managed and controlled by a dedicated team leader.

Once the problem has been addressed, the responder can move to the next step (following black arrows) in the MARCH algorithm to look for other time critical concerns. All time critical concerns must be constantly reassessed and reported clearly when care is handed over to the next team in the patients journey from roadside to critical care.

If there are no signs of life under Respiratory or Circulation assessment, follow the dotted red arrows to initiate a Traumatic Cardiac Arrest Protocol

Whilst some time critical of these may be treated on the spot, many will require transporting the patient and this should not be delayed. For example, getting a patient to the operating theatre for internal haemorrhage control must be considered a resuscitation measure, not necessarily definitive care. Attempting to "get them more stable" with intravenous fluids or blood transfusion is utterly misguided and will simply result in worsening blood loss, hypothermia and coagulopathy.

SAFE APPROACH

MASSIVE HAEMORRHAGE?

AIRWAY PROBLEM?
- Unconscious
- Obstructed
- Airway at risk
- Cx spine: if 2 persons

RESPIRATORY PROBLEM?
- RR >20/min or <8/min
- Reduced SpO2
- Difficulty in breathing
- Major Thoracic Trauma

NO SIGNS OF LIFE

CIRCULATION PROBLEM?
- Confusion / Reduced GCS
- CRT >3secs
- Radial pulse absent or >120/min
- Poor response to fluid bolus
- Evidence internal haemorrhage

NO SIGNS OF LIFE

HEAD /SPINAL/OTHER INJ
- Reduced GCS
- Focal Neurological Deficit
- Neurogenic Shock
- Major burns
- Others eg. Drowning, crush, paeds

Re-assess again!

Traumatic Cardiac Arrest Protocol

TIME CRITICAL

HAEMORRHAGE CONTROL
- D.D.I.T.
- Wound Packing
- Haemostatic Agents

AIRWAY MANEOUVRES
- O2 15l/min
- Jaw Thrust (chin-lift)
- NP/OP/iGel LMA
- BVM/C-Circuit
- Consider RSI
- ETT x 2 attempts
- iGel / LMA
- Surgical Airway

RESPIRATORY SUPPORT
- Extrication/Rescue
- Pain Relief
- Seal / Drain/ Thoracostomy
- BVM/C-circuit
- NIV
- Consider RSI
- Lung Protective Ventilation

CIRCULATORY SUPPORT
- Recheck D.D.I.T.
- 'Scoop and run' approach
- Gentle handling
- Pelvic strap
- Damage Control Resuscitation
- Temperature Control
- Tranexamic Acid (if <3hrs)

DISABILITY SUPPORT
- Oxygenation/ventilation
- Immobilise/splint
- Temperature Control
- Pain Relief
- Consider tertiary centre
- Consider RSI
- Neuroprotective strategy
- Burns protocol
- Crush protocol

The ATACC Algorithm

The MARCH Assessment

Traditional resuscitation and trauma courses often use the ABC approach (which stands for Airway, Breathing, and Circulation) to aid the responder in identifying and managing life-threatening issues. ATACC adopts a modified approach which has two significant changes. First, ATACC focuses initially on the life-threatening issue of massive external haemorrhage before addressing ABC concerns. Secondly, ATACC includes an additional step in its algorithm to check for other serious injuries.

The ATACC approach to care can be remembered using the acronym MARCH, which stands for:

• Massive external haemorrhage control
• Airway management
• Respiratory management
• Circulatory management
• Head trauma and other serious injuries

The MARCH approach is the initial Primary Survey Assessment in Trauma. It is intended as a rapid assessment and includes all the same components as the classical ABC first aid approach, shares the same principles as the updated, alternative cAcBCDE approach, which adds the concepts of catastrophic haemorrhage, cervical spine, disability, and exposure/examination. The cAcBCDE mnemonic may be difficult to remember and confusing with three C's. While the principles remain the same, we believe that the MARCH algorithm is the simplest and easiest method to follow.

Massive Haemorrhage

The term massive external haemorrhage refers to a major bleed that is rapidly life-threatening (e.g. a lacerated femoral artery which can bleed at 1 litre/min). Spurting arterial bleeds, blood soaked clothing, or pools of blood collecting on the floor should prompt a rapid assessment and immediate assessment and management.

Massive haemorrhage must be aggressively addressed before any other casualty assessment takes place. The body has only a limited volume of circulating blood (5 litres), and once a large amount is lost (approximately 3 litres), as the Damage Control Resuscitation chapter will explain, it cannot be simply or effectively replaced with intravenous fluids or even blood transfusion. The ATACC method of care focuses on circulation preservation (aiming to preserve circulation rather than replace it).

Massive external haemorrhage control should be a solo responder's initial focus.

However, it should be completely under control using the DDIT method within one minute to enable the responder to move onto the next step in the MARCH algorithm.

In a team, one member of the team can be assigned to massive haemorrhage management, whilst the rest of the team continue through the algorithm, working simultaneously.

Airway

Airway compromise is the major cause of preventable deaths in prehospital trauma. Loss of the airway can deprive the brain and organs of vital oxygen and can lead to death in minutes.

Unconscious?

The airway is normally open and clear in a conscious patient. If the patient is totally unresponsive, or only responding to Pain, their airway is at risk and they are time critical.

Obstructed?

The ability to talk normally immediately tells you that the airway is clear. This assessment tool is particularly useful for a casualty trapped beyond your site or at a distance.

In a partially obstructed airway, the flow of air to the lungs is restricted, resulting in a harsh, high pitched noise known as stridor. The casualty may also cough and gag (which indicates that some air is passing around the obstruction) and speech will be severely impaired or even impossible.

In a totally obstructed airway, no sounds of breathing effort can be heard, and no air is able to move in or out, despite good respiratory effort. Speech is impossible, but the casualty may have a silent cough. If the airway is completely obstructed, the patient will lose consciousness rapidly.

When an oxygen mask is used, the mask will fog every time the casualty breathes out through an open airway. This method of airway assessment allows the responder to count the respiratory rate and may be especially useful if access to the casualty is limited.

Patients further down the line of care, may have End Tidal CO2 monitoring. The waveform trace is an invaluable gold standard in airway monitoring.

At Risk?

Airway risk stratification is covered in detail in the Airway Chapter.

Examples include:

- Facial burns (e.g. redness, blistering, peeling skin)
- Loss of facial hair
- Swelling of the lips or mouth
- Soot in the airway (smoke inhalation)
- Blood in the mouth
- Foreign bodies in the mouth (e.g. broken teeth, scene debris)

Respiratory

During the initial MARCH assessment, the goal is rapid identification of time critical injuries. A full thoracic examination using the RU In SHAPE mnemonic may follow during the secondary, head-to-toe examination or, if you pick up evidence of a respiratory problem at this stage, a rapid RU In SHAPE examination should ensue to rule out any immediate life threatening pathology that needs to be addressed before continuing the primary assessment.

Repiratory Rate

Ask yourself, is this casualties respiratory rate normal for the environment you are in? Generally speaking, an Adult with a respiratory rate out of the normal, i.e. >20/min or <8/min should prompt you to a more thorough RU In SHAPE examination.

Tachypnoea is frequently only down to anxiety and pain, but you should always exclude serious pathology.

Normal Paediatric Respiratory Rates	
Age	Rate
Infant (birth–1 year)	30–60
Toddler (1–3 years)	24–40
Preschooler (3–6 years)	22–34
School-age (6–12 years)	18–30
Adolescent (12–18 years)	12–16

Reduced SpO2

The portable pulse oximeter has become ATACCs "handshake." Whilst it notoriously under-reads, a normal SpO2 reading >94% is very reassuring. An incredibly simple device, it should be the very first piece of monitoring placed on the patient. Low SpO2 readings should be investigated fur-

ther. Whilst it may simply represent hypo-perfusion from cold, it could indicate more serious respiratory or cardiovascular insult.

Difficulty in Breathing

Casualties with serious chest trauma often experience obvious distress or difficulty breathing. Listen to the casualty's ability to speak. If the casualty is unable to complete full sentences, then this should raise concerns and justifies a more thorough assessment. Consider the situation 'time critical' if the casualty can only speak a few words at a time or only in gasps.

Listen for any wheezing sounds or bubbling noises in the chest, which may indicate existing medical conditions such as asthma or heart failure. In trauma cases, these sounds may indicate serious chest injury and should be thoroughly assessed.

Paediatric Pulse Rates		
Age	Low	High
Infant (birth–1 year)	100	160
Toddler (1–3 years)	90	150
Preschooler (3–6 years)	80	140
School-age (6–12 years)	70	120
Adolescent (12–18 years)	60	100

A stethoscope is not needed to listen to chest sounds; if these sounds indicate serious concerns, they will be easily seen or detected. The stethoscope in the prehospital or emergency room setting is throughly unreliable.

Major Thoracic Trauma

Look for obvious signs of blunt or penetrating chest trauma. Where there are injuries to the upper chest, remember that the first and second ribs are very strong and well protected. Therefore if these ribs are fractured, it strongly suggests underlying serious trauma and the potential for numerous other internal injuries, particularly circulatory.

Wounds in the neck or around the clavicle can indicate serious internal injury and may be challenging to manage in the field due to the presence of large blood vessels, nerves, and the trachea. When injured, these structures can retract into the chest or up the neck, making management difficult.

If No Signs of Life

If no signs of Life, a traumatic cardiac arrest protocol should be immediately followed.

A traumatic Cardiac Arrest is NOT the same as a medical cardiac arrest.

Circulation

Just as with respiratory compromise. During the primary MARCH assessment, use the following quick indicators to guide you into a more focused clinical examination, which is contained in the Circulatory/Shock Chapter. Any indication of time critical circulatory pathology should immediately prompt a gentle, but rapid "scoop and run" towards definitive intervention aimed at haemostasis.

Reduced GCS

Whilst a reduced level of consciousness may represent a traumatic brain injury or hypoxia, in the presence of trauma, always consider blood loss. A pitfall to be cognitive of, is the combative or annoying patient, who appears intoxicated. Always use this as a prompt to check their blood pressure and consider major blood loss.

Capilary Refill Time

The capillary refill time should be less than two seconds. If it is delayed, and certainly if it is greater than three seconds it warrants further investigation. Whilst peripheral capillary refill will be reduced in cold states, reduced central capillary refill, taken by pressing a thumb into the forehead or chest and timing the blanching return to normal, is very sensitive of a hypoperfusion state.

Radial Pulse

If a radial pulse is absent, it indicates that the systolic blood pressure may be less than 80mmHg or that circulation is compromised to that limb. It is one of the simplest indicators of time critical circulatory pathology, particularly in the presence of reduced consciousness.

If there is evidence of tachycardia, this could be down to anxiety or pain, but a sustained tachycardia in an otherwise healthy individual is blood loss or circulatory compromise until proven otherwise.

Evidence of Internal Haemorrhage

Signs of internal haemorrhage are highly subjective until definitive radiological or surgical confirmation. Always think about the mechanism of injury during your primary survey. For example, a person who has had their abdomen driven over is at imminent risk of internal haemorrhage and should be managed as time critical until major internal haemorrhage is definitively ruled out.

Consider cavities into which circulatory volume can be lost - thorax, abdomen, pelvis and even around long bones. An acute abdomen in trauma, is an indication to treat the casualty as time critical.

Absent Signs of Life

If there are no signs of Life, a traumatic cardiac arrest protocol should be immediately followed.

A traumatic Cardiac Arrest is NOT the same as a medical cardiac arrest.

Head/Spinal Injury / Other Injuries

In the last component of the primary trauma survey, we consider head, spinal and other life threatening injuries.

Reduced GCS

Whilst a reduced GCS or a patient only responding to pain or totally unconscious could be an indicator of a range of pathologies, including intoxication, it should prompt rapid investigation and be considered time critical.

Remember that drunks don't get drunker without alcohol! A patient in your care that is seemingly getting more and more intoxicated needs to be investigated further.

Focal Neurological Deficit

Think FAST !

Subtle lateralising signs will be missed during a primary survey, but do actively seek out similar pathology commonly found in stroke. Sometimes the only indication of a rapidly expanding intracranial bleed is unilateral limb weakness.

Neurogenic Shock

Neurogenic shock is a distributive type of shock resulting in hypotension, occasionally with bradycardia, that is attributed to the disruption of the autonomic pathways within the spinal cord.

• Hypotension occurs due to decreased systemic vascular resistance resulting in pooling of blood within the extremities lacking sympathetic tone.

- Bradycardia results from unopposed vagal activity and has been found to be exacerbated by noxious stimulus such as hypoxia and endobronchial suction.

Neurogenic shock can be a potentially devastating complication, leading to organ dysfunction and death if not promptly recognised and treated. It is not to be confused with spinal shock. Spinal shock is a neurological deficit as opposed to a cardiovascular state.

Major Burns

Major burns are a circulatory emergency. Any burn that potentially meets the Referral to Burns Centre criteria, should be managed as a time critical circulatory emergency.

Criteria for transfer to a regional burns centre include:

- 10% partial thickness burns in the under 12s or over 60s
- 15% partial thickness burns in 12 – 60 year olds
- 5% full-thickness burns
- Burns to special areas mentioned above
- Circumferential burn
- Inhalational burn
- Chemical, radiation, or high-voltage electrical burns

Other Life Threatening Pathologies

Immersed, Submersed, trapped, suspended, inverted and crushed individuals should be considered time critical until proven otherwise.

Exposure

Full head to toe exposure has been, and is likely to remain a vital cornerstone in trauma assessment. The timing of the secondary survey however requires a fundamental rethink.

What are the priorities for the particular patient in front of you at this moment in time?

Some examples to consider:

- In an internally haemorrhaging patient, it matters more that a patient goes to theatre, rather than establishing that they have a potentially fractured limb.

- Exposure of the patient is not usually appropriate in the entrapment situation. Consideration must be given to the delay that this will effect on the extrication evolution, and the quality of examination possible in a confined space.

- Fully examining a patient prehospital, is almost always impossible. Whilst it may

be necessary to assess for major haemorrhage or to manage life-threatening or open injuries, it certainly is not acceptable to cause hypothermia and worsen a coagulopathy.

• An unconscious patient is notoriously challenging to adequately examine. A full examination may have to be delayed until they are alert and orientated.

• Full "Pan Scan" CTs are very rarely looking at all the extremities. They certainly are not fully diagnostic, missing in particular tendon and nerve injuries. They frequently impart a false sense of security, that there are no other significant injuries.

Delaying full exposure and secondary examination for definitive life threatening treatment makes sense. However protocols need to be in place to ensure:

• All clothes are removed at the earliest opportunity, preferably in hospital, to minimise the hazard of injury to patient and staff from items concealed within clothing.

• Hand over of responsibility for carrying out this head-to-toe assessment is part of a hospital wide policy.

• The examination is indeed thorough and well documented.

Adopting a delayed examination approach is acceptable, however bear in mind that an intensive care specialist may not necessarily be the right specialist to examine for and manage minor injuries. Whilst this might not seem important whilst a patient is intubated and ventilated for their traumatic brain injury, not picking up an on something simple early, may lead to permanent disability down the line.

In some centres, the responsibility for full head-to-toe examinations remains with Emergency Medicine, even if the patient has already been through theatre and is now in a ward environment.

References

1. Kanakaris NK, Giannoudis PV. Trauma networks: present and future challenges. BMC Medicine. BioMed Central Ltd; 2011 Nov 11;9(1):121.

2. Subcommittee of the American College of Surgeons Committee on Trauma: Trauma Performance Improvement Reference Manual. Chicago, IL, USA: American College of Surgeons; 2002.

3. http://www.rcseng.ac.uk/news/docs/FINAL%20trauma%20statement%207%20sept%2007.pdf/

Team Approach

Introduction to the Team Approach

In this chapter we will consider some of the issues that will affect the working of your trauma team, whether at the roadside in the ER, theatre or ICU. There are many quotes relating to team-work but this one nicely sums up the benefits and the importance of the team and its members.

'The strength of the team is each individual member. The strength of each member is the team'

This quote comes not from the trauma world but Phil Jackson, an American basketball coach - however it perfectly sums up many of the key messages in this chapter. Everyone has a part to play in the team and must be fully aware of their role and also empowered to deliver in that role.

As a team we can achieve incredible things, which would be impossible working alone.

BMW offshore tri-hull racing yacht

Sailing

This is the fastest yacht in the world, but to reach those speeds reliably and safely requires a well-led, well-trained team working in total harmony.
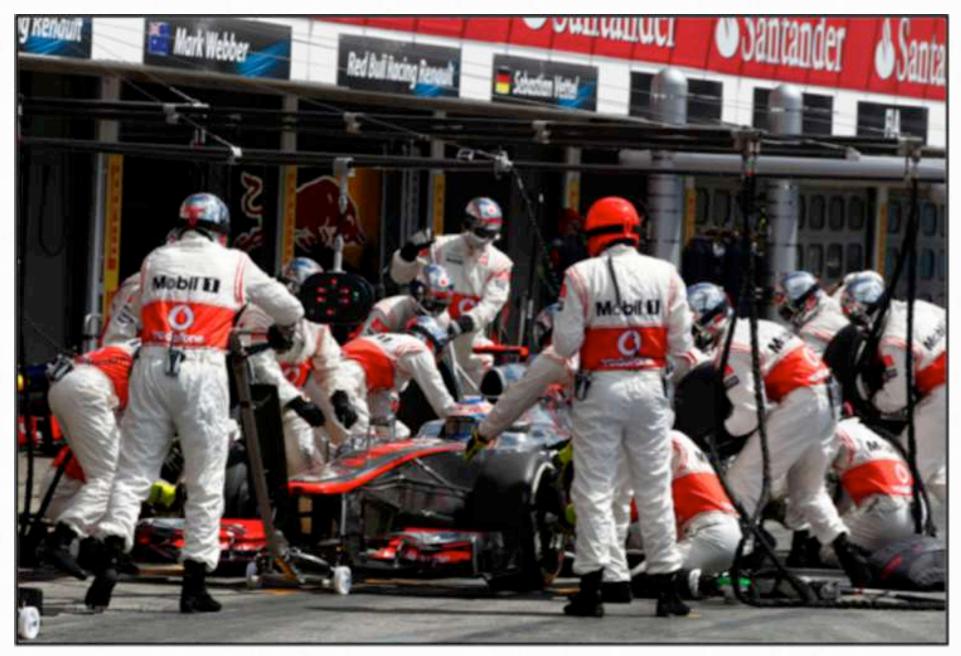
But these vessels are also potentially extremely dangerous, with their huge sail area and their immense power, things can very quickly become dangerous or even fatal. In any such life threatening situation a good team and leadership becomes even more important. Trauma is much the same, as a complex system that requires a skilled leader, who can direct and focus the team members, who each have their own individual skills. When it all comes together, even the most challenging cases will be well managed and potentially salvaged, but without effective team-work and guidance, even a highly skilled team will manage the simplest of cases badly.

Formula 1 Pit crews

If we consider another expert team that you are all familiar with - a formula 1 pit crew. There have been numerous comparisons with anaesthetic, critical care and trauma teams and lessons that can be learnt from this precision, high performance industry.

One of the best such teams is the McLaren team. In a recent season, during an actual race they broke all previous records and completed a pit stop, including 4 wheels changed and refueling in just 2.31 seconds. It has to be seen to be believed, but what you will observe is a highly trained team , who all know exactly what they have to do under the direction of a good team leader.

Clearly they have practiced endlessly to achieve this standard and this training and development of 'muscle memory' does undoubtedly have a significant effect, but without the leader or the other team members none of that would matter. In simple terms, the wheels

McLaren record breaking Formula 1 pit crew

cant be changed if the jack doesn't lift the car, or the lollipop man lets it drive off too soon etc. Remarkably, even this time has now been surpassed by the [Red Bull Team in 1.923 secs](#) in the 2013 US Grand Prix.

Looking at the pit-stop in a slightly different way, we could argue that each team member has just one simple job to focus on and this role is entirely predictable. But, if we consider the trauma team, then we can also identify that within each individuals role there are only a limited number of specific skills required for each person. The anaesthetist can concentrate on securing the airway, the orthopaedic surgeon can concentrate on fractures and the surgeon on the abdomen.
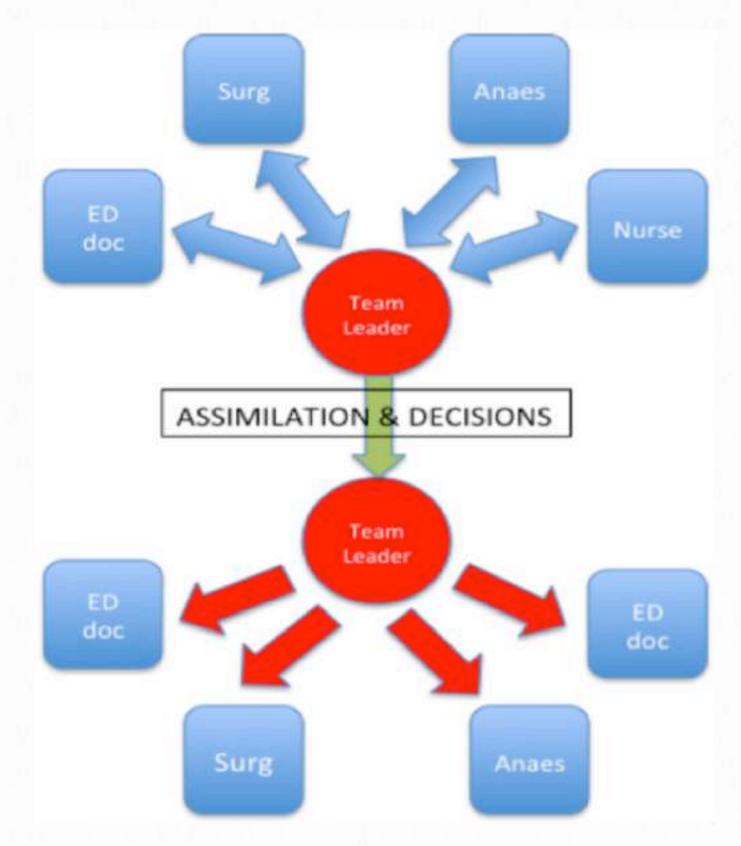
In addition, since the advent of ATLS we manage trauma in a clear and systematic manner every time, no matter what the case i.e. CAcBC or MARCH. As such we can train the team in a similar manner to optimise their individual roles and performance. This is clearly a key role and objective for clinical simulation.

Another key part of the pit-crew is the team leader, He or she stands back and takes an overview of the team and the process. The leader is clearly identifiable (red helmet, whereas the others are all wearing chrome ones) and also has communications with all team members. No one should speak unless essential and all messages must go through the leader.

Team Leaders

Considering the team leader a little further, the role requires someone with the skills and ability to gather all the information before them, including visual information, findings from team members and other feedback. They must then assimilate and process the information and sift out the key details. From these important decisions are then made and fed back to the team.

This is summarised in the diagram overleaf, which demonstrates several other important factors. The Leader, does not have to have all the answers and does not have to be an 'expert' in everything. Further advice and guidance can be obtained from the team members to support the decision making (two-way arrows in upper field).

Assimilation of information and decision making in the trauma team

Insignificant information must filtered out and ignored and this can be difficult, particularly if a team member believes that their issue is vitally important and a priority. They must be able to express that importance clearly to the team leader but it may well not be such a priority in the 'bigger picture' and the team leader must filter and sort the information gathered. From this summated and filtered information a decision must be made.

This decision will dictate the subsequent actions, which are fed back as instructions to the team members. As these actions are performed, the effects are monitored and the process continues.

Although we have suggested that the Team leader is best placed, stood back in an overseeing position, they are still part of the 'team' and must remain approachable and involved with the process. This can be a challenge and suitable leadership skills are essential, striking a balance between listening, assertiveness, aggression, calm and numerous other traits.

Without good leadership and team skills your Trauma Team will operate to an inferior standard and in the worst cases will result in poor management, patient deterioration and potentially even death.

The Aviation Industry

Anther area commonly quoted when considering teams is the aviation industry. Crew Resource Management or CRM is the system adopted by pilots and their crews to interact effectively and safely during flight operations. From the minute the crew prepare for flight the process commences and whilst a clear rank and leadership structure exists, there is also a defined process for raising concerns or questioning decisions, which can be escalated in various pre-determined ways, such as becoming more formal in terms of names, changing the pitch of your voice or making clear statements.

In routine operations, at times when key decisions are being made, such a pre-flight checks, there should be minimal or no distraction ('Sterile cockpit') and use of challenge checklists.

The processes and systems avoid errors, forgotten details, distractions and also provide a systematic approach that can be adopted when things start to go wrong or the stress of the situation is greatly elevated. Communication is optimised to avoid time wasting, whilst keeping all essential team members fully briefed. We will look at this in the section below.

If we consider one of the most famous recent aviation adverse incidents, that of American Airlines, Flight 1549, a large passenger plane, which ditched in the Hudson river after a bird strike stalled the aircrafts engines, within minutes of take-off.

If we listen to the inflight 'blackbox' flight recorder we can hear how Capt 'Sully' Sullenberger managed to save all 155 people on board his plane, avoid a major city with

Flight 1549 ditched in the Hudson River, USA

a plane full of fuel and land safely in a remarkably challenging place.

There is no doubt that what he achieved was miraculous in the eyes of most people, however 'Sully' remains very modest and claims that he merely followed procedures and used skills developed in the flight simulator during his training.

So was he a one in a million pilot or did he just 'follow procedure'. Well let's consider some of the issues in this incident.

The 'Team' included the pilot (Team Leader), co-pilot, cabin crew and control tower staff from 3 different airports. As you can see, the last members of the team are likely to be people that Sully had never met, which might be considered unusual for a team until we con-

sider many other teams that come together in the face of an emergency situation, including trauma teams. Often, these individuals will have never met or worked together before, but with good quality standardised training and communication skills then this does not have to be a major obstacle to effective team work.

If we listen to the radio messages, or read the statements from the cabin crew, we can see that despite the short timescales involved and the likely fear and stress of the situation, that all communications were clear, calm and concise following rapid decision making by the Flight crew.

Below are a short selection of these messages from the Pilot to the Air Traffic Control and as you can see they are as short and concise as possible, giving the absolute essential information only and nothing more or less:

'We're unable, may end up in the Hudson'
'Unable'
'Yes'
'We can't do it'
'We're gonna be in the Hudson'

What is even more remarkable is that whilst flying the plane, evaluating the situation and all possible options, the pilot also managed to attempt a full engine re-start,

a return to the departure airport, divert to alternative airports, briefing the cabin crew and finally selection the safest place to land.

Interestingly, whilst this was a truly remarkable performance by everyone involved, a few potentially serious mistakes were still made, such as the completely wrong aircraft number being used by one controller.

That said, there was very little unnecessary delay or excessive communication. In fact the controller from La Guardia starts to quiz the pilot about which engine has failed and then suddenly stops himself, his training kicks in and he realises that such information is irrelevant to him and he immediately confirms that he has got enough information and detail with the simple phrase 'Got it'

Finally, the option to divert and the options for alternative airports were offered and selected at incredible speed. In fact the final comment from the chief controller at Teterboro, when told that he want to emergency divert to their airport, was simply 'Check'. A single word that clearly confirms that a message has been, received, understood and agreed. This reply is a key part of the RSI and other challenge-response checklists.

So whilst Sully and his team performed in a truly commendable fashion, he was indeed correct when he modestly suggests that the procedures that he followed had been practiced and ingrained in the simulator during training, supported by their emergency cockpit checklists.

The Role of Simulation

Such simulation has been a mandatory part of aviation training for decades and it is used to establish effective crew interaction. This has been termed Crew Resource Management or CRM and this is now playing an increasing part in medical training and clinical improvement.

It is well known that humans make mistakes under various circumstances – it is human to err! These errors can occur under severe stress or conversely even when performing un-stimulating routine tasks, where concentration may wander.

In a simulator the crew will work through simple and mundane day to day tasks, learning to avoid errors when concentration may be easily lost. They also learn to identify critical steps in their operations when all distractions should be removed. They term this a 'sterile cockpit' and if any interruption does occur then the checklist or drill is re-commenced.

Another crucial part of simulation based training is critical incident management. At NASA the astronauts in training spend hour upon hour in the simulator for months, being tested and challenged for absolutely any eventuality or failure that may occur. In doing this they aim to reduce risks as much as possible and to also instill in the crew a systematic approach to managing problems without panic or irrational decision.

Astronauts and pilots have a term called 'Boldface' which is used for procedures that are absolutely essential in the event of various major misadventures. For these, they would expect no thought or need to read a check-list, but simply a mere reflex response in the shortest possible time. Once completed the rest of the emergency checklist and other actions can be continued.

In trauma care, we can also adopt check lists and CRM to prepare the equipment and the team for safe and effective operations. In addition, when more challenging and complex incidents occur then the team can utilise challenge checklists and even bold face (e.g. for a cardiac arrest) to optimise their response and minimise risks.

Simulation plays an ever increasing part in such training and as the quality of the simulation becomes increasingly 'immersive' then the potential benefits can increase. However, the evidence to support the benefits of simulation remains somewhat limited, although most would agree that both the experiences of the aviation industry and a degree of common sense suggest that it must improve performance, especially in adverse situations. What is clear is that simply increasing the complexity of the 'simulator' or mannequin is not enough, the scenario, the environment and the organising team must all play a part in creating a high quality simulation, that will achieve the necessary objectives.

This is reflected in prehospital trauma training, where the environment and a live actor, with good cas-sim/wounds, may actually be a more powerful educational tool than a hugely expensive hi-fidelity simulator or Sim-centre

Sim-man in a clinical skills lab can lose the potential of simulation

Medical Simulation

We have all seen experienced training with resuscitation mannequins and they undoubtedly have a part to play. They have now reached remarkable levels of sophistication but if simply placed in a training room, with a few bits of medical equipment then they lose so much of the true potential of simulation.

This sort of basic environment is acceptable for teaching core skills and basic procedures, but for full team training and complex incident management, we need a far better location and scenario, because if it doesn't feel 'real' to the candidates then much of the benefit and true effect will be lost.

Similarly, whilst dedicated multi-million pound simulation centres are very useful training tools, they lose something as they are not the real environments in which the staff work. They are often rarified environments, remote from the rest of the hospital clinical areas and without the same support services and as such they just don't feel entirely 'real' enough sometimes for experienced teams.

We can create exact replicas of clinical areas such as emergency rooms or theatres, but they only feel real to the staff from those areas and become more constrained. What ATACC prefer is 'Applied simulation' delivered in the real clinical environments, with the real equipment and staff, in their normal locations. In this way, we not only challenge the clinical skills and team CRM, but we also build in the integration into the normal working environment, with the regular staff in those areas. We also build in a degree of 'memory' in terms of where kit and emergency kit is located. Problems and logistical issues are identified and solutions or procedures can be tested. Scenarios can embrace not only clinical scenarios but other drills and system testing such as fire evacuations, power failures, floods etc.

However, clinical pressures often make this difficult to organise within working hours, so we are now looking at new technologies for the answers. As huge supporters of simulation ATACC are constantly trying to augment and improve the immersive quality of our training. We have not only looked to the medical and aviation world but also to the military, maritime and other industries and the sports world to optimise what we deliver. As such we have learnt that all types of simulator have a value, but that they need to be carefully selected for the situation or training involved. Our latest projects include projection cubes to create a diverse range of fully immersive and interactive environments at the click of mouse. This may be the most practical, cost effective next step in clinical simulation and education.

Communication

When we investigate any critical or unto-ward incident, as we even saw with Flight 1549, there is virtually always some degree of communication failure identified and as such, this must play and essential part in any training that we provide. The team must relay messages to all who need that information and this process must be two-way, with inputs and outputs, all directed through the Team Leader.

The Team leader gathers the information and then processes it with a simple 'sift and sort' into what matters and what doesn't as we saw earlier in the chapter.

In this section we will describe several methods that have been established to im-prove communication in the healthcare and industry environments.

ISBAR handovers

Communication aids can be a great help and a key one often used in healthcare is the 'ISBAR' handover based on the follow-ing terms:

• Introduction
• Situation
• Background
• Assessment
• Recommendations

In simple general terms these refer to the following:

Introduction: Who exactly is everyone and there roles

Situation: a succinct statement about the situation or problem

Background: is a short history of what is has been happening in the pre-ceding time, this may include details of past medi-cal history or just the development of the new problem and how it has developed

Assessment: your current evaluation of the situation, including your findings, obser-vations and the key priorities or concerns

Recommendations: is a summary of the advice given to manage the situation.

ISBAR COMMUNICATION TOOL

I Identify	➤ **Yourself:**
	    ☐ name,
	    ☐ position,
	    ☐ location
	 ➤ **Receiver:** Confirm who you are talking to
	 ➤ **Patient:** name, age, sex, location

S Situation	➤ State purpose "The reason I am calling is……." ➤ If urgent — SAY SO, Make it clear from the start ➤ May represent a summary of Assessment and Requirement

B Background	➤ Tell the story
	 ➤ Relevant information only:
	    ☐ history,
	    ☐ examination,
	    ☐ test results,
	    ☐ management
	 ➤ If urgent: Relevant vital signs, current management

A Assessment	➤ State what you think is going on, your interpretation
	 ➤ Use ABCDE approach
	    ☐ Airway
	    ☐ Breathing
	    ☐ Circulation
	    ☐ Disability
	    ☐ Exposure
	 ➤ State any interventions e.g applied oxygen

R Requirement	➤ What you want from them — BE CLEAR
	 ➤ State your request or requirement
	    ☐ Urgent review (state time frame)
	    ☐ Give approval / recommendation for further course of action while awaiting attendance eg. ECG, bloods
	    ☐ Give opinion on appropriate management

Modified from Southern Health

ATMIST

Other examples include the ['ATMIST' handover](#) for prehospital providers:

- A = Age of patient
- T = Time since injury
- M = Mechanism of injury
- I = Injuries identified or suspected
- S = Symptoms, signs and clinical observations
- T = Treatment provided

They all establish a systematic approach, to assist the individual in focusing and organising the key information, even in very complex situations, whilst also providing an aide memoire to prevent important details being forgotten. In addition, these systems keep communication short and brief, avoiding long winded, rambling explanations, which simply waste valuable time.

Check-lists are playing an ever increasing part in our everyday healthcare lives and they are not just about handing over information, but are about safety and ensuring that all systems are in place and prepared. Consider the WHO safe surgical check-list.

The WHO Safe Surgical Checklist

[The WHO checklist](#) has been introduced to improve safety in the surgical environment where, even in the most experienced hands doing everyday surgery, serious and potentially life changing or life threatening mistakes can be made for the reasons already discussed in this chapter.

The check-list begins with a 'Time out' which is the equivalent to the sterile cockpit approach of the pilots. This should focus the staff and suspend all other activity. All staff should introduce themselves at the beginning of the list. In trauma terms, this is typically on arrival in the ED or theatre. A series of questions, that demand a response from key individuals then follows. The check-list can be modified for local requirements, but the principle and method should be the same.

For many, who work in theatres, the surgical checklist is considered something of a delay and a nuisance, but these individuals have completely failed to appreciate that such complacency is why incidents occur on routine lists. In addition, when emergencies occur, it ensures that the team are at least in some way prepared to manage that situation. The whole team will know something about the patient, the nature of the procedure and if what they subsequently see is an unexpected event. Check-lists and challenge check-lists for trauma take this element and process a step further.

SURGICAL SAFETY CHECKLIST (FIRST EDITION)

Before induction of anaesthesia ►►►►►►►► **Before skin incision** ►►►►►►►►►►► **Before patient leaves operating room**

SIGN IN

- PATIENT HAS CONFIRMED
  - IDENTITY
  - SITE
  - PROCEDURE
  - CONSENT
- SITE MARKED/NOT APPLICABLE
- ANAESTHESIA SAFETY CHECK COMPLETED
- PULSE OXIMETER ON PATIENT AND FUNCTIONING

DOES PATIENT HAVE A:

KNOWN ALLERGY?
- NO
- YES

DIFFICULT AIRWAY/ASPIRATION RISK?
- NO
- YES, AND EQUIPMENT/ASSISTANCE AVAILABLE

RISK OF >500ML BLOOD LOSS
(7ML/KG IN CHILDREN)?
- NO
- YES, AND ADEQUATE INTRAVENOUS ACCESS AND FLUIDS PLANNED

TIME OUT

- CONFIRM ALL TEAM MEMBERS HAVE INTRODUCED THEMSELVES BY NAME AND ROLE
- SURGEON, ANAESTHESIA PROFESSIONAL AND NURSE VERBALLY CONFIRM
  - PATIENT
  - SITE
  - PROCEDURE

ANTICIPATED CRITICAL EVENTS

- SURGEON REVIEWS: WHAT ARE THE CRITICAL OR UNEXPECTED STEPS, OPERATIVE DURATION, ANTICIPATED BLOOD LOSS?
- ANAESTHESIA TEAM REVIEWS: ARE THERE ANY PATIENT-SPECIFIC CONCERNS?
- NURSING TEAM REVIEWS: HAS STERILITY (INCLUDING INDICATOR RESULTS) BEEN CONFIRMED? ARE THERE EQUIPMENT ISSUES OR ANY CONCERNS?

HAS ANTIBIOTIC PROPHYLAXIS BEEN GIVEN WITHIN THE LAST 60 MINUTES?
- YES
- NOT APPLICABLE

IS ESSENTIAL IMAGING DISPLAYED?
- YES
- NOT APPLICABLE

SIGN OUT

NURSE VERBALLY CONFIRMS WITH THE TEAM:
- THE NAME OF THE PROCEDURE RECORDED
- THAT INSTRUMENT, SPONGE AND NEEDLE COUNTS ARE CORRECT (OR NOT APPLICABLE)
- HOW THE SPECIMEN IS LABELLED (INCLUDING PATIENT NAME)
- WHETHER THERE ARE ANY EQUIPMENT PROBLEMS TO BE ADDRESSED
- SURGEON, ANAESTHESIA PROFESSIONAL AND NURSE REVIEW THE KEY CONCERNS FOR RECOVERY AND MANAGEMENT OF THIS PATIENT

THIS CHECKLIST IS NOT INTENDED TO BE COMPREHENSIVE. ADDITIONS AND MODIFICATIONS TO FIT LOCAL PRACTICE ARE ENCOURAGED.

Example of the WHO Surgical Checklist

Aviation checklists

When flying a plane the check-list can be kept the same every time for each type of air-craft. In addition, they are a designed for a series of very specific statements, which simply require a confirmation response – 'CHECK'. As we saw earlier with Flight 1549, this pro-vides a clear message that the information has been received, understood and agreed. This process also provides an even greater level of certainty and reassurance as com-pared to the WHO surgical checklist, where some of the questions are 'open' and may re-sult in quite extensive responses to questions such as 'Do you have any concerns?'.

This is the difference in the 'Challenge –response' checklists. A clear pre-determined ques-tion demands a clear and predetermined response, which is rarely more than one or two words.

Flying plays a major part in some aspects of emergency medical care such as Air ambulance work and those involved will be well aware that even for an emergency take off the pilot will complete a pre-flight check list. What this demonstrates is that these lists are considered of absolute importance but if necessary they can be done very quickly, without undue delay and without compromise if part of a well established and rehearsed routine.

If we consider air ambulance work at little further, it is also a confined space environment, with limited resources, which can also move in all three dimensional planes, suddenly and at high speed. This is a truly challenging environment to manage even the simplest of casualties. As such the team CRM needs to be as good as possible, Clear communication and leadership are essential and just as important, if not more important as good clinical skills.

Anaesthetic Checklists

Just like the pilot pre-flight challenge check list we also have an anaesthetic one for pre-hospital or emergency anaesthesia. There is little excuse not to follow such an approach and all leading Trauma and pre-hospital systems around the world are adopting such a system. Interestingly, whilst most people would consider that the

most high risk airway management situations occur prehospital or in the resus room, the NAP4 study demonstrated that the highest risk area was actually the intensive care unit, yet very few in the UK currently use a challenge check-list and good CRM as part of their standard approach to emergency airway management.

The ATACC RSI challenge checklist has been based on a number of other similar ones drawn from specialist teams from around the world, but they are all very similar. The checks take approximately three minutes, which also provides time for good simultaneous pre-oxygenation. Each challenge or question demands a clear response or 'CHECK'.

The grey shaded areas are for use on the rare occasions where the patient is not breathing adequately or is deteriorating rapidly requiring immediate intubation. This is obviously a degree of compromise but constitutes the absolute minimum pre-anaesthetic checks for out of theatre RSI.

As we suggested earlier for us to establish all of these principles of team working, leadership, CRM, checklists etc we need to gather the whole team regularly and get them to train together, We cannot expect a group of strangers to come together on an

infrequent basis and suddenly perform like an F1 team. We therefore need to identify the team members, encourage them to fully adopt a systematic approach to trauma care. The team members then need to come together with all their core skills, on a regular basis to practice, full integration as team and to ensure that all SOPs actually work.

Ideally, the team should gather each day or each shift for a personal introduction and a briefing about the work ahead and any issues relating to kit, facilities or team members. This is typically called the 'Team Brief'

The Scribe

Finally, we have already mentioned the importance of good communication, but that largely referred to verbal communication, but there also needs to be a written record of events. Whilst the Trauma Team are working it is essential that a robust and reliable record is being kept of all key findings, interventions and observations. Always remember the legal position that: if it hasn't been written or recorded then it didn't happen.

Prehospital and in a small team then someone still needs to keep a record, but at the very least you should keep a record of your own actions. Be aware of everything.

In a hospital trauma team, everyone will be busy with their active designated roles and the Team Leader often has far too much to concentrate on, but a scribe based at the shoulder of the team leader can keep this record, based on details and information 'snippets' fed to them by the Team Leader.

The scribe does not have to be a senior clinician or nurse, but they do need to have some understanding of the language used and the technical terminology, or they will miss key facts and details, whilst recording unimportant information.

The records of the scribe, provide the Team Leader and other team members with a rolling record of all the patient details from admission, which can be consulted at any time to ensure that nothing has been missed. This should ideally all be recorded in Trauma Patient pathway, which follows the patient and ultimately is secured in the case notes.

Fire & Rescue Team Approach

We have already discussed the role of the Team leader, but every member of the team needs to know their role, because everyone is an important element of the team. The Fire and Rescue services adopt

a team approach for every incident and as such, when they arrive, they can immediately set to work on their particular task, under the supervision and guidance of the team leader or Officer in Charge (OIC).

For example at a road traffic collision they will adopt the following roles:

- Officer in Charge -Team Leader
- Technical - Stability, glass management & extrication tool operators
- Medical - Casualty Carers

The roles are dictated and shared to significant degree by the number of team members available. Other roles will also be required such as Safety Officer, communications, command support and these will be identified and established as the incident develops and more help arrives. This is a dynamic team, which can adapt and change, under the direction of the Team leader, as required. For example, if they arrive first on scene and find four seriously injured casualties. In this situation, scene safety, triage and casualty care become the greatest priorities and before any further assistance arrives, the majority of crew members will stick with a casualty care role. The Officer in Charge will still try and stand back and keep an overview.

Consider an RTC or complex trauma incident, even in the hospital, a large number of individuals will arrive. Everyone wants to help or get involved but the team leader must keep the team one step back wherever possible and decide who does actually need direct involvement. A doctor on scene may need to rapidly evaluate a casualty and manage a life threatening problem, or he or she may have already done this and is now simply in the way!

If it is not essential to be immediately hands on the casualty then take a step back, move your equipment if it is causing an obstruction and let the extrication proceed. If you do need to be actively involved then do everything that you can to keep the process moving along. Avoid constant stops to perform unnecessary medical interventions, give the firefighters space to operate their tools to free the casualty and ideally adopt a process of 'continuous activity' with casualty care and extrication working hand in hand simultaneously. This is a skill that you will hopefully learn and adopt during the ATACC course extrication and rescue training.

Basically, always ask yourself, is this procedure or assessment essential or are you simply getting in the way and delaying the

extrication and rescue. Work as part of the team, not against it!

Military MERT Teams

Earlier in the chapter we considered the work of a team on an air ambulance and many less experienced medics or teams and even transfer training courses will teach that you that you cannot achieve a great deal in terms of casualty interventions 'en route'. They would suggest that all actions and key procedures should be completed before departure in a place of safety. However, whilst this is undoubtedly true for critical care transfers, trauma transfers are different, especially from the scene, where there is a real degree of urgency and we don't have the luxury of a safe, warm, well lit hospital environment to stay and better prepare our patient for the transfer. As such these transfers, even from a Trauma Unit to a Major Trauma Centre, may be far more active and involved, with ongoing resuscitation and care in transit. Some things must be done before you leave, but many others can be done during the transfer to save time.

But what can realistic be achieved in transit? Well with a suitably trained, and equipped team it is remarkable what levels of trauma care that can be delivered. Consider the Military MERT (Medical Emergency Retrieval Teams) working in Afghanistan. They are working in a confined space, often with low light in an aircraft frequently performing evasive manoeuvres, to avoid incoming live fire with some of the most major trauma found anywhere on the planet. Despite this, their team deliver a highly advanced level of invasive trauma care and perform airway skills, chest decompression, thoracostomies and other skills successfully to save lives. They can do this, because they have focused on the task in hand and then trained and simulated the realities of the challenge they face to best prepare their teams.

As a trauma care provider, if you are involved in pre-hospital or transfer of these patients, consider what level of care that you can realistically perform 'on the move'. You do not want to compromise patient safety and some procedures such as RSI and intubation typically demand a static stable environment, but what if you work on ships? Then you need to consider what challenges that you face and simulate and train with you team.

Similarly, there may be safety issues to consider for example a combative patient will need to settled before helicopter transfer or a chest may need to be decom-

pressed before a high altitude transfer. Consider the nature of your work, plan ahead and be prepared.

The Team Approach - Summary

Team work and CRM are huge subjects and extend far beyond this chapter, but we have tired to highlight a number of the key issues and various ways in which they can be addressed.

Any team, but especially a trauma team needs good leadership. The team must be appropriate and clearly identified to other members and they must come together, not only for real trauma cases, but also to train and simulate live incidents as much as possible.

When working with other agencies and teams, we must consider the importance of their role and interact with them accordingly, without unnecessarily inhibiting them in their task. A good example is the interaction between medical and fire & rescue staff at incidents where medical care should not interrupt the extrication unless absolutely essential or impossible without the firefighters stopping.

Finally, the key to a well functioning team is good Crew Resource Management, which encompasses all of the above and also many other issues relating to team work, performance evaluation and most of all communication. CRM is a whole specialist field in itself and during the ATACC course we will constantly highlight CRM issues, introduce you to some of the key elements and demonstrate how you and your team may be improved in whatever environment you work.
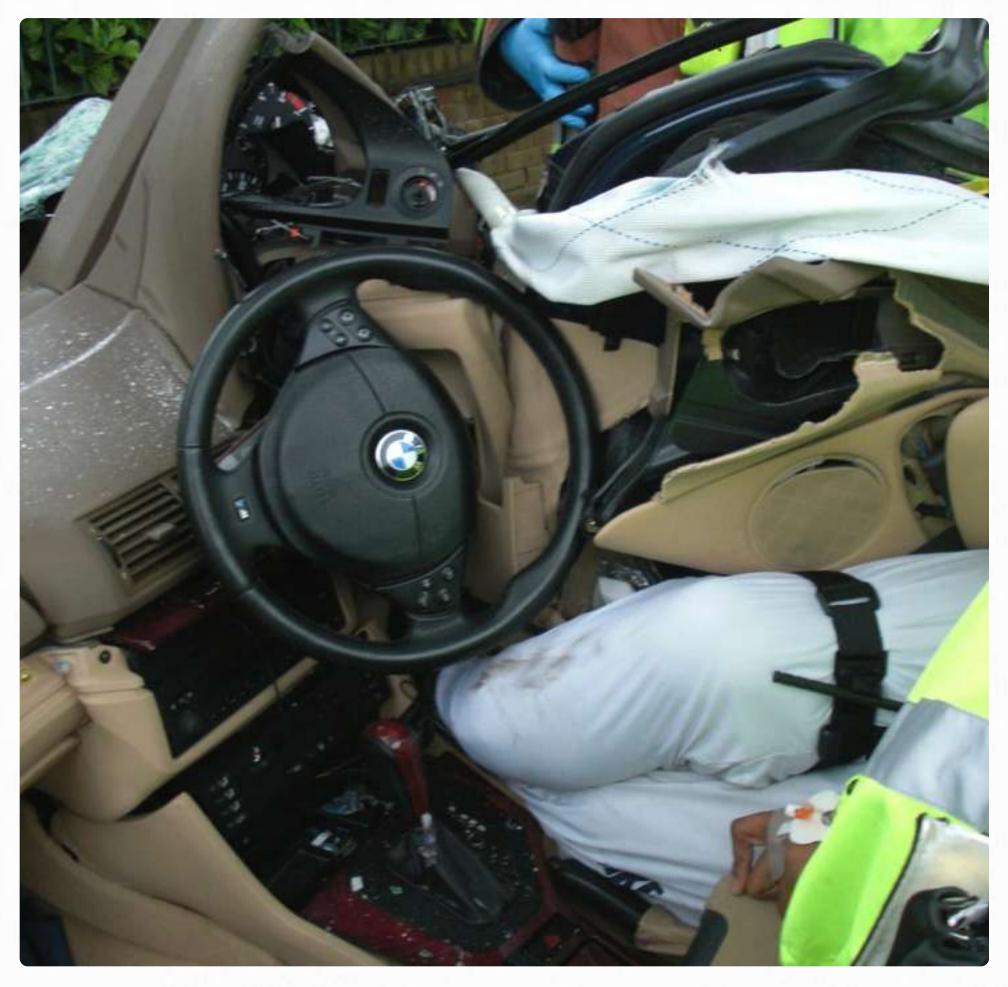
References

1. http://www.infiniti-redbullracing.com/article/sebs-great-eight-states

2. Global aviation Training – CRM for aviation course

3. National Transportation Safety Board: Accident report- US Airways, Flight 1549, NTSB/AAR-10/03 – May 2010

4. 'To human is err' – Alexander Pope

5. www.Laerdal.com - Human simulation

6. Velji, K; Baker GR, Fancott C et al. (2008). "Effectiveness of an Adapted SBAR Communication Tool for a Rehabilitation Setting". Healthcare Quarterly(Longwoods) 11 (Spec.): 72–79. PMID 18382165.

7. MIST handover - ATMIST modification
http://www.swast.nhs.uk/Downloads/Clinical%20Guidelines%20SWAT%20staff/CG05_ATMIST_Patient_Pre-Alert.pdf

8. WHO - safe surgical checklist
http://www.who.int/patientsafety/safesurgery/ss_checklist/en/index.html

9. RSI challenge checklist -
http://nswhems.files.wordpress.com/2011/09/rsimanual2-0final.pdf

10. J R Army Med Corps. 2010 Dec;156(4 Suppl 1):365-9. Simulation, human factors and defence anaesthesia. Mercer SJ, Whittle C, Siggers B, Frazer RS.
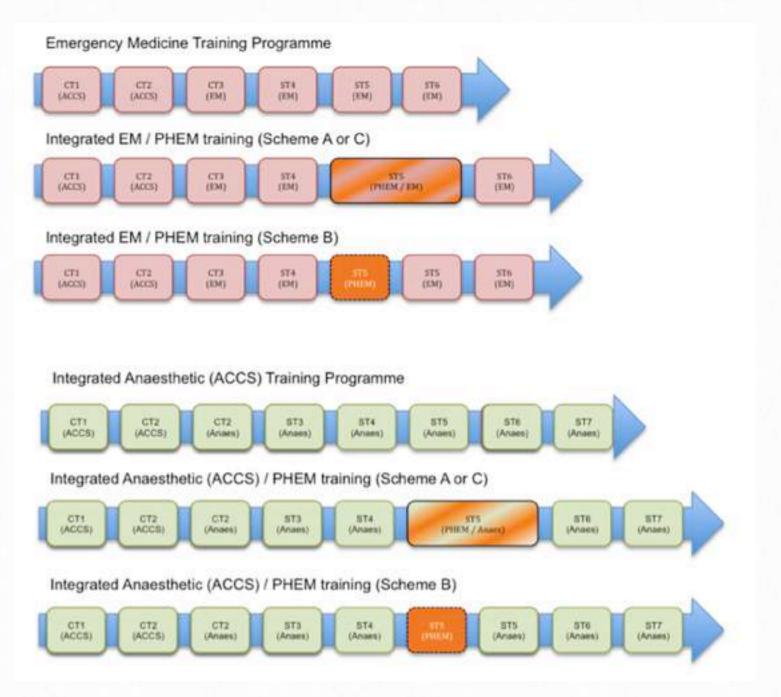
3

Prehospital Care

The Speciality

Emergency Medicine Training Programme

CT1 (ACCS)	CT2 (ACCS)	CT3 (EM)	ST4 (EM)	ST5 (EM)	ST6 (EM)

Integrated EM / PHEM training (Scheme A or C)

CT1 (ACCS)	CT2 (ACCS)	CT3 (EM)	ST4 (EM)	ST5 (PHEM / EM)	ST6 (EM)

Integrated EM / PHEM training (Scheme B)

CT1 (ACCS)	CT2 (ACCS)	CT3 (EM)	ST4 (EM)	ST5 (PHEM)	ST5 (EM)	ST6 (EM)

Integrated Anaesthetic (ACCS) Training Programme

CT1 (ACCS)	CT2 (ACCS)	CT2 (Anaes)	ST3 (Anaes)	ST4 (Anaes)	ST5 (Anaes)	ST6 (Anaes)	ST7 (Anaes)

Integrated Anaesthetic (ACCS) / PHEM training (Scheme A or C)

CT1 (ACCS)	CT2 (ACCS)	CT2 (Anaes)	ST3 (Anaes)	ST4 (Anaes)	ST5 (PHEM / Anaes)	ST6 (Anaes)	ST7 (Anaes)

Integrated Anaesthetic (ACCS) / PHEM training (Scheme B)

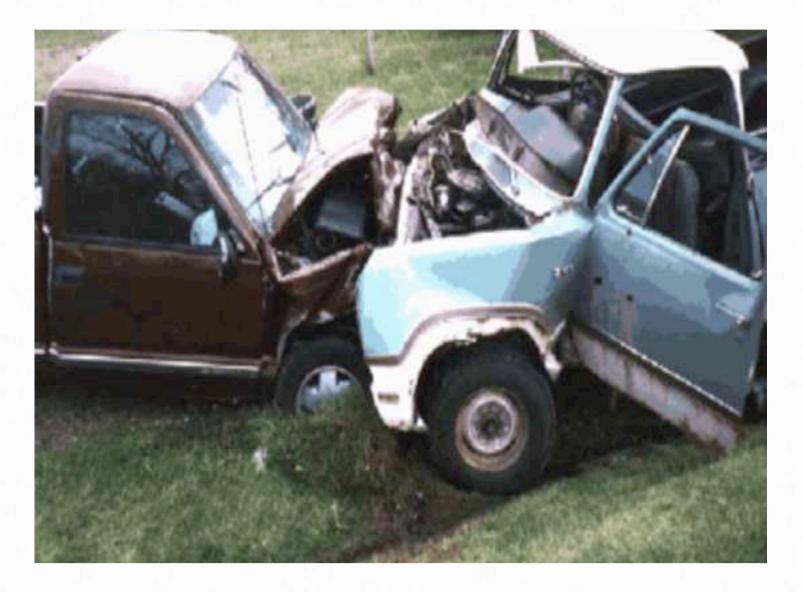| CT1 (ACCS)	CT2 (ACCS)	CT2 (Anaes)	ST3 (Anaes)	ST4 (Anaes)	ST5 (PHEM)	ST5 (Anaes)	ST6 (Anaes)	ST7 (Anaes)

Traditionally, the 'Forward Aid Call' was a daunting experience for many medical & nursing staff. After receiving an urgent call down to the emergency department, a hastily assembled team of hospital doctors and nurses was given a variable amount of poorly fitting high-visibility/protective clothing and the forward aid medical/anaesthetic equipment, before being transported to the scene of an accident at high speed in an emergency ambulance.

If a hospital team had been requested by the ambulance service then, the incident was likely to be serious — involving entrapment, prolonged extrication or complicated injuries. You would suddenly find yourself working in an unfamiliar environment, often with sub-optimal equipment, poorly designed for out-of-hospital work, alongside a group of highly trained emergency service personnel, who clearly know their role, and were expecting the hospital team to manage medical issues beyond

their level of skill. This inevitably led to a range of Human Factors and Crew Resource Management problems, frequently to the detriment of the patient.

This haphazard approach is thankfully becoming a thing of the past. Pre-hospital Emergency Medicine (PHEM) is now a recognised sub-specialty in the United Kingdom. A formalised training program, run by an Intercollegiate Board for Training in Pre-hospital Emergency Medicine, encompasses the underpinning knowledge, technical skills and non-technical (behavioral) skills required to provide safe pre-hospital critical care and safe transfer.

The role of the Pre-hospital Emergency Medicine Practitioner is uniquely challenging. The tempo of decision making, the threats posed at incident scenes, the relatively unsupported and isolated working conditions, the environmental challenges, the resource limitations and the case mix all make this a very different activity compared to in-hospital Emergency Medicine and Anaesthetic practice.

Mechanism of Injury

Kinematics is the study of how matter moves and interacts in collisions, whether this is a person in a car, a person against a floor or the thoracic contents striking the inside of the rib cage. Understanding the forces generated in collisions and how quickly they act on casualties involved in an incident allows us to understand potential mechanisms of injury and have a high index of suspicion for potential occult injuries

Physics

The first step is to understand how the forces are generated and dissipated in collisions, to do this we need to understand several key fundamental physics principles. The first principle we need to consider is Newtons first law of motion which states that an object moving at a constant velocity (or at rest) will continue at that velocity unless acted on by an external force. Further more the force (F) required is pro-

portional to the product of the mass (m) times the acceleration (a).

$$F = ma$$

Consider an occupant of a car travelling at 60 m.p.h., the car strikes a tree providing a force large enough to stop the car, the person however is still travelling at 60 m.p.h and requires a force to stop them (typically from the seat belt) and finally even when the person is stopped their internal organs continue to move, giving rise to shearing injuries.

The second consideration is the amount of energy liberated at the moment of impact. The kinetic energy of a moving object can be found from:

$$KE = \frac{1}{2} mv^2$$

(where KE = kinetic energy, m = mass and v = velocity)

From this expression it can be seen that the most important aspect of the equation is the speed, by doubling the speed we quadruple the kinetic energy liberated - the road safety message "Speed Kills" is a valid one. However, a very heavy object moving slowly (such as a tanker) also has an enormous amount of energy. The final thing we need to consider when evaluating the incident is to assess the rate that the momentum changed either by acceleration or deceleration; this leads us to the impulse momentum theorem.

$$F = \frac{\Delta P}{\Delta T}$$

An example of this can be considered by thinking of two different situations, firstly where a person falls onto a concrete surface and secondly the same person landing on a soft mattress. In the first example the deceleration is very rapid and therefore a lot of force is generated as the change of momentum (ΔP) occurs over a very short time (ΔT). In the second example the mattress cushions the fall - in effect slowing the rate at which momentum is lost. As a result, less force (F) is applied to the victim.

Airbags and vehicle crumple zones are an attempt to extend the impact and change of momentum time thus reducing the force generated on an object. Seat belt pretensioners pull tight against the body to make sure that the body is immediately restrained and does not accelerate forward and then stop suddenly. Seatbelts are now even constructed with rip apart sections to extend the length whilst decelerating and therefore the prolong the amount of time to stop the body.

An interesting consequence of these safety innovations is that a newer car will look more damaged following an impact when compared to a structurally sound older one, the important difference though is that the newer car has extended the impact deceleration time and absorbed a lot of the energy (see picture at start of section).

Road Traffic Collisions

Using the principles of kinematics we now have an understanding of the forces involved in collisions and we can relate this to the injury patterns we see, or suspect. Road traffic collisions involving vehicles will broadly fall into the following categories;

Car versus:

• Pedestrian
• Cyclist
• Motorcyclist
• Other road user (e.g. horse & rider)

Car versus Car or other large vehicle (Casualty Entrapment):

• Head on frontal impact
• Side impact (T-Bone)
• Rear Impact

Car/Cycle/Motorcycle versus Stationery Object (e.g. tree):

• Head on frontal impact
• Side impact

Following any of the above collisions, the vehicle can come to rest in the following orientations:

On its wheels*
On its side
On its roof

*Remember the fact that a car is on its wheels does not mean that it has not also rolled over. Think Kinematics!

The commonest incident will involve a frontal impact to a car on all four wheels, with a single occupant. This allows us to consider and focus our training.

Pedestrian

During an impact with a pedestrian, the vehicle will usually suffer minimal damage but the pedestrian (who has little effect on the speed of the vehicle) suffers from a large transfer of momentum with massive acceleration from near zero. This force can obviously cause massive trauma to any internal structures.

The sequence of events is important to understand as the pedestrian goes through (usually) 3 separate collisions:

- The initial impact with the vehicle e.g. front bumper causing lower limb trauma (in an adult)
- Secondary impact as they strike the bonnet of the vehicle causing head, neck and chest injuries
- Tertiary trauma is caused when they contact the ground resulting in head, spinal and abdominal injuries
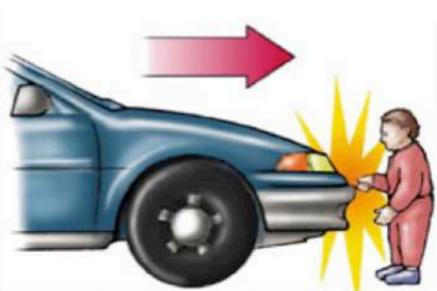
Summary of Typical Pedestrian Injury Patterns:
- Sheer injuries to kidneys, liver and spleen
- Lower limb injuries
- Hyperextension/hyperflexion injuries to the neck
- Head Injuries
- Chest injuries
- Pelvic fractures

N.B. The comments above assume a standard sized adult being hit by a small to medium sized car. The pattern of injury in the paediatric population or in an adult hit by a large vehicle or truck will change, as the angle of impact and the amount of energy transferred as a result will differ. An anal-ogy is hitting a golf ball with an iron versus a wood club. The iron (in this case, a standard size car) gets under the ball and elevates it. In the same way, a frontal pedestrian impact will result in the victim rolling over the bonnet and the roof of the car. In contrast if a golf ball is hit with a wood, a higher amount of energy is imparted and the trajectory of the ball is flatter. In the case of a pedestrian victim, this equates to a greater forward momentum and different pattern of injury. In the same way that an adult will not roll over the roof of a semi-articulated lorry if they are hit by one, a small child who is hit by a normal sized car will also be thrown forward rather than rolled over the bonnet/roof. The energy transfer (and hence damage) is greater in these impacts for three reasons - firstly the mass of the vehicle is larger, so by the kinetic energy equation shown above the energy of the moving vehicle is higher. Secondly, the angle of impact results in a higher transmission of energy (think of a direct blow vs a glancing impact). Thirdly, the energy transfer per kilogram body weight is higher, so the forces imparted can do more damage to the victim for the same amount of energy transfer/absorption.

Patterns of injury change depending on the size of vehicle and victim. The adult on

the left will roll over the bonnet and then the roof, whereas the child on the right will be thrown forwards in the direction of the car.

Cyclists and Motorcyclists

Cyclists and Motorcyclists may fall, collide with other vehicles or collide with stationary objects. The nature of injuries sustained by riders of cycles/motorcycles varies considerably. It is also very dependent on the level of personal protection worn by the rider. Without the presence of crumple zones or protective airbags to decrease the amount or rate of energy transmission, the rider is subject to the entire kinetic energy of the collision. Without restraints the rider is also exposed to rotational forces, which can have a devastating effect. If the energy is dissipated by the rider sliding or rolling down the road then they are likely to suffer less severe injuries than if they have

a sudden deceleration from hitting a wall or a tree for example. Almost any type of injury is possible within this group and as much detail about the kinematics should be gained immediately. It goes without saying therefore that this group of road users are at very high risk and require an appropriate high index of suspicion.

Frontal / Head-on

Approximately 70% of all collisions are frontal resulting in rapid deceleration and large forces transmitted to the occupants of the vehicle.

Unrestrained passengers in the vehicle move forward, their knees striking the dashboard, head hitting the roof and then the windscreen (look for bulls-eye on the inside of the windscreen) with a high chance of ejection from the vehicle, through the windscreen. Thoracic and up-

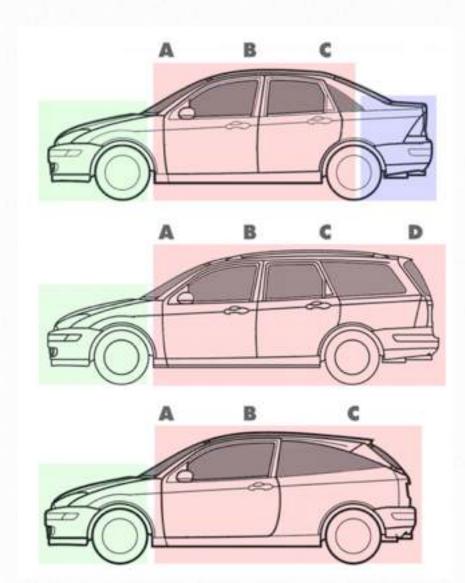per abdominal injuries occur from collision with the steering wheel.

The prevalence of airbags in vehicles reduces the effects of some blunt force trauma, but not all and the passengers still tend to move forward and can receive serious injuries unless wearing a seatbelt. If the A post deforms during the impact then this can also cause head injury.

The airbag deployment itself can cause injuries, the talcum powder used to cover the air bag results in a fine dust which can aggravate respiratory problems. Burns and grazes can occur on arms and faces plus arm injuries can occur as the airbags deploy. Casualties may also be temporarily deafened from the noise of the explosive deployment in the confined space of the care. With an impending collision the driver tends to brace them selves for the impact holding their breath. This can result in the paper bag effect type injury where pulmonary injury occurs, as well as fractures or dislocations to the wrists and/or forearms.

Summary of injury patterns:
- Shear injuries to kidneys, liver and spleen
- Hyperextension of the lumber spine
- Chest injury
- Pelvic fracture

- Leg injuries
- Hyperflexion of the neck
- Axial compression of the spine
- Ejection if unrestrained
- Chance fracture and associated injuries

Location of A, B and C posts on various vehicle body types

A Chance fracture is an anterior wedge fracture commonly occurring at the thoracolumbar junction (T12 to L3) or mid-lumbar region. This fracture is traditionally associated with lap belt restraints as a sudden forward flexion of the spine causes a wedging of the spine, occasionally associ-

ated with a posterior transverse fracture of the spine. Whilst the anterior wedge fracture itself is managed non-operatively unless there is either associated spinal cord compression, disruption of spinous ligaments or column damage, they are associated with mesenteric, pancreatic or duodenal injuries in 50% of cases in some series, and should raise the index of suspicion of occult intra-abdominal injury.

Chance fracture of T9 and T10

This also assumes that any seatbelt restraint systems are worn correctly and the patient is the appropriate size to sit in the seat. The effect of "submarining" (sliding down and forward, out of a restraint system) can also lead to prolonged entrapment, lower limb and pelvic fractures and axial loading fractures of the spine.

Rear Impacts

Rear collisions are less common but present a higher risk of cervical spine injury than frontal collisions. The initial collision forces the occupants backwards stretching the body backwards and upwards leading to neck injuries especially in cars without (or with badly adjusted) head rests. When the car stops moving the occupants are then forced forwards and may have similar injuries to the frontal collision.

Summary of injury patterns:
- Hyperextension of the lumber spine
- Hyperextension and hyperflexion of the neck
- Shear injuries to kidneys, liver and spleen

Side / T bone Collision

Despite the increase in strength in modern vehicles the side of a car has less room and mass with which to absorb energy and we still see side impacts resulting in far more serious injuries and more entrapments. The close proximity of the vehicle construction means the occupant will more likely suffer a head injury, typically from the B post. Other injuries are typically all down the side of the impact but may

also occur from hitting the other passengers or fittings inside the car. Of course the exact injury pattern depends on the impact point and the location of the occupant (driver/passenger or front/rear seat).

During a side impact the occupant initially moves towards the direction of the incoming force. Immediately after this the reaction forces then move the occupant in the opposite direction. Remember it is possible that two occupants may impact upon each other in the event of a side impact.

Summary of Injury Patterns:

- Head injury through impact with the side of the car (then possibly with adjacent occupant)
- Shear injuries to kidneys, liver and spleen
- Pelvic and long bone injuries
- Pulmonary contusion and diaphragmatic injury
- Lateral hyperextension of the neck
- Arm and leg injuries

Rollover

Although statistically rare, rollovers often produce the most severe injuries. Only 2.4% of crashes involved rollovers but they accounted for one third of occupant deaths. Restrained passengers will normally remain in their seat but will be struck by unrestrained objects/occupants in the vehicle. If casualties are unrestrained they may be ejected from the vehicle leading to a higher rate of mortality.

Rollovers can occur end over end, following a frontal/rear collision or side rollover following a T-bone collision. It is important to remember that a car that has rolled over may come to rest on its wheels, roof or side, and this is where the principles of kinematics are extremely valuable. Look for deformation of the roof, multiple body panels and damage to the environment.

Due to the nature of rollover incidents almost any injury is possible and occupants require a high index of suspicion.

Falls

Falls from height may produce major injuries, and are not infrequently involved in successful suicides. Clearly the most important factor influencing the severity of injury is the height fallen. However, as described above, the rate of deceleration is also important, as are the parts of the body striking the ground. Falls from greater then six metres are a major cause for concern. In addition, serious injury has been reported in falls from much lesser heights, and many triage systems use 2 metres or three times the patient's body height as a cut off for a 'major' response. Injuries associated with a fall from height onto feet typically include calcaneal fractures, lumbar spine blowout fractures and possibly unstable vertical shear pelvic injuries.

Blunt trauma

Blunt trauma is essentially any traumatic injury that does not involve an object entering the body. It may be further subdivided depending on either the mechanism (assault, RTC (Road Traffic Collision), fall from height etc) or clinical symptoms and signs that appear as a result (bruising, laceration, fracture of bones). Typically blunt trauma is more common than penetrating (essentially stabbings or shootings) and will form the bulk of the workload that the NHS trauma services see. Internationally, blunt trauma is the leading cause of traumatic death in the USA, and the most common cause of death within the blunt trauma subset is RTC's.

In contrast to penetrating trauma which may have a single bleeding point or involve only a limited area, patients who are involved in a severe blunt trauma such as a rollover or ejection RTC will present with a multitude of injuries involving remote regions of the body. Blunt traumatic injuries usually have a worse outcome in comparison to penetrating injuries as there is a higher kinetic energy transfer in most cases, and a higher resultant area of tissue damage. This causes the release of various cytokines and pro-inflammatory substrates such as DAMPs (Damage Associated Molecular Proteins - e.g. mitochondrial DNA, HMGB1, Syndecan-1), the magnitude of which can be a marker of amount of tissue damage and oxidative stress. If there is a large amount of bleeding and tissue damage, the microcirculatory system is damaged (specifically the glycocalyx of cells) leading to hypoxic/hypotensive cell death. This protein and glucose extracellular layer has been implicated in many cell regulatory processes and its destruction is a marker of poor tissue perfusion and under-resuscitation, which may cause a

systemic inflammatory response syndrome (SIRS). If we can protect microcirculation and by extension the glycocalyx, we may optimise resuscitation.

Penetrating Trauma

Penetrating trauma from stabbing or shooting can represent a vast spectrum of pathology depending on what cavity is penetrated, with what instrument, with what degree of force and what organs or tissues are injured. Ballistic trauma is considered elsewhere in this text, but generally speaking with low velocity trauma such as stabbing, source control is usually surgically more straightforward. Once the appropriate vessel is ligated and systemic flow restored, as long as the period of hypoperfusion and poor oxygen delivery has not been too extreme then the systemic sequelae of low velocity penetrating trauma is not too severe. The obvious caveat to this is that if a vital structure such as the heart, major thoracoabdominal or peripheral blood vessels have been injured then the likelihood of this being the clinical picture declines significantly, as does the presence of profound hypotension for a prolonged period of time.

We specifically discuss Ballistics in the Specialty Care Chapter "Tactical Medicine"

Summary

By understanding the magnitude of the forces involved in collision and the direction they occur we can deduce the potential injuries that the casualty may suffer. In particular:

• Pedestrians are at high risk of serious injury if struck by moving vehicles and should be considered to have such injuries until proved otherwise.

• Patients ejected from their vehicle or involved in a roll over are at increased risk of death.

• Falls from height carry a risk of major injury, especially from above six metres.

• The external wounds sustained by victims of shootings may not reflect the internal damage.

Further Reading

Road Traffic Collisions:

http://ftp.rta.nato.int/public/PubFullText/RTO/EN/RTO-EN-HFM-113/EN-HFM-113-06.pdf - Overview of G-load tolerance and human survival on impacts

http://www.euroncap.com/home.aspx - comparative car safety in various collisions

http://www.holmatro.com/en/vehicle-rescue/news/204-available-now-vehicle-extrication-techniques-training-book-by-holmatro.html

http://blog.holmatro.com

Safe Approach

The traditional approach to a training incident of "Scene safe?"; a nod, a tick in a box and then move on to the meat of the scenario is standard in simulation training, however in ATACC, as in real life, ensuring your safety and that of your team is a key priority.

Simply verbalising that you 'thought' about safety for a brief moment at the beginning without actively managing risk will put you and your team at great risk

Thankfully, most places in which we work are designed to be safe - hospitals are well lit and spacious, security guards around the corner and without trip hazards, traffic, unprotected heights or exposed wires.

However when we look at trauma patients 'from roadside to critical care' we move out of a hospital environment and the considerations change. Trauma, by its nature is unplanned, unpredictable and has a cruel sense of humour: your stab victim

will be in the smallest room at the back of a house during a wild party, or the painter who falls off his ladder will never land conveniently on the front driveway - he will be on the upper level of the enclosed back yard.

To manage these incidents we need to develop skills other than clinical skills, and these need to be applied throughout all phases of the incident:

- Teamwork
- Communication
- Hazard identification and risk assessment
- Scene/situational awareness

Before the Call

Your employer or agency defines what role you have at a scene. At a rescue the fire service is in charge of the scene, and will have an Incident Commander who will declare the scene safe or unsafe to enter, whilst the Police will control access to the scene. Rescuers from all organisations must follow these directions.

No organisation wants individual practitioners to take risks that might compromise their safety. You need to be familiar with your own workplace or team health and safety rules - examples include long

sleeved high visibility jacket and helmet at all road traffic collisions, buoyancy aid when working near water etc.

Under UK workplace health and safety legislation, both employers and employees have responsibility to maintain safe work practices, to identify hazards and to take steps to lessen the risk and to provide and use safety equipment.

 "It is an employer's duty to protect the health, safety and welfare of their employees and other people who might be affected by their business. Employers must do whatever is reasonably practicable to achieve this."

"Workers have a duty to take care of their own health and safety and that of others who may be affected by your actions at work. Workers must co-operate with employers and co-workers to help everyone meet their legal requirements."

These laws apply at all times, and breaking them can have serious consequences for the organisation or the individual, including fines and imprisonment.

Every organisation with 5 or more employees must have a written Health and Safety Policy document, and you should familiarise yourself with yours.

Your own safety must be your number one priority at all times, your second priority is the safety of your team and your next priority is the safety of your equipment.

The patient is never more important than the safety of you and your team. A hero mentality - "I'll save him at any cost" is foolhardy and never supported by your employer.

Zero Harm is a relatively new concept in the EMS world, though it has been around a long time in the oil and mining industries. When Zero Harm and EMS are mentioned together people laugh and say "no way, that's totally unachievable" and perhaps they are correct because we are human beings working in unplanned and unpredictable situations. However accepting an attitude that we leave our family every day and a certain percentage of us of will not come home, or come home injured, is not acceptable. We should certainly aim for Zero Harm, take every measure and follow every step that will keep us safe and well while doing our job.

Preparation is the first step to safe practice, and to keeping yourself and your team safe.

- Frequent kit checks have many benefits - you know that you are fully stocked, where all the equipment is and how it is laid out.

- Preparing your kit well also presents an opportunity to prioritise safety - placing safety equipment appropriate for a task with the tools for that task, for example having safety glasses with the laryngoscope and a sharps container with the cannulae.

- Practicing with your team develops relationships and helps team members identify strengths and weaknesses within the team, which ultimately lead to a stronger and safer team.

Ideally we would all be totally familiar with all of our team, our workplace and our equipment however that isn't always achievable in the real world. We need to be aware that when a given situation is less than ideal, we need to make an extra effort to keep safety a priority.

When you find yourself in a situation where you feel less than prepared, communicating with your team to identify the hazards, assess the risks, make safe plans, and share the mental model will make the job safer.

Communications

Communication systems and command and control systems are different in every service and in every area. You need to be familiar with the system in your own area in order to

- Receive tasking and confirm response to an incident

- Alert the control room if you need urgent assistance. All communication systems have a way of contacting them urgently if you are in danger. This made be a specific verbal radio message or a code to input into the radio

- Send and receive information about an incident

- Maintain a record of the response to an incident

Personal Protective Equipment

A huge range of expensive equipment is available. Your role needs to be clearly identifiable, and your PPE should give you adequate protection for the task to be undertaken but remember that your PPE is not a superman suit. Gloves and safety glasses offer a layer of protection but you may be dealing with hazards that are far greater than the protection offered by a layer of plastic.

Your employer should dictate which PPE you are required to use. How you organise your equipment is what makes it really effective. Having the safety glasses in your intubation bag is a great idea, you will definitely need them then, but if you arrive at an arterial bleed you may not have time to open zips and search through bags to find them. Having another pair in your pocket or tucked behind your collar will mean you have them quickly and not feel pressured into taking the risky gamble of doing without them.

The same thought process should go into each item. Safety glasses, a torch, a trauma shears, extra gloves (a size bigger if you are sweaty) can all be in your pockets. Your helmet and debris gloves should be located where you can access them easily, or you can describe exactly where it is to somebody who can fetch them for you.

Your hi-vis jacket should always be close at hand and be zipped up when worn to ensure you are visible from the front.

Driving

Emergency driving and navigating are difficult skills to master. They both take a lot of focus. When a team is responding 'lights and sirens/code one/hot' to an incident the driver and passenger are both busy with tasks, more so in heavy traffic or built up areas.

The passenger assists with identifying hazards and checking for oncoming cars from the left. As the vehicle enters an intersection the passenger looks left and says "clear left" when it is clear to proceed, but the ultimate responsibility lies with the driver and this trust only comes with time and experience.

The driver and passenger need to remain focused on safe driving and should only listen to or participate in the team planning and comms when it is safe to do so.

In all cases the driver should drive within their own skill and ability and their employer's driver training guidelines. Red Mist is defined as "A mental and physiological state which drivers experience when they are so determined to achieve some non-driving objective that they are no longer capable of assessing driving risks realistically".

Remember the key phrase – 'Drive to arrive'

The passenger should communicate with the driver if they are concerned about their safety and remind them that the priority is safety not speed. Ambulances crash, even more often while on emergency responses, so ensure that all passengers are wearing seatbelts at all times, and that all loose items and heavy equipment is securely stowed.

Leadership

Who is in charge?

The fire service is the agency responsible for overall scene command in most instances. The fire service will designate a scene commander and he or she will be identifiable by a tabard, and will usually be wearing a white helmet. This is the officer in charge or OIC, responsible for declaring the scene safe for rescuers to enter and medical responders must take guidance on safety issues from the fire service. Medical responders will be required to wear appropriate PPE and have identification before they will be granted access to the scene.

The police remain responsible for crowd control and traffic management, and overall command will pass to the Police Commander in the event of serious crime, firearms or terrorist incident.

Medical responders to the scene make contact with the Fire OIC and then identify themselves to the Ambulance Incident Officer (AIO), who in the initial stages may be a member of the first ambulance crew to arrive on scene. As a complex incident matures the AIO may be identified by a tabard.

360 Degree Survey

Safe scene assessment begins with a 360 degree survey and rescuers should not proceed until all hazards are identified and communicated. The process of controlling the hazards then begins. At simple scenes, a 360 degree survey can be quickly accomplished; the more complex the scene the more effort should be put into a thorough scene assessment and as further help arrives it maybe sectorised.

Dynamic Risk Assessment

Dynamic risk assessment is the continuous process of identifying hazards, assessing the risks that these hazards present and taking action to eliminate or reduce risks while you work at an incident.

A hazard is a danger, something that has to potential to cause you harm.

The risk is a measure of the likelihood of that hazard actually causing you harm and how great that harm might be.

If the hazard is considerable and the risk is high then immediate action to remove the hazard or reduce the risk must be taken.

This may involve staging or holding back at an area or Rendezvous point (RVP) until the scene is made safe. Even if the hazard is small and the risk is low action should still be taken; this may be simply identifying and communicating the danger to your team mates, or making a change in the plan.

At a busy or potentially hazardous scene a safety officer should be appointed. This person will remain focused on team and patient safety at all times. Having a safety officer still doesn't mean that the rest of the team can forget all safety considerations! Everyone has a duty for their own safety and the safety of the team.

Exit strategy

As you enter a scene you should be planning an exit strategy, should the need arise to leave the scene quickly.

• A slow controlled entry, will raise your awareness of the whole scene and will allow you time to gather information and prepare – including where are the safe exits? Who else is on scene and where are they positioning themselves? Can the scene be made safer e.g. turning on lights, opening blinds, removing dogs, positioning vehicles to fend off traffic, moving people or debris and obstructions?

• Positioning yourself on the exit side of the patient, requesting that the door be left open or unlocked, knowing which level you are going to and coming out of in a high rise building or even nominating a team member just for safety

- In the event of a hurried exit kit bags can be a big hindrance- if you have concerns about the safety of a scene as you approach it will justify you leaving your kit bags outside the door. Never put your kit bag behind you- you will trip over it and look like an idiot. If there is a real threat to your life then just abandon your kit if it is going to slow you escape.

- When possible vehicles should be parked in such a way that that they do not have to be turned around in the event that a rapid exit is required.

- Your instinct for danger or tension on scene develops as you gain more experience, listen to your gut feelings and keep yourself safe. If the crusty old paramedic suggests that everyone leave in a hurry - go with him!

If it's not safe, don't approach

Snatch rescue

Care under fire, is extremely dangerous and there is very little that can be done other than simple haemorrhage control or positioning (rolling them on to their side). Ideally, we need to simply extract the casualty as quickly as possible e.g. a snatch rescue. This is a military concept - when a soldier is injured in a battlefield his colleagues may elect to rescue him by quickly grabbing him and moving him to a safe place where treatment can begin. In the civilian world we would not normally choose to put ourselves in a position of risk to save a patient, however if you are with a patient and the scene becomes unsafe for any reason you may elect to quickly take the patient with you as you move to safety. A snatch rescue may be performed by the fire service where persons are reported in a fire e.g. someone collapsed in the hall of a domestic property, just inside the front door.

Such rescues may involve moving the patient before they are fully packaged, or before they are stabilised, but is useful in the extreme situation where leaving them behind to die is the only other option.

A more likely scenario is where the scene is potentially unstable such that only the most urgent issues are addressed before the patient is moved to a safer environment.

Manual handling

Ambulance Service of New South Wales Multi Purpose Vehicle, used for bariatric taskings.

It's better to be clever than strong- use the lifting and moving skills and equipment- not brute strength

- Recruit help for lifting

- Wearing a hi-vis jacket does not make you stronger. Back and shoulder injuries account for 46% of injuries to paramedics

- Good communication within your team when lifting and moving patients is vital.

- Good communication within your team when lifting and moving patients is vital.

ATACC recommend the following approach to patient moving:

- Declare the command e.g. we will slide on the command 'slide'

- Position your team

- Ask 'Is anyone NOT ready?' – no one should reply unless not prepared

- Ready, brace, SLIDE

Infection Control

Patients and staff are safest when we follow strict infection control guidelines, this is the case in the prehospital setting as well as the hospital setting.

- Before you attempt an invasive procedure you need to prepare and lay out your equipment, prepare the patient (remember a trauma patient may often be physically dirty and an area may need to be cleaned prior to attempting to make it sterile)

- Wear your PPE

- Plan for how you will dispose of sharps and contaminated items.

Outside of hospital all these steps and stages take a lot more time that you would expect if you are used to working in a well laid out hospital department.

The decision to spend that time performing an invasive procedure where there are infection and safety implications for the patient or the staff, should be a balance between the clinical need, how quickly or easily it could be achieved against the effects if postponed until later in hospital. For example: safely setting up for and getting IV access on a stable patient lying on a dark wet road side is a lengthy process, giving intranasal analgesia and moving the patient into a clean bright ambulance removes lots of unnecessary risks.

However a patient on the same dark road with a tension pneumothorax requires immediate intervention, and having your kit

well prepared and your torch close at hand will mean you can act more quickly and safely.

RTC Safe Approach

- Approach the scene slowly and carefully to avoid creating a hazard for people already at the scene

- Park the vehicle in a 'fend off' position to create a barrier between your work area and the passing traffic

- Wear your PPE

- Report to the scene commander

- Listen to the ongoing safety discussions going on around you

Be aware at all times that the road my be 'live', that means traffic may pass you at any time

RTC Safety Equipment

Sharp Edge Protection Covers go over the cut metal to protect rescuers and patients

Chocks and Blocks are pushed under the vehicle to stabilise it while rescuers work.

Protection Shields are flexible strong plastic boards used for protecting patients and rescuers while glass or metal is being cut nearby.

Airbag covers are placed over un-deployed airbags. An airbag may deploy unexpectedly during a rescue even if the battery has been disconnected and cause serious harm to the patient or rescuer.

During Transport

While few published data exist in the United Kingdom, figures from the US show that Emergency vehicles have a higher incidence of fatal road traffic collisions when compared to other road traffic.

Rear compartment occupants are more likely to be injured or killed in a collision than the driver. This may be due in part to the tendency of medical personnel to not wear seatbelts when treating patients in a moving ambulance. Unrestrained equipment and patients are also hazardous in the event of a collision.

An ambulance trolley with a Ferno® 5 point safety harness seatbelt. These seatbelts are tested to 10G in 6 directions.

An ambulance trolley with the equipment safety secured on a trolley bridge.

In-hospital Safety

• The trauma patient did not plan to come to hospital, they may have dangerous tools or weapons in their pockets

• Broken glass and dangerous debris often gets trapped in the patients clothes and packaging

• Patients who are involved interpersonal violence may still be targets even after they arrived in hospital and staff can get caught in the crossfire

Debrief

Taking part in a debrief has many benefits for both the individuals and the organisations involved. Discussions surrounding safety issues give people the opportunity draw attention to hazards, and the lessons learned become collective rather than just individual.

Team members may have different experiences even at the same incident. During a debrief you can realise how much you missed when you were focused on one task or one patient. This can make you realise that you can be extremely vulnerable, and that you need your team to take responsibility for your safety.

Post traumatic stress disorder is a real condition, and working in the unpredictable pre-hospital environment certainly puts you and your colleagues at risk. Knowing the symptoms and knowing how to seek help is valuable, supporting and having the support of your colleagues is invaluable. Understand that this type of work is never simple, that there is no single correct answer, accept that you did your best under trying circumstances and accept that you may have learned a lesson you will use next time.

Talking through and debriefing all of your incidents and especially the more challenging ones will not only educate but also help to reduce the stress of poor decision making or unpleasant incidents. Safety must be your priority at all times, plan and practice for safe working habits, communicate with your team and take every opportunity to avoid hazards and reduce risks.
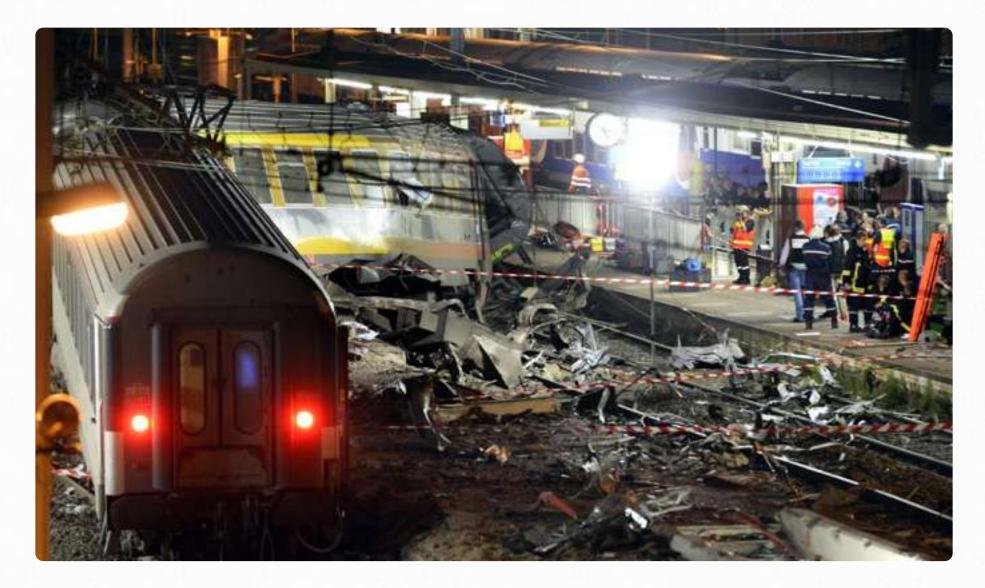
Always Aim for Zero Harm

References

1.
   http://www.hse.gov.uk/workers/employers.htm. Accessed 21/2/14

2.
   http://www.hse.gov.uk/workers/responsibilities.htm. Accessed 21/2/14

3. Roadcraft: the police driver's handbook: The Essential Police Driver's Handbook. Philip Coyne, Penny Mares, Barbara MacDonald. Stationery Office Books, 2007

4. Queensland Employee Injury Database http://www.deir.qld.gov.au/workplace/documents/showDoc.html?WHS%20Publications/healthcommunity%20-%20ambulanceofficerparamedic. Accessed 20/02/2014

5. Teri L. Sanddal , Nels D. Sanddal, Nicolas Ward, Laura Stanley. Ambulance Crash Characteristics in the US Defined by the Popular Press: A Retrospective Analysis Emerg Med Int. 2010; 2010: 525979.

6. Kahn CA1, Pirrallo RG, Kuhn EM. Characteristics of fatal ambulance crashes in the United States: an 11-year retrospective analysis. Prehosp Emerg Care. 2001 Jul-Sep;5(3):261-9.

Mass Casualty Management

During a Mass Casualty Event, the goal is to maximise chances of reducing mortality and morbidity, through efficient use of resources. A major incident is any event where the number of casualties and the rate at which they occur cannot be handled within routine service arrangements.

METHANE Report

On arrival on scene of a major incident, a METHANE report should be given to control. This allows for the activation or standby of a major incident plan and is the most efficient way of giving control pertinent information so that they can start organising a systemic response. It also ensures that no steps are missed in getting an initial report logged. METHANE reports can be repeatedly used at major incidents as more information is gained to give a brief and structured update to supervising officers.

METHANE report

M My call-sign, or name and appointment
Major incident STANDBY or DECLARED

E Exact location
  • grid reference, or GPS where available

T Type of incident

H Hazards, present and potential

A Access to scene, and egress route
  • helicopter landing site location

N Number and severity of casualties

E Emergency services present and required

Triage

Triage is the process of determining the priority of patients' treatments based on the severity of their condition. This rations patient treatment efficiently when resources are insufficient for all to be treated immediately. The term comes from the French verb trier, meaning to separate, sift or select. Triage may result in determining the order and priority of emergency treatment, the order and priority of emergency transport, or the transport destination for the patient.

During the triage process, only immediate life saving interventions, that are non-time consuming, should be performed. For example this may include application of a tourniquet for massive haemorrhage; but certainly not trying to control massive haemorrhage with direct pressure or bandaging.

Triage may also be used for patients arriving at the emergency department, or telephoning medical advice systems, among others.

The term triage may have originated during the Napoleonic Wars from the work of Dominique Jean Larrey. The term was used further during World War I by French doctors treating the battlefield wounded at the aid stations behind the front. Those responsible for the removal of the wounded from a battlefield or their care afterwards would divide the victims into three categories:

• Those who are likely to live, regardless of what care they receive

• Those who are likely to die, regardless of what care they receive

• Those for whom immediate care might make a positive difference in outcome

As medical technology has advanced, so has modern approaches to triage which are increasingly based on scientific models. The categorisations of the victims are frequently the result of triage scores based on specific physiological assessment findings. Some models, such as the START model may be algorithm-based. As triage concepts become more sophisticated, triage guidance is also evolving into both software and hardware decision support products for use by caregivers in both hospitals and the field.
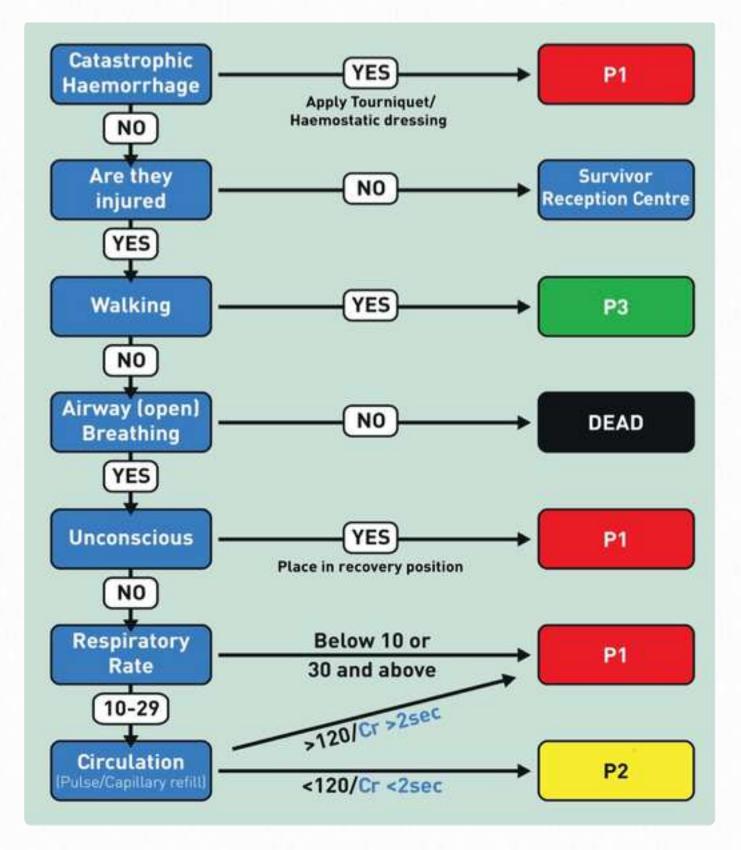
Triage Sieve

START is a simple triage system that can be performed by just about anyone. Whilst it originally was never intended to supersede trained medical judgment, it was quickly demonstrated that during mass casualty events, better outcomes in mortality and morbidity occurred when lay persons performed START without emotion or thinking.

Triage Sieve separates the injured into four groups:

• The expectant who are beyond help

• The injured who can be helped by immediate transportation

• The injured whose transport can be delayed

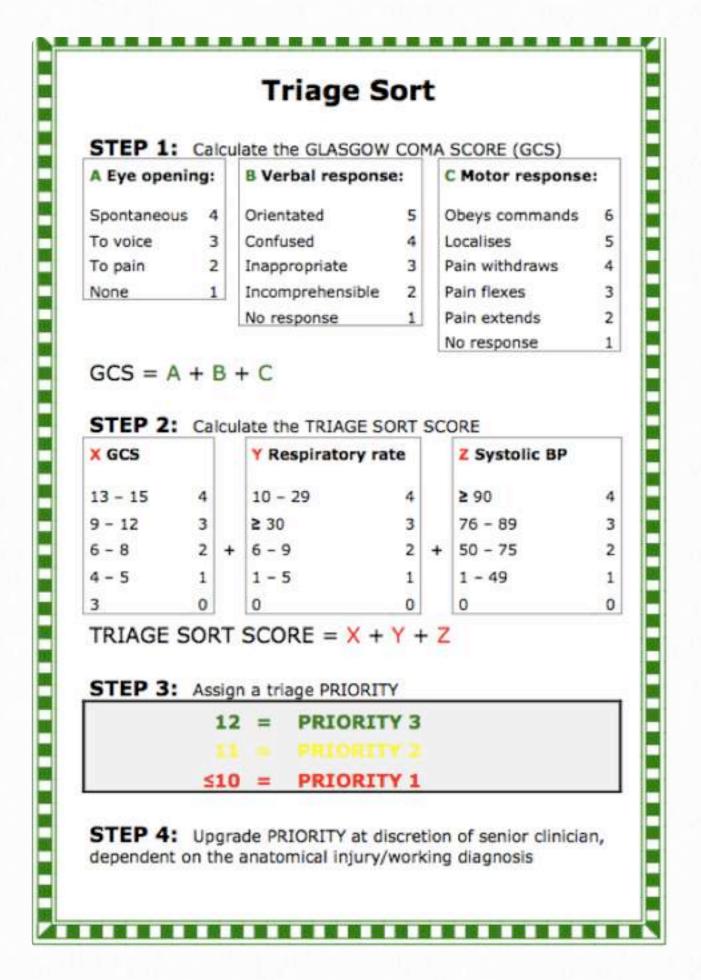• Those with minor injuries, who need help less urgently

A number of triage systems are now also including massive haemorrhage as the first assessment in the Triage Sieve.

If a casualty has massive haemorrhage, a tourniquet is applied where applicable and then the assessor simply moves on to the next casualty. There is no place for direct pressure, unless the casualty can apply this to themselves – 'self-aid'

The above is the triage sieve that ATACC recommends, however some organisations use a sieve that does not include massive haemorrhage.

If a casualty has massive haemorrhage, a tourniquet is applied where applicable and then the assessor simply moves on to the next casualty. There is no place for direct pressure, unless the casualty can apply this to themselves – 'self-aid'
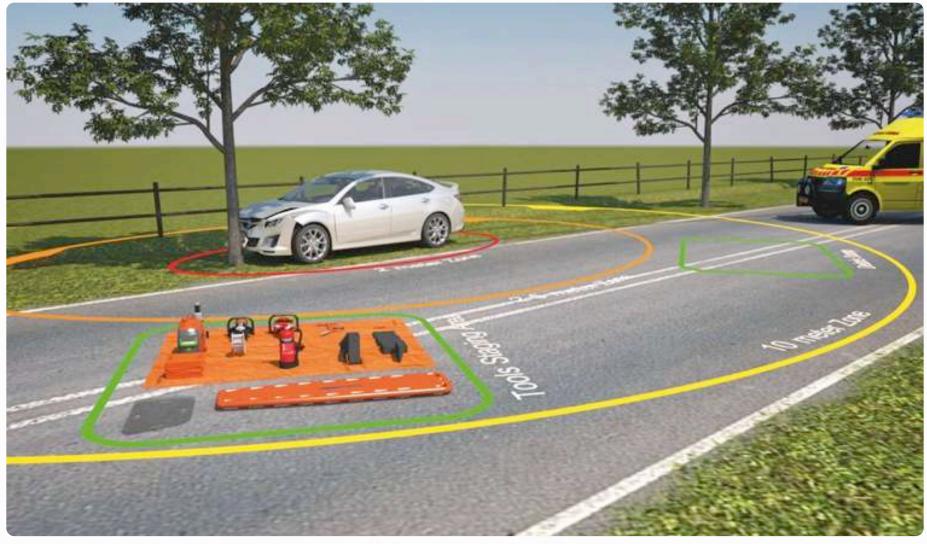
Triage Sort

STEP 1: Calculate the GLASGOW COMA SCORE (GCS)

A Eye opening:		B Verbal response:		C Motor response:	
Spontaneous	4	Orientated	5	Obeys commands	6
To voice	3	Confused	4	Localises	5
To pain	2	Inappropriate	3	Pain withdraws	4
None	1	Incomprehensible	2	Pain flexes	3
		No response	1	Pain extends	2
				No response	1

GCS = A + B + C

STEP 2: Calculate the TRIAGE SORT SCORE

X GCS		Y Respiratory rate		Z Systolic BP	
13 – 15	4	10 – 29	4	≥ 90	4
9 – 12	3	≥ 30	3	76 – 89	3
6 – 8	2	6 – 9	2	50 – 75	2
4 – 5	1	1 – 5	1	1 – 49	1
3	0	0	0	0	0

TRIAGE SORT SCORE = X + Y + Z

STEP 3: Assign a triage PRIORITY

12	=	PRIORITY 3
11	=	PRIORITY 2
≤10	=	PRIORITY 1

STEP 4: Upgrade PRIORITY at discretion of senior clinician, dependent on the anatomical injury/working diagnosis

Triage Sort

• Triage Sort sets priorities for evacuation and transport as follows:

• Deceased are left where they fell. These people are not breathing and an effort to reposition their airway has been unsuccessful.

- Immediate or Priority 1 (red) are evacuated as soon as resources are available. These people are in critical condition and would die without immediate assistance.

- Delayed or Priority 2 (yellow) can have their medical evacuation delayed until all immediate persons have been transported. These people are in stable condition but require medical assistance.

- Minor or Priority 3 (green) are not evacuated until all immediate and delayed persons have been evacuated. These will not need advanced medical care for at least several hours.

Road Traffic Collision Extrication

Safety is the priority on scene

Although modern cars are considerably stronger than they were a decade ago, a road traffic collision where a casualty is trapped is still a very common occurrence and will form the major proportion of work for anyone who deals with trauma in the pre hospital environment. Vehicles are now designed to absorb energy and redirect forces around the passenger call. There are usually a minimum of six airbags in new vehicles and the use of boronated steel makes the structure much stiffer and stronger yet much lighter. Despite these advances, high speed collisions still lead to occupants being trapped and suffering major polytrauma.

The extrication process is a complex mixture of medical and technical expertise and requires a high degree of on scene communication and dynamic planning. It is important to understand that a casualty can be trapped in any of the the following manners:

Casualty is Physically Trapped

• The casualty cannot be physically removed due to the structure of the vehicle preventing extrication e.g. high speed frontal impact resulting in the driver trapped by the dashboard and steering wheel

Casualty is Medically Trapped

• The casualty although not physically trapped by the structure of the vehicle, cannot extricate themselves due to their medical condition, e.g. relatively low speed frontal impact with the driver complaining of severe pain in the neck/back.

In the most severe collisions of course; the entrapment will be both physical and medical meaning that space will have to be created by the technical rescue team (usually the fire and rescue service).

Fire Service Role

The technical rescuers will work to what is known as 'The Team Approach'. This is a six phase process:

1) Safety and scene assessment

As soon as the technical rescue team arrives on scene they will perform a 360 degree survey and ensure that a safe working area is established. They will identify the following:

• Spills/leaks

• Broken glass

• Undeployed airbags

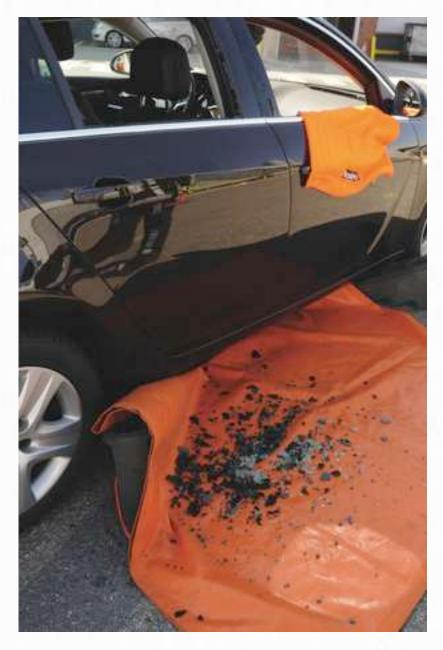• Vehicle fuel type (e.g. petrol, electric/hybrid)

Inner and outer cordons will be established. The inner cordon will be the area immediately around the vehicle (within 5m). To enter this area, full PPE must be worn.

2) Stability and initial access

The vehicle will be stabilised in order to prevent unnecessary movement and assist medical interventions. Then Access can be gained to the inside of the vehicle for the rescuers.

3) Glass management

Glass will be systematically removed to reduce the hazard

The glass will be systematically removed and controlled to reduce the hazard on scene as well as allowing access into the vehicle for personnel and equipment

4) Space Creation

Hydraulic tools are used to create space

Where possible the technical rescue team will first try and create an Emergency Exit route in the event that a casualty rapidly deteriorates and literally needs to be pulled from the vehicle, e.g. Cardiac or respiratory arrest. This is called the "B-Plan"

Hydraulic rescue equipment will be used to gain access, move vehicle construction and often remove the roof for full access.

5) Full Access

An example of full access

Full access such as a roof removal gives rescuers more access, making rescue safer, easier and quicker. Maximum space is also more sympathetic to the casualty. The "A-Plan" is the exit route that keeps the casualty 'in-line' therefore minimising rotation and any avoidable spinal compromise.

6) Immobilisation and Extrication

Throughout the earlier phases the casualty should be 'packaged' and ready for extrication as soon as full access is completed. This can involve KED devices and pelvic splints. The casualty will normally be extricated by use of a long (spinal) board.

Medics Role

In most collision situations the technical rescue team (fire and rescue service) will be responsible for formulating the Extrication Plan. This can only be contrived by gathering critical information from the casualty via the medic. In addition to the medical information, the technical rescuers will assess the vehicle and decide (with medical advice) on the best extrication route. This will, where possible, be the most sympathetic way of extricating the casualty from the vehicle in the least amount of time.

Once you have completed your initial primary/secondary survey, pass this information onto the fire service incident commander (IC). This allows the plan to be contrived.

It is possible that a medic arrives on scene first, in which case SCENE SAFETY training is important to ensure the ability to work safely and efficiently. However it may be that medical personnel arrive on scene after the technical rescue has begun. In this instance it is important to quickly receive information from the fire service incident commander (IC). The IC can mostly (but not exclusively) be identified by the presence of a white helmet.

If in doubt ASK!

The medic must slip seamlessly into the process and not delay the extrication.

Upon arrival, consider the following basic principles:

- Establish who is in charge (fire service IC)

- Establish current level of medical resource on scene and obtain handover (if applicable)

- Consider if more resources are required

- Initial patient survey – communicate your findings to IC

- What is the plan? Is this plan conducive to your findings?

Communication

The importance of on scene communication cannot be over emphasised, this is principally due to safety, but moreover it ensures a timely, effective and casualty centred rescue. When you arrive must obtain a handover from the current medical person on scene (where applicable), this may be:

- A passer-by with no medical training

- A police officer with varying level of medical training

- A fire officer with varying level of medical training

- An ambulance technician

- A paramedic

A doctor who is not trained in pre hospital care

Are you Prepared to work in here?

There will be varying levels of medical information handed over to you, so be clear what information you need.

It is essential that the on scene medic gives regular updates to the IC as any change in the casualty's condition will require a change in the technical aspect of the plan. This must be reciprocal communication and the IC of the technical rescue team must give regular updates on their progression. In essence what needs to be clearly conveyed is:

- Immediate Extrication = Immediate

- Rapid Extrication   = < 5min

- Urgent Extrication   = < 20min

If in doubt ASK!

It is important that you have a clear idea of how long it will take to reach the FULL ACCESS stage of the team approach, so you can make the right clinical decision

In addition, the medic must also communicate with the technical rescue team who are working in or around the vehicle. If their actions in any way compromise the casualty, communicate this and they will find an alternative. As the team approach demands the completion of tasks that can traumatic for the casualty (breaking of glass, spreading/cutting of metal etc) the use of verbal warnings is common. This allows the medic to prepare the casualty and make the process less traumatic.

Examples of verbal warning are:

- Breaking glass! Breaking glass now!

- Cutting! Cutting Now!

- Impact, Impact now!

- Spreading! Spreading Now!

Methods of Extrication

The purpose of this text is to outline the variety of methods available and how the casualties state influences the choice of technique (see table, overleaf).

Route 1

Considered as the extrication gold standard. This is the commonest, and usually the safest approach to front seat occupants, for a vehicle on all four wheels. This serves to demonstrate the basic principles of the long board as an extrication tool. The technique is easily modified and adapted for other types of entrapment.

- Casualty in seat (front, although can be adapted for rear seat passengers).

- Head manually immobilised.

- Hard cervical collar is applied.

- Roof-flapped or removed.

- Shoulders supported from each side, gently moved forward or better supported whilst seat reclined just enough to introduce long board.

- Clean Teardrop slid behind casualty by fourth person, to facilitate entry of board long-board slid in between patient-back and seat-back.

- Seat-back lowered further (if possible).

WALK OUT (Assisted Extrication)

If it is identified that the occupant has no injuries and they are not physically trapped it may be possible to consider walking them out. This can only ever be considered on the advice of a qualified medical professional.

DANGER ZONE

If the casualty is physically trapped by the deformed structure of the vehicle, e.g. dashboard and steering wheel, they cannot be removed in an emergency (e.g. cardiac arrest or vehicle fire). Removing this physical entrapment MUST be the priority.

IMMEDIATE

In the event of the casualty suffering an immediate event, e.g. cardiac arrest/respiratory arrest, they can be immediately removed from the vehicle so medical intervention can continue. The priority here is the dire need for medical assistance that cannot be performed whilst they are inside the vehicle, e.g. chest compression/intubation.

RAPID
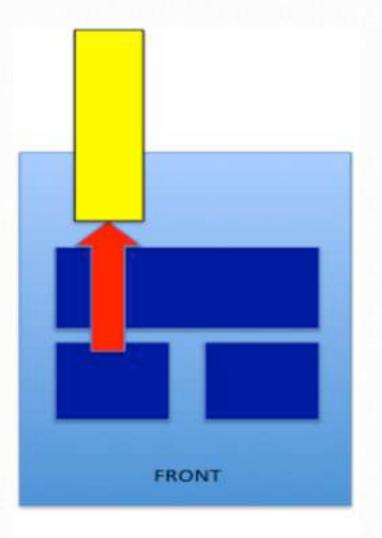
If during medical assessment it is noticed that the casualty's medical condition is worsening, there may be a need for a rapid extrication, e.g. within 5 minutes. This will allow medical intervention to continue and should be completed as soon as possible This extrication must prioritize the clinical needs of the casualty over the extrication procedures adopted for a Gold Standard removal.

URGENT

If the casualty remains relatively medically stable we can extricate using our preferred 'Gold Standard' method. Where possible, the casualty should be kept inline (straight) therefore minimizing rotation of the spine/pelvis. Ideally the extrication should be completed in 20 minutes or less.
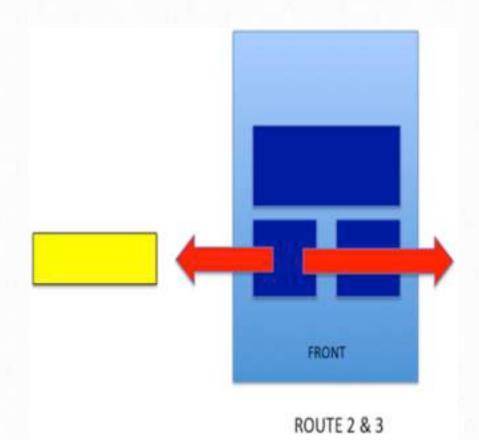
- If seat-back will not recline, then it can be cut out (caution with air-bags).

- Minimum of five casualty handlers, including a leader who takes the head.

- Controlled, progressive slide up the board in a series of moves.

- Once fully onto the board, it can be leveled and removed from the vehicle.

- Head blocks and straps are usually left until after casualty has been removed from the car.

- Secrets of this technique are; good access, good communication and plenty of pairs of hands!

This method requires optimal space creation and usually means roof removal and may require good side access and dashboard role. It is best understood when practically demonstrated and can easily be modified for vehicles on their side or roof.
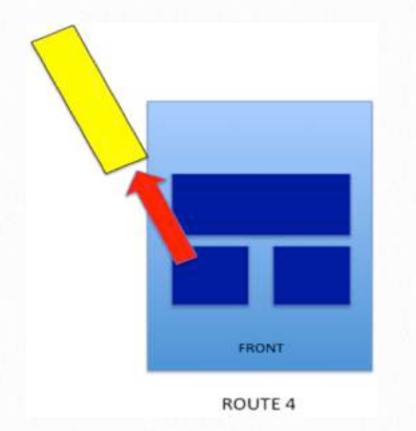

ROUTE 1

Route 2 & 3

Considered a Rapid or Emergency exit route and forms part of the B-Plan out either side door. This usually involves some degree of rotation of the casualty and ideally should be avoided in suspected spinal and pelvic injuries

ROUTE 2 & 3

Route 4

This method is useful when it is difficult or undesirable to remove or flap the roof fully. A large amount of side access is required and this usually entails a 'B-Post Rip'. In this cut the top and bottom of the B-post is cut and the whole side of the car opened out. A long board is then brought in behind the patient in the 10 o'clock position. A small amount of controlled rotation is required to bring the patient in line with the board. They can then be slid obliquely backwards and out of the vehicle, onto the board, with relatively little twisting.

FRONT

ROUTE 4

Rapid Controlled Extrication

Where the casualty has a 'time critical injury' that cannot be managed within the car, then they will need to be extricated rapidly.

If some temporary measures can be put in place, then a rapid controlled extrication can be performed. If it is not possible to get control of the situation or they continue to deteriorate to an immediately life-threatening sate then, a Snatch Rescue or Emergency Extrication will need to be performed.

A typical example of rapid patient extrication is by the use of Route 2 or 3. This technique may not require any cutting, if the door can be opened, and there is no absolute entrapment. The following steps should be followed:

- Manual immobilisation of the neck from behind or one side.

- A second person applies a hard collar.

- A long board is slid under the patients buttocks.

- While control of the c-spine is maintained, one person supports the torso and a third frees the patients legs and feet, but ideally more hands should be utilized.

- Clear commands must be given by a designated leader (usually the person at the head).

- It may be necessary for control of the head and c-spine to be changed be-

tween individuals as the move progresses, as door pillars or headrests frequently get in the way.

- The legs are raised and swung across the transmission tunnel and the patients whole body is rotated in line.

- When full rotation is complete the patient is lowered backwards onto the long board.

- If necessary the casualty can be moved out onto the board in a lateral position and then moved to supine once clear of the vehicle.

- Once the patients torso is on the board, he or she can be slid up the board in a series of small steps either on his side or back. When the patient is correctly positioned on the spinal board, with the person at the head of the board giving the commands, the patient can then be lifted clear of the car.

This method involves compromises. The casualty has to be rotated at some point, which loads the spine. The legs and knees often have to be bent-up or angled to clear the transmission tunnel which may also load the lumbar spine, as well as effecting intra-abdominal organs. The casualty may have to be removed on their side. All of these issues must be considered in a rapid risk vs benefit assessment by the rescue team.

Once clear of the vehicle it is important to act on whatever condition required the rapid extrication, which may simply involve scooping and running for hospital. Each rapid extrication will pose its own problems as vehicles and their occupants vary in size and shape but the above outline is a basis on which to plan an extrication.

Emergency Extrication

If the patient is so unstable that immediate intervention is required and it can only be provided outside of the vehicle, then a snatch rescue will be required. It may also be required if the patients physical safety is threatened by, for example, imminent fire. The fire brigade will achieve the "B-Plan", gaining some access to the patient and remove any absolute entrapment. The patient will then be pulled from the vehicle. As many people as possible should help, and it may still be possible to avoid gross movement of the cervical spine. But it must be remembered that at this point, the focus is on saving life.

Alternative Extrication Devices

Although the spinal long board is the extrication device of choice for an entrapped patient, especially if they require spinal sta-

bilisation, it may be impossible to use it in the confined space of a crashed car, especially if cutting tools are not available, or will take too long. This maybe a time to consider using a vest-type extrication device an example of which is the Kendrick Extrication Device (KED) or newer devices such as the Vac-KED.

The Kendric Extrication Device (KED)

In-line immobilisation is established and a rigid collar is applied by an assistant. The patient is then positioned in an upright position with a small space between the patient and the back of the seat. The device is the slipped down between the patient and the vehicle seat. When the vest is correctly in position with the top level with the top of the patients head, the torso straps are secured by the buckles and then tightened. Over tightening will restrict ventilation. The groin straps are then passed under each thigh. Finally, head pads are placed and the forehead strap and chin strap secured. The chin strap is positioned over the rigid collar. Once the neck strap is positioned, immobilisation is completed and the head, neck and torso should be secured. Manual immobilisation of the neck can be released and simply holding the top of the device will support the patient. The patient can then safely be moved to the spinal board by a multi-person lift. Avoid utilising the "lifting straps" on the KED, focusing on lifting the person instead.

If a solo responder, the use of certain adjuncts such as the "Speed Board" may be useful. These are not full KEDs, rather a ½ longboard with the ability to secure the

head to the board. Whilst frequently misunderstood as a "rapid extrication device" in practice it is a useful tool to secure an unconscious or semi-conscious patient in a neutral position until further help arrives, or indeed to simply free up a member of the team from holding the c-spine. During Emergency Extrication, the device offers a limited degree of spinal protection. The casualty must be lifted bodily and not by the board, as it will become displaced. It does however provide a much better degree of protection to the patient during rapid moves. During more controlled extrication, it can aid in sliding the patient up a longboard, and can easily be removed afterwards.

On Scene Practical Considerations

Remember you are attending the scene of a road traffic collision. The factors that caused the incident may also impact on the safety of your approach (e.g. weather, road conditions) so ensure a safe, slow controlled approach when you near the scene.

The position which you decide to park is crucial. You must be close enough to have ready access to any equipment you may need but DO NOT block the access for

other emergency vehicles. Once vehicles are parked it simply is not practical (in many circumstances) to get them moved.

Do not attempt to enter the inner cordon without full PPE. In UK the inner cordon is controlled by the fire and rescue service and they will not give you access if you are not correctly dressed. Working on a patient inside a vehicle is not easy. It is essentially a confined space and you are in a noisy and stressful environment. It is essential that you and your equipment are well prepared.

Are you Prepared to work in here?

Training and Preplanning

It is important that anyone attending the scene of a road traffic collision is well trained and prepared. As extrication is a multi-agency process there should be a degree of preplanning with other services to establish working practices and on scene

priorities. Fire and rescue services very often train for such incidents and it is recommended that medics approach their local services to facilitate a multi-agency approach to training. This kind of partnership will promote a more efficient on scene working relationship by giving a greater understanding of each other's requirements and limitations.

Summary

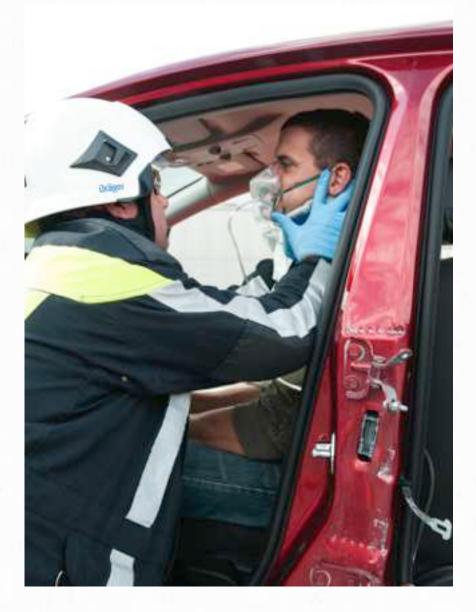Safety MUST be the priority in all pre hospital environments

If you arrive on scene first, your priorities are:

- Safety – If in doubt, wait for assistance

- Think kinematics!

- Identify/triage/complete primary & secondary survey of casualty(s) if safe to do so

- Brief oncoming rescue personnel (fire and rescue/Paramedic)

- Liaise with IC regarding extrication plan

- Communication – update technical rescue team

If you are not first to arrive on scene, your priorities are:

- Safety – If in doubt, wait for assistance

- Think Kinematics

- Identify IC from fire and rescue service (if incident is an entrapment)

- Liaise with on scene medic (receive handover)

- Assess the level of response required on scene, are you actually required?

- Liaise with IC regarding extrication plan

- Communication – update technical rescue team

Obtain a handover from the medic who is already on scene

- What further medical resources do you require? e.g. air ambulance

- Package patient and supervise extrication phase of the rescue

REMEMBER:
Safety
Establish Communication
Maintain Communication
If in Doubt, ASK!

Trauma Retrieval

Primary retrievals involve treatment of the patient at the scene of an incident, and transport to an appropriate receiving facility. Some services may undertake 'modified primaries' where the patient is retrieved from a (usually rural) healthcare facility where minimal or no intervention has taken place prior to the arrival of the retrieval team.

Secondary (Interhospital) transfers are between hospitals. Intrahospital transfers take place within an institution (e.g. from ICU to CT).

Retrieval medicine is well established in certain parts of the world, particularly in Australia. Dedicated retrieval teams, immediate care schemes and air ambulance services are now providing critical care transfers on a more regular basis in the UK. The development of Trauma Networks has led to an increased need for timely transfers of trauma patients

Properly trained and equipped teams can safely perform critical care transfers, and specialised transfer teams improve the quality of care provided during transfer. Despite this, adverse events can occur during transport. Risks to patient safety during critical care transfers can be grouped into four categories - equipment, team, organisation and patient factors.

Care of the critically ill patient during transport can be challenging, and an appreciation of the environment, equipment and process is vital to safely perform critical care retrievals.

Primary and secondary transfers pose different challenges, but many of the organisational elements are similar.

Packaging Considerations

Patient packaging is a critical part of the transport process, and a balance must be struck between careful packaging and a reasonable time spent doing the packaging. Retrievalists have different preferences about how packaging is done, but no evidence exists to recommend one method of folding blankets over another. So long as the patient is warm, secure, has pressure areas protected and the team has access to IV injection ports then the packaging is adequate, however neat packaging por-

trays a well planned, controlled and professional transport team.

Patient position should be optimised e.g. head up in traumatic brain injury if spinal injury has been excluded. If spinal immobilisation is required, a vacuum mattress should be used – hard devices such as spinal boards and scoop stretchers are not recommended.

Packaging and preparation for transfer can be broken down into steps, and the order in which these steps are completed may vary from case to case.

Constantly moving patients from ambulance trolleys to Emergency Department Trolleys, to the CT table, to theatre trolleys, to theatre tables, etc. adds multiple possibilities for adverse events, inadvertent monitoring or therapy disconnection.

The ideal situation sees the patient comfortably packaged in such a manner as to protect them from further harm and onto a mattress that they will not need to be moved off as their care progresses through the pre-hospital and in-hospital phases until definitive diagnostic procedures (i.e. CT scanning) is completed and the patient returned either to the Emergency Department, ICU or the Operating Theatre table.

Pre-transfer decision making

For a discussion on the merits of clearing the cervical spine (if possible) and current thinking on the use of cervical collars in transfer of trauma patients, please see the cervical collar section in the Spinal Care chapter. ATACC currently recommends that c-collars should be correctly sized and fitted if they are to be used in patients with a high suspicion or confirmation of cervical spine injury. However, in patients with a suspected head injury who is receiving a neuroprotective anaesthetic, the collar should be loosened to allow adequate

cerebral venous drainage and optimise ICP.

Vacuum mattress

The vacuum mattress results in lower pressures to potential pressure areas, and has been shown to provide both reduced movement and increased patient comfort when compared to immobilisation on a spinal board or scoop using a standard strapping technique., While a recent randomised controlled trial found the spinal board to be superior to the vacuum mattress, the methodology of the trial has been questioned, and the preferred ATACC method for spinal immobilisation is to use a vacuum mattress. We consider the spinal board to be an extrication device and the scoop a transfer device (from one location or device to another), not a transport device, however clinical judgment may be applied in the case of haemodynamically unstable patients with very short transport times.

In UK trauma systems the scoop stretcher is currently being promoted and used as a transport device. This is useful for gathering a patient off the floor and effective in transferring patients with minimal movement. It has been shown to be more comfortable than a spinal board but it is still a metal or plastic board and the risk of pressure areas remains.

There are claims that as log rolling can be avoided with careful application of a scoop, then internal bleeding can be reduced, especially from an unstable pelvic fracture. ATACC remains unconvinced about this, as there is no evidence to support this view and any suspected unstable pelvis should have a pelvic binder applied BEFORE the casualty is placed in the scoop. In addition, poorly trained providers will often end up log rolling twice, to the left then the right when inserting the scoop blades.

We must also consider that a log roll is likely to be an essential part of many casualty assessments.
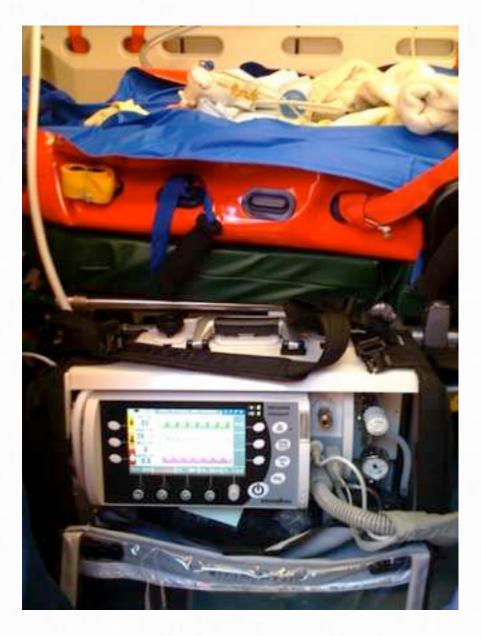
Care must also be taken to minimise hypothermia if transporting patients on a cold metal device and the plastic alternatives are preferred, with all clothes cut away and a 'scoop to skin' approach. However, removing all of the clothes may not be necessary or practical in some situations and then the scoop can be difficult to apply.

For rapid emergency transfers of <10 minutes, then we would simply transport on whatever device the patient is on. To start scooping off a board for such a short distance is not necessary and confers no clinical advantage that we can identify and simply introduces a delay.

For prolonged transport times (>10 minutes) then we would recommend transfer to a vacuum mattress.

When the vacuum mattress is not available, ATACC considers transporting the patient on the ambulance trolley to be an acceptable option, in line with Australian practice.

Whichever device is used, care must be taken to compensate for the anatomical differences seen in young children, those with underlying spinal deformity such as scoliosis or abnormal body habitus and patients wearing personal protective equipment such as equestrian body protectors or motorcycle leathers.

Monitoring

Minimum monitoring standards include ECG, NIBP and SpO2. Ventilated patients should have continuous capnography and invasive blood pressure (IBP) should be monitored in haemodynamically unstable patients and those requiring cardiovascular support. The IBP transducer can be taped to the patient's arm at the correct level for the duration of transport. Appropriate alarm limits should be set prior to departure.

Patients with IBP monitoring should have a NIBP cuff in place to mitigate against malfunction of IBP equipment, and backup (e.g. handheld) SpO2 and EtCO2 monitors should be available in the event of monitor failure.

If the need for defibrillation/cardioversion/transcutaneous pacing is predicted the pads should be placed prior to final packaging to obviate the need to do this during transport. Electrical therapy (and NIBP measurement) place extra demand on battery life, and if the facility to plug in the monitor is not available extra batteries should be carried.

Patient access

A well-prepared and packaged patient should not need to be 'unwrapped' during transport. A venous access port should be easily accessible by the retrieval team, without having to undo their seatbelts. This should be clearly marked and identified by both practitioners pre-departure. Holes can be cut in the sheet/blanket to facilitate this. The arterial line transducer should also be accessible to flush the line if necessary.

Restraint

The patient should be secured to the stretcher using the stretcher straps per the manufacturer's instructions. All equipment must be secured using restraining brackets or an equipment bridge. Transporting patients with unsecured equipment, or equipment secured with tape is an unsafe practice.

Secondary (Interhospital) Retrieval

The standard of care the patient receives during transport should be the same or greater than that provided to the patient in the referring facility. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) and the Intensive Care Society (ICS) have published guidance documents for interhospital critical care transfers, including minimum monitoring standards, and teams providing critical care transfers should be familiar with these recommendations.

The team composition varies from service to service, and may be doctor-nurse/ doctor-paramedic/ doctor-ODP/ paramedic-nurse/ paramedic-paramedic depending on the area and the planned mission.

Pre mission

Training should be provided to staff who will undertake critical care transfers to ensure that they are prepared to work safely and effectively in the retrieval environment.

Familiarity with the kit and how it is laid out is a vital part of the retrieval process. The team must be trained in the operation of all of the retrieval equipment, and must be capable of troubleshooting in the event of malfunction during the transfer. A daily checklist driven system for maintenance of drugs and equipment ensures both that the kit is in order and that staff are familiar with it.

The retrieval and particularly the prehospital environment exposes the patient and the crew to hazards, and suitable personal protective equipment must be available.

Time of tasking – interhospital

An ideal transfer system will facilitate a conference call involving the referring, receiving and transfer teams at the time of tasking. This updates all parties on the current condition of the patient and allows for a jointly agreed treatment plan to be made, ensuring optimal patient management and continuity of care. Any concerns regarding the time critical nature and patient suitabil-

ity for transfer or timing of the transfer can be discussed.

This conference call is useful to help the transfer team mentally prepare and plan for the mission, as well as to plan practical issues such as drug and oxygen requirements. As a general rule the calculated requirements should be doubled to allow for delays or unforeseen circumstances. Local staff can be requested to draw up the required drugs and infusions while the retrieval team is en route, minimising delays on scene.

The transfer team should plan to rationalise treatment during transfer. Two infusions (sedation ± circulatory support) are usually sufficient, and rarely are more than three infusions required during transport. This minimises the number of pumps/syringe drivers that need to be carried and secured, and minimises the risk of equipment failure.

The key to safe transfer is preparation and planning of each phase

While en route to the referring facility the retrieval team should discuss a detailed plan, including actions in the event of an adverse event. The roles and tasks that each member will undertake on scene should be agreed to optimise efficiency.

Arrival on Scene

The tasking authority should be notified of the arrival of the retrieval team at the patient. Ensure that all the required equipment is brought to the patient. The first few minutes at the patient's bedside are crucial, both to form an initial impression of the patient's condition and to initiate a relationship with the referring staff. If there are no immediately life-threatening issues, take the time to actively listen to a detailed handover while assimilating other information from monitors etc.

If retrieving a patient from a rural centre remember that the staff may not have access to the same level of investigation, intervention or equipment as in a tertiary centre, and may not have experience with high volumes of critically ill patients. It is important to maintain a professional attitude and to be respectful of the efforts of the local team prior to your arrival. This can be a useful educational experience for them, and you may have the ability to influence

the management of future patients with judicious advice and feedback.

On Scene

Crew resource management is an important skill on scene. Many steps need to be completed in the shortest time possible so it is vital to provide leadership to the local staff and enroll their help to complete multiple tasks simultaneously.

"Share the mental model"
-Cliff Reid, Sydney HEMS

Decide what needs to be done for this patient at this particular point in time, within overall context of the management plan. (e.g. a time critical isolated traumatic brain injury may require an arterial line, but perhaps not a central venous cannula prior to transport for neurosurgical intervention)

Decisions about the level of intervention required for a given patient may be different in the transport environment than in ED/ICU; for example the threshold for intubation is lower to avoid the need to intubate in sub optimal conditions during transport, particularly if aeromedical transport is required.

Interventions such as enteral feeding should be stopped during transport, and

antibiotics etc should be given either before transfer or after arrival the receiving facility if possible. Catheter bags, NG drainage bags etc should be emptied and volumes recorded. Chest drain underwater seals should be changed to a closed system with a flutter valve.

Pre departure

Communicate with control centre to update your ETA at the receiving facility, and ensure that the destination (e.g. ED/ICU/CT etc.) and receiving clinician is known and documented.

Before leaving the relative safety of the referring site, a final head to toe check of the patient and equipment should be made to ensure:

- A: Secure, backup airway available

- c: Appropriate spinal injury management if required

- B: Effective ventilation, optimised ventilator settings

    - Adequate oxygen supply until arrival at the ambulance/aircraft

    - EtCO2 within accepted limits

- C: Heart rate and blood pressure within accepted limits

    - IBP functioning correctly

- D: Patient adequately sedated and paralysed

    - Drug infusions running, all clamps open

    - Documentation and imaging

- E: Extra drugs available as required

- F: Adequate fluids/blood products for the duration of the transfer

Drugs

Sufficient drug and fluid volumes must be available for the transfer, as well as adequate calculated reserves. In general, ventilated patients should remain paralysed for the duration of transport.

'Emergency drugs' should also be predrawn and easily accessible.

Ventilation

The patient should be set up on the transport ventilator for a period prior to leaving the referring unit to ensure adequate ventilation pre departure. If an arterial line is in place, correlate PCO2 with EtCO2 to allow for accurate ventilation targets. If the patient is PEEP dependent remember to clamp the ETT prior to switching ventilators.

A Bag-Valve-Mask must always be immediately available in the event of failure of the ventilator

Emergency Equipment

Immediate access must be available to emergency airway equipment in the event of accidental extubation. Oxygenation and ventilation must continue with a supraglottic airway or BVM until the patient can be reintubated. Pneumothorax is a risk in any ventilated patient, and a scalpel and forceps should be available to perform a thoracostomy if necessary.

People

The patient's family should be considered, and should be allowed to see the patient before departure. A parent may be permitted to travel with a paediatric patient, but they must be adequately briefed, and may require anti emetic prophylaxis (as may the medical team). The family should transport luggage or belongings separately wherever possible.
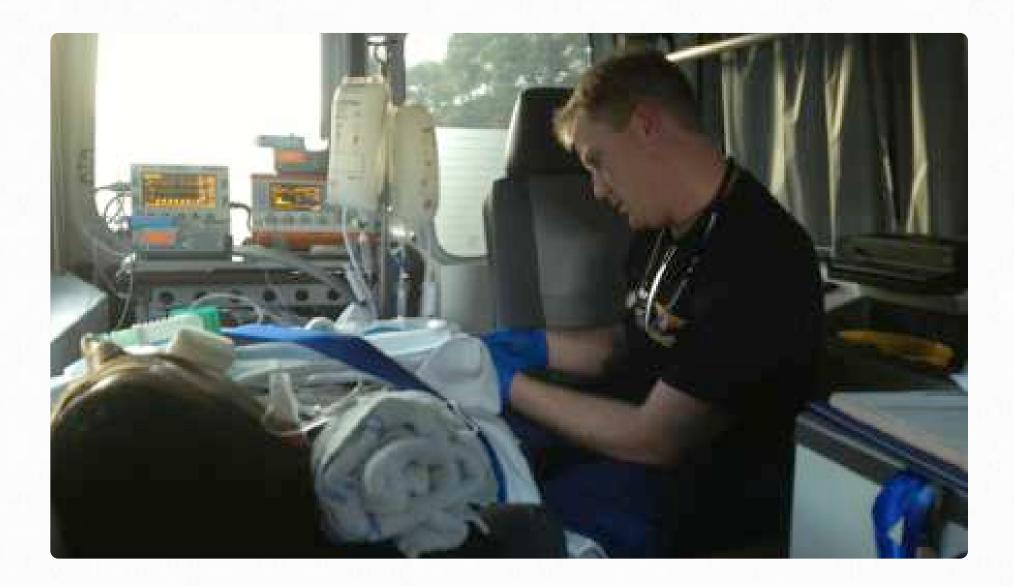
The transfer team should take the opportunity to use the bathroom before departure

En route

Even when transferring an unstable patient, a properly planned transfer of a well packaged patient should mean that the re-

trieval team does not need to get out of their seatbelts en route - ideally the ventilator, monitor, infusion pumps and IV access port should be within reach. Vital sign trends should be closely monitored, and physiological parameters should be manipulated to remain within target ranges. The ventilator should be connected to the vehicle oxygen supply, and electrical equipment should be plugged in where possible.

Further boluses of neuromuscular blocking drugs should be given at appropriate time intervals. This is particularly important in traumatic brain injury to avoid transient rise in intracranial pressure from coughing etc. Infusions should be actively managed to avoid the need to change syringes at high risk times such as loading or unloading the vehicle, and to avoid running out prior to changing over to the receiving facility's infusions.

While some transfers are time critical, safety is paramount. Rapid acceleration and deceleration may affect physiological parameters. Any potential time saving attributable to aggressive high speed driving can more easily achieved by being organised and efficient during the packaging process. Police escorts have risks associated with them, and may not confer a sig-

nificant time saving. The risks and benefits should be carefully considered on a case-by-case basis.

Handover

On arrival the receiving hospital inform the control centre of your arrival. The portable oxygen cylinder pressure should be checked to ensure there is sufficient supply to get to the final destination. The patient should be carefully unloaded, with a 360-degree check that no lines or tubes have become entangled in any parts of the vehicle during transport.

On arrival in the receiving unit identify the person responsible for receiving the patient and give a structured handover, taking care to communicate any new or time critical issues that have arisen since the previous conference call. Complete all documentation and leave a copy with the patient's records.

Post transfer

All of the reusable kit should be collected, cleaned and checked before departure, ensuring that nothing is left behind. Any drawn up controlled drugs must be disposed of. The case

should be discussed within the retrieval team as part of a hot debrief, and any immediate issues should be dealt with. Any identified areas for improvement should be discussed at a service level governance meeting.

Primary retrievals

While the same principles apply to primary trauma retrievals as to interhospital transfers, some other considerations apply. Emphasis must remain on quality critical care despite the sub-optimal conditions often encountered on scene. Communication with the tasking agency when arriving on and departing from scene is vital, as is communicating with the receiving Emergency Department in order that the trauma team/theatre/radiology/massive transfusion protocol can be activated

Time of tasking/en route

Planning for primary missions will take place en route to the scene. Often information will be limited or incorrect, so possibilities should be discussed and roles designated but it is important to retain the ability to react to a dynamic situation. Accurate and up to date information from the scene is vital to help the team decide how best to manage logistics – for example it may be advantageous to meet the ambulance at a rendezvous point rather than at the scene in order to minimise the total prehospital time.

On scene

Safety is paramount, and the retrieval team should not enter the scene until it has been declared safe. Good crew resource management is vital and clear introductions are vital to establish an effective working relationship with the other providers on scene. Priorities should be identified with the scene commander, ensuring that accurate triage is completed, and that sufficient resources have been allocated to the scene.

The retrieval team may provide interventions, decision making and transport decisions that differ from the management provided by the Ambulance Service or Fire & Rescue providers. The rationale behind

these differences should be verbalised to avoid confusion and conflict.

It is important to utilise resources efficiently to minimise scene time. As many tasks as possible should be allocated to other providers, allowing the retrieval team to focus on the tasks that only they can provide.

Momentum can easily be lost, for example the entire team may stop to watch an RSI, while there are other tasks such as preparation for splinting and packaging that need to be completed before leaving the scene. Clear requests for help with the next stage of the treatment/packaging plan with helps to maintain the mission trajectory.

Packaging is a vital part of prehospital critical care. Avoidance of hypothermia is an important consideration in prehospital care, and prehospital anaesthesia is associated with hypothermia. May different methods are used to maintain body temperature in the prehospital environment including blankets, foil, bubble wrap and active heating devices. The focus should be on maintaining temperature as opposed to rewarming a cold patient.

Laying a patient on sheets/blankets and folding them around the patient facilitates examination of the patient while exposing only small areas at a time. The secondary survey may be more effective if performed in the heat and light of the ambulance as opposed to on the ground.

In common with packaging for interhospital transfers, the patient must be warm, secure, have pressure areas protected and the team must have access to a reliable IV injection port.

Clinical and logistical factors will influence the interventions required and the appropriate destination for each individual patient, but the focus must remain on contributing useful interventions while minimising the time spent in the prehospital phase. Some interventions (e.g. tranexamic acid) and examination (e.g. eFAST) may in some circumstances be more appropriately performed en route than on scene.

Handover to the receiving team should be structured, concise, and ideally should be mentally rehearsed prior to arrival at the Emergency Department. Accurate and comprehensive documentation must be completed and a copy include in the patient's notes at the receiving facility.

Post mission

The team should ensure that all equipment is cleaned or replaced before informing the tasking agency of its availability for the next mission.

A debrief should be completed with the team involved, and each case should be reviewed by the system's clinical governance procedures. If possible feedback should be given to the ambulance crews who assisted on scene.

Complex transfers

Complex transfers of patients requiring advanced cardiovascular support in the form of Intra Aortic Balloon Pump (IABP) or Extra Corporeal Membrane Oxygenation (ECMO) can be safely achieved. The normal retrieval crew composition may need to be augmented by specialists (e.g. perfusionist; surgeon to gain vascular access for ECMO), and the retrieval service should plan and train for these before performing such complex transfers

References

1. Major trauma care in England. National Audit Office 2010.

2. Lahner D, Nikolic A, Marhofer P, Koinig H, Germann P, Weinstabl C, Krenn CG. Incidence of complications in intrahospital transport of critically ill patients - experience in an Austrian university hospital. Wien Klin Wochenschr 2007, 119:412-416.

3. Wiegersma JS1, Droogh JM, Zijlstra JG, Fokkema J, Ligtenberg JJ.

4. Quality of interhospital transport of the critically ill: impact of a Mobile Intensive Care Unit with a specialized retrieval team. Crit Care. 2011;15(1):R75.

5. Bellingan G1, Olivier T, Batson S, Webb A. Comparison of a specialist retrieval team with current United Kingdom practice for the transport of critically ill patients. Intensive Care Med. 2000 Jun;26(6):740-4.

6. Beckmann U , Gillies DM , Berenholtz SM , Wu AW , Pronovost P . Incidents relating to the intrahospital transfer of critically ill patients. An analysis of the reports submitted to the Australian Incident Monitoring Study in Intensive Care. Intensive Care Med. 2004 Aug;30(8):1579-85.

7. Fanara B, Manzon C, Barbot O, Desmettre T, Capellier G. Recommendations for the intrahospital Transport of critically ill patients.Crit Care. 2010;14(3):R87.

8. Stiell IG, Wells GA, Vandemheen KL, Clement CM, Lesiuk H, De Maio VJ, Laupacis A, Schull M, McKnight RD, Verbeek R, Brison R, Cass D, Dreyer J, Eisenhauer MA, Greenberg GH, MacPhail I, Morrison L, Reardon M, Worthington J. The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA. 2001 Oct 17;286(15):1841-8.

9. Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. N Engl J Med. 2000 Jul 13;343(2):94-9. Erratum in: N Engl J Med 2001 Feb 8;344(6):464.

10. PHECC CPG spinal decision tree. http://www.phecit.ie/Images/PHECC/Clinical%20Practice%20Guidelines/2012%20Edition%20CPGs/Advanced%20

Paramedic%20CPG%202012%20Version.pdf. Accessed 05/02/14.

11. Stiell IG, Clement CM, McKnight RD, Brison R, Schull MJ, Rowe BH, Worthington JR, Eisenhauer MA, Cass D, Greenberg G, MacPhail I, Dreyer J, Lee JS, Bandiera G, Reardon M, Holroyd B, Lesiuk H, Wells GA.The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med. 2003 Dec 25;349(26):2510-8.

12. Michaleff ZA, Maher CG, Verhagen AP, Rebbeck T, Lin CW. Accuracy of the Canadian C-spine rule and NEXUS to screen for clinically important cervical spine injury in patients following blunt trauma: a systematic review. CMAJ. 2012 Nov 6;184(16):E867-76. doi: 10.1503/cmaj.120675. Epub 2012 Oct 9. Review.

13. Gill DS, Mitra B, Reeves F, Cameron PA, Fitzgerald M, Liew S, Varma D. Can initial clinical assessment exclude thoracolumbar vertebral injury? Emerg Med J. 2013 Aug;30(8):679-82.

14. James CY, Riemann BL, Munkasy BA, Joyner AB. Comparison of Cervical Spine Motion During Application Among 4 Rigid Immobilization Collars. J Athl Train. 2004;39:138–145.

15. Podolsky S, Baraff LJ, Simon RR, Hoffman JR, Larmon B, Ablon W. Efficacy of cervical spine immobilization methods. J Trauma. 1983;23:461–465.

16. Connor D, Greaves I, Porter K, Bloch M; consensus group, Faculty of Pre-Hospital Care. Pre-hospital spinal immobilisation: an initial consensus statement.Emerg Med J. 2013 Dec;30(12):1067-9.

17. Sheerin F, de Frein R. The occipital and sacral pressures experienced by healthy volunteers under spinal immobilization: a trial of three surfaces.J Emerg Nurs. 2007 Oct;33(5):447-50.

18. Chan D, Goldberg RM, Mason J, Chan L. Backboard versus mattress splint immobilization: a comparison of symptoms generated. J Emerg Med. 1996 May-Jun;14(3):293-8.

19. Luscombe MD, Williams JL. Comparison of a long spinal board and vacuum mattress for spinal immobilisation. Emerg Med J. 2003 Sep;20(5):476-8.

20. Mahshidfar B, Mofidi M, Yari AR, Mehrsorosh S. Long backboard versus

vacuum mattress splint to immobilize whole spine in trauma victims in the field: a randomized clinical trial. Prehosp Disaster Med. 2013 Oct;28(5):462-5.

21. McDonald N, Webster M, Orkin A, Vanderburgh D, Johnson DE. The Long Backboard vs the Vacuum Mattress. Prehosp Disaster Med. 2014 Jan 16:1-2

22. Del Rossi G, Rechtine GR, Conrad BP, Horodyski M. Are scoop stretchers suitable for use on spine-injured patients? Am J Emerg Med. 2010 Sep;28(7):751-6.

23. Krell JM, McCoy MS, Sparto PJ, Fisher GL, Stoy WA, Hostler DP. Comparison of the Ferno Scoop Stretcher with the long backboard for spinal immobilization. Prehosp Emerg Care. 2006 Jan-For Mar;10(1):46-51.

24. P J Shirley, S Hearns Retrieval medicine: a review and guide for UK practitioners. Part 1: Clinical guidelines and evidence base. Emerg Med J 2006;23:937– 942.

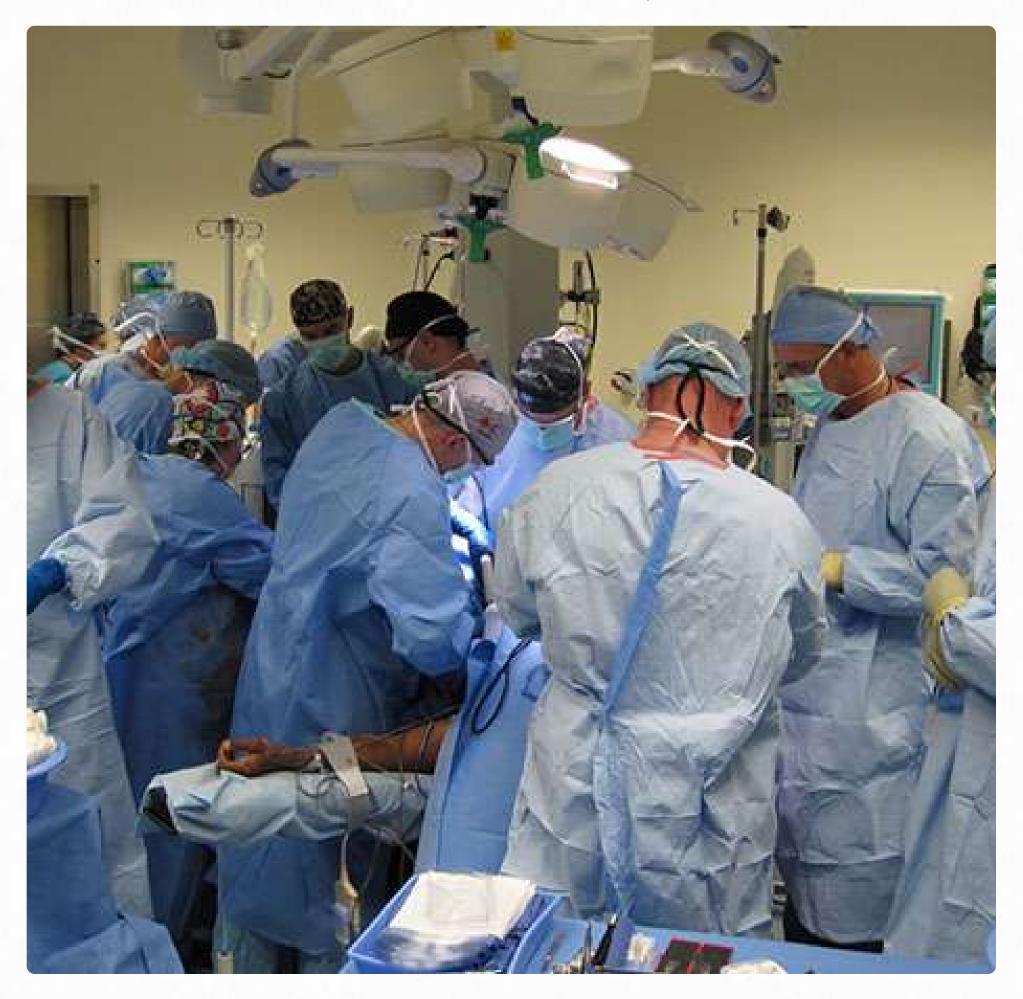25. AAGBI Safety Guideline, Interhospital transfer. Association of Anaesthetists of Great Britain and Ireland, London, 2009

26. Intensive Care Society. Guidelines for transport of critically ill adults. London: Intensive Care Society, 2011

27. Doring BL, Kerr ME, Lovasik DA, Thayer T: Factors that contribute to complications during intrahospital transport of the critically ill. J Neurosci Nurs 1999, 31:80-86.

28. Burns B, Reid C, Habig K. Review of aeromedical intra-aortic balloon pump retrieval in New South Wales.Eur J Emerg Med. 2013 Feb;20(1):23-6

29. Dorlac GR, Fang R, Pruitt VM, Marco PA, Stewart HM, Barnes SL, Dorlac WC. Air transport of patients with severe lung injury: development and utilization of the Acute Lung Rescue Team. J Trauma. 2009 Apr;66(4 Suppl):S164-71
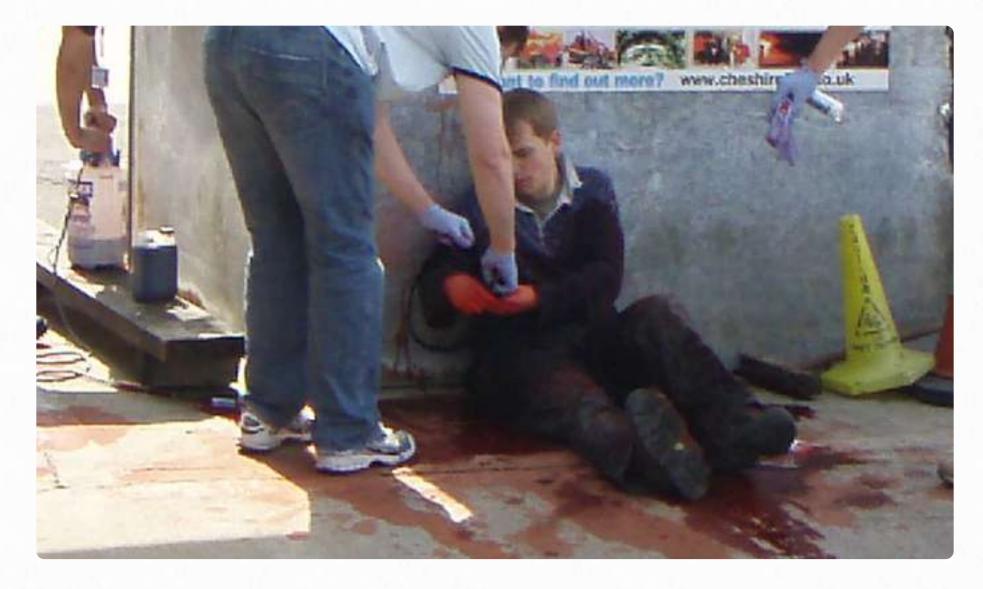
4

Initial Management

Massive Haemorrhage Control

Lessons from History

In deaths from trauma, exsanguination accounts for over 50% of those deaths. This can be as a result of massive external bleeding (which should be obvious) or massive internal bleeding which may not be immediately apparent. In the absence of obvious massive external blood loss, signs of shock should be actively and quickly considered elsewhere:

BLOOD ON THE FLOOR AND FOUR MORE

In other words, in addition to what may be obvious externally, or 'on the floor' it is vitally important to remember that significant amounts of blood can be lost from bleeding in the chest, abdomen, pelvis and long bones (but not into the skull once the sutures have fused in early childhood).

Many of the methods of controlling massive haemorrhage have been informed by developments in the treatment of battlefield injuries with most recent techniques coming from the wars in Iraq and Afghanistan. During the Vietnam war 50% of battlefield deaths occurred as a result of blood loss with extremity wounds accounting for over 50% of potentially preventable deaths in combat. Whilst a frequent occurrence in theatres of war, injuries causing catastrophic life threatening bleeding are comparatively rare in the civilian pre-hospital arena, but that is not to say it does not happen. Internationally events such as the shootings in Mumbai and the Westgate Mall in Nairobi posed many challengers to rescuers faced with casualties suffering from multiple gunshot wounds and massive blood loss, whilst the Boston Marathon bombing had rescuers dealing with multiple limb amputations. Closer to home the Home Office reports over 7000 gun related crimes and over 50,000 victims of knife related injuries every year. High profile cases such as the stabbing of Stephen Lawrence and the Cumbria shootings highlighted the need for improved training and techniques when dealing with these situations.

The Stephen Lawrence case is particularly relevant as the enquiry that followed found failings in that:

- "Officers on scene assumed that Stephen was being cared for by others, and like others, relied on the imminent arrival of the ambulance"

- "They wrongly thought that the best thing to do was to leave him where he was and allow the paramedics to take over, once the ambulance arrived"

- The ambulance took 12 minutes to arrive, by that time Stephen was already dead.

Thankfully those days are long gone and pre-hospital care has learnt much about treating catastrophic haemorrhage from the conflicts in Iraq and Afghanistan.

So, what can we do pre-hospital?

Preserve, Don't Replace

Currently blood isn't carried routinely pre-hospital, with few exceptions such as London HEMS, which carries units of O-Negative blood. At best fluid resuscitation MAY buy a window of opportunity but the delay in transporting the patient whilst these interventions are carried out WILL increase mortality.

Saline DOES NOT carry oxygen.

Cannulation and fluid resuscitation DOES NOT stop bleeding. "PRESERVE, DON'T REPLACE"

Following the ATACC algorithm, massive haemorrhage control is carried out as the first step in the primary survey. Remember to carefully consider kinematics and look closely at the mechanism of injury. Penetrating trauma with signs of massive blood loss needs early surgical intervention. As mentioned above massive external haemorrhage should be obvious but once you have assessed the scene and considered the kinematics of the incident you MUST have a high index of suspicion for potential internal blood loss. The seriously deteriorating patient with signs of shock, and no obvious injuries should alert you to the possibilities of serious internal injuries, which WILL need surgical intervention if the patient is to survive.

Whilst the options for treating massive internal haemorrhage are limited, there is much we can do for catastrophic external bleeding. What is required is 'AGGRESSIVE' control of the obvious massive external bleed.

Management of Massive External Haemorrhage

The casualty, if conscious, may be frightened, uncooperative and possibly aggressive. If they are to survive, your approach must be firm and direct. Approach any catastrophic haemorrhage to limbs using the DDIT method:

- **D- Direct Pressure**
  Expose the source of bleeding and apply firm direct pressure at the site immediately

- **D- Direct Pressure (more!)**
  If bleeding does not stop and soaks through the dressing then apply another dressing with pressure directly on top of that point. Make sure that the pressure is well directed and focussed but avoid repeated removal of the dressing to 'have a look'

- **I-Indirect pressure**
  Applying pressure to the artery proximal to the bleeding point

- **T - Tourniquet**
  Applied above the site of massive haemorrhage. Tighten until bleeding stops. Be aware that if done properly, this WILL cause extreme pain in the conscious casualty but will effectively stop major life threatening bleeds.

Massive external haemorrhage will be obvious

1st Attempt - Direct Pressure

After PPE is donned, the first step of the DiD-IT technique involves the application of direct pressure to the wound. This is achieved by using a sterile dressing, which is either held in place by the responder or through the use of a bandage. However, this is rarely the time for bandaging and we would not recommend the use of elastic combat dressing at this time, unless you have other injuries to manage. These

dressings are very good and highly effective but they take time to apply, especially for the inexperienced and this delay will greatly increase blood loss. We would prefer to simply maintain firm direct pressure and re-consider a combat dressing/bandage in 'C" – circulation, when we re-assess wounds and bleeding.

The dressing should be of a suitable size to cover the wound and must be absorbent. Common field dressings have a bandage with an absorbent dressing fixed to one end. If a sterile dressing is not immediately available, the thumbs or heel of a gloved hand may be used.

If a bleeding point is small and obvious (such as a damaged artery), then even a single thumb or finger can often control the bleeding. Thumb or finger pressure can also be used for bleeds in small children. If this initial pressure effectively controls the bleeding, then no further steps are required.

2nd Attempt - Direct Pressure (again)

If bleeding continues and blood soaks through the dressing, attempt to very quickly re-assess that you are pressing in the correct place and you are applying pressure directly to that point (avoid the desire to repeatedly have a look). Do not simply pile more and more dressing on top of the existing ones, simply use one or two to apply focussed pressure than consider other options such as tourniquet or haemostatic agents. Blood soaking into dressings is blood lost to the system.

Be sure to strive for adequate control of haemorrhaging within one minute. If the first dressing saturates with blood within seconds, have a tourniquet ready to apply before attempting indirect pressure.
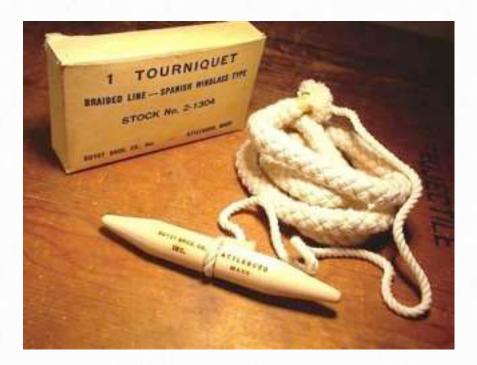
Indirect Pressure

Apply pressure proximal to the wound where the main supplying artery runs close to or over a bone. In the arm it will be where the brachial artery runs over the humerus in the crease between the biceps and triceps. For bleeds to the legs the pressure needs to be applied to the femoral artery, in the inguinal canal. The combination of direct pressure, with or without indirect pressure, if applied correctly will control up to 90% of external bleeds.

Tourniquets

Tourniquets are not a new idea and can be traced back as far as Roman times and were used extensively during the 1800's and 1900's.

During the Vietnam War the view of the military was that the use of tourniquets led to an increase in limb amputations.

"Some people saw them as lifesaving, and others said they were the instrument of the devil," said Dr. John F. Kragh Jr., an Orthopaedic surgeon with the U.S. Army's Institute of Surgical Research in Texas.

Over the years tourniquets have gone in and out of fashion but from lessons learned from the recent conflicts in Iraq and Afghanistan have seen tourniquets become standard equipment in the arsenal of the pre-hospital provider. It has been recorded that no pre-hospital device de-ployed during the second Gulf War had saved more lives than tourniquets. There are several different designs in use today, each with their pros and cons.

SOFT Tourniquets

Perhaps the most recognisable and widely used is the Combat Application Tourniquet (CAT). Aside from the countless military lives saved during recent conflicts, the CAT is now being carried extensively by many members of the emergency services.

The majority of ambulances will have the CAT tourniquet as part of their standard response kit as well as more specialised teams such as HART (Hazardous Area Response Team). CAT's are also carried by some fire and rescue services and have become standard issue to police tactical firearms teams.

To date there are already numerous incidents in which CAT tourniquets have been used effectively , and saved lives, in the UK civilian pre-hospital setting.

Improvised Tourniquets

Although not as effective as the purpose made tourniquets mentioned above, a degree of haemorrhage control can be provided by the application of an improvised tourniquet. However, military studies have found that the average time it took to apply and improvised tourniquet was approximately 4.4 minutes.

With a haemorrhage rate of 1 litre per minute from a femoral artery, this would lead to total exsanguination

Although it would be extremely difficult to generate enough pressure with an improvised tourniquet around the thigh to completely occlude the femoral artery occlu-

sion of the brachial artery by this method has been seen to be successful.

That considered, recently however lives were saved during the Boston marathon bombings by the public using this method.

A retrospective study carried out by the Israeli Military looked at 550 casualties treated by military personnel between 1997 and 2001

91 of the survivors had been saved by having tourniquets applied to catastrophic limb haemorrhage. There were 125 deaths, but NO death from uncontrolled limb bleeding. The study concluded that tourniquets were effective in controlling life threatening bleeding.

Haemostatic Agents

Whilst tourniquets can be used to control massive haemorrhage from limbs, injuries to areas such as the abdomen, groin and axillae can also be fatal if not treated quickly and effectively as would the wound to the neck area shown below.

The introduction of haemostatic agents has enabled this category of wound to be treated more effectively than the traditional technique of wound packing with dressings alone. There are numerous commercial presentations of haemostatic agents from powder, granules and gauze to more expensive alternatives such as fibrin sealant glue.

Again having proven their worth in austere and hostile environments haemostatics such as Quikclot, Celox and Hemcon have become part of the civilian pre-hospital practitioners' equipment.

Chitosan

Celox products are impregnated with chitosan, which is extracted from the powdered shell of shrimps, If used in the powder form, chitosan breaks down in the body and forms a gel-like plug with bleeding controlled after pressure for 3-5 minutes.

The various Celox agents and gauzes also work without active clotting factors and even in the presence of heparins or warfarin. Unlike early products, which heated up significantly to over 40 degrees when used, which would cause superficial burns and even blisters. However, the newer products such as Celox and the latest Quikclot now have no exothermic reaction or heat generation.

Some haemostatic agents have been packaged and marketed to be applied to narrow penetrating wounds, such as gunshot or stab wounds, via a syringe like applicator. ATACC does not support or recommend this unpredictable use without more data. We suggest that carers stick to the either the powder or even better packing with gauze. Larger wounds will need to be packed with haemostatic dressings. The new generations of Quicklot products are now impregnated with an inert mineral called kaolin which contains no animal or human proteins or botanicals and is unlikely to cause any adverse protein-mediated reactions..

To enable good haemorrhage control of deep or cavitating wounds products such as QuikClot combat gauze, or Celox Rapid come in lengths of up to 12 feet and are most effectively applied using two people. One feeds the gauze whilst the other packs it tightly into the wound, whilst maintaining direct pressure. Full haemorrhage control should be achieved after 3-5 minutes of direct pressure.

Future Developments
Abdominal Aortic Tourniquet

Unlike a haemorrhage in the arm or leg where a tourniquet can be used to shut off bleeding, there was no way until recently to do the same for wounds in the lower torso, which can kill a person in a matter of minutes. The AAT is an inflatable tourniquet that buckles around a victim's abdomen, and when pumped with air becomes a wedge shape that puts about 80 pounds of pressure on the abdominal aorta, cutting off blood flow to the pelvis Studies to test the efficacy of the device using Dop-

pler ultrasound and healthy soldiers under the age of 25 have shown that this device has proven effective in the control of blood flow in the pelvis and proximal lower limbs.

It has also been used to stem [major haemorrhage from a gunshot wound](#) to the axilla.

We have tried these as a Faculty on test subjects and in our limited experience with healthy volunteers with normal blood pressures, the device was ineffective. They may however work better in a shocked individual.

At present we are unable to recommend this device until further studies are performed and usage data is collected.

Novel Tourniquet Designs

Other new tourniquets with ratchet mechanisms and quick-twist application collars are now reaching the market as many find the CAT Velcro tricky to use, especially

when wet, dark or under pressure, which most major bleed situations are! Once again, we have only limited anecdotal experience of these devices at present.

Novel Haemostatic Dressings

New haemostatic agents are still being developed and this one consists of dressings with haemostatic pellets actually within the dressing. There are also pellets which will hugely expand to fill any potential space, such as a wound cavity e.g. RevMedx XSTAT developed as part of a project by the United States Special Operations Command. These have been tried with and without haemostatics in early trials and they appear to work just as well with or without suggesting that the pressure effect of the expanding pellets is the key. Practically the pellets come in an applicator, which is plastic and quite bulky, which theoretically allows delivery deep into a penetrating wound. We have yet to see any good data to support this approach.

The ITclamp

This is another small mechanical device, which takes a 'bite' of the tissue on either side of the wound with small teeth and then a strong spring mechanism pulls the wound edges together.

Although there were initially some concerns about ongoing concealed bleeding within the wound cavity and lack of actual pressure effect, there are some small studies that already suggest that for the right wounds this is an effective device.

It has been suggested that the wound could be packed or filled with haemostatic and then held closed with the itclamp for good effect, but once again we are unaware of any case reports or recorded use in this manner.

Anecdotal individual case reports suggest that they are useful for many wounds, especially on the scalp, but also even larger vessels in marginal areas. This has been

[demonstrated in pigs](#) and there is increasing human work, which includes reports of the device not being actually that painful to have applied (quoted at 4/10 for a deep groin wound in one report).

Next Steps

So what action can be taken once the obvious external bleed has been managed and things are just getting worse?

Consider the mechanism and the findings during the primary survey, has anything alerted you to the possibility of massive internal bleeding? Have you maintained a high index of suspicion for unrevealed blood loss? If so then at this point the only thing that will save this patients life is surgery and this is not going to happen at the roadside. What is needed now is rapid transportation to definitive care and at this point definitive care is a major trauma centre and with the relevant specialties to deal with this patient.

During Transit:

- Maintain adequate perfusion, infuse just enough to maintain a perfusing blood pressure confirmed by the presence of a palpable radial pulse. (see [shock chapter](#))

- Gentle handling, the first clot is the best clot, try not to undo the good work already done by the healing process and dislodge clots that have started to form.

- Apply a pelvic binder, do NOT spring the pelvis, if you are suspecting a pelvic fracture bind the pelvis and leave well alone.

- Alert the trauma centre to activate their major transfusion procedure, give Tranexamic acid if indicated and available and "scoop and run".

- IV fluids, unless they are essential to restore a radial pulse have a very limited role in pre-hospital care. They may buy time but too much WILL certainly make things worse. Use the MINIMUM amount to maintain 'adequate' blood pressure to support essential end organ perfusion.

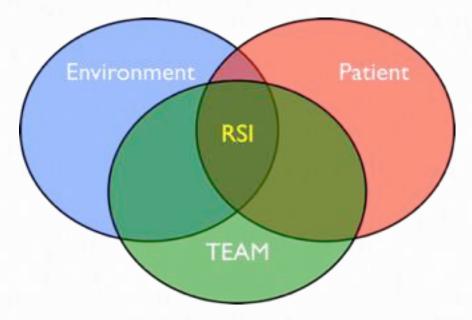- If available, consider giving blood products/ FFP.

Airway

Advanced airway management including rapid sequence induction (RSI) of anaesthesia is a fundamental component of critical care. Securing airway patency and protection is an essential skill in caring for the multiply injured or severely ill patient. It maximises oxygenation, enables safe transport, facilitates neuroprotection as well as rapid in-hospital investigation and definitive care. The extra time spent on scene securing an airway (even by skilled clinicians) is one of the greatest controversies in prehospital care. This time delay is normally offset by time saved during the transport and in-hospital phases of care, provided it is performed safely and expeditiously. Prehospital and Emergency Room RSI times of less than twenty minutes are safely and easily achievable and should be the target during training.

Out-of-theatre RSI should only be carried out only by appropriately governed, trained and equipped teams. Apart from the obvious emergent nature of the procedure, it is considered more 'high risk' than in-theatre RSI because of crew, resource, environment and patient factors. Every effort must therefore be made to minimise this risk and to ensure the safety of the procedure. In aviation and military settings it is well accepted, that the higher the acuity of the situation, the greater the need to remove individual procedural preference and the greater the need to adhere to a standard operating procedure.

This text has been developed to be straightforward, with patient safety at its core. The underlying philosophy is to promote a pre-planned laryngoscopy strategy that maximises first-pass success. The vast majority of patients can have a controlled, safe procedure, with just a few minutes of preparation time before the pre-determined treatment steps. There also exists an exceptionally small group of patients who are in or near respiratory or cardiac arrest, or who have agonal respiration, that require immediate intervention with little or no time for preparation. Training should prepare the team for either situation.

Indications for Out of Theatre RSI

The decision to proceed with an out of theatre RSI must be based on an informed assessment of the risk of the procedure versus the clinical benefits regardless of the indications listed. The team must take the following factors into consideration:

Factors in favour of RSI	Factors against RSI
Impaired airway maintenance and/or protection	Time critical surgical lesion (e.g. penetrating trauma with shock)
Hypoxaemia or hypoventilation, or hyperventilation in patients requiring neuroprotection	Morphology or pathology that may hinder successful intubation (e.g. laryngeal fracture, morbid obesity).
Fluctuating or deteriorating level of consciousness	Short distance from more appropriate environment, e.g. Resus or Operating Room.
Thermal injury to airway (at risk)	Paediatric patients- particularly <5 yo
Penetrating neck injury	Hostile environment
Long road or air transfer with risk of deterioration	Poor team dynamics
Polytrauma with requirement for multiple interventions and/or operative procedures	If a cricothyroidotomy (as a rescue surgical airway) is deemed to be impossible
Combativeness	Poor operator skills
High cervical lesion with diaphragmatic breathing	

Although simple airway manoeuvres and adjuncts such as airway suctioning, jaw thrust, chin-lift, oropharyngeal and nasopharyngeal airways may be essential initial measures to open and maintain a non-patent airway, these should be regarded as temporising measures. Such patients will ALL require a secure or 'definitive' protected airway at some point during their care and this should be considered as soon as it can be done safely and expeditiously.

Failure of Airway Protection

An unconscious patient with an easily maintained airway and adequate ventilation is still at significant risk of passive regurgitation and aspiration of stomach contents, secretions or blood, particularly if transport times to or within hospital (particularly journeys to CT scan) are prolonged. A patient with an unprotected airway is best defined by their inability to prevent aspiration of secretions, blood or vomitus and is indicated by an absence of spontaneous swallowing and/or failure to spontaneously clear blood, saliva or mucous from the oropharynx and loss of an effective gag reflex.

Failure of ventilation/oxygenation

Patients with acute ventilatory failure or failure to maintain adequate oxygen saturation despite supplemental oxygen and simple corrective measures should be considered for emergency anaesthesia and intubation. Such patients may have diminished respiratory drive due to head injury, critical chest injuries impairing ventilation or fatigue due to prolonged periods of increased work of breathing.

Protective ventilation – Damage Control

Typically in a head injury where a secure airway allows control of both oxygenation and often more importantly CO_2 control and to a degree ICP.

Anticipated Clinical Course

This indication refers to the patient who can be predicted to deteriorate (e.g. head injuries, inhalational/thermal injuries or spinal injuries) or where emergency anaesthesia will be important in removing the work of breathing in the face of multiple major injuries. In the case of major trauma patients, whose management is certain to in-

clude a complex and potentially painful series of procedures and diagnostic evaluations including emergency surgery; early anaesthesia and intubation should be considered.

To Facilitate Safe Transportation

A sub-group of patients will require emergency anaesthesia to ensure safe transportation particularly in rotary-winged or fixed-wing aircraft and/or where transport times are prolonged. These patients include agitated or uncooperative head injured patients or those with severe psychiatric disturbance.

Preparing to RSI

Once the decision to RSI has been made, a standardised and "automated" series of events should be triggered, interrupted only for true life saving procedures such as managing catastrophic haemorrhage. Interventions aimed at speeding up access to definitive care can take place simultaneously, provided personnel not directly involved in the RSI process perform them and they do not delay or hamper the process. Those involved should create a "sterile cockpit" environment. Make it clear to everyone around you that whilst this critical intervention is being performed the team should not be disturbed unless there is a critical issue.

Personnel

Ideally four people are required to perform an RSI, but certainly a minimum of three. It would be considered exceptional if this minimum team was not in place. It is recognised that many practitioners work in unique isolated environments. In exceptional circumstances a PHEM Physician might find themselves having to weight up the risk of not performing the procedure against less than ideal conditions, or with only two personnel.

- 1 x Laryngoscopist
- 1 x Airway assistant
- 1 x Drug administrator
- ± 1 x Person for In-line cervical spine control

The delegation of roles and appropriate briefing of staff is an essential task. From experience, the most experienced physician should be free to lead the team, administer drugs and provide situational awareness. A range of personnel can perform laryngoscopy; as part of a first pass airway failure plan, the brief should include the option of changing the laryngoscopist to the most experienced. If a difficult airway is anticipated or adequate pre-oxygenation is difficult, then the most experienced laryngoscopist should perform the

laryngoscopy and delegate situational awareness and leadership to someone else.

On arrival to an Emergency Department, the pre-hospital team should maintain control over the patient and assume leadership of the resuscitation team up until complete handover of the patient. The emergent nature of the patient's condition might necessitate induction immediately on arrival. In this instance, the handover of care should only take place after RSI is complete.

Clinical Area - 'Kit Dump'

The establishment of a clinical environment in a non-clinical area is the hallmark of good quality out-of-theatre RSI. Ensure adequate access to the casualty. Intubation is rarely practical in the position in which the injured or ill person is found and intubation on the ground should be avoided if at all possible. By default monitoring, equipment and airway assistant are usually located to the right of the patient, with all additional personnel on the left.

360-Degree Access

The first task after deciding to intubate is to locate the most appropriate place to perform the procedure. In the emergency department environment, it is frequently more practical to move a patient out of an enclosed majors cubicle to a dedicated resuscitation area, ensuring good 360-degree access, skilled staffing and standardised equipment. Many hospitals now have a successful policy of "scoop and run" from wards, clinics or diagnostic areas to a dedicated stabilisation area, usually theatre recovery or ICU. It not only allows a full, skilled team to get ready to receive a patient, it focuses the limited personnel with the patient to concentrate on pre-oxygenation. The risks incurred by postponing intubation to transfer the patient to an area of safety are smaller than the risk of performing the procedure in an unfamiliar environment, with hastily assembled equipment and limited monitoring.

In the pre-hospital environment, the ideal situation to perform RSI is on an ambulance trolley, outside of the ambulance with good lighting, but away from direct sunshine. Weather conditions may dictate alternative arrangements. In an extrication scenario, preparation for RSI should take place in a safe area away from the patient. This team approach allows rescue to take place without unnecessary medical interference and has been demonstrated to greatly speed up time to definitive care. This may have to be facilitated by analgesia or sedation.

In-Ambulance RSI

In-Ambulance RSI requires practice. Monitoring is usually clipped into its bracket on the ambulance and is therefore on the left of the patient. Airway Equipment is placed on the forward most seat, to the right of the patient, with the seat positioned directly forwards. The area next to the emergency exit door must be completely clear of all obstructions. The Airway assistant stands to the right of the patient, passing equipment with their left hand and providing external laryngeal manipulation (ELM) or other support with their right. It is important to have the 'manual in-line immobiliser' stood to the patient's right, or from behind the airway assistant. This is to ensure easy swapping of laryngoscopists in a confined space. In older ambulances, the trolley being moved from against the wall may assist this. It may be an option to open the side door of the vehicle to create more space and use the area immediately adjacent to the ambulance if necessary. An IV line with an extension set and access port will help create space for drugs to be administered without having to be stood over the patient in a confined space.

RSI on the ground

Laryngoscopy is the process of bringing the glottis into alignment with the eyes of the operator and as such the laryngoscopist must lay completely prone to maximise their position for laryngoscopy. It should not be considered as a primary option. If the patient cannot be moved onto an ambulance stretcher, attempt to move them on a longboard, scoop or vacuum mattress onto a raised platform off the ground. If there is no other choice, the following may be helpful:

- Kneeling is probably the most versatile position on rough or soiled ground but often difficult to obtain a good view. The use of proper knee padding on PPE facilitates this.

- Lying prone gets the eye line right, but may put the laryngoscopist at a mechanical disadvantage.

- Left lateral position provides a good eye line and mechanical advantage. It is best performed with the patient already in a vacuum mattress. Comfort for the laryngoscopist may be improved with padding under the left elbow (such as a folded SAM splint). It has an added advantage in that it enhances drainage of fluid from the oropharynx. This is an advanced skill and must however be really well practiced in training to ensure optimum and consistent performance.

In-Aircraft RSI

If onwards transport is required in an aircraft and airway compromise is a possibility or if the patient is a potential threat to the aircraft (e.g. combative), a low threshold for RSI prior to take-off is required. Prior planning and preparations is essential to prevent at all costs, emergency airway management in an aircraft.

A multitude of distinctly different HEMS platforms are currently available. Some have very limited access to the whole patient, with only their head and upper torso visible (e.g. Eurocopter EC 135), some have good access to the whole patient, but in certain set up, limited access above the head (e.g. Augusta Westland 139). Others used in Coast Guard and Mountain Rescue Environments are more spacious than most hospital resuscitation bays (e.g. Sikorsky S-92 or Westland Sea King). Likewise, a multitude of different fixed-wing aircraft platforms exist, too numerous to mention. For ease of description in this text, we consider aircraft platforms in two categories, confined or non-confined airway access.

EC 135 Helicopter with limited access to body

AW 139 Helicopter with limited access to head

Should a patient deteriorate in flight in a confined access aircraft and RSI is required; the standard checklist is to be initiated and best efforts made to pre-oxygenate whilst the helicopter or aircraft lands to facilitate space creation and laryngoscopy on the ground. In truly life-

threatening situations, Pharmacologically Assisted Laryngeal Mask Placement (PALM) may be considered (see below section). In smaller helicopters, often all that is required is the door of the aircraft to be opened and the laryngoscopist to stand on the skid or ground. In many fixed wing platforms, the patient may need to be manoeuvred out towards the door on the sled to gain full access to the head.

Manoeuvring patient to the door of a Citation II Jet to RSI

In-flight RSI in larger platforms should still be avoided, unless the patient has been rescued from a place of danger into the helicopter, in which case RSI may be a suitable planned intervention. In-flight deterioration in larger aircrafts does not mandate landing, but landing should certainly be considered and discussed with the pilots.

Spacious working platform within a S92 Helicopter

Optimising Pre-Oxygenation

Pre-oxygenation is essential for safe anaesthesia and RSI. It should commence from the time the decision to RSI has been taken, and continue throughout the preparation phase. Pre-oxygenation denitrogenates and establishes an oxygen reservoir in the lungs, blood and tissues. If effective it will allow several minutes of apnoea without O_2 desaturation and the need to ventilate the patient. All patients should also be commenced on high flow oxygen at 15L/min by nasal cannulae from the onset. This has been proven to greatly extend the safe laryngoscopy window in terms of oxygenation. There are two considerations when pre-oxygenating depend-

ing on the patient's ability to maintain effective self ventilation.

Positioning

Patient positioning is critical, particularly in respiratory failure, the obese, or heavily pregnant. If a patient has positioned themselves in a sitting position out of necessity to maintain ventilation they should remain as long as possible in this position, whilst pre RSI checks are being conducted.

It has been well demonstrated that simple atlanto-occipital extension pivots the tongue backwards, and does not adequately open the airway. Head elevation improves jaw mechanics, allowing for jaw distraction to lift tongue and jaw upwards, opening the airway. The tragus of the ear should be at the same horizontal level as the sternum, so called "Ear to Sternal Notch Position". Placing a small pad under the occiput assists this ideal position in adults; e.g. a SAM splint or the padding used to secure blocks on a longboard. In contrast, the younger the child, the proportionally larger the occiput, may necessitate no pad or even a rolled towel placed under the shoulders to achieve a similar positioning.
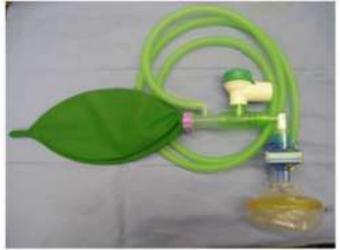
Apnoeic Diffusion Oxygenation (ADO)

Studies have demonstrated that during the apnoea post muscle relaxant administration in RSI, passively supplying oxygen to a patent airway can significantly prolong safe apnoea time before desaturation occurs. This phenomenon is well described in brainstem death testing protocols where normal oxygenation can be maintained for long periods without ventilation, when oxygen is supplied to apnoeic patients via the tracheal tube.

The physiology is relatively straightforward; during apnoea, oxygen is continuously absorbed into the blood from the lungs functional residual capacity, at a rate of approximately 250mL/min. CO_2 continues to accumulate in the blood, only slowly diffusing into the aveoli at a rate of approximately 10mL/min. This differential creates sub-atmospheric alveolar pressure and a net inflow of gas into the lungs of around 240mL/min in the average anaesthetised human, provided the airway is kept patent. If oxygen (rather than room air) is supplied then the oxygen reservoir in the lungs can be maintained for extended periods.

In the pre-hospital setting this is best accomplished by administration of high-flow oxygen at 15L/min by nasal cannula and

left on until successful tracheal intubation is accomplished. The airway is naturally opened during laryngoscopic attempts and this can be augmented by airway adjuncts or jaw thrust. It is also worth noting that the use of nasal cannula in addition to simple facemask or Mapleson C Circuit ventilation for pre-oxygenation; has been shown to significantly increase FIO2 in spontaneously breathing patients by washing out ventilatory dead space with oxygen in-between spontaneous breaths and thus increases the effectiveness of pre-oxygenation

Nasal Cannula in situ as part of an Apnoeic Diffusion Oxygenation Strategy

Mapleson C Circuit (AKA Waters' Circuit)
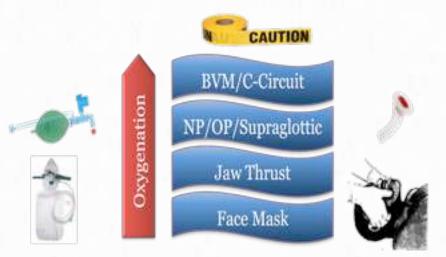
Adequate Ventilation

Patients who are able to maintain effective ventilation on their own or with the addition of simple airway adjuncts such as nasal or oral airways should be left to breath spontaneously on the nasal cannula and a tight fitting non-rebreathe (trauma) mask or Waters' circuit until the RSI Checklist is complete. The advantage of this approach is it completely frees a member of the team from manually attending to the airway. Also in when comparing this approach to different types of bag-valve-mask (BVM) devices commonly used, the FiO2 may be substantially lower than with a trauma mask or Water's circuit:

On 15Lt/min SV:

FiO2 96%

FiO2 70%

FiO2 55%

All >95% FiO2 with PPV

A jaw thrust, with or without Nasopharyngeal and/or oropharyngeal airways should next be considered if there is difficulty at

first maintaining an open airway. Instrumenting and suctioning the airway should be avoided, if at all possible, to prevent regurgitation and unnecessary rise in intracranial pressure. If completely obtunded, consider a supraglottic airway device.

Stepwise Approach to Optimising Pre-Oxygenation

Inadequate Ventilation

A Bag-valve-mask (BVM) has been the standard for assisted ventilation since the 1950s. It is immensely useful in the pre-hospital setting, providing positive-pressure ventilation without an oxygen source. However, we consider it to be a backup device, used for failure of oxygen supply only. It requires considerable effort to self ventilate through the device and thus is only really effective in completely apnoeic patients. There is good evidence, particularly from Resuscitation literature, of its inconsistency to provide guideline consistent ventilation. Even in experienced

hands, it offers little visual clue to the actual tidal volumes being delivered and it is far too easy to hyperventilate with resultant hypocapnoea or barotrauma.

A Mapleson C-Circuit (Waters Circuit) for adults, and a Mapleson E-Circuit (Ayres T-Piece) for children, should be carried as standard equipment for patients with inadequate ventilation. In addition to the nasal cannula the circuit is set up to include a heat moisture exchange (HME) filter, ET-CO_2 monitoring, and held on with a face-mask to provide 100% oxygen during the checklist.

In some instances, patients may already be on a trial of Non-Invasive Ventilation, these patients should remain on the system for pre-oxygenation.

Ayre's T-piece

Effective use of Jaw Thrust + Oral airway + C-Circuit + ETC02 monitoring to Pre-oxygenate

Failure to Adequately Preoxygenate

Combative, agitated or uncooperative patients are a particular challenge. This is commonly encountered and is usually as a result of unmanaged hypoxia and pain following a head injury. Premedication can dramatically improve seemingly helpless situations, allowing the team to gain control and composure. Careful consideration must be given to identifying and resolving a respiratory, as opposed to airway, cause for failure to adequately pre-oxygenate.

In certain critically ill patients, even with effective measures described above, oxygen saturation may remain suboptimal and the intubation may need to proceed regardless, provided all reasonable measures to correct hypoxia have been instituted. This

is a matter for clinical judgement on the part of the team.

Pharmacologically Assisted Laryngeal Mask Placement

The Concept of Pharmacologically Assisted Laryngeal Mask (PALM) placement fits into a failure to adequately pre-oxygenate scenario, particularly where a patient is trapped, such that access to their airway is suboptimal, making endotracheal intubation unsafe.

Some patients falling into this category may benefit from supraglottic airway management with the clear aim of promoting oxygenation and improving ventilation. These are usually patients who are extremely agitated from hypoxia, drugs or head injury, but are not suitably obtunded to tolerate a supraglottic airway device. A consensus meeting in the UK was held with a range of stakeholders supporting this concept for the 'non-RSI competent provider'. The notable non-signatory was the Royal College of Anaesthetists.

When all other airway manoeuvres, adjuncts and most importantly pain management strategies have failed to oxygenate, and the patient is clinically deteriorating, the pharmacological blunting of airway reflexes sufficient to tolerate a supraglottic

airway device has been advocated as a rescue technique. There is no consensus as to the most appropriate agent, but both ketamine and midazolam have been described in the literature. It is worth noting that although laryngospasm is rare (1-2% of sedations), most regular prescribers of ketamine will have seen it.

Pre-Medication

Anxiety and pain are inter-related and each may exacerbate the manifestation of the other. As a result, managing a patient will become more difficult unless each is managed appropriately. Any drug treatment must be symptomatically targeted. This text makes an intentionally clear distinction between analgesia and anxiolysis. Sedation is not a substitute for satisfactory analgesia. It is recommended that if both an analgesic and a sedative drug are being considered, the analgesia be given first and allowed to become maximally effective before any sedative is administered. Opioid analgesics are themselves sedating to a varying degree.

i-Gel LMA with distal suction and oxygenation ports.

Analgesia

As a neuro-protective strategy, Fentanyl should be considered in haemodynamically stable patients with raised intracranial pressure or in patients with underlying cardiovascular disease at a dose of 1-3 µg/kg at least 3 minutes before intubation. Fen-

tanyl is 100 times more potent than morphine with a peak analgesic effect approximately six minutes following intravenous administration. It is therefore best given very early. The usual duration of analgesic action is no more than 30 to 40 minutes after a single intravenous dose.

Alfentanil, at a dose of 5-30 µg/kg, has a far more rapid onset profile and is immensely useful if potent analgesia is required just before laryngoscopy, although it may not fully obtund the CVS response to laryngoscopy. It is however not always easy to obtain out of the operating theatre environment and can easily produce profound respiratory depression for short periods.

Ketamine is a far more useful analgesic agent than opioids for limb ischemia associated with entrapment, as it is a potent NMDA Receptor antagonist. Pre-medication with ketamine (0.1-0.5 mg/kg increments) can be very effective means of facilitating further assessment, monitoring and pre-oxygenation. This has been described in the literature as "Delayed Sequence Intubation." Owing to their highly lipophilic properties and resultant ease of absorption, fentanyl and ketamine are particularly useful for administration via the oral transmucosal or intranasal routes.

Anxiolysis

Out-with psychosis, it is very uncommon not to achieve adequate anxiolysis with appropriate analgesia. Sedation as a purposeful delayed sequence intubation pathway is however increasingly described in the literature, largely for humanitarian reasons. Ketamine fulfils both analgesia and dissociative rolls with this regard, and would be our first choice for delayed sequence intubation. Small doses of short acting benzodiazepines, such as midazolam or alternatively small doses of your chosen induction agent will be just as effective. Note this should not be confused with PALM, but rather as a continuum of sedation into RSI, during the challenge-response checklist.

Fluid Bolus

Adequate fluid preloading may help to mitigate hypotension that can occur during RSI as a result of reduction of systemic vascular resistance and positive pressure ventilation. A crystalloid fluid bolus of 10ml/kg immediately prior to RSI should be considered, unless an active decision for permissive hypotension has been taken.

RSI Check-List

READ EVERY WORD AND WAIT FOR RESPONSE BEFORE MOVING TO NEXT LINE

What is indication for RSI ?	Response
PRE-OXYGENATE – Consider Nasal Prongs……….........................	**Check**
Oxygen cylinder more than 1/2 full or Wall source available…......................	Check
Spare cylinder available under trolley …………………………………….	Check
Water's Circuit connected to Oxygen…………………………………….	Check
Face Mask, filter and End Tidal CO_2 Monitoring connected into circuit…..…	Check
SUCTION working………………………………………………........	**Check**
Hand-held back-up suction available……………………………………	Check
BVM available…………………………………………………………	**Check**
Oropharyngeal & 2 nasopharygeal airways available...................................	Check
LMA available if there is failure to intubate on 2nd attempt?......................	Check
Surgical airway kit available……………………………………………	Check
IV Running with access port connected..	Check
2nd cannula in situ or IO available …………………………………	Check
Drugs for Induction, what dose?..	Response
Paralysing agent, what dose?..	Response
What maintenance drugs are we using? ……...........................	Response
Emergency drugs available?……...	Response
LARYNGOSCOPE working………………………………………	**Check**
Back-up Laryngoscope working…………..........................	Check
Bougie……………………………………………………………	Check
Tube size ……………………………………………............	Response
Alternate tube size ………………………………………	Response
Test tube cuffs…………………………………………..................	Check
Tube tie or tape ………………………………………………	Check
Check Monitoring, what is the **O2 Saturation?**...........................	Response
What is the **Blood Pressure?**..	Response
What is the **Heart Rate?**..	Response
Brief drug administrator………………………………………	Briefing
Perform In-line immobiliser brief……………………………………	Briefing
Perform Cricoid brief…………………………………………………	Briefing
All listen for failed airway plan………………………………………	Briefing
Optimise patient position…..…………………………………	Ready
Check Complete…………………………………………………	Proceed

ATACC RSI Checklist

RSI Challenge – Response Checklist

The RSI Challenge-Response Checklist is handed to someone to read. This person has the words in red "READ EVERY WORD AND WAIT FOR RESPONSE BEFORE MOVING TO NEXT LINE" clearly pointed out to them. This checklist is designed to

take 3 minutes in a well-drilled team, facilitating the requirement for pre-oxygenation and collation of equipment. It must be followed in its entirety, with each preparation line checked or responded to and all procedural briefings completed.

Equipment

A separate Airway Equipment Appendix at the end of this text covers individual equipment items

All equipment should be laid out ergonomically, to facilitate ease of access and checked against the RSI Checklist. Equipment bags can act as a useful crowd barrier if set up as a cordon prehospital. All equipment must be to hand and made ready for the worst-case scenario. Avoid unnecessary or unrelated equipment cluttering the working space. The first equipment to be made ready is everything associated with pre-oxygenation.

Whilst a spare oxygen cylinder is intuitive in the prehospital environment, in-hospital it is frequently not always underneath the trolley, ready to transfer the patient onwards to theatre or CT; hence its inclusion in the checklist. Always ensure that it is available, or on its way, to avoid unnecessary delays.

Suction is frequently neglected until it is too late or fails due to catheter blockage. It is recommend that two forms of suction are always available, with the suction catheter placed to the right of the patient's head, for easy access during laryngoscopy.

Safe Clinical Area for RSI being Prepared

Monitoring

Full monitoring as per AAGBI guidelines is mandatory in all patients. It should ideally have already been in place from the Primary Survey, prior to RSI decision-making. Minimum monitoring standards widely accepted include:

- SPO_2

- NIBP (set at three minute cycles or less)

- ECG

- Waveform $ETCO_2$

Waveform ETCO$_2$ should be placed in line, behind the HME filter, when setting up the Mapleson C Circuit. It is mandatory, prior to drug induction to allow or support the patient to ventilate through this circuit, to confirm that Waveform ETCO$_2$ is fully functioning.

A Mapleson C (Water's) Circuit with inline HME and Waveform ETCO$_2$

Situational awareness throughout the process is essential. Ideally a person dedicated to scribing should be appointed to record and monitor all events. This aught not to be the most junior member of the team, but ideally a senior member of nursing or paramedical team who is empowered to speak up and alert the team leader to deteriorating trends, unsafe or unorthodox practice. Standardised patient report forms pre-hospital or dedicated resuscitation proformas have been shown to not only improve note taking, but the overall performance of the team.

Monitoring alarms must be pre-set with limits clearly agreed to in advance, particularly when adopting permissive hypotensive strategies. Alarms, which have to constantly be silenced, pose significant risk of being ignored during a significant adverse patient event.

Emergency Drugs

Inotropic agents should always be to hand prior to induction e.g. Ephedrine, adrenaline or metaraminol (aramine) and atropine for example.

Team Briefing

In-Line Immobiliser Brief

The C-spine should be routinely protected in all blunt trauma patients and this should be performed by an assistant holding the head from the left side of the patient. If a cervical collar is already in situ, it should be open and the mandible freed of any restrictions for intubation. Experienced operators should consider obtaining the minimal acceptable view that allows intubation as this may reduce cervical movement, though this should not endanger the success of the procedure.

In many patients the cervical collar causes significant neck extension and this is exaggerated in those with a large body habitus. This can be corrected by placing a folded

towel or SAM splint beneath the occiput to maintain a neutral head position. This may reduce the tendency of the laryngoscopist to "suspend" the head from the laryngoscope and flex the neck. If cervical spine precautions are not needed (for example some burns patients or following submersion incidents) the patient should be positioned "Ear to Sternal Notch."

Cricoid Brief

There is no evidence that even well applied cricoid pressure (Sellick's Manoeuvre) prevents passive aspiration. It is most commonly poorly performed. There is evidence that cricoid pressure may reduce tone at the lower oesophageal sphincter, significantly impair laryngoscopic view and cause unwanted movements of the cervical spine. It should no longer be routinely applied. If the clinician decides to use cricoid pressure for the RSI they must ensure the cricoid operator is briefed appropriately and cricoid pressure removed if laryngoscopic view is difficult.
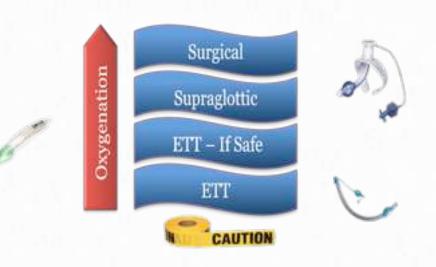
External Laryngeal Manipulation (ELM) or Backwards, upwards and right-wards pressure (BURP) have been demonstrated to improve laryngoscopic view and should be used whenever initial view is suboptimal. The laryngoscopist manipulates the thyroid cartilage to maximise their view and an as-sistant can be directed to hold the thyroid cartilage in position during intubation. It is important to recognise the difference between this technique and cricoid pressure.

Failed Airway Plan

The team, as part of the RSI checklist, must agree upon the plan for failed intubation. Pre-hospital failed intubation protocols deviate from the Difficult Airway Society algorithm based on the premise that in the emergency environment intubations are carried out due to either failed anatomy or failing physiology and the option of waking the patient up simply do not exist in most cases.

For many years the original algorithm promoted by London HEMS consisted of surgical airway for "repeated failed" intubations. This led to a surgical airway rate of around 2%, approximately half of which followed failed intubation and half performed as primary procedures. This compared well with emergency department surgical airway rates for severely injured patients.

Alternative algorithms promoted in UK and Australian prehospital services gives the choice of the iGel or similar suitable supraglottic airway device as an acceptable early alternative. This has translated well into hospital practice, and the below sim-

ple algorithm has been modified with hindsight of the UK NAP4 study, highlighting the exponential dangers of >2 attempts at intubation together with evidence pertaining to airway soiling in trauma being predominantly from upper airway bleeding.

First failure of direct laryngoscopy

Where an adequate view cannot be obtained a further attempt may be undertaken provided deliberate steps have been taken to identify and rectify the problem causing the failure and that oxygenation can be maintained between attempts. These are the thirty second drills, so named because they should be easily performed long before a normal pre- oxygenated patient begins to desaturate.

30 sec Drills prior to 2nd attempt at direct laryngoscopy

• Consider Changing Operator

• Consider Removing Cricoid if used and perform External Laryngeal Manipulation (ELM) or BURP.

• Optimise operator position

• Optimise patient position (small pad under the head with neck in neutral position)

• Use better suction where secretions or blood block the view

• The laryngoscope can be inserted deeply and slowly withdrawn until identifiable anatomy is seen

• Consider changing laryngoscope blade size or type

Second failure - MANDATORY supraglottic

If two failed endotracheal attempts at intubation occur in the field, the patient should be transported into the emergency department with a supraglottic airway or a surgical airway in situ. A supraglottic device should be used in preference to BVM (Bag – Valve – Mask) ventilation to prevent gastric inflation and an increased risk of aspiration. It may rarely be inserted blindly into trapped patients. It is a useful option, if the experienced operator feels that further attempts at intubation will likely be futile and

that expediting care to hospital the safer option.

This is a "can't intubate – can ventilate" scenario and must be accepted as such with no further attempts at obtaining a definitive airway pre-hospital. A call must be made to pre-alert the Emergency Department to ensure a Consultant Anaesthetist is on hand to receive the patient. A number of options exist in hospital, which include converting the Supraglottic device by means of a fibreoptic scope into a definitive airway.

If at any time a "Can't intubate – Can't ventilate" scenario occurs, it is mandatory to proceed immediately to a surgical airway, without any further delay.

If two failed endotracheal attempts at intubation occur, a supraglottic airway should be inserted and no further intubation attempts made until the patient has access to facilities and expertise to fiberoptically convert a supraglottic airway to a definitive airway. The most important consideration always being the maintenance of oxygenation.

Can't Intubate - Can't Ventilate

If at any point a can't intubate, can't ventilate scenario is encountered, a surgical airway must be performed without delay.

Human factors research and high profile cases such as the "Elaine Bromley Case" have clearly demonstrated that performing a surgical airway in this instance is an absolutely essential, mandatory procedure, that must be a mere simple reflex response in the shortest possible time. In preparing for any RSI, the surgical airway must always be considered, with equipment readily to hand.

The likelihood that a practitioner would have to carry out this procedure *regularly* in their clinical career, is slim to none. It is imperative that it is regularly trained for in a simulated environment. A number of purpose designed commercial emergency cricothyroidotomy kits exist, each with their own merits and pitfalls. It is increasingly being recognised that the emergency cricothyroidotomy is the most reliable approach and the one that uses equipment familiar around the world. It is not the same procedure as a planned surgical tracheostomy.

A needle cricothyroidotomy is a lifesaving technique that will buy you time, but will only be truly effective using a jet-ventilator to provided effective gas delivery through the cannulae. Ideally the cannulae should be armoured to avoid kinking and it can be inserted directly through the cricothryroid membrane. If there is any swelling of the

neck, make a small incision and then dissect a little to identify the cartilage. NAP4 has demonstrated there is a significant failure rate if the membrane is not properly identified. If a needle cricothyroidotomy is performed you must ensure that gas can escape the chest by mouth or another cannula and at the earliest possible opportunity, this should be converted to a definitive airway.

Definitive Airway Management

Induction Agent

Ketamine is the out of theatre induction agent of choice. In comparison to other currently available induction agents, Ketamine has a much safer haemodynamic profile in the emergent patient. Ketamine is a safe and effective drug for patients with traumatic brain injury and intracranial hypertension. Whilst studies from the 1970s raised concerns with regards to an association between ketamine and increased ICP, in more recent studies no statistically significant increase in ICP was observed following the administration of ketamine in patients with head injury. With more recent increase in use and further study, there is emerging evidence of a net increase in CPP following ketamine administration.

Ketamine for induction and on-going maintenance of anaesthesia should be carried in the more concentrated 50mg/ml doses to avoid dose confusion and to enable multiple routes of drug administration. In all Paediatric patients, Ketamine must be reconstituted to a concentration of 5mg/ml

An emergency situation is however not the time to practice with new or unfamiliar pharmacological agents, so the agents used most regularly to perform rapid sequence induction of anaesthesia by the team, is _usually_ the most appropriate. Propofol is usually avoided in shocked or major trauma patients, however if they are cardiovascularly stable and this is the drug with which you are most familiar than it can be used with caution. Remember that the cardiovascular stimulation-inhibition balance of any induction agent, ketamine included, may be altered by hypovolaemia and severe critical illness. The dose of any induction agent should be significantly reduced in the critically ill.

Paralysing Agent

In the out-of-theatre environment, intubations are carried out due to either failed anatomy or failing physiology. The option of waking the patient up simply does not exist. High dose rocuronium (1.2mg/kg) is therefore our muscle relaxant of choice. Ro-

curonium has some additional [practical and theoretical advantages] over suxamethonium including a better safety profile, longer duration of action, and a longer desaturation time.

Suxamethonium does convey the advantage of a slightly faster onset (45 vs 60sec). The presence and cessation of fasciculation is an additionally useful indicator of muscle relaxation in most patients. Suxamethonium should be avoided in burns patients after 24 hours, crush injuries and patients suffering denervation. It may cause a slight rise in ICP that is considered by most to be clinically unimportant.

Direct laryngoscopy

Approximately 45-60 seconds after muscle relaxant administration, the jaw should be tested for flaccidity and laryngoscopy attempted. There is always time to perform laryngoscopy gently and carefully.

Bougie or Stylet Introducer

The use of an intubating bougie or Stylet Introducer is associated with higher success rates, particularly on first attempt. It reduces the chance of being unable to pass a tube when the glottis is well visualised and may reduce cervical movement required to perform intubation.

Two well described signs of tracheal placement of a gum elastic bougie have recently been studied.

- Clicks (produced as the tip of the bougie runs over the tracheal cartilages)

- Hold up of the bougie as it is advanced (when the tip reaches the small bronchi)

No clicks nor hold up occur with the bougie in the oesophagus. Clicks were recorded in 89.7% of tracheal placements of the bougie. Hold up at between 24-40 cm occurred in all tracheal placements. Whilst these signs may be useful, unrecognised traumatic oesophageal and tracheal perforations are a well documented risk of bougie use. Gentle insertion and control of the introducer at all times is paramount. The Royal College of Anaesthetists recently recommended that the hold-up sign should no longer be used with single-use bougies, following findings that It is possible to apply a force [at least five times] greater than the force required to produce significant trauma with a Frova single-use bougie. They additionally cautioned eliciting this sign using the more flexible Eschmann reusable bougie.

Confirmation of Tube Placement.

There are a number of Subjective signs that have been used to confirm endotracheal tube placement:

- See the tube passing through the cords

- Palpation of tube movement within the larynx and trachea

- See the chest expand equally with each ventilation

- Auscultation of breath sounds

- Absence of epigastric sounds with respiration

The above clinical signs are notoriously unreliable, particularly in the prehospital environment. The AAGBI recommend that all endotracheal intubations are routinely confirmed Objectively with:

- Waveform End Tidal CO_2 monitoring (capnography).

- Disposable qualitative $ETCO_2$ detectors may be used as a backup in case of failure of waveform or quantitative capnography. These devices undergo a colour change when expired CO_2 passes across their surface. The colour change is from purple (room air) to yellow (4% CO_2). At least six tidal volumes should be given before these detectors are used to confirm tracheal tube position.

Waveform Capnography Is a Basic Standard of Care

Emergency Surgical Airway

A surgical cricothyroidotomy involves making a surgical incision through the skin and cricothyroid membrane and inserting a tracheostomy (or ET tube if unavailable), through the incision and into the trachea.

Tools:

- Scalpel

- Forceps

- Bougie

- 6.0 ETT

Approach:

- Extend the patient's neck fully and locate the cricothyroid membrane

- Using a scalpel, make a stab incision through the skin/cricothyroid membrane until there is a loss of resistance

- Using a forceps, grab the scalpel on either side of the blade until both the forceps tip and scalpel are in the trachea.

- Remove the scalpel and dilate the passage into the trachea using the forceps.

- Before removing the forceps, Insert a bougie into the passage, aiming towards the patient's feet

- Railroad a size 6.0 ET tube over the bougie into the trachea

- Inflate the ET tube cuff and confirm tube position with capnography/auscultation

This technique is ideally performed with two team members. It is important to make sure that the scalpel and forceps are not prematurely removed from the incision

made as the tissues will rapidly close, causing loss of the passage into the trachea. If the patient has a short neck or is over-weight, correct identification of landmarks may be difficult. Finally, the incision into the neck will cause bleeding, which must not distract from the timely placement of the tube. Bleeding tends to be tamponaded by a tube in situ. If bleeding persists, again it is much more easily managed with direct pressure applied around a tube in the trachea.

Post RSI Care

It is important to avoid a post-intubation lull in tempo and vigilance. This is the time when serious errors are most likely. It is possible for the tube to become dislodged, monitoring to become disconnected, ongoing sedation to be forgotten or no one remember to bag-ventilate the now paralysed patient. The team must remain vigilant to avoid such errors.

Be very aware of post RSI complications such as bradycardia (from hypoxia or repeated suxamethonium doses) and hypotension (over sedation), development of tension pneumothorax and inadequate or hyperventilation.

Targets

The aim is to provide a high standards of Intensive Care, regardless of the patients location.

Parameter	Target
MAP	> 80
SpO2	> 95%
ETCO2	4 – 4.5 KPa

Maintenance of Anaesthesia

Vigilance for evidence of awareness post RSI should be maintained, actively excluding signs such as:

- Tachycardia

- Hypertension

- Eye Watering

- Sweating

- Symmetrical pupil dilatation

It should be noted that many patients will actively improve their pre-RSI conscious levels with good quality ventilation and blood pressure control. An anaesthetic maintenance plan needs to be initiated before transferring the patient. In planning any journey, sufficient analgesia, hypnotic and paralysing agents should be to hand.

Ketamine

A single 500mg vial of Ketamine will satisfy most patient's analgesia and hypnotic requirements from Prehospital RSI to hospital and even beyond to CT or operating theatre. It is therefore a very useful single agent. Once induction of anaesthesia is complete, it should be remembered that 50mg/ml is a very concentrated dose of Ketamine and should be used with caution.10mg/ml is a safer alternative for the less experienced.

Alternative Hypnosis

Drugs usually delivered via syringe driver add an additional layer of complexity to out of theatre practice and should be avoided. Boluses of Propofol on the way down the corridor for example, create huge margins for both catastrophic hypotension and awareness.

Midazolam is a useful hypnotic agent to bolus. In larger doses it reaches steady state, increasing its length of therapeutic effect, whilst maintaining a relative degree of cardiac stability.

Analgesia

Morphine titrated regularly in small 0.02 – 0.05mg/kg boluses remains a very safe agent for ongoing analgesic requirements of an intubated and ventilated patient.

Again, the use of shorter acting agents via infusion pump adds a layer of complexity that should be carefully considered.

Paralysis

To keep the number of agents used to a minimal, if using rocuronium at induction, muscle relaxation should be maintained by bolusing rocuronium at a dose of 0.6mg/kg (~50mg) every 30-40 minutes. Alternatives include vecuronium and pancuronium, depending on the length of paralysis intended. Atracurium can also be used, but it is very unstable out of the refrigerator

Ventilation

Overzealous positive pressure ventilation (particularly in hypotensive patients) by increasing intrathoracic pressure and reducing venous return will reduce cardiac output. It is well established that inappropriately high peak airway pressures (PAW) only serve to inflict barotrauma and add to the risk of ARDS further along the patients journey.

Lung Protective Ventilation Strategies should be initiated from the onset of paralysis. Avoid hand ventilation where at all possible, but if unavoidable, use a Mapleson C or F Circuit, which provides for far greater guideline consistent ventilations than BVM.

End-tidal CO_2 should be kept within physiological values around 4-4.5 kPa., particularly in head injured patients. Increasing PEEP, whilst frequently necessary, will only serve to reduce cardiac output in the haemodynamically compromised patient and should be kept as low as possible.

If a patient desaturates following intubation and ventilation, displacement of the tube, obstruction somewhere in the breathing circuit, pneumothorax and equipment failures such as mal-assembly or malfunction should be checked.

Gastric decompression with an orogastric tube should also be considered particularly in children and in patients who have had a period of bag-valve-mask ventilation.

Airway Equipment Appendix

This section is intended as a more general guide to various pieces of airway equipment referred to in this text for the novice reader. It covers a basic description and the relative advantages or disadvantages of each device.

Bag-Valve-Mask (BVM)

A BVM is a ventilation device designed to provide positive-pressure ventilation. It consists of a mask which forms a tight seal over the patient's face, and a self-inflating bag with a one way valve which is squeezed to inflate the lungs. Depending on make some BVM devices may also have:

- Heat-moisture exchanger (HME) filters
- Pop-off valves to prevent over-inflation of the lungs
- Oxygen reservoir

Advantages:
- Self-inflating (Does not need an external oxygen supply)
- Can give supplemental oxygen to patient if oxygen supply available
- Readily available

Disadvantages:
- Unsuitable for spontaneously breathing patients
- Risk of over-inflating lungs/stomach if bag squeezed too hard

Mapleson C-Circuit/ Waters' Circuit ("Anaesthetic Circuit")

A C-circuit is a ventilation device that can provide both positive-pressure ventilation or allow the patient breath spontaneously. It consists of tubing which connected to an oxygen supply, a reservoir bag and an Adjustable-Pressure Limiting (APL) Valve.

The APL valve can be manually adjusted to increase or decrease the amount of pressure in the circuit before the excess pressure escapes through the valve.

By squeezing the reservoir bag, the patient will be given positive pressure ventilation. If you find the reservoir bag is emptying too quickly/not re-inflating quickly enough, usually the problem is one of four things:

- Inadequate/absent oxygen flow rate
- Poor seal around patient's airway
- APL valve is in the fully open position
- Rarely, a crack/hole is present in the C-Circuit

In a spontaneously breathing patient, one can see the bag inflate/deflate as the patient breaths. By adjusting closing the APL valve, one can give the patient some positive-end-expiratory pressure (PEEP) to help keep their airways open in expiration.

Advantages:
- Can be used in spontaneously patients as well as for positive pressure ventilation
- The "feel" of the bag can be used to gauge lung compliance
- The APL valve can be used to alter the amount of pressure used to inflate the lungs
- The spontaneous inflation/deflation of the bag can be used to detect a patient breathing for themselves

Disadvantages
- Will not function without an oxygen supply
- Not as readily available as a BVM circuit

Supra-Glottic Airway

Supraglottic airways refer to a group of airway devices that sit over the glottis (the opening into the trachea) and allow both positive pressure ventilation and spontaneous ventilation depending on patient condition. They are suitable for patient who are deeply comatose/have no airway reflexes.

There are multiple makes of supraglottic airways from different manufacturers. Common makes include Laryngeal Mask Airway (LMA), Proseal LMA and iGel.

The main components are:

- A soft mask which sits over the glottis and provides a seal
- An airway tube which connects to a gas-supply

Depending on the model, there may be other modifications to the device such as:

- A gastric port to allow suctioning/bypass for gastric contents/air
- An inflatable cuff to provide a better seal
- A bite-block to prevent occlusion of the airway channel
- A separate oxygenation port for passive oxygenation

Supraglottic airways sizing is based on the patient's weight. However, if the patient's is over/under-weight, the sizing should take into account the patient's ideal body weight as well.

Insertion of the supraglottic airway, involves opening the patient's mouth and inserting the lubricated device over the tongue and down the throat until the mask sits easily in the patient's throat. If resistance is felt, rotation of the device can sometimes aid insertion. Other causes of difficulty of insertion include:

• The patient have a present gag reflex

• Using too large an airway

• Lack of lubricant

• Anatomical abnormalities prevent insertion

Supraglottic airways can be used to give positive-pressure to ventilate the patient. However care must be taken as too high a pressure can cause air to leak around the mask due to the high pressure overcoming the seal. This air can either audibly leak out the patient's mouth or over-inflate the stomach which may cause the patient to vomit.

Supraglottic airways are not definitive airways as they do not provide reliable airway protection from aspiration. However, they do provide a certain level of airway protection by way of the mask sitting over the glottis. Certain modifications to the airway such as a gastric port are designed to further decrease the risk of aspiration.

Advantages:

• Relatively easy to insert

• Can be used in spontaneously patients as well as for positive pressure ventilation

• Provide an element of airway pressure

Disadvantage:

• Not a definitive airway

• Can easily dislodge

• Patient needs to be deeply unconscious to tolerate

• May not be able to adequately ventilate a patient with poor lung function

Endotracheal (ET) Tube

An ET tube is an airway device that is inserted into the trachea. It is an example of a definitive airway (defined as "an inflated cuffed tube in the trachea"). There are mul-

tiple types of ET tube but the standard oral ET tube consists of:

• A plastic tube

• An inflatable cuff with a pilot cuff

• A connector for a ventilator device

The end of the tube with the inflatable cuff is inserted through the vocal cords, usually under direct vision using a laryngoscope. When the tube and cuff are past the cords, the cuff is inflated to provide a seal. This seal prevents leakage of secretions down the trachea and also prevents air leaking around the tube when the patient is ventilated.

Sizing of the ET tube is done according to age. The formula Age (years) /4 + 4 is the most widely accepted for sizing children. Adult females usually take size 7 – 7.5 ET tubes while adult males usually take 8-8.5 ET tubes

There are multiple pitfalls that can happen when inserting an ET tube:

• Being unable to insert an ET tube into the trachea

• Inserting the ET tube into the oesophagus and not the trachea (fatal if not recognised)

• Inserting the ET tube too far down into one lung

• Not inserting the ET tube far enough and the tube dislodging

Insertion of both the laryngoscope and/or ET tube into the mouth is extremely stimulating to a patient. Hence, to tolerate insertion the patient will either have to be:

• In cardiac/severe respiratory arrest

• Anaesthetised +/- Paralysed

• Deeply comatose

• Have local anaesthetic applied to their airway

Advantages:

• Is a definitive airway that protects from aspiration

• Can ventilate the patient effectively with high airways pressures

Disadvantages

• Takes skill to insert safely

• Usually requires an anaesthetic to allow insertion

• Can be sometimes difficult/impossible to insert into the patient (i.e. difficult intubation)

Laryngoscope

A laryngoscope is a device that facilitates insertion of an ET tube. There are multiple makes of laryngoscopes in use but the most common types in use are:

• Mackintosh/"Mac" blade (The most common laryngoscope with a curved blade)

• Miller (A straight blade laryngoscope used commonly in paediatrics)

• McCoy (Curved blade with hinged tip used sometimes in difficult intubation)

McCoy laryngoscope blade

The laryngoscopes consist of three main parts

• A handle

• A blade

• A light source

The handle is held in the left hand while the blade in inserted into the mouth to the right of the tongue. The tongue is scooped to the left and the blade is advanced for-

ward until the epiglottis is visualised. By lifting up with the laryngoscope barrel, the vocal cords can be further visualised. Once the vocal cords are visualised, an ET tube can be inserted through.

Avoid the temptation to lever back on the laryngoscope in the mouth in order to improve the view as this can damage the patient's teeth and actually make the view of the vocal cords worse.

Bougies & Intubating Stylets

"Eschmann tracheal tube introducer" is more commonly referred to as a "Gum-Elastic Bougie." A bougie is effectively a long thin plastic (traditionally hard rubber) tube that can be shaped to allow easy insertion into a person trachea if difficulty is encountered while inserting an ET tube due to difficult patient anatomy.

By shaping the tip of the bougie, it can allow the bougie pass underneath the epiglottis/negotiate difficult angles of insertion.

To use the bougie, perform laryngoscopy and insert the bougie either through the vocal cords or blindly underneath the epiglottis. Signs of successful insertion of the bougie into the trachea include feeling for clicks against the tracheal rings or hold up of the bougie at the bronchial tree.

Once in place, maintain your view of laryngoscopy and ask your assistant to insert an ET tube over the end of the bougie. Railroad the ET tube over the bougie into the trachea and then ask your assistant to remove the bougie. If resistance is felt on trying to pass the ET tube over the bougie, often withdrawing the ET tube and rotating counter clockwise before reinserting again in successful.

Intubating with a bougie

Respiration

Introduction

Supporting ventilation is our primary means of controlling acid-base homeostasis. Patho-physiology of acute lung injury and failure of ventilation is multifactorial. Bruising of the lung results from passage of a shock wave through the tissue. Microscopic disruption occurs at any air-tissue interface, of which the lungs have plenty.

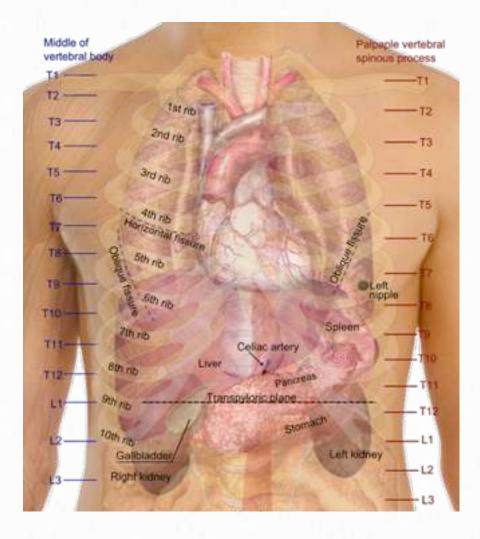**A key goal in supporting oxygenation and ventilation, is not to make matters worse.**

In 2002, CHEST published a "Top 10 list in Mechanical Ventilation." Whilst not new, the concept of lung protective ventilation strategies have became progressively more main-stream.

ATACC advocate a stepwise approach to Oxygenation and Ventilation, constantly mindful that immediate restoration of "normality" may be to the long term detriment.

Mechanism of Injury

The anatomy of the thoracic cage is well covered in numerous anatomy texts but it is worth remembering that when examining the chest it has a front, back, two sides, an upper limit including the root of neck and shoulder girdle and a lower limit that overlaps with the upper abdominal organs. Function depends on integrity of both the boney thoracic skeleton and the organs within.

Adequate oxygenation and ventilation depends on an intact chest wall. Ribs 1-3 are relatively rarely fractured because of the protection provided by the upper limb girdle, unless there is also a lower cervical spine injury. Their fracture, along with those of the scapula and sternum require high-energy transfer and suggest a severe injury involving the head and neck. Ribs 4-9 are most commonly injured and 10-12 fractures may involve the underlying liver and spleen.

Significant injury with fracture and muscular disruption may allow direct injury to the underlying lungs, heart, great vessels and upper abdominal viscera. In addition, respiration may be seriously impaired by ineffective or paradoxical motion of a portion of the thoracic cage (as in flail chest) and the result is respiratory insufficiency leading to failure. It is worth bearing in mind that the elasticity of the thoracic skeleton changes with age and degree of ossification from the easily deformed but rarely broken ribs of children to the more rigid brittle bones of the elderly.

Fractured ribs in a child indicate a high energy transfer and a potentially serious injury but a lack of fractures does not rule

out significant intra-thoracic injury. In the elderly a simple low energy fall may result in multiple displaced rib fractures and significant disruption of thoracic function.

In pregnancy, more of the abdominal contents are beneath the front margin of the thoracic rib cage

Penetrating wounds of the chest (gunshot or stab wound) may cause comminuted fractures of a rib, with bone fragments driven into the lung substance. The most common manifestation of penetrating trauma to the visceral and parietal pleura is disruption of normal negative intrapleural pressure resulting in pneumothorax. Penetrating wounds cause both direct injury to structures encountered by the weapon and indirect injury. The amount of indirect tissue damage remote from the track of a penetrating object depends on the energy transfer from the object to the tissues as it traverses the tissue. High levels of energy transfer can cause damage at a significant distance from the track. Therefore the extent of internal injuries cannot be judged by the appearance of a skin wound.

Blunt forces applied to the chest wall cause injury by three mechanisms:
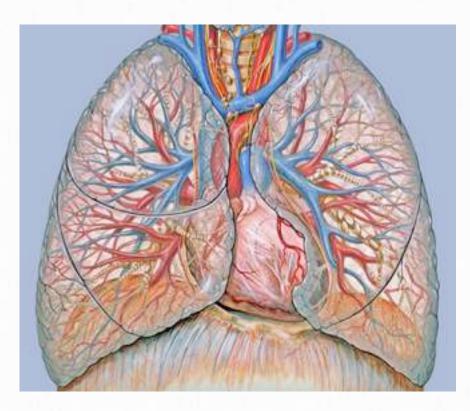
- Rapid deceleration
- Direct impact
- Compression.

Rapid deceleration is the usual force involved in high-speed motor vehicle accidents and falls from a height. The degree of external trauma may not fully predict the severity of internal injuries and clinical suspicion of cardiac and vascular trauma should be heightened.

Direct impact by a blunt object can cause localised fractures of the ribs, sternum or scapula with underlying lung parenchyma injury, cardiac contusion or pneumothorax. Sudden dynamic anterior-posterior compression forces place indirect pressure on the ribs, causing lateral, mid-shaft fractures. Lateral compression forces applied to the shoulder are common causes of sternoclavicular joint dislocation and clavicle fractures. Massive blunt injury to the chest wall may comprise elements of deceleration, direct impact, and dynamic compression to yield multiple adjacent rib fractures. In this setting, a free-floating segment of the chest wall can move paradoxically with respiration causing ineffective ventilation.

Continued static compression of the chest by a very heavy object, which prevents respiration, causes marked increases in pres-

sure within veins of the upper thorax. Not only will it result in worsening respiratory acidosis, it may result in traumatic asphyxia. Compression of the chest in entrapped patients, requires urgent removal of the entrapping force before any further steps in the extrication evolution take place.

As well as respiratory insufficiency thoracic trauma may cause haemorrhagic shock due to haemothorax and rarely haemomediastinum. Haemothorax is common in both penetrating and non-penetrating injures to the chest. If the haemorrhage is severe, it may cause not only hypovolaemic shock but also dangerously reduced vital capacity by compressing the lung on the involved side.

Persistent haemorrhage usually arises from an intercostal or internal thoracic (internal mammary) artery and less frequently from the major hilar vessels. Bleeding from the lung generally stops within a few minutes, although initially it may be profuse. In some cases haemothorax may come from a wound to the heart or from abdominal structures such as the liver or spleen if the diaphragm has been lacerated. The thoracic cavity is far more vascular than you think.
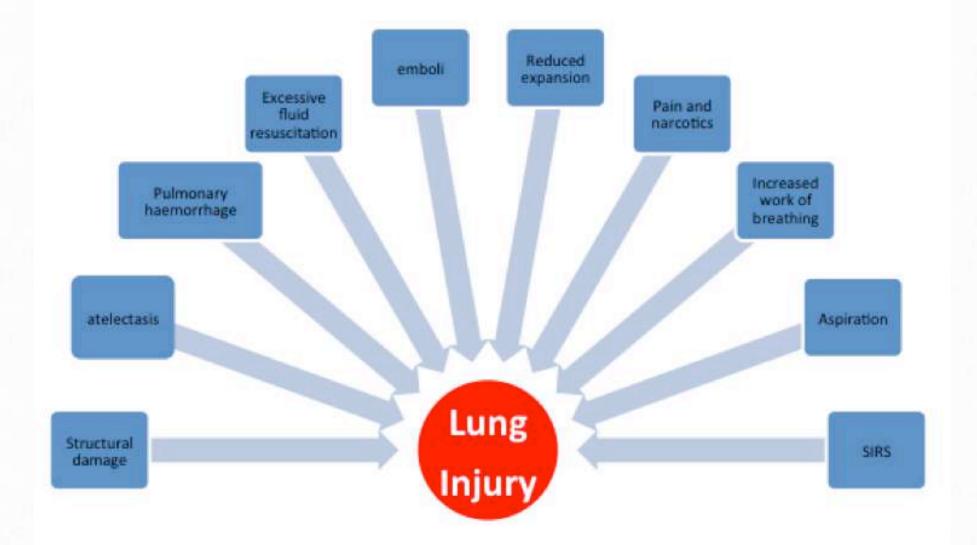

In addition, hypovolaemic shock and haemomediastinum can derive from thoracic great vessel injury that may be result of penetrating or blunt trauma. The most common aetiology is from penetrating trauma; however, the descending thoracic aorta, the innominate artery, the pulmonary veins, and the vena cava are all susceptible to deceleration injury and rupture from blunt trauma.

Pulmonary Contusion

Pulmonary Contusion occurs in 30–75% of severe chest injuries. With an estimated mortality rate of 14–40%. Bruising of the lung results from passage of a shock wave through the tissue. Microscopic disruption occurs at any air-tissue interface where energy is dissipated (of which the lungs have

Pathophysiology of
Acute Lung Injury

plenty). Injuries involving rapid high energy transfer rather than slow crushing are more likely to cause pulmonary contusion.

The concussive loss of vessel integrity results in intraparenchymal and alveolar haemorrhage, decreased pulmonary compliance and increased shunt fraction. Pulmonary contusion frequently manifests itself as hypoxaemia and dyspnoea but this may develop over the first 24-48 hours. 50-60% of patients with significant pulmonary contusions will develop bilateral Acute Respiratory Distress Syndrome (ARDS).

There are few immediate clinical signs. Early chest x-ray is likely to be non-diagnostic. Pulmonary contusion presents as a diffuse non-segmental airspace consolidation on the

antero-posterior chest radiograph. Spiral CT is more sensitive than chest X-ray for picking up early pulmonary contusions. Haemorrhage into the tracheobronchial tree occurs in approximately 10% of patients with pulmonary contusion.

Lung function in trauma becomes impaired primarily by direct parenchymal damage (from blood or tissue disruption) resulting in activation of systemic inflammation that causes alveolar capillary barrier dysfunction and increases extravascular lung water. All these pathophysiological events, in addition to excessive bronchial sections and inability to adequately clear them through pain suppressed cough, can promote alveolar collapse, ventilation perfusion mismatch, lung consolidation and lung infections.

The incidence of post-traumatic Acute Respiratory Distress Syndrome (ARDS) is quoted between 8% and 37%. The highest percentage occurs between 48 and 72 hours post injury. The associated mortality reported range between 16% to 29%.

Management

The goals of treatment are oxygen therapy, positive pressure either with a CPAP (Continuous Positive Airway Pressure) or BiPAP (Bilevel Positive Airways Pressure) mask or by intubation and mechanical ventilation with PEEP (Positive End Expiratory Pressure). Splinting from the pain associated with rib fractures requires aggressive pain management, i.e. parenteral narcotics, intrapleural local anaesthetics or epidural narcotics/local anaesthetics. The contused lung is prone to capillary leak and therefore careful fluid management is suggested, but other treatment modalities for pulmonary contusion such as fluid restriction, diuretics and corticosteroids have not shown any proven benefit. The main method of management of haemorrhage into the tracheobronchial tree is frequent tracheal tube aspiration. Lung isolation procedures may be required in 33% of patients with airway bleeding. Positive end expiratory pressure will improve hypoxaemia by alveolar recruitment but does not change the underlying contusion. Rotational/kinetic therapy beds may improve oxygenation and aid clearance of secretions.

Flail Chest

A flail chest occurs when there are two or more adjacent ribs fractured in two or more places, to result in a free or floating segment of chest wall. The presence of the classical paradoxical motion of the chest wall depends on the location of the fractures. Fractures in the lateral or anterior regions of the chest wall are relatively poorly

supported. As a result, during spontaneous ventilation the floating segment moves in the opposite direction or paradoxically to the rest of the chest wall. Posterior and postero-lateral fractures are better supported by surrounding muscle and may not exhibit this paradoxical movement. Flail chest, therefore, is a purely clinical diagnosis that can only be made during spontaneous ventilation. Confirmation of a flail segment is made on the x-ray. The incidence of flail chest in patients with blunt thoracic trauma presenting to the operating room is 23.1%. Because of the pliant nature of a child's ribs, flail chest is rarely seen in the paediatric population. Flail chest is almost always associated with an underlying pulmonary contusion because of the high-energy transfer needed to fracture multiple ribs. Splinting of respiration from pain inhibits ventilation, the underlying pulmonary contusion compromises oxygenation by intrapulmonary shunting and increased work of breathing and haemothorax from multiple fractured ribs contributes to hypovolaemia.

Assessment

Using a standard "medical assessment" of the respiratory system in trauma will miss significant life threatening injuries. Respiratory assessment in trauma is notoriously skimmed over and frequently lacking in suf-

ficient detail to be of any real benefit. "Vital signs" are notoriously unreliable. Just as in the circulatory assessment in the following chapter, ATACC recommended to separate out, and clearly document both a "subjective" as well as "objective" findings, leaning your decision making towards more evidenced based practice.

Subjective

As part of a subjective assessment, ATACC have a very thorough pneumonic for respiratory assessment in the trauma patient, RU IN SHAPE?

- **R**espiratory Rate

- **U**nequal Movements

- **IN**juries

- **S**earch

- **H**ands on - feel

- **A**uscultate

- **P**ercuss

- **E**verywhere

Regardless of your preferred method of respiratory assessment in trauma, a careful physical assessment of the ventilatory function of the thoracic trauma victim should include inspection for respiratory rate, pres-

ence of paradoxical or unequal movement of the chest wall and obvious chest injuries.

Palpation of the chest should identify pain, crepitus or subcutaneous emphysema as clues to underlying pathology. Be thorough, search everywhere using your hands, not forgetting the axilla, neck and back.

Auscultate literally means listen! Do not just rely on a stethoscope, listen to the patient speak, listen for sucking wounds, listen to the work of breathing. The auscultate of the lung fields may detect a pneumothorax or haemothorax, but is notoriously subjective.

Percussion, although theoretically of use in differentiating between pneumothorax and haemothorax, may be practically difficult in the atmosphere surrounding a typical resuscitation bay. Do ask for silence when examining the chest.

When you've finished, ask yourself the question… Have I covered everywhere? Frequently missed is the back of the thorax. If not performing a log role for haemodynamic reasons, if you've missed a part of the chest examination, be sure to document this and come back to the area.

Objective

As Part of an objective assessment, ATACC advocate that following observations be recorded and trends monitored throughout the patients journey:

• pH

• SpO2

• ETCO2

• Tidal Volume (Vte)

• Minute Volume (MVe)

Ultrasound has an increasing role in the assessment of Thoracic Injuries. It has a similar diagnostic yield to CT for ruling out Pneumothorax and is very useful as a rapid check for pleural fluid in the right hands. The presence of a sliding pleural sign and B-Lines (or Comet Tail) effectively rule out a Pneumothorax on that view. They extend from pleural line and reach the distal edge of screen. On M-Mode, a "Seashore sign" further confirms a pneumothorax rule out, whilst a "Stratosphere sign" is highly suggestive of a pneumothorax. Be sure to cover all areas using a standardised technique. Pleural fluid is obedient to the law of gravity, so pleural fluid will collect in the dependent portion of the thorax (unless loculated).

CT Scanning remains the gold standard of objective thoracic injury assessment.

Immediately Life Threatening

A useful mnemonic for 6 immediately life threatening injuries is:

All Trauma Consultants Occasionally Miss Fractures.

- All: Airway obstruction (See Airway Section)
- Trauma: Tension Pneumothorax
- Consultants: Cardiac tamponade (See Circulation/Shock Section)
- Occasionally: Open chest wound
- Miss: Massive Haemothorax
- Fractures: Flail chest

These six pathologies will prove fatal if not addressed and controlled rapidly.

Respiratory Support Ladder

ATACC's concept of a Respiratory Support Ladder in Trauma is novel. It Promotes a logical stepwise approach to ramping up of care from Roadside to Critical Care as opposed to a pathology based approach. We purposefully separate initial resuscitative respiratory management from that of managing Thoracic Trauma, which we've included in the Specialist Care section of this manual.

The Respiratory Support Ladder:

- Rescue
- Positioning
- Analgesia
- Oxygen
- Non-Invasive Ventilation
- Chest Decompression
- Positive Pressure Ventilation
- Oscillation
- Interventional Lung Assist
- ECMO

Rescue

Fire and Rescue personnel play a key role in respiratory care, but frequently don't appreciate the importance of the extrication evolution in supporting respiratory physiology.

The physical rescue of a patient could be our primary means of controlling acid-base homeostasis.

If an entrapping force is physically restricting chest wall movements, it is obvious, but even, patients who are unable to ambulate are at potential risk of ventilatory fail-

ure, depending on how they are positioned. Body habitus contributes significantly to risk, particularly centrally obese or indeed pregnant patients. With diminished tidal volumes, carbon dioxide levels (P_aCO_2) can rise, effecting a respiratory acidosis and a detrimental chain of events.

One of the first considerations in an extrication evolution is the ability to achieve the "B plan", or route of immediate extrication, if a patient deteriorates. Part of this process is both space creation, and removal of any physical entrapping force. This early respiratory intervention in the extrication evolution is potentially life saving and its urgency should be communicated as soon as it is discovered to any Officer in Charge of an extrication.

Positioning

Patients will always attempt to position themselves in the most ergonomic posture to effectively ventilate. In trauma management, we frequently immobilise patients flat on a board, with chest straps, which is counterintuitive in the presence of any chest pathology, pre-existing medical condition, obesity or pregnancy.

See section on Clearing the Spine with regards to early spinal clearance. If at all possible, always attempt to sit a patient up in their most comfortable position to breathe effectively. Just as airway takes ultimate priority over over cervical spine protection, impending or actual ventilatory failure needs to take priority over immobilisation. If the spine can not be cleared, a number of options exist:

- Neutrally immobilise the patient, using a vacuum mattress, in a semi-recumbent position. Particularly if no lumbar spine injury is present or suspected.

- Place the scoop or longboard in a head up position, but be cautious of orthostatic hypotension.

- Progressively move up the Respiratory Support Ladder

Rib Fractures

The most common blunt thoracic injury in both adult and paediatric age groups is rib fractures. The incidence of fractured ribs in patients presenting to the operating room for emergency trauma surgery is 67.3%. Rib fractures are frequently associated with other injuries such as flail chest, pulmonary contusion and pneumothorax. Unmanaged pain will lead to atelectasis and secretion retention. The Journal of Trauma reported in 1994 that multiple rip fractures were associated with pulmonary complica-

tions in more than 1/3 of cases and pneumonia in as many as 30% of cases. Pain management is frequently the primary means of preventing deterioration and controlling acid-base homeostasis.

In conscious patients fractured ribs can be easily detected with physical examination. Chest radiography is extremely unreliable in detecting fractures, with CT being the gold standard if there is any concern regarding associated injuries. The only intraoperative concern regarding rib fractures is that a fractured rib edge may cause a pneumothorax when the patient is placed on positive pressure ventilation after the induction of anaesthesia. Most anaesthetists would feel safer with the placement of a prophylactic chest tube in patients with fractured ribs undergoing anaesthesia with positive pressure ventilation.

Analgesic Options

See Thoracic Analgesia Section of this manual for more detail.

- Simple analgesia (Paracetamol)

  Note Paracetamol is now available in a licensed i.v. preparation for management of acute pain.

-

- NSAIDS

  Care needed with renal function in hypovolaemia but very effective if normovolaemic with no ongoing bleeding

- Opiates (Oral, Parenteral, OTM, IN)

  Traditionally gold standard analgesia in trauma but best in conjunction with other techniques if possible as can contribute to hypoventilation

  Prehospital, in the awake and coherent patient, patient controlled analgesia with Fentanyl lollipops are very effective.

- Regional Analgesia

  - Intercostal Block

  - Intrapleural Block

  - Paravertebral Block

  - Thoracic Epidural (+/- opiate)

- Low dose Ketamine infusion. (22)

  0.05-0.1mg/kg i.v. loading dose followed by 0.05-0.2mg/kg/hr infusion in a monitored "HDU" environment. Confusion or agitation should be managed by reducing the dose of the infusion .Very good in combination with any or all of the above techniques

Oxygen

Under normal circumstances, a person's body can operate efficiently using the amount of oxygen found in normal room air. However, the amount of blood lost after a traumatic injury could mean that insufficient oxygen is delivered to the cells of the body, which can result in shock, death, or serious long term disability. Administering supplemental oxygen to a casualty increases the amount of oxygen delivered to the cells of the body and often makes a positive difference to the casualty's outcome.

Simple face mask oxygen should be used to treat hypoxaemia. A pulse oximeter should be used to ascertain the blood oxygen saturation level. Most commonly the pulse and oxygen saturation will be recorded, but newer models may provide other information as well such as carboxyhaemoglobin.

Oxygen is now viewed as a drug and must be prescribed appropriately by a clinician. A generic prescription or SOP/SIS can be written by a Medical Director for emergency care providers who have been suitably trained.

There is often some confusion produced by the British Thoracic Society Guidelines about oxygen administration but in major trauma our guidelines are very clear:

All casualties suffering major trauma should receive high levels of supplemental oxygen through a reservoir mask at a rate of 10 to 15 L/min.

As the casualty becomes stable, you may consider reducing the oxygen flow rate and aim for a target saturation of between 94–98% in the average adult casualty, but if in doubt, then the higher flow rate should be used.

The oxygen saturation is considered 'Time critical' if below 92% on air or 94% on supplementary oxygen.

Prehospital Oxygen Equipment

Oxygen is compressed and stored in portable cylinders. Standard oxygen cylinders are filled to 137 barometers, newer Kevlar wrapped lightweight cylinders are charged to 200 to 300 bar pressure, depending on the manufacturer or supplier. In older style cylinders, a gasket is used between the pressure regulator/flowmeter and the cylinder; this ensures a tight seal and maintains the high pressure inside the cylinder). In the new style cylinders, the pressure regulator and flowmeter are an integral part of the cylinder apparatus and no assembly is needed prior to oxygen administration.

All oxygen supplies should be checked on each shift, including contents, regulator valves, and flow meters. For cylinders with a separate regulator, if it is necessary to change the regulator, ensure that the valves are free of grease, oil, and dust. Before fitting the regulator, the cylinder should be opened slightly for a few seconds to clear any dust or debris from the outlet port.

Oxygen does not burn or explode by itself. However, it can quickly turn a small spark or flame into a serious fire. Therefore, all sparks, heat, flames, and oily substances must be kept away from oxygen equipment. Smoking should never be permitted around oxygen equipment.

The pressurised cylinders are also hazardous because the high pressure in an oxygen cylinder can cause an explosion if the cylinder is damaged. Oxygen cylinders should be stowed securely to protect the cylinder and regulator/flowmeter. At the scene of an incident, ensure the cylinder is handled carefully and not exposed to any damage during an extrication attempt.

It is important to note the amount of oxygen remaining in the cylinder to ensure that it will not run out while treating a casualty. A normal portable oxygen cylinder contains between 340 and 1,000 L. Responders should monitor the gauge and seek additional cylinders before they are needed. If further cylinders are not available, then providers may reduce the flow rate to 10 L/min (or less), though this will significantly reduce the percentage of oxygen being delivered to the casualty. In this case, the casualty should be monitored with a pulse oximeter if one is available to ensure that the oxygen saturation is kept greater than 90%.

Non-Invasive Ventilation

A recent systematic review and meta-analysis of noninvasive ventilation in chest trauma found that Non Invasive Ventilation (NIV) significantly increased arterial oxygenation and was associated with a significant reduction in intubation rate; reducing

the incidence of overall complications and infections.

ATACC advocate the careful use of NIV in the management of acute respiratory failure due to chest trauma.

NIV is not without its risks and complications. The key to success is being patient focused and not simply treating NIV as a prescription:

Define clinical goals
- What constitutes NIV failure in this patient? Document clearly and hand over to all staff when intubation and IPPV need to be considered BEFORE you start NIV.

- Never wait until the patient is critically unwell.

- What are we defining as success? Ie. When do we expect the patient to no longer require NIV.

Analgesia is paramount to success
- If the patient is not entirely comfortable they will not settle and you will fail.

- Anxiolytics are a poor excuse for adequate analgesia and simply increase risk of complications

Rule out contraindications before you begin

Absolute
- Non-compliant patient

- Apnoea

- Haemodynamic Instability

- Inability to protect own airway

- Significant altered mental status

- Pneumothorax

- Gastric, laryngeal or oesophageal injury

- Significant facial fractures (in particular those involving the cribriform plate)

- Inadequate staff to closely monitor the patient.

Relative
- Nausea and vomiting

- Agitation

- Cardiac Injury, ischaemia or arrhythmia.

Start small - aim small
- NIV can be incredibly uncomfortable and frightening.

- Start with very basic settings which genuinely provide comfortable support. Let the patient become used to the mask and sounds first.

- Increase support in very small increments. NIV to work well takes more staff time than intubation and ICU admission!

- Concentrate on the support before you ramp up the PEEP.

- Permissive hypercapnoea needs to be considered, i.e. small wins for longterm gain. Don't just go for normal numbers

Decompress the gut
- A fine bore NG tube to prevent uncomfortable gastric distension should be passed early.

Know your delivery system
- Face-masks are not all the same.

- Tightly fitting masks often leak more!! Modern masks are designed to "float" on a cushion of air and move with facial movements accepting a small leak. They will not work if forced into the face.

- Consider full hoods. Many patients find these much easier.

- Consider new modalities such as high flow nasal CPAP systems

Know your ventilator!
- Ensure the ventilator is in spontaneous mode!

- Know where the ramp settings are and match the machine to the patients respiratory efforts (i.e. not the other way around!)

- Where is the trigger? Set it too sensitively and every movement will trigger a support breath, the patient will start to fight the ventilator. Set it too high and the patient will feel like its suffocating them.

- On most machines a respiratory rate (V_f) can be set to guarantee breaths delivered if the patients efforts fall below a set frequency. Be careful that this is not set above the patients own, settled ventilatory rate. Use this as an emergency setting only.

- Concentrate on providing support (IPAP) before you provide PEEP. PEEP feels very suffocating at first. Provide oxygen (FiO_2) first, then slowly increase the PEEP to reduce your FiO_2 requirements over time.

- Understand the alarms. If a machine is alarming constantly - sort it out, YOU are doing something wrong! It's unlikely to 'settle down as the patient settles down.'

- Humidification of inhaled gases is important if the integrity and function of the respiratory tract is to be maintained and various options exist to humidify gases in non-invasive circuits.

NIV terminology

CPAP (Continuous positive airways pressure):

- The provision of positive airway pressure throughout all phases of spontaneous ventilation.

- This increases the functional residual capacity of the lungs by holding airways open and preventing collapse. The application of CPAP also causes the patient to breathe at higher lung volumes, making the lungs more compliant.

- CPAP is particularly useful for improving oxygenation.

Inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP):

- These terms are commonly used in reference to BiPAP. IPAP is the pressure set to support the patient on inspiration. EPAP is the pressure set for the period of expiration.

- The actual airway pressure during inspiration is independent from the expiratory airway pressure. For example, BiPAP ventilation using IPAP 15 and EPAP 8 is equivalent to conventional pressure support delivering pressure support of 7 above PEEP of 8.

Bi-level positive airway pressure (BiPAP©):

- This is the trade name for the machines most commonly used in the UK to provide two levels of airway pressure. Positive pressure is maintained throughout the respiratory cycle, with a higher pressure during inspiration.

- BiPAP results in reduced work of breathing and an improvement in tidal volume and CO_2 removal; it is therefore particularly useful in the treatment of type 2 respiratory failure.

- Spontaneous modes are similar to use of pressure support ventilation (or assisted spontaneous breathing) on invasive ventilators, whereas timed modes are analogous to conventional mandatory ventilation.

- Note that bi-phasic positive airways pressure (BIPAP) is different to BiPAP and less commonly encountered. The patient breathes at a preset level of CPAP and at timed intervals (not synchronised to the patient's inspiratory efforts) the level of CPAP is reduced to a lower level. The intermittent reduction in CPAP leads to a large expiration and therefore increases CO_2 elimination.

Chest Decompression

A pneumothorax is an abnormal collection of air or gas in the pleural space that separates the lung from the chest wall. Any pleural injury communicating with the atmosphere will lead to a pneumothorax. Pneumothorax in blunt trauma may result from alveolar rupture with perivascular spread of gas to the hilum and connection of a distal airway to the pleural space. More usually the air leak is secondary to laceration or puncture of the visceral pleura by bone fragments of fractured ribs. Large volume continued air leak in the context of a pneumothorax represents major laceration of lung parenchyma or damage to major airways.

Chest Decompression does not fit exactly into any one particular rung in the ventilatory support ladder. However it needs to be discussed at this point when we are considering artificially ventilating patients.

A review article in the EMJ in 2005, highlighted significant misunderstandings in our knowledge and common misconceptions in our current thinking of how to manage a pneumothorax, or air artificially in the pleural cavity. The "classic signs" of life threatening tensioning of a pneumothorax, on review of the literature, are actually not that classic.

There are multiple case reports of diagnostic difficulty or missed diagnosis because of an absence of "classic signs". In addition, the management of the spontaneous pneumothorax is fundamentally different from that of a traumatic pneumothorax and the two are frequently misinterpreted as a single entity.

There are two distinctly different patient populations that need to be considered as separate entities:

- The Spontaneously Ventilating.

- Artificially Ventilated.

The Haemothorax is a pathology distinct from the pneumothorax, independent of ventilation mode, and is considered as a third subset of patients in this chest decompression section of the manual.

Pneumothorax - Spontaneously Ventilating

Under normal physiological conditions:

- Transpulmonary pressure (difference between alveolar pressure and intrapleural pressure) is always positive.

- Intrapleural pressure (pressure within the chest cavity between the lungs and the chest wall) is always negative and relative large

- Alveolar pressure (pressure within the air spaces of the lungs themselves) moves from slightly positive to slightly negative as a person breathes.

- The intrapleural pressure ranges between -5 to -8 cm H_2O.In order for air to enter artificially into the pleural space, a one-way valve or communication must exist. Any pleural injury communicating with the atmosphere via a one way valve that opens on inspiration and closes on expiration will lead to an expanding pneumothorax. This initially causes an increase in intrapleural volume which augmented by subsequent raised intrapleural pressure causes ipsilateral lung collapse and mediastinal displacement to the opposite side. Pulmonary collapse results in hypoventilation and hypoxaemia.

- When a patients lung (or indeed lungs) completely collapse within an air filled pleural cavity, a pneumothorax will begin to tension, ultimately accumulating in positive intrapleural pressures throughout the respiratory cycle where a patient literally is unable to generate a negative pressure to inspire, and thus suffers complete and very sudden ventilatory failure. **Self-ventilating patients tension slowly** within but a tension pneumothorax (i.e. positive intrapleural pressures through

the respiratory cycle) is still very much a final and fatal event.

Universal 99%	Common 50-75%	Occasional <25%	Rare <10%
Chest Pain	Tachycardia	↓ SpO2	Cyanosis Hyper-resonance
Respiratory Distress	Ipsilateral ↓ AE	Tracheal deviation	Hyper-expansion Hypo-mobility
		↓ BP	Cardiac apical displacement
			Sternal resonance epigastric pain

Signs and Symptoms of a Tension Pneumothorax. **From Leigh-Smith S et al. Tension Pneumothorax – time for a rethink? EMJ 2005**

Needle Aspiration

Needle Aspiration of a traumatic pneumothorax will considerably relieve pain and discomfort, and is a useful procedure in settling a patient prior to insertion of a formal tube thoracostomy. Insertion of Intercostal chest drains coveys an 11% empyema risk if performed outwith the sterile environment of an operating theatre. Mindful of what has previously been discussed about the risk of a pneumothorax tensioning, in the vast majority of incidences, aspirating a pneumothorax whilst waiting to move a patient to a sterile environment for insertion of a formal intercostal drain is logical.

The approach is to insert at least an 8cm needle attached to a syringe with a bit of normal saline in it, in the second intercostal space in the mid-clavicular line. Aspirat-

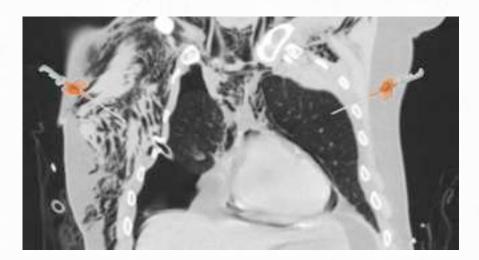ing whilst advancing until air begins to bubble through the saline.

Needle aspiration or needle thoracocentesis must be followed by a formal tube thoracostomy even if there is no evidence of air rushing out on first insertion of the needle. If the needle has hit lung tissue, a simple pneumothorax would have been created, which may tension under IPPV.

The diminishing role of Needle Thoracocentesis

Needle thoracocentesis has a 40%-50% failure rate. It still has a limited place in the emergency management of decompensating patients but mindful of this failure rate and associated morbidity and should not be used indiscriminately. Morbidity includes laceration of the lung, haemorrhage, cardiac tamponade and infection.

If you have suspect a tensioning pneumothorax then do not wait for a chest X-ray to confirm this diagnosis, a rapid release of air on needle thoracocentesis confirms the diagnosis. In case of thoracocentesis with a large calibre cannula, try and slide the plastic cannula in (this can be very difficult).

The approach is identical to needle aspiration in the second intercostal space in the mid-clavicular line, but involves the insertion of a cannula into the pleural space and leaving it in place until a formal chest drain can be secured. Bear in mind that the cannula may kink or not even reach the pleural space in large patients. A standard 14G i.v. cannula is only 4.5cm in length and there is good evidence that to reliably reach the pleura in the average male, you require an 8cm needle. Other reasons for failure of this technique include obstruction of the cannula with blood or tissue, inadequate calibre of the cannula and failure to hit a localised pneumothorax.

Tube Thoracostomy

This must be a carefully planned and very controlled procedure, which will be taught on the course. It must be performed with full universal infection control procedures. It carries between a 1% and 25% Empyema risk if performed in the Emergency
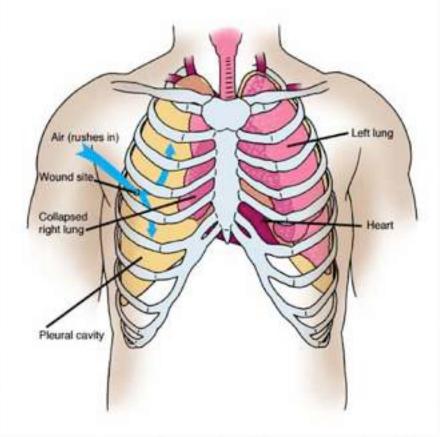
Department with current universal precautions.

What is clear that there is no justification for rushing to put in a chest drain, especially in a spontaneously ventilating patient, unless there is an emergent need for RSI, and even then, a finger thoracostomy post RSI is a far less risky procedure from an infection control perspective.

Open Chest Wound - Spontaneously Ventilating

This automatically produces a pneumothorax on the side of the injury. If it is a simple, non-sucking wound in a spontaneously ventilating patient, ATACC recommend completely sealing the wound and monitoring the patient for signs and symptoms of a tensioning pneumothorax.

In Spontaneously Ventilating patients - Seal and Monitor small wounds

Large Open Chest Wound - Spontaneously Ventilating

If the size of the wound is greater than the ⅔ tracheal diameter, air preferentially enters the chest through the chest wound in inspiration. This eventually leads to lung collapse. Whilst there will be respiratory compromise, it will not tension unless the hole is sealed, because during expiration, air will preferentially move out of this wound.

In a spontaneously ventilating patient - Apply a valved chest seal to large wounds

Chest Seals

If available, a special dressing for this type of injury, known as chest seal, should be applied.

- Assess the chest wall and identify any open wounds (front, sides or back)

- Open the chest seal dressing pack

- Dry the area around the hole with the gauze swab in the pack to give the dressing the best chance to stick to the skin around the wound

- Peel off backing to expose adhesive side of chest seal and apply over the wound

- Ensure a good seal of the dressing. If it does not fully cover the wound or is not big enough then a sheet of cling-film or a damp saline soaked swab can be used

- Defibrillator pads are very useful to completely occluding large wounds in a wet, chest where nothing else will stick.

If there are multiple wounds or you have no seal then apply an improvised 3 sided dressing:

- Place a square of non-porous material (such as plastic dressing packaging) over the wound.

- Tape the dressing on the top and both sides using adhesive tape

- Leave the bottom of the dressing free, to act as a one-way flap valve and allow any blood to drain out of the chest.

- Completely seal all but one of the holes if they are all sucking (i.e. have one three sided dressing and multiple completely covered four sided dressings)

Pneumothorax - Ventilated Patient

In a ventilated patients by definition, positive ventilatory pressure is artificially created throughout the respiratory cycle. In the presence of a pneumothorax, without any means for this intrapleural air to escape the thoracic cavity, the lung will very rapidly collapse and a tension pneumothorax will rapidly develop.

Artificially Ventilated Patients Rapidly Tension

Universal 99%	Common 50-75%	Inconsistent <20%
↓ Sp02	High Ventilation Pressures	Venous Distension
↓ BP & ↑ HR	Ipsilateral ↓ AE Hyper-expansion Hypo-mobility	Cardiac apical displacement
Surgical Emphysema	Tracheal Deviation	

Signs and Symptoms of a Tension Pneumothorax. Ventilated Patient - Regardless of Chest Wound or Not.
From Leigh-Smith S et al. Tension Pneumothorax – time for a rethink? EMJ 2005

In the ventilated patient the rapid decrease in cardiac output is due to a combination of events including ventricular or great vessel compression, decreased venous return, hypoxaemia and decreased pulmonary blood flow through the collapsed lung. Profound hypotension results and if uncorrected will result in cardiac arrest.

Finger Thoracostomy

If a patient tensions while already receiving IPPV or is in a peri-arrest situation and about to be intubated it is more effective to decompress the chest with simple finger thoracostomy rather than needle decompression.

This is not however a suitable technique for a spontaneously breathing patient for obvious reasons. Whilst the principles of the procedure are the same as the standard technique to place a chest drain there are certain points to note and reinforce: The approach should be via the 4th or 5th intercostal space as when placing a drain. However the presence of surgical emphysema and flail segments can make placement of the incision over the 4th or 5th intercostal space difficult. To ensure safe placement the incision should always be above the nipple line (in males).

Bleeding from the subcutaneous tissues in the axilla would normally be compressed by the drain and sutures. This effect is not present in simple thoracostomy. Blunt dissection is essential to limit the effect of any bleeding. Skin prep and sterile gloves should be used at all times and a drain inserted as soon as is practical.

The hole through intercostal muscles should allow free insertion of a finger without pushing. This may require some of the intercostal muscle being stripped off the rib. This will allow adequate rapid decompression of the chest while the chest drain trolley is still being prepared allowing the lung to re-expand and potentially tamponade off bleeding intercostal vessels. Significant blood loss from a haemothorax should be controlled by occlusion of the thoracostomy with a dressing pad until the drain is available.

This is a very good technique in the pre-hospital setting with a ventilated poly-trauma patient and avoids the problems of drain insertion, displacement or kinking during transport. However thoracostomy in the pre-hospital setting is more difficult with a patient lying on the floor in less than ideal conditions of access, lighting and asepsis. It should only be performed by experienced medical staff comfortable with the procedure and its complications in-hospital. It is not suggested that it should be performed by prehospital paramedic personnel - the complications can be very serious. Pre-hospital thoracostomy should only be performed for the following indications:

Absolute Indications
- Traumatic Arrest or peri-arrest
- Tension Pneumothorax

Relative Indications:
- IPPV + Chest signs to hemithorax
- Reduced air entry
- Surgical emphysema
- Unilateral wheeze
- Bony crepitations (rib fractures)
- Hypoxia or hypotension with Chest signs

Contra-indicated:
- Simple pneumothorax
- Unilateral chest signs without hypoxia or hypotension
- Spontaneously breathing patient

Following a Finger thoracostomy, ATACC advocate sealing the wound with a chest seal. Further evidence of tensioning within the chest may be relieved by insertion of a sterile gloved finger through the thoracostomy.

Open Chest Wound - Ventilated Patient
In ventilated patients, open chest wounds should simply be left uncovered, until surgi-

cal washout and closure can be performed and intercostal chest drain placed.

Haemothorax

Bleeding from intercostal muscle damage or torn intercostal vessels from rib fractures is commonly the cause of simple haemothoraces. Small haemothoraces may expand slowly and become quite significant over several days even with simple uncomplicated rib fractures. Repeat chest X-ray may demonstrate accumulation of blood and this is at risk of infection and empyema formation. It is wise to drain haemothoraces with a tube thoracostomy when they are detected.

The incidence of haemothorax in adult trauma patients presenting to the operating theatre for emergency surgery is 26%. Bleeding may be minimal or massive as a hemithorax can accommodate 30 to 40% of circulating blood volume. Such a massive haemorrhage may indicates injury to the heart, great vessels or the root of the lung but the usual aetiology is a tear in an intercostal vessel secondary to rib fracture. A massive haemothorax is defined as more than 1500 ml of blood in the pleural space and will cause hypovolaemic shock. (>20% of circulating volume)

>1500mls of blood from a chest drain or thoracostomy? The patient needs a thoracotomy

Diagnosis of Haemothorax

Decreased breath sounds and reduced chest movement on the affected side. Mediastinal shift and tracheal deviation away from the affected hemi-thorax may be a late sign. The volume of blood that may be contained in the haemothorax will certainly result in cardiovascular embarrassment or collapse.

The diagnosis is confirmed with ultrasound. A supine film will demonstrate an increased density over the affected hemithorax but no definite fluid level.

Haemothorax Management

Intravenous access must be gained before draining a massive haemothorax. Loss of the tamponade effect of the blood in the pleural space as it is drained may precipitate further massive haemorrhage in an already hypovolaemic patient. Tube thoracostomy is the treatment of choice to allow re-expansion of the lung. Insertion of a chest tube results in evacuation of the haemothorax improving ventilation and allows for monitoring of any ongoing thoracic blood loss. Re-expansion of the lung to approximate to the parietal pleura will tam-

ponade bleeding intercostal vessels in most instances. Continued bleeding indicates inadequate lung expansion, predominantly intercostal arterial bleeding, direct lung laceration or great vessel injury. Ongoing massive blood loss will require surgical control. Clamping the chest drain has very little effect on slowing blood loss and will cause further problems with inadequate lung expansion.

Clamping the chest drain may be indicated to allow conservation of shed blood prior to connection of a cell salvage system if facility and protocol permits. Autologous cell salvage and retransfusion of blood shed into chest drain bottles may be possible with some drainage systems (e.g.the Ocean system) to reduce the problems associated with massive allogeneic blood transfusion.
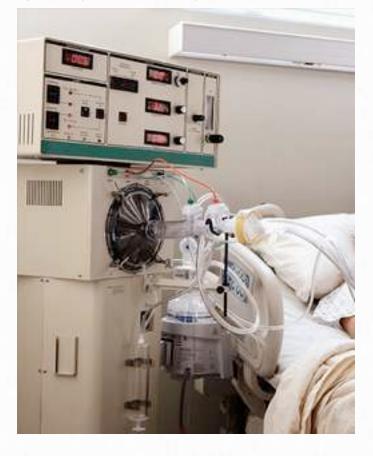
Positive Pressure Ventilation

Traditional approaches to mechanical ventilation used tidal volumes of 10 to 15 ml per kilogram of body weight. These volumes are larger than those in normal subjects at rest (range, 7 to 8 ml per kilogram), but since atelectasis, contusion and oedema reduce aerated lung volumes in patients with acute lung injury, they were frequently necessary to achieve normal values for the partial pressure of arterial car-

bon dioxide and pH. The result however is further disruption of pulmonary epithelium and endothelium, lung inflammation, atelectasis, hypoxemia, and the release of inflammatory mediators. The release of inflammatory mediators increases lung inflammation and cause generalised systemic dysfunction. Thus, the traditional approach to mechanical ventilation actually exacerbated or perpetuated lung injury.

Ventilating to normal parameters, regardless of the pressure used has thankfully, by enlarge become a practice of the past. Protective lung ventilation is the current standard of care for mechanical ventilation. It is synonymous with low tidal volume ventilation (4-8 mL/kg) and often includes permissive hypercapnia. 6 mL/kg predicted body weight is most commonly quoted, as this was the intervention arm of the practice defining ARDSNet ARMA trial and is physiologically normal for a healthy person.

Conventional wisdom holds that hypercapnoeic acidosis (HCA) is bad and is a consequence of relative hypoventilation. HCA per se has protective effects on the pathogenesis of Acute Lung Injury (ALI). Secondary analysis of the original ARDSnet data showed that HCA itself was protective in the 12 ml kg−1 tidal volume group. There was no additional benefit in the 6 ml kg−1

group. HCA is protective in many models of ALI. Beneficial effects include attenuation of lung recruitment, pulmonary and systemic cytokine concentrations, cell apoptosis, and free radical injury. Reassuringly, humans are remarkably tolerant of even extreme hypercarbia and the accompanying acidosis. How HCA will translate into beneficial clinical practice remains to be seen, with issues such as timing and patient recovery remaining to be resolved.

Protective lung ventilation should be used for all mechanically ventilated trauma patients. It can be combined with an open lung approach (i.e. higher PEEP and recruitment manoeuvres) and even High Frequency Oscillatory Ventilation.

Oscillation

The potential benefits of high-frequency oscillatory ventilation (HFOV) include small tidal volumes, higher mean airway pressure, and maintenance of a constant airway pressure. This should translate to less shear stress and barotrauma. HFOV has been shown to improve oxygenation, albeit temporarily, but has not been shown to improve mortality. The few studies that have been performed compared HFOV with conventional ventilation with tidal volumes in the range of 8–10 ml kg have really not shown any practical benefit.

Despite modern lung protective ventilation strategies, some patients have refractory hypoxaemia and hypercarbia. These patients have renewed interest in salvage or rescue therapy for non-responsive ALI/ARDS. Although many of these therapies are aimed at increasing oxygenation, improved oxygenation does not correlate

with improved outcome. The goal of rescue strategies for hypoxaemia is to support the patient without inducing further injury, specifically to the lungs.

Interventional Lung Assist

Along the continuum of lung protective ventilation therapy is the concept of ultra-protective ventilation utilising pumpless extracorporeal lung assist and near-static ventilation. The Novalung membrane ventilator allows O_2 and CO_2 gas exchange via simple diffusion. The membranes are bio-compatible and provide a non-thrombogenic surface. It is designed to work without a mechanical pump in an arteriovenous configuration, thus requiring an adequate mean arterial pressure to drive flow. Flow rates are typically 1–2 litre min−1, or ~15% of cardiac output. CO_2 clearance is controlled by varying the oxygen flow rate into the membrane. Oxygenation is variable and might not be sufficient in severe hypoxic disorders. Compared with conventional extracorporeal membrane oxygenation (ECMO), the Novalung is a simple, pumpless, and, very importantly, portable device. Anti-coagulation requirements are much reduced and bleeding complications and blood product requirements are subsequently less.

Tidal volumes ≤3 ml kg−1, low inspiratory plateau pressure, high PEEP, and low ventilatory frequencies are all possible with the Novalung in situ, causing less Ventilator Induced Lung Injury (VILI) and subsequent remote secondary organ failure.

ECMO

ECMO has been used successfully for severe respiratory failure in trauma. It is highly specialised, resource intensive and expensive, and hence limited to specialised centres. It also allows for ultra-protective lung ventilation as described with the Novalung, but has significantly more side-effects, higher cost, and less portability.

Circulation - Shock and Traumatic Bleeding

In this chapter we will be considering traumatic bleeding in terms of shock and how it should be managed, based on current evidence.

Despite a major change to traditional approaches some years ago, it is quite disturbing how many frontline clinicians still hang on to many of the historical methods when it comes to managing shock. As we will see, this is of particular concern, as not only will they fail to help the patient but they are highly likely to make them worse.

Shock has been defined in many ways, amongst these are the following:

"Acute circulatory failure resulting in the inability of the body to deliver essential oxygen and substrates to the major organs"

There are many others, some more expansive or science based and others shorter and more succinct such as:

"Inadequate end organ perfusion"

The end organs of particular concern being the kidneys, lungs, brain and heart etc. These definitions ultimately mean the same thing. As a result of a failure in the circulation, from real or relative loss of blood volume or pump failure, the substances that are being carried in the blood, to and from all of the essential tissues and organs, do not get there and as a result the body starts to potentially die.

Types of shock

There are obviously a number of different types of shock and the first thing that a layperson may consider it the 'emotional shock' of some particular trauma, but this is not the clinical state they we refer to in trauma. Our definition is an actual physiological failure or pathological state and there are a number of familiar causes.

The classic list describing the types of shock will include the following:

- **Hypovolaemic**: this is the commonest type seen in trauma and is typically as a result of blood loss, although spinal shock can mimic it with similar signs. The blood pressure is low, heart rate high and there are signs of reduced circulating volume.

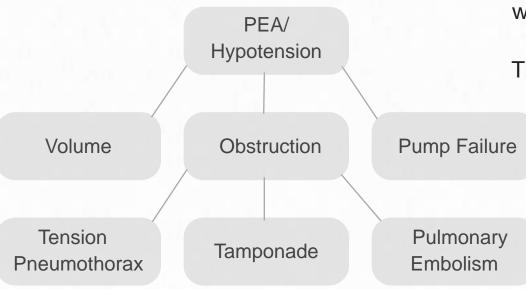- **Cardiogenic**: this is typically associated with medical causes such as myocardial infarction, but a direct cardiac injury such as a contusion or a cardiac tamponade may present with it.

- **Septic**: not usually associated with the early management of trauma, however it may occur during later stages of trauma care as a result of infection. The patient is typically, hot, flushed, with a low vascular resistance and a high cardiac output.

- **Anaphylactic**: again, not usually associated with trauma but may occur as a result of drugs given or even latex gloves. Produces major vasodilation and low blood pressure

- **Neurogenic/spinal**: result from injuries to the nervous system and either affect the vasomotor control centre in the brain or the sympathetic nerve supply from the spinal cord. This may produce marked vasodilation and potentially cardiac failure with the resulting low blood pressure. This may appear to be as a result of hypovolaemia, but no actual volume has been lost as it is a 'relative hypovolaemia' with no blood lost.

- **Tension pneumothorax**: is always included in the list, but this is not a 'type' of shock, but is obviously a cause of life threatening shock.

Hypovolaemic shock can be obvious in some cases, where there is massive blood

loss at scene. Alternatively, there may be minimal apparent blood loss, but there may be swelling or obvious haematoma developing e.g. fractured femur.

Shocked states in trauma resulting in profound hypoperfusion or in extreme cases, PEA, are best thought of as either a volume, pump or obstructive problem.

```
                    PEA/
                 Hypotension

    Volume      Obstruction    Pump Failure

  Tension                       Pulmonary
Pneumothorax    Tamponade       Embolism
```

Causes of hypotension in trauma

This allows for targeting of therapeutic interventions that are more likely to succeed than trying to remember the 4 H's and 4 T's taught on life support courses, and should prompt the practitioner to relate the kinematics and presenting symptoms and signs to the pathophysiology that may be occurring

Finally, there may be little or no sign of blood loss, but developing signs of occult blood loss and shock. This would strongly suggest that there is internal bleeding into one of the large cavities of the body such a chest, abdomen or pelvis.

Bleeding in trauma

Bleeding remains a major killer in trauma and NCEPOD 2007 stated that it resulted in up to 30-40% of the deaths in trauma, within the first 6 hours of injury.

There are two key reasons for this:

- •Failure to identify active bleeding: typically this is occult bleeding into a major body cavity which, if not appreciated from the mechanism of injury or developing signs, will results in rapid deterioration or death

- Failure to control active bleeding: for many years we have taken and almost blasé view of external bleeding, knowing that we can replace it with IV fluids and transfused blood.

However, these are both poor alternatives as compared to your own blood and preservation of your circulation is now a fundamental part of bleeding and shock management. This may be as simple as a pressure dressing or tourniquet or may be as com-

plex as urgent laparotomy or angiography for internal bleeding.

In our MARCH assessment, if we have not seen any massive external haemorrhage we may then pick up signs of more occult bleeding as we start to assess the other systems such as A- airway and R- respiration or breathing.

The breathing may be rapid and shallow and there is no better single sign of an unwell patient than increased respiratory rate, but this could be from a number of causes but should not be ignored. The developing acidosis which occurs in the face of worsening shock will result in a signs of 'air hunger' where casualties are gasping to get their breath and trying to hyperventilate to compensate in respiratory terms for the metabolic acidosis and rising lactate.

The oxygen saturation may be poor, simply from lack of signal and general peripheral shut down, low blood count or even increasing ventilation /perfusion mismatching in the lungs.

Interestingly, these are also features, which may also suggest a pneumothorax, but this should hopefully have been excluded during the respiratory assessment, before we get to circulation.

Classical signs of shock

This is the classic table representing the signs of increasing blood loss and it would be wonderful if every patient had read this and followed it to the letter!

Signs	Compensated shock 25% blood loss	Uncompensated shock 25-40% loss
Tachycardia	+	++
Capillary refill	+	++
Skin	COOL/PALE	MOTTLED
Respiration	+	++
Mental state	ANXIOUS	CONFUSED
Blood pressure	NORMAL	NORMAL/ FALLING

There is a gradual change in all of these parameters as suggested by the columns, but it can often be confused as the body works hard to compensate or other injuries accelerate the change.

Classically, ATLS and other courses teach that tachycardia is shock until proved otherwise and this is true. However, we also have other useful signs of developing shock and failure in compensation mechanisms such as skin colour, capillary refill time and conscious level.

Conscious level is particularly useful as a practical indicator of the level of brain perfusion. Tachycardia may be present and signify blood loss, but if the patient is coherent and not confused then they are clearly adequately perfusing their brain at present and do no warrant resuscitation, especially with crystalloids.

Respiratory rate inevitably increases with shock but the blood pressure is typically ignored and treated as an unreliable tool as it doesn't fall until a significant volume of blood has been lost (typically >25% circulating volume). At this point the shock is often described as 'uncompensated' and there will clearly be increasing signs of hypo-perfusion. However, it is overly simplistic to assume that we can simply 'fill this patient up again' back to normal.

Whilst blood pressure alone will not demonstrate early signs of shock, we must consider all of the information and the signs that we have and build up an overall picture of the severity and speed that shock is developing. Once the blood pressure does fall 'significantly', or in practical terms, the radial pulse becomes thready and eventually disappears, then we need to take some serious action to stop the bleeding and the restore the circulation.

What we mean by a 'significant' fall in blood pressure is the big question. How much shock is too much? How low can the blood pressure go and for how long? These are important questions that still need more answers.

In the meantime, what is very clear is that whilst tachycardia is an indicator of shock, it should no longer be considered a 'target for resuscitation'. In other words, for a bleeding patient with a high heart rate, the traditional approach would be to avoid anaesthesia or to continue resuscitation with fluids until the tachycardia has resolved. We now know that this approach is fundamentally flawed when a patient is still actively bleeding.

Blood pressure as a sign

Hypotension as we have already said is a relatively late sign and by then, we are potentially running into serous compromise from hypo-perfusion. An American retrospective review of 115,000 trauma patients demonstrated that by the time that the blood pressure fell the base deficit was already as much as >20 and the subsequent mortality > 65%. Some would now question this study as we will actually temporarily accept low blood pressure and degrees of shock in our patients for short periods – 'Permissive Hypotension'. What is impor-

tant is what is done during that time in terms of ongoing care. If we simply pour in fluids then things will just get worse and if we stand still and don't work towards some form of definitive haemorrhage control things will still get worse. What we should be doing is rapidly getting control of the bleeding by whatever means in necessary in the shortest possible time.

Other early indicators of bleeding

If we are looking for more reliable indicators of major bleeding that may manifest themselves earlier then consider:

- Mechanism of injury
- Arterial blood gases – especially lactate trends and base deficit
- Haemoglobin – falling level
- CT pan scan

Note that vital signs, FAST scanning and invasive monitoring are not in the list or terribly reliable on review. Whereas the CT pan scan is remarkably sensitive and may detect up to 20% of injuries without any vital sign abnormalities.

This may seem a little confusing as we are now saying that patients with major bleeding would be better left in a state of shock, whilst we try and stop the bleeding. Simply trying to fill them up or 'chase the tachycar-

dia', whilst still bleeding will not address the cause and is likely to increase bleeding and make things worse. However, we must be very clear that leaving a patient 'shocked' and especially one in uncompensated shock with a low blood pressure is a serious compromise and needs some justification.
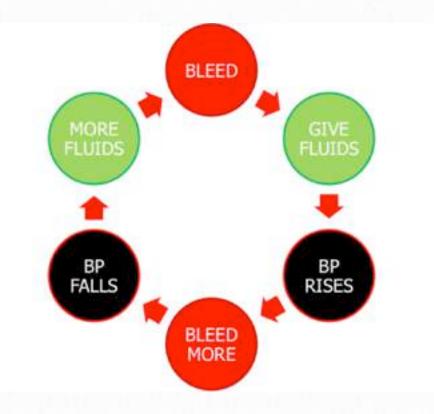
Permissive Hypotension

Let's consider why we wouldn't want to just fill the patient up again. In 1994 Bickell and Mattox published something of a landmark paper, which compared immediate versus delayed resuscitation for hypotensive patients with penetrating torso trauma.

They compared a traditional aggressive fluid resuscitation regime to correct tachycardia and maintain blood pressure and a low fluid volume approach, which allowed the blood pressure to fall to an agreed predetermined 'shocked' level.

When the patients finally got to theatre the blood pressures were no different, but the early resuscitation group had received 2,500ml of fluid as compared to the delayed group that had minimal 'resuscitation' with only 375mls of fluid, but still had the same vital signs. The obvious question is how could that possibly be?

Mattox gives this simple yet clear explanation. If you have a patient who is actively bleeding and demonstrates a degree of shock and give them fluids then this will raise the blood pressure. However, if the 'hole is not plugged' the rise in blood pressure and hydrostatic pressure in the vessels, will simply increase the bleeding still further. The blood pressure will fall again, which will encourage the provider to give yet more fluids and the cyclical process simply continues. He calls this 'Cyclic hyper-resuscitation' and it results in nothing more than a greatly increased fluid resuscitation volume being delivered, with no change in outcome, instead of the better approach of simply stopping the bleeding and then restoring the blood pressure and circulation.

Mattox's Cyclic hyper-resuscitation model

Is there a problem with high volume fluid replacement?

Fluid administration in such large volumes is not without consequence. Some of the fluid in the circulation may be lost as blood, some may be lost in the urine, but by far the majority will be lost into the tissues or what has been previously termed the 'third space'. This fluids 'leaks' from the small vessels in increasing amounts as a result of the traumatic injury and the subsequent SIRS reaction that follows.

Using a traditional ATLS approach to fluid resuscitation, huge volumes of fluid were administered to patients through large bore lines and rapid infusors. This continued from the resus room, through theatre and beyond and after surgery in the critical care unit, the patient would inevitably look massively swollen and often unrecognisable, even to their families as a result of there gross positive fluid balance, almost like a 'Michelin man. In addition, it would appear that the type of fluid, such a crystalloid, colloid, starches etc makes very little difference.

In addition to the increased bleeding from open wounds, wherever a clot has formed or a vessel has gone into spasm, the increased hydrostatic pressure following aggressive fluid resuscitation will dislodge

the clot. This is often described in trauma terms as 'popping the clot' and as a result bleeding will re-commence and increase greatly.

This phenomenon was well described after the Falkland's war, where soldiers with severe injuries and open wounds would be found alive but significantly shocked on the battlefield, often hours after injury. Their bleeding would seem to have stopped, until they were evacuated to the medical centre, where they were 'resuscitated with large volumes of warmed IV fluids. As soon the blood pressure started to improve the bleeding would re-commence and the casualties would often actually get worse. This was a classic demonstration to the British military of the concept of 'popping the clot' and over-zealous resuscitation before bleeding has been controlled.

Crystalloid resuscitation

The idea of simply 're-filling' the circulation and correcting shock seems superficially like common sense. After all, a patient with significant hypoperfusion and poor oxygen delivery for prolonged periods will ultimately not do well, so what research or evidence exists to support this concept or was it nothing more than a good idea, without any real evidence?

If we look back through the literature one the key landmark papers was written by Tom Shires et al, who has often been described as the 'Father of crystalloid fluid resuscitation' – this may well now be a tittle that he wishes to avoid!
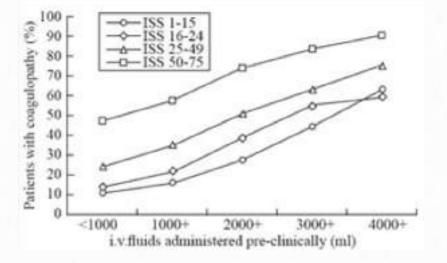
In his paper in the World J Surg, May 2001 he describes how early resuscitation reduces inflammatory markers and also improves survival- some very bold claims! But if we look at the paper more closely, even Shires identified that casualties that received large volumes of crystalloid (e.g. 80-100ml/Kg) had a significantly increased mortality.

These were typically the patients who have the most severe injuries, the greatest blood loss and degree of shock. Casualties with lesser injuries, not surprisingly did do better with correction of their 'shock' at the earliest possible opportunity, but there was clearly far more to the process of resuscitation than simply 'filling' and replacing any identified blood loss.

Since then countless papers have been published around the topic of fluid resuscitation and many of these are summarised in two large review articles, one looking at all of the animal trials published in J trauma 2003 and the other looking at all

the [human studies, considering timing and volume of fluid administration](#), published by the Cochrane Review Library. The conclusion of both review articles, based on all of the evidence that they could find at the times was that:

'there is no significant evidence to support early or large volume fluid resuscitation in major trauma patients with potential active bleeding'

If we actually look at these reviews we find that not only do they fail to improve outcome but, large volumes of crystalloid or any resuscitation fluid, can actually make things worse. Increasing blood loss, diluting all elements in blood, leaking from capillaries, worsening coagulopathy, causing lung injury, impairing healing and the immune system and producing a hyperchloraemic acidosis.

What appears to be a better, but not perfect concept is to accept that the patient is

'shocked' to a degree, but also accept that to try and fully resuscitate them is at worst futile and at best likely to potentially increase their morbidity. However, it also implies that this period of hypotension should be as short as possible to avoid unnecessary effects of shock and under perfusion.

In light of this shocking realisation that the traditional ATLS approach to shock 'chasing the tachycardia' and using large bore lines and high crystalloid volumes, may be detrimental, a new UK consensus statement was made by [Revell, Porter and Greaves in 2002](#).

They stated that:

• Trauma victims with a radial pulse do no require fluid until haemostasis is achieved

• If radial pulse is absent then a 250ml bolus of normal saline is given

• In penetrating torso trauma the presence of a major pulse e.g. femoral is considered adequate

They explained that this approach must be combined with rapid transfer to theatre or control of bleeding but they did also identify a number of exceptional situations:

- Head injuries - they were unsure what best evidence recommended

- Children – titration to a brachial pulse was recommended

As we highlighted earlier, one of the greatest concerns with this new approach is that we are leaving a casualty underperfused and we know that this will ultimately result in serious compromise or death. However, in an actively bleeding patient, we are caught between a rock and a hard place, as to raise the blood pressure will increase bleeding and does not ensure improved survival.

The process is clearly more challenging and complex, especially until bleeding is stopped. We clearly cannot fully resuscitate the casualty, whilst bleeding, but is there a 'best' compromise?
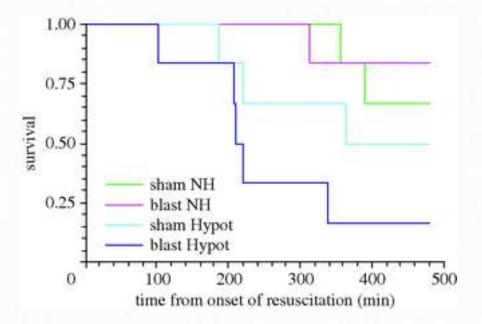
Hybrid or Targeted Resuscitation

One option that has been trialled and published with apparent success in animals by the MOD is a model called 'Hybrid' or 'targeted' resuscitation. This aims to offer the best compromise and is also based on the principle that it is not blood 'pressure' that matters, but rather blood 'flow' and perfusion of tissues.

They adopted the following strategy:

- Maintain a Systolic BP of 80mmHg for the first hour of care

- Then increasing the Systolic BP to 110 mmHg for the remaining period until haemostasis is achieved

- Then fully restoring normotension with blood and blood products

By doing this they reduce the overall level of base deficit, the degree of coagulopathy and systemic inflammation, so this may well be the sort of compromise that we will adopt in future years

When this model was used on a blast model in rats, survival was much improved in the group that had permissive hypotension followed by slow resuscitation to normotension rather than persistent hypotensive resuscitation.
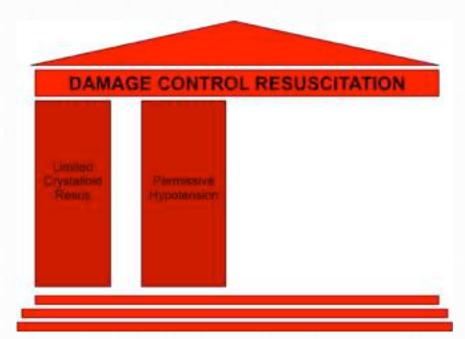
As yet we just do not have all of the answers. However, we have at least demonstrate some things that will definitely make casualties worse:

• Excessive volumes of resuscitation fluid whilst still bleeding

• Delays in achieving control of bleeding

Looking in the literature for human clinical trials, there are some coming through. One of the largest is currently being conducted in Houston by Mattox et al. They have targeted a mean arterial blood pressure of 50 mmHg until bleeding had been controlled.

In J Trauma 2011 they published their preliminary results, which demonstrated both reduced transfusion requirements and an improved survival at 24 hours with this approach.

So if we reconsider our foundation pillars for Damage control resuscitation we can see that two fundamental ones are limited crystalloid resuscitation and the adoption of permissive hypotension. However, we must always remember that any form of damage control is a short terms stop-gap measure 'to get the ship home' or until bleeding is controlled.

The Pillars of damage control resuscitation

If the period of DCR and particularly the hypotension and shock is prolonged then the benefits may be reduced.

The discussion so far has been within the first stages of trauma care in the pre-hospital and immediate hospital resus room phase. We must now consider what we do once in hospital or once blood products of various types are available. Which are best and how much do we use in what ratios?

In-hospital On-going care
Many of the key principles remain the same such as speed to control of bleeding and preservation of circulating volume. This is highlighted every year by leading groups such as the Multi-Disciplinary Task Force for Advanced Bleeding in Trauma, which was first established in Europe in 2005.

They published in 2007 some key recommendations and guidelines and the main beneficial factor that they identified in such cases, was to achieve the shortest time to theatre.

They also highlighted the importance of circulation preservation with measures such as:

- Damage control

- Early recognition of bleeding

- Pelvic stabilisation

- Consideration of early angiography and embolisation for pelvic bleeding and

- Multi-disciplinary approach.

In a later review in the same Journal, Critical Care (2010) a number of recommendations were made based on the evidence at the time:

- Target a systolic blood pressure of 80-100 mmHg

- Target haemoglobin of 7-9 g/dl

This haemoglobin appears a little low when other papers are considered and in an actively bleeding patient a target of 10g/dl seems to be more favoured.

However, once haemostasis is achieved and we are in the critical care phase then any level >7g/dl seems to be adequate (see below)

Continuing all the same key principles started in the pre-hospital arena we avoid excessive crystalloid, rapidly assess and investigate before making a decision and moving forward to scan or theatre etc.

Circulation preservation remains a key priority by whatever means is necessary and at this point we should note that immobilisation devices such as the Pelvic binders or traction splints should be considered as part of 'C' circulation management, as their application will potentially greatly reduce blood loss.

Finally, do not forget that simple measures such as 'gentle patient handling' can also have a profound effect in terms of minimising further blood loss. The increased use of the scoop stretcher has little firm supporting evidence, except one study suggesting improved comfort compared to a long board, but anecdotally it may reduce sudden internal blood loss and pain by avoiding the need for log-rolling.

However, others would argue that a 'log-roll' will have to be performed anyway to thoroughly check the back of the patient and it can be performed without compromise once the pelvis is strapped.

There is also the issue of poor technique, whereby the insertion of the scoop can actually involve two log rolls, first to the left and then to the right, if not properly understood!

As it stands at present the scoop is favoured over the long-board by UK ambulance services. Ultimately, a vac-mat is probably a far better device than both of these for the casualty, but this does have both cost and practical limitations.

Insignificant blood loss – every drop counts!

Throughout the early time in hospital it is essential to look for any signs of ongoing uncontrolled bleeding. If identified then this should be managed quickly and effectively to preserve all circulation. We should not be over-reliant on the lab and transfused blood, as it is not the patient's blood and will never be as good as their own.
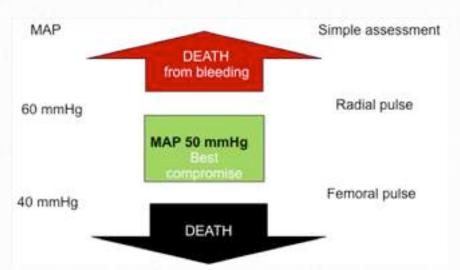
Severe shock – very low blood pressure

What do you do if the blood pressure is too low? At some point, wherever you set your resuscitation threshold, there comes a time where you may have to give some fluid, simply to keep the patient alive. As we have seen, the recommended trigger threshold for commencing fluid replacement varies, but we are far clearer on how much fluid.

There is a general consensus view that if blood is not available, then 250ml boluses of normal saline (ideally warmed) are the best compromise, titrated to response and kept to a minimum.

Other fluids such as hypertonic saline with a starch or dextran, have simple practical advantages and work in very small volumes. However, a review by NICE was unable to demonstrate any effective advantage to justify the cost and therefore cannot support their use, although they may have a place in shocked head injuries and they certainly have a value in the management of raised intracranial pressure and ICP control.

The debate about when to commence fluid replacement in a bleeding patient continues. In 2012 Rick Dutton attempted to sum up the current position with the simple diagram below. This suggests that a mean arterial pressure of 50mmHg is the best compromise target pressure, anything more will increase bleeding to a serious degree and with anything less, the shock will become life threatening.

Dutton guide to fluid resuscitation in bleeding major trauma patients

How we identify the 'best compromise' pressure depends on your setup at the time. He relates to the MAP but at times we may have to adopt a rapid simple assessment. For example, the presence or absence of pulses, even though the direct correlation between pulses and BP is actually quite poor, but at least it offers a simple and quick, practical assessment of perfusion.

What about traumatic brain injuries and shock?

Dutton's and similar papers provide us with a clear target for us to work to for a patient with trauma and major bleeding, but what about such a patient that also has a head injury?

The neurosurgeons would like 'normotension' and a rapid delivery to their care. However, there is actually little quality research to guide us in this area.

Animal work suggests that a MAP of 50-65 mmHg is the best compromise.

What is clear is that high volumes of fluid will raise ICP, impair oxygen delivery in the brain and reduce survival. Interestingly, a number of studies also suggested that maintenance of brain perfusion with the use of vasopressors will result in a poor outcome, in fact in one study all of these patients died. Clearly more work is needed in this field and a compromise between increasing bleeding and reduced brain perfusion must be achieved.

Resuscitation with blood

What if we have blood? An increasing number of studies suggest that we should avoid all crystalloid and go direct for blood and blood products.

This is supported by the fact that blood is pretty safe these days, for example according to the Serious Hazards of Transfusion (SHOT) report of 2007, there was only 1 death directly as a result of a blood transfusion in the previous 12 months.

However, this is not the perfect solution as stored blood is seriously impaired in a number of ways:

- The blood is rarely 'fresh' and if stored for more than 14-21 days then there is a significant increase in thrombotic complications following transfusions.

- Oxygen carriage is poor as a result of low 2,3, DPG levels

- Oxygen delivery is poor because stored red cells start to become stiff and move poorly through capillary beds

- We rarely get 'whole' blood and as such all of the essential clotting factors have been removed

In addition, if we consider the great deal of research conducted on the 'ideal' target haemoglobin following transfusion it is typically 7 g/dl, suggesting that transfusion is not the answer and in fact normovolaemic haemodilution may be a safer approach.

These optimal transfusion triggers have also been demonstrated in critical care patients, trauma patients, neuro trauma and even paediatric patients.

If we transfuse to higher levels then there is no reduction in 30 day mortality and a good chance of increased complications such as multiple organ failure and length of stay.

The bottom line is that best blood to be circulating in your vessels is your own and we should do everything that we possibly can to preserve that volume.

Inevitably some exceptions do exist and the obvious ones quoted, as most likely to require the optimum delivery of oxygen are ischaemic heart disease or stroke patients. In fact, even back in 1992 publications had demonstrated that in ischaemic heart disease the mortality was higher for patients with a haemoglobin of 7-9g/dl as compared to >10g/dl.

However, as ever in resuscitation, things are not as simple as they may seem, as another study demonstrated that in post coronary artery bypass patients, if the Hb is higher than 10g/dl then there is a significantly increased rate of myocardial infarction, which is probably related to the viscosity of blood.

Such low targets are fine if the patient is now being stabilised in the critical care unit, after bleeding has been controlled, but what do we do in the early stage when bleeding is active and very rapid? If we set the bar as low as 7g/dl then by the time that we respond and commence transfusion, the level of haemoglobin will have fallen dangerously low.

As a result we need yet another compromise position, not wishing to fully transfuse back to the normal levels, but avoiding a life threatening dip in oxygen delivery capacity with very severe anaemia.

Interestingly there is another factor, which will influence our decision. A number of recent studies have clearly demonstrated that red cells play an essential part in the clotting process. As such a haemoglobin concentration >10g/dl seems to stabilise the clot and a reduced haematocrit appears to inhibit platelet aggregation.

In view of this the current best compromise during active bleeding is considered a target Hb concentration of 10g/dl

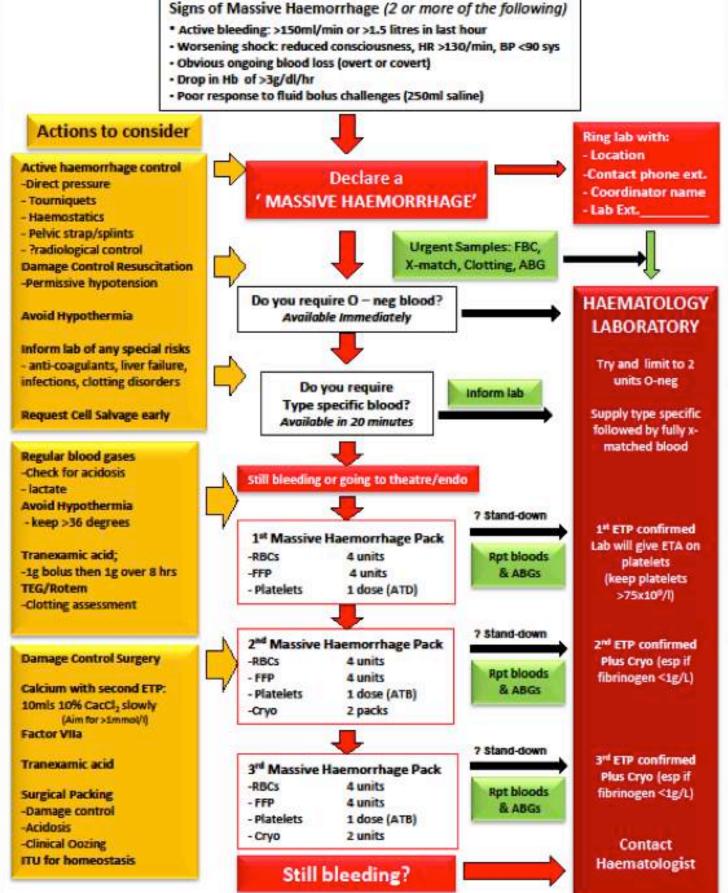If we try and summarise the last few sections in a series of consensus statements:

- Avoid crystalloid/colloid resuscitation unless there is no other option

- Give blood is the Systolic blood pressure is < 80mmHg or the MAP <50mHg

- Try and avoid raising the MAP >60mmHg

- Keep the patient in the this hypotensive state for the shortest possible time, with control of bleeding achieved in less than 1 hour

As we have now identified that if we need to give fluid replacement, then the best option, although not ideal, is blood and blood products. This must be obtained at the earliest possible opportunity, which may even be pre-hospital in certain parts of the UK e.g. London HEMS.

Massive Haemorrhage Policy

At the earliest possible opportunity, which again may even be pre-hospital, or at the very least in the resus room, we should activate a predetermined massive haemorrhage policy with the lab. This is a pathway, with locally agreed protocols, quantities of blood products and on-going re-assessment. Clearly this offers a lot more than just blood as remember that we are not using 'whole' blood but typically 'packed red blood cells' with platelets and the clotting factors removed. This alone will result in dilutional coagulopathy and a worsened situation so we must be provided with supplementary clotting factors as early as possible.

Management of Massive Haemorrhage

Signs of Massive Haemorrhage *(2 or more of the following)*
- Active bleeding: >150ml/min or >1.5 litres in last hour
- Worsening shock: reduced consciousness, HR >130/min, BP <90 sys
- Obvious ongoing blood loss (overt or covert)
- Drop in Hb of >3g/dl/hr
- Poor response to fluid bolus challenges (250ml saline)

Actions to consider

Active haemorrhage control
- Direct pressure
- Tourniquets
- Haemostatics
- Pelvic strap/splints
- ?radiological control

Damage Control Resuscitation
- Permissive hypotension

Avoid Hypothermia

Inform lab of any special risks
- anti-coagulants, liver failure, infections, clotting disorders

Request Cell Salvage early

Regular blood gases
- Check for acidosis
- lactate

Avoid Hypothermia
- keep >36 degrees

Tranexamic acid;
- 1g bolus then 1g over 8 hrs
TEG/Rotem
- Clotting assessment

Damage Control Surgery

Calcium with second ETP:
10mls 10% CaCl₂ slowly
(Aim for >1mmol/l)
Factor VIIa

Tranexamic acid

Surgical Packing
- Damage control
- Acidosis
- Clinical Oozing
ITU for homeostasis

Declare a 'MASSIVE HAEMORRHAGE'

Ring lab with:
- Location
- Contact phone ext.
- Coordinator name
- Lab Ext. _____

Urgent Samples: FBC, X-match, Clotting, ABG

Do you require O – neg blood?
Available immediately

Do you require Type specific blood?
Available in 20 minutes

Inform lab

Still bleeding or going to theatre/endo

1st Massive Haemorrhage Pack
- RBCs — 4 units
- FFP — 4 units
- Platelets — 1 dose (ATD)

? Stand-down

Rpt bloods & ABGs

2nd Massive Haemorrhage Pack
- RBCs — 4 units
- FFP — 4 units
- Platelets — 1 dose (ATB)
- Cryo — 2 packs

? Stand-down

Rpt bloods & ABGs

3rd Massive Haemorrhage Pack
- RBCs — 4 units
- FFP — 4 units
- Platelets — 1 dose (ATB)
- Cryo — 2 units

? Stand-down

Rpt bloods & ABGs

Still bleeding?

HAEMATOLOGY LABORATORY

Try and limit to 2 units O-neg

Supply type specific followed by fully x-matched blood

1st ETP confirmed
Lab will give ETA on platelets
(keep platelets >75x10⁹/l)

2nd ETP confirmed
Plus Cryo (esp if fibrinogen <1g/L)

3rd ETP confirmed
Plus Cryo (esp if fibrinogen <1g/L)

Contact Haematologist

Example of a Massive haemorrhage policy

Typical predictors for activating the MHP

Pre-hospital:
- Low blood pressure
- Increasing tachycardia
- Mechanism of injury
- Poor response to a fluid challenge

In-hospital:
- Positive FAST
- Low systolic blood pressure
- Increasing heart rate
- Penetrating trauma

We can also use some blood results as triggers, but this will often come somewhat later.

Blood results:
- Fall in haemoglobin or haematocrit
- Deranged clotting
- Low ph. (acidosis)

The in-hospital criteria described are both sensitive (up to 75%) and also predictive (86%).

As we move through the trauma system with an actively bleeding patient we must continue our 'circulation preservation'. Even in theatre, until the bleeding is surgically controlled, we adopt a degree of permissive hypotension (or maybe a 'Hybrid' approach), small volume resuscitation and speed.

Damage Control Resuscitation

A damage control resuscitation and surgical approach will be adopted and the shortest time possible taken to stop the bleeding. Once we have achieved control we can then rapidly restore the circulation and correct the shock and hypoperfusion.

During the Damage Control Surgery other factors will help to minimise further blood loss including use of a cell saver system, anti-fibrinolytic agents and other blood product such as FFP, platelets and cryoprecipitate.

During the surgery in theatre we may be tempted to reach for many of the numerous critical are devices traditionally associated with assessing resuscitation, cardiac output and hydration. In reality, few if any of these offer much value until bleeding is controlled and we then enter the true resuscitation and correction of coagulopthy phase.

Even relatively simple monitors such as an arterial line, can delay transfer and control of bleeding. As such, as we do in the pre-hospital domain, before every action we should consider, do we really need to do this now?

Failure to respond to resuscitation

For some patients, we may attempt to maintain them at our ideal target MAP, but they continue to deteriorate and do not respond in a 'normal' way to filling with blood or other fluids.

This may obviously be as a result of such rapid and massive bleeding that we are simply falling further and further behind with worsening shock but in the worst of these cases, there maybe no response to any filling, no matter how aggressive and

we must then consider have we missed something? Go back and review your patient and consider these options:

- Tension pneumothorax
- Cardiac tamponade
- Adrenal insufficiency or crisis
- Hypocalcaemia – especially after large volume transfusion of stored blood
- Hypoglycaemia
- Spinal injury

Alternative Agents

In this final section, we will consider any other options that may exist or be on the horizon for damage control resuscitation and replacement of major blood loss.

Two key products that have been extensively explored are:

- Synthetic haemoglobins
- Perflourocarbons

Synthetic haemoglobins

Synthetic haemoglobins have been around for over 20 years, but a number of the early ones such as haem-assist and haemolink were withdrawn from clinical trials in 1998 because of increased mortality from complications associated with their use.

However, the next generation of products such as HBOC-201 and Hemopure seemed far more promising. They appear to offer the ideal solution for blood transfusion:

- They are immediately to hand and have a long shelf life.
- They are universally compatible with all blood groups
- They are effective in small volumes and are moderately hypertonic and ideal for resuscitation
- They carry and delivery oxygen effectively, although they have a higher p50 which means that they do not 'give up' their oxygen as readily as normal blood

This all sounds very good, but in JAMA, Oct 2008 there was a large meta analysis of all the synthetic haemoglobin studies and it was identified that many had been abandoned or stopped prematurely with lots of lost or undeclared data. The FDA went as far as to describe a 'secret science' associated with these products.

All types were associated with a significant increase in the risk of death and MI. This

would appear to be related to Nitric oxide scavenging, increases in SVR, reduced blood flow and/or platelet aggregation with thrombotic complications.
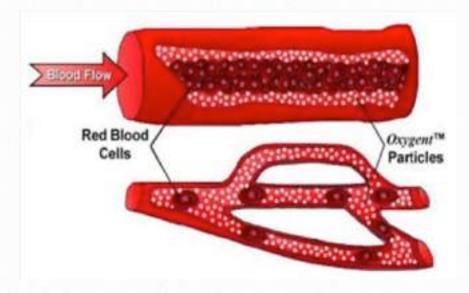
However, it should be noted that these agents are still being used effectively in parts of South Africa and there do appear to be some significant industry competition issues. As such, at present we cannot recommend the use of these agents, but we should also not totally dismiss them for the future.

Perflourocarbons

Many of you will have seen perflourocarbons in use in the film the 'Abyss' where a diver descends to incredible depths using a liquid breathing. This all looked a little like science fiction, but these materials actually exist and will carry oxygen sufficient for life.

In the 80s and early 90s this technique was adopted for injured lungs in critical care with a product called Exosurf, but the results were poor and its use was abandoned.

However, in recent years new alternative perfluorocarbon solutions for intravascular, rather than inhaled use have been developed. One such agent is 'Oxygent' which effectively delivers oxygen combined with

micro-particles much smaller than red blood cells. This particle size greatly improves capillary level delivery of oxygen.
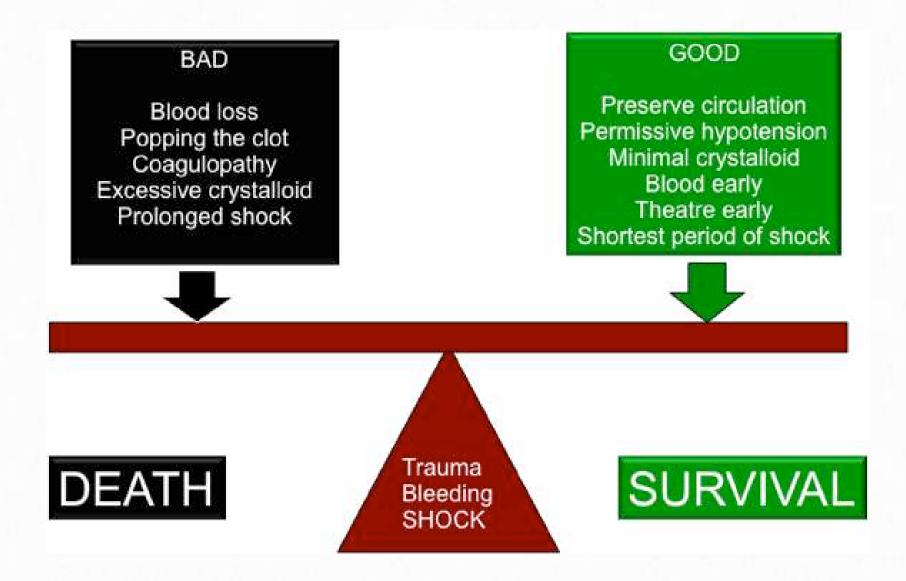
Oxygen perfluorocarbon micro particles

They offer a simple injectable solution for improving oxygen carriage without the problems of blood grouping and antibodies.

Phase III trials are underway in the US and Canada and they appear to have an excellent side effect profile so far, simply removed by the liver and kidney without causing compromise. At this stage it is difficult to say anymore other than 'watch this space'

Stem Cell Blood:

Finally, there is another alternative based on actual human blood, but actually grown from stem cells, from an umbilical cord. The US military research facility DARPA

have invested 2 million dollars into this project and can already produce 20 units of blood from one umbilical cord in just three days.

Unfortunately, each unit currently costs $5,000 but this will come down. This has already been sent for FDA approval and is being considered for military use.

Summary

In summary, shock and fluid resuscitation seems so simple if considered as a leaky bucket that just needs re-filling. However, what and how much you fill it, especially before the leak is fixed are hugely complex questions when it comes to trauma resuscitation and blood loss.

Allowing bleeding to continue, popping the clot, complications from overzealous use of crystalloid and prolonged periods of shock are bad and will result in an increased mortality of morbidity. These must all be carefully balanced with circulation preservation, permissive hypotension, minimal crystalloid use and early control of bleeding before full resuscitation to achieve the best chances of survival.

This is a very challenging balance to achieve and as yet we do not have all of the answers.

References

1. Definition of Shock:
   http://en.wikipedia.org/wiki/Shock_(circulatory)

2. NCEPOD 2007 – 'Trauma who cares'

3. Classical signs of shock: Greenfield's Surgery Scientific Principles and Practice, 5th edition
   https://www.inkling.com/read/greenfields-surgery-scientific-principles-practice/chapter-8/types-of-shock

4. Systemic hypotension is a late marker of shock after trauma: a validation study of Advanced Trauma Life Support principles in a large national sample.
   http://www.ncbi.nlm.nih.gov/pubmed/17161083 Parks JK, Elliott AC, Gentilello LM, Shafi S. Am J Surg. 2006 Dec;192(6):727-31

5. N Engl J Med. 1994 Oct 27;331(17):1105-9. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. Bickell WH1, Wall MJ Jr, Pepe PE, Martin RR, Ginger VF, Allen MK, Mattox KL.

6. Permissive hypotension – 'cyclic hyper-resuscitation' Traum.org archives, Mattox 2002

7. J R Army Med Corps. 2007;153 Suppl 1:74-7. Lessons from the Falklands campaign. Crawford IP, Abraham P, Brown M, Stewart JB, Scott R.

8. World J Surg. 2001 May;25(5):592-7. Improved survival with early fluid resuscitat ion following hemorrhagic shock. Santibanez-Gallerani AS1, Barber AE, Williams SJ, ZhaoB S Y, Shires GT.

9. J Trauma. 2003 Sep;55(3):571-89. Fluid resuscitation strategies: a systematic review of animal trials. Mapstone J1, Roberts I, Evans P.

10. Volume and timing of fluid administration in bleeding trauma patients, head injuries and critical care – 2000, 2002 and 2014 Cochrane reviews
    http://onlinelibrary.wiley.com/cochranelibrary/search

11. Emerg Med J. 2002 Nov;19(6):494-8. Fluid resuscitation in prehospital trauma care: a consensus view. Revell M1, Porter K, Greaves I.

12. J Trauma Acute Care Surg. 2012 Apr;72(4):835-43. doi:

10.1097/TA.0b013e318248347b. Targeted resuscitation improves coagulation and outcome. Doran CM1, Doran CA, Woolley T, Carter A, Male K, Midwinter MJ, Mahoney PF, Watts S, Kirkman E.

13. J Trauma. 2011 Mar;70(3):652-63. doi: 10.1097/TA.0b013e31820e77ea. Hypotensive resuscitation strategy reduces transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: preliminary results of a randomized controlled trial. Morrison CA1, Carrick MM, Norman MA, Scott BG, Welsh FJ, Tsai P, Liscum KR, Wall MJ Jr, Mattox KL.

14. Crit Care. 2007;11(1):R17. Management of bleeding following major trauma: a European guideline. Spahn DR1, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E, Gordini G, Stahel PF, Hunt BJ, Komadina R,Neugebauer E, Ozier Y, Riddez L, Schultz A, Vincent JL, Rossaint R; Task Force for Advanced Bleeding Care in Trauma.

15. Crit Care. 2013 Apr 19;17(2):R76. [Epub ahead of print] Management of bleeding and coagulopathy follow-ing major trauma: an updated European guideline. Spahn DR1, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E, Filipescu D, Hunt BJ, Komadina R, Nardi G,Neugebauer E, Ozier Y, Riddez L, Schultz A, Vincent JL, Rossaint R.

16. Br J Surg. 2012 Jan;99 Suppl 1:21-8. doi: 10.1002/bjs.7731. Resuscitative strategies to maintain homeostasis during damage control surgery. Dutton RP.

17. Shock. 2013 Oct;40(4):247-73. doi: 10.1097/SHK.0b013e3182a3cd74. Combined hemorrhage/trauma models in pigs-current state and future perspectives. Hildebrand F1, Andruszkow H, Huber-Lang M, Pape HC, van Griensven M.

18. J Trauma Acute Care Surg. 2013 Dec;75(6):968-74; discussion 974-5. doi: 10.1097/TA.0b013e31829a021a. Differential effects of fresh frozen plasma and normal saline on secondary brain damage in a large animal model of polytrauma, hemorrhage and traumatic brain injury. Hwabejire JO1, Imam AM, Jin G, Liu B, Li Y, Sillesen M, Jepsen CH, Lu J, deMoya MA, Alam HB.

19. Serious Hazards of Transfusion, UK 2007 http://www.shotuk.org/

20. Vox Sang. 2002 Aug;83 Suppl 1:387-96. The TRICC trial: a focus on the sub-group analysis. Hébert PC1; Transfusion Requirements in Critical Care.

21. Transfusion. 1992 Jan;32(1):17-22. Autologous blood donation: hemodynamics in a high-risk patient population. Spiess BD1, Sassetti R, McCarthy RJ, Narbone RF, Tuman KJ, Ivankovich AD.

22. Anaesthesist 2006 Nov;55(11):1142-56. [Tolerance to perioperative anemia. Mechanisms, influencing factors and limits]. Habler O1, Meier J, Pape A, Kertscho H, Zwissler B.

23. J Trauma. 2008 Oct;65(4):951-60. doi: 10.1097/TA.0b013e318187e15b. Preconditions of hemostasis in trauma: a review. The influence of acidosis, hypocalcemia, anemia, and hypothermia on functional hemostasis in trauma. Lier H1, Krep H, Schroeder S, Stuber F.

24. Warrington & Halton Hospital Foundation NHS Trust/MHP policy/2012

25. J Trauma. 2009 Feb;66(2):346-52. doi: 10.1097/TA.0b013e3181961c35. Early prediction of massive transfusion in trauma: simple as ABC (assessment of bloodconsumption)? Nunez TC1, Voskresensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA.

26. Crit Care. 2012 Jul 20;16(4):R129. [Epub ahead of print] Predicting ongoing hemorrhage and transfusion requirement after severe trauma: a validation of six scoring systems and algorithms on the TraumaRegister DGU® Brockamp T1, Nienaber U, Mutschler M, Wafaisade A, Peiniger S, Lefering R, Bouillon B, Maegele M; TraumaRegister DGU.

27. Curr Drug Discov Technol. 2012 Sep;9(3):158-65. Crosslinked, polymerized, and PEG-conjugated hemoglobin-based oxygen carriers: clinical safety and efficacy of recent and current products. Jahr JS1, Akha AS, Holtby RJ.

28. J Trauma. 2009 Feb;66(2):365-76. doi: 10.1097/TA.0b013e3181820d5c HBOC-201 vasoactivity in a phase III clinical trial in orthopedic surgery subjects--extrapolation of potential risk for acute trauma trials. Freilich

D1, Pearce LB, Pitman A, Greenburg G, Berzins M, Bebris L, Ahlers S, McCarron R.

29. JAMA. 2008 May 21;299(19):2304-12. doi: 10.1001/jama.299.19.jrv80007. Epub 2008 Apr 28. Cell-free hemoglobin-based blood substitutes and risk of myocardial infarction and death: a meta-analysis. Natanson C1, Kern SJ, Lurie P, Banks SM, Wolfe SM.

30. Br Med Bull. 1999;55(1):277-98. Red blood cell substitutes: fluorocarbon emulsions and haemoglobin solutions. Remy B1, Deby-Dupont G, Lamy M.

31. Transfus Med Rev. 1993 Apr;7(2):84-95. Perfluorocarbon-based red blood cell substitutes. Biro GP.

32. Artif Cells Blood Substit Immobil Biotechnol. 2005;33(1):47-63. Under-standing the fundamentals of perfluoro-carbons and perfluorocarbon emul-sions relevant to in vivo oxygen deliv-ery. Riess JG.

33. Stem Cells Dev. 2011 Sep;20(9):1639-47. doi: 10.1089/scd.2011.0078. Epub 2011 May 11. Generation of red blood cells from human induced pluripotent stem cells. Dias J1, Gumenyuk M, Kang H, Vodyanik M, Yu J, Thomson JA, Slukvin II.

34. Curr Opin Hematol. 2012 May;19(3):163-9. doi: 10.1097/MOH.0b013e328352240a. Ex-vivo generation of human red cells for transfusion. Anstee DJ1, Gampel A, Toye AM.

Head Injury
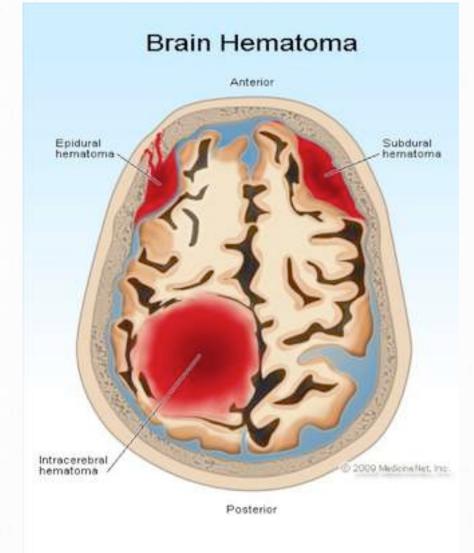
Morbidity and mortality from severe head injuries presents a significant problem to the healthcare systems of all developed countries. Head Injury is the commonest cause of death in all patients under the age of 24 years in the U.K. and many survivors of severe head injuries, struck down in their youth or the prime of their working lives, are unable to re-take their place in society, consuming healthcare resources, rather than contributing to them. In the course of any year, up to 1.5 percent of the population in the UK will attend hospital with some sort of head injury. Only a tiny fraction of these patients will have a life-threatening problem, but identifying these serious cases may not always be completely straightforward. Once identified, optimal management for such patients can reduce mortality and severe morbidity significantly. A reliable system is needed for assessment and subsequent treatment.

Mechanism of Injury

The cause of severe head injuries varies with the age of the victims. In the young, road traffic accidents predominate as the cause, either as pedestrians or occupants of a vehicle. Assaults are also common in this age group, and over 50% of these patients will have other extracranial injuries. In the elderly, falls predominate as a cause of head injury. Chronic alcohol abuse is a significant related factor, and the injuries may be related to relatively minor trauma and separated from presentation by a significant period of time.

Pathology
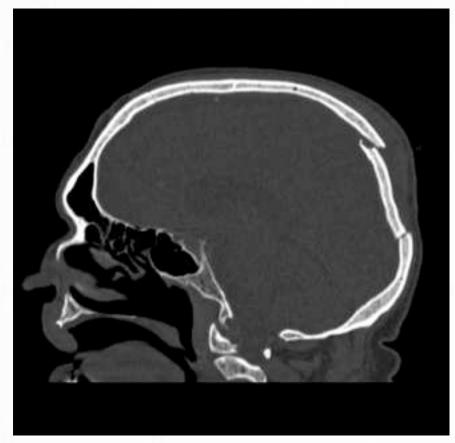
The most important consequence of head injury is brain damage. Crucially, it must be realised that this may arise as a direct result of the primary impact on the head, but also due to avoidable secondary insults at a later stage; chiefly hypoxia, hypercarbia, hypotension, or a combination of all three. Apart from superficial damage to the scalp, head injuries include skull fractures, focal brain injuries, diffuse brain injuries, and secondary brain damage.

Brain Hematoma

(Ref: http://images.medicinenet.com/images/illustrations/brain-hematoma.jpg)

Skull Fractures

Skull fractures are common, but do not cause neurological disability in themselves. They do however, provide an indicator of the force of impact, and thus identify patients at higher risk of significant neurological damage. If the fracture is compound, it may be associated with a CSF leak and with the risk of the entry of air and bacteria.

Depressed fractures usually require surgery if compound, or if displaced inwards more than 1cm.

Battle's sign is bruising over the mastoid.It is seen several days following a basilar skull fracture.
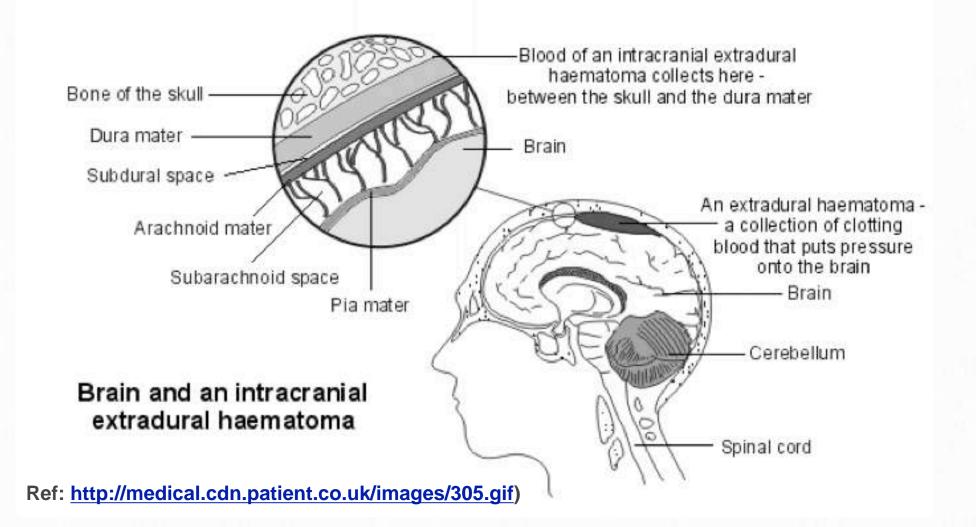
Focal Brain Injuries

Are classified as lesions where macroscopic damage occurs in a localised area. They comprise contusions and hae-

matomas. Emergency surgery may be required because of the mass effects of these lesions. 30% of head injury patients in coma have an intracranial haematoma.

Contusions
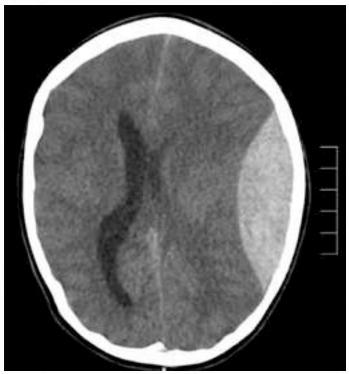
Contusions are caused by contact between the surface of the brain and the interior ridges of the skull. The brain is normally well protected against these impacts by the cushioning effect of the CSF by which the brain's weight (1200g) is reduced to effectively only 50g by floating in the fluid. Rapid accelerations caused by impacts to the head or decelerations from falls, RTCs etc., will result in shock waves travelling through the soft substance of the brain. This leads to contusions under the site of impact or remote from it (contracoup).

Extradural Haematomas

Extradurals are almost invariably associated with a tear in a dural artery, usually the middle meningeal. Primary brain damage can be minimal, and evacuation without delay minimises secondary problems from cerebral compression. Studies have shown that about one third of patients with fatal head injuries were talking at some time after their injury. 75% of such patients have been found to have an intracranial haematoma at post mortem.
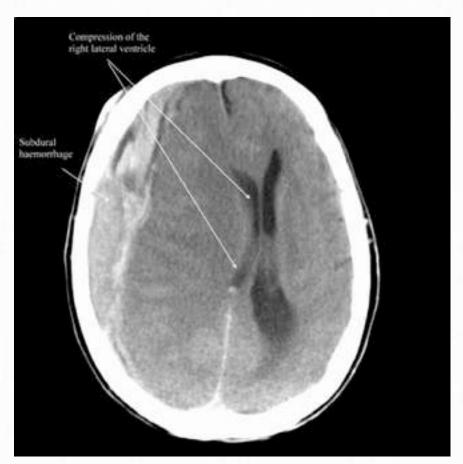
Brain and an intracranial extradural haematoma

Labels:
- Bone of the skull
- Dura mater
- Subdural space
- Arachnoid mater
- Subarachnoid space
- Pia mater
- Blood of an intracranial extradural haematoma collects here - between the skull and the dura mater
- Brain
- An extradural haematoma - a collection of clotting blood that puts pressure onto the brain
- Brain
- Cerebellum
- Spinal cord

Ref: http://medical.cdn.patient.co.uk/images/305.gif)

The classical presentation of an extradural haematoma is of a loss of consciousness followed by a 'Lucid period', then leading to a lapse back into unconsciousness associated with development of an ipsilateral dilated pupil, and a contralateral hemiparesis. Unfortunately, only about 45% of extradurals present like this.

Extradural Haematoma: Note the bi-convex appearance.
(Ref: http://cdn.lifeinthefastlane.com/wp-content/uploads/2010/08/VAQ_20101_8.jpg)

Acute Subdural Haematomas

More common than extradurals, these lesions are caused by the rupture of veins which bridge the space between the cerebral cortex and the dura. Increasing cerebral atrophy with advancing age, and chronic alcohol abuse tends to open up this space, making these lesions more common in the elderly. In the young, associated primary brain injury is often more severe, resulting in coma from the outset. Early evacuation (within 4 hours) has been shown to improve outcome.

Acute Subdural Haematomas (Ref: http://cdn.lifeinthefastlane.com/wp-content/uploads/2010/04/CT-Head-SDH-acute.jpg)

Diffuse Brain Injuries

The rapid acceleration forces which are transmitted to the brain following an impact to the head, result in pressure waves travelling through the brain substance, these can cause stretching and tearing of axonal tracts resulting in widespread disruption of brain function. Mild forms of this, usually termed concussion, may result in temporary unconsciousness and amnesia. Severe and diffuse axonal injury results in prolonged coma. Little can be seen at a macroscopic level, even with sophisticated imaging. Post-mortem however, microscopic examination of the brain reveals widespread tearing of the tracts within the white matter. Cerebral oedema is an inevitable accompaniment to this type of injury, given the characteristic appearance of swollen brain on CT scan. Progressive development of oedema leads to a rise in intracranial pressure which may cause severe secondary injury or death.
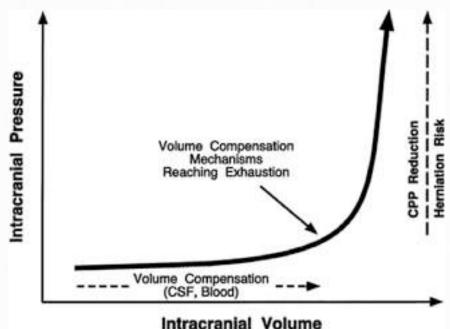
Secondary Brain Injury

While the primary injury has already occurred, it may be possible to prevent some of the secondary injury. This is the objective of head injury management.

Intracranial Pressure Considerations

The cranium forms an essentially rigid box which is in communication with the vertebral canal. The normal pressure inside the box is 3-10mmHg and is the result of a dynamic relationship between the volumes of the various intracranial contents:
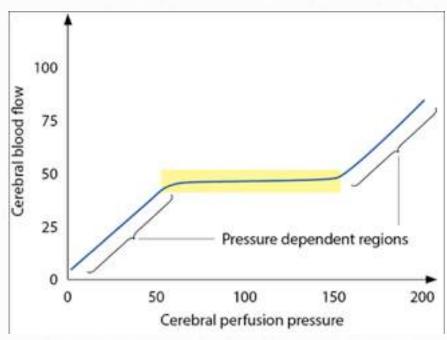
Because the container is rigid, a rise in volume of one component must be compensated by a fall in one of the others, or the pressure will rise. This is the Monroe-Kelly Doctrine. Only the volumes of blood and CSF inside the head can be regarded as reducible. If a blood clot or excess oedema occurs, CSF can be displaced into the spinal subarachnoid space, but once this compensation is exhausted pressure can rise very quickly.

Pressure Volume Curve

Modifications in cerebral blood flow can cause major changes in pressure inside the skull once this situation is reached. The partial pressure of CO_2 in the arterial blood is a major regulating factor in cerebral vasodilatation and constriction. A raised P_aCO_2 will cause cerebral vasodilation and increase the intracranial pressure:

	Volume (ml)	% Total
Brain Tissue	1200-1600	70-80
CSF	130-150	10
Blood	100-150	5-10
ECF	<100	<5

Cerebral Auto-regulation Graph

It has been shown that, in patients with documented hypoxia and/or hypotension, the proportion of poor outcomes (death or persistent vegetative state) rises alarmingly. The aim of management in these patients however must be to maintain normal levels of oxygenation and an adequate Cerebral Perfusion Pressure (CPP). CPP is defined as the difference between Mean Arterial Pressure (MAP) and Intracranial Pressure (ICP):

CPP = MAP - ICP

Appropriate figures for CPP are:

Adults and Children over 12 years	70mmHg
Children 4 to 12 years	60mmHg
Children under 4 years	50mmHg

Whilst it is quite easy to obtain a reliable estimate of any patient's mean arterial pressure at any time in hospital, measurement of intracranial pressure requires specialist invasive equipment. In the early stages of resuscitation and management of these patients therefore, it is imperative to make the assumption that any patient with a head injury who has a depressed level of consciousness and/or neurological deficit may have raised intracranial pressure.

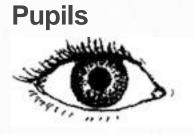**Assume an ICP of 30mmHg**

Initial Assessment

As much information as possible must be obtained from witnesses, paramedics etc. about the patient's level of consciousness at the scene of the accident to gain a feel for the general trend in the neurological status. Prior to RSI and subsequent sedation and paralysis, make a note of the neurological state of the patient.

A Neurological Assessment has three basic constituents:

• Level of Consciousness

• Pupils

• Focal neurological assessment.

Level of Consciousness

At its simplest, this can be assessed using the AVPU scale. It can also be measured on the Glasgow Coma Score, which allows accurate information to be conveyed to distant clinicians by telephone. Repeated assessments give valuable trending information to allow decisions to be made regarding ongoing management.

Pupils

Pupils are assessed for their equality and response to a bright light. Their speed of response must be recorded. A sluggish response may indicate a developing intracranial injury. Remember that a difference in size between pupils of up to 1mm may be normal.

A unilaterally dilated and fixed pupil is a sign of significantly raised ICP with a focal mass lesion. Bilaterally fixed dilated pupils is a ominous sign of bilateral brain stem compression. Remember also, that small, unresponsive pupils are abnormal and may indicate mid brain pathology. All such pupillary abnormalities are an indication for immediate consultation with a neurosurgeon.

Focal Neurological Deficit

Spontaneous movements are observed to assess whether they are equal on both sides or not. If there is no spontaneous movement, then painful stimuli must be applied. A clear difference on one side may indicate an evolving intracranial bleed. Score the best side in the motor component of the GCS.

Glasgow Coma Score

The Glasgow Coma Score provides a quantitative measure of a patient's level of consciousness. It is the sum of the scores from three areas of assessment: eye opening, verbal response and best motor response. The lowest score is 3, the highest 15.

Response	Score
Eye opening	
Opens eyes spontaneously	4
Opens eyes in response to speech	3
Open eyes in response to painful stimulation (eg, endotracheal suctioning)	2
Does not open eyes in response to any stimulation	1
Motor response	
Follows commands	6
Makes localized movement in response to painful stimulation	5
Makes nonpurposeful movement in response to noxious stimulation	4
Flexes upper extremities/extends lower extremities in response to pain	3
Extends all extremities in response to pain	2
Makes no response to noxious stimuli	1
Verbal response	
Is oriented to person, place, and time	5
Converses; may be confused	4
Replies with inappropriate words	3
Makes incomprehensible sounds	2
Makes no response	1

Categories of Head Injury

Head injuries can be categorised according to GCS. Patients in coma are defined as having no eye opening (E = 1), no ability to follow commands (M = 1 to 5) and no ability to form words (V = 1 to 2) i.e. patients with a GCS of 8 or less are in coma.

- GCS ≥8 Severe Head Injury
- GCS 9-12 Moderate Head Injury
- GCS 13-15 Mild Head Injury

Immediate Management for Severe Head Injury

Patients with a GCS of 8 or less on admission have a severe head injury and are in coma. Immediate intubation and ventilation is required before more detailed investigation can take place. The following guidelines may seem obvious to some, but this does not diminish their importance.

Patients with a GCS of 8 or less require urgent intubation and ventilation to reduce unacceptably high risks of both aspiration and secondary brain injury.

Common Pitfalls

1. Beware of associated cervical spine injury. Have an assistant perform in-line stabilisation.

2. Perform a rapid airway assessment to ensure that you are confident that you can intubate the patient without problems. Failed intubation may involve a significant secondary insult to the brain. A delay to summon senior help may be justified if the airway can be maintained.

3. A full rapid-sequence induction is mandatory. Suxamethonium is known to elevate intracranial pressure but the risk is outweighed by those of aspiration and failing to achieve optimal intubating conditions.

Rocuronium proves a much better alternative, See Airway Management Chapter.

4. Ventilation must be assisted after intubation. Adequate sedation and paralysis with an appropriate non-depolarising relaxant are mandatory at this stage. A mechanical ventilator should be used. Blood gases should be checked as soon as possible.

5. End Tidal CO_2 must be used, to confirm intubation, ensure adequate ventilation and guard against disconnection.

Resuscitation Targets

In the early phase of resuscitating the head injured patient, it may not be fully apparent what their injuries are. This makes it difficult to decide what blood pressure to try and achieve. As outlined elsewhere, aggressive fluid therapy before haemostasis may be detrimental. However, hypotension is detrimental to head injured patients. With this in mind, a compromise systolic blood pressure of 100mmHg should be achieved early on. Once haemorrhage has been controlled, or found to be minimal, a higher blood pressure can be sought, based on the CPP target. E.g. adult patient, target CPP is 70mmHg, assume an ICP of 30mmHg, produces a target MAP of 100mmHg. Achieving this may well require the use of vasopressors or inotropes.

Continued Management

For patients with isolated head injuries, further investigation and management is directed by a protocol based on their initial and subsequent performance in repeated neurological assessments. Patients with multiple injuries must undergo resuscitation and stabilisation before any transfer for imaging can take place. This may include going to theatre for life-saving surgery (e.g. splenectomy).

CT Scanning

All patients in coma, as well as those with focal neurology, or with open injuries will require CT scanning. Intra-hospital transfers to the scanner should be regarded in the same way as transfers out of the hospital, and carefully planned. Full anaesthetic monitoring should be applied to the patient and, during the scan, the anaesthetist must be positioned where he can see both the patient and the monitoring clearly. Restless or agitated patients may not tolerate scanning without sedation to keep them still. This must be accompanied by intubation and ventilation.

Head injury patients must not be sedated without a definitive airway!

Neurosurgical Consultation

Following the results of scanning, all patients with severe head injuries will require consultation with a neurosurgeon. The duty neurosurgeon will need as full a picture as possible about the patient, and a successful consultation will require a telephone history containing the following elements:

- Basic patient details (age/sex etc.)
- Very brief relevant past medical history
- History of incident
- GCS at scene (if available)
- GCS on admission, pupils, focal neurology
- Other injuries
- Initial Management
- Basic investigation results (bloods, gases, c/spine films)
- CT scan results
- Current neurological status
- If possible, transmit the CT images to the neuro-centre as soon as they are available (most DGH x-ray departments can do this).
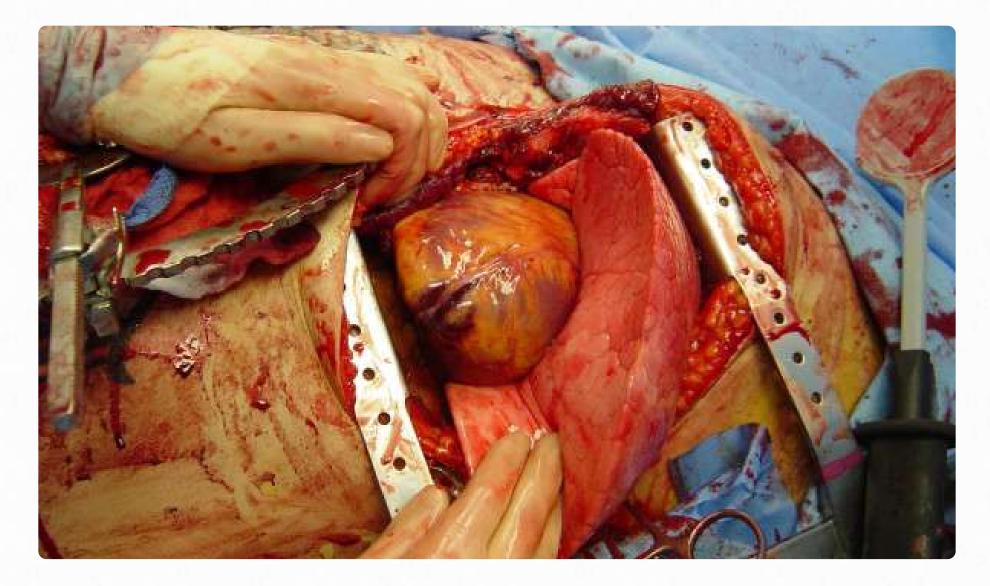
Summary

In Patients with Head injury:

	Primary	Secondary
Blood Pressure	>100mmHg systolic	CPP >70
Oxygenation	SpO2 >95%	PaO2 > 13 kPa
Ventilation	Clinically Adequate	PaCO2 4.5 kPa

• Management is directed towards prevention of secondary brain injury

• Initial Management, as always, is driven by an ABC approach

• Repeated and systematic neurological assessment guides further management

• Rapid and safe transfer for surgery improves outcome in patients with focal lesions.

• ITU management should be guided by maintenance of an adequate Cerebral Perfusion Pressure.

Traumatic Cardiac Arrest

Cardiac arrest associated with trauma is associated with a poor outcome. Blunt traumatic arrest is worse than arrests as a result of penetrating trauma.

Typical survival rates quoted range from 0-3.7%. However, the Royal London HEMS team has had greater success with a more aggressive approach to early resuscitative thoracotomy (RT). In 2006 Lockey et al published the following data:

• 81% died out of hospital or in transfer

But:

• **14% survived to discharge from ED**

• **7.5% survived to discharge from hospital**

This can only be achieved with early surgical intervention, which may include the ED or even pre-hospital. As the years have passed, HEMS have modified and simplified their approach to give the best chance

of a good outcome to the inexperienced provider. They are also lowering their threshold for RT and their results continue to improve.

Audit data, HEMS London, G Davies, 2013

	Penetrating Trauma	Blunt Trauma
Best Survival Rates	60%	10%
Average Survival	40%	2%
Overall Survival	For All Groups 10%	

As you can see from the table, the survival rates are hugely variable. A patient with a single penetrating stab wound, and deterioration from a tamponade, managed early in the ED with RT has a good chance of survival, as compared to casualties who collapse from blunt trauma. Overall, the survival rate is 10%, which is still a significant improvement on previously reported data.

ONE THING THAT IS VERY CLEAR IS THAT THIS IS NOT ALS!

So when should we consider Resuscitative Thoracotomy?

We need to try and identify the casualties who will have the best chance of survival

and the best time to perform RT. The current indications include:

- **Witnessed loss of output following penetrating trauma**
- **>1500mls from a chest drain or thoracostomy**
- **Persistent BP <70mmHg despite resuscitation**

Other things to consider:
- Trauma Hospital more than 10 minutes away following pulse loss
- If there is loss of output 'at scene' then it is usually too late to commence RT in the ED
- Having a rhythm but no output improves outcome
- Good for abdominal/chest junctional injury
- Knife injury better results than guns

Other factors that improve outcome:

- Tamponade physiology has the best outcome
- Theatre >ED>prehospital
- Cardiac versus lung injury

- Single chamber injury versus multiple

- Right ventricle rather than left

Acknowledgement for much of this material must be made to the Faculty of the PERT (Prehospital Resuscitative Thoracotomy) course, Royal College of Surgeons, London.

Practical approach
Before commencing a thoracotomy the airway must be secured with an endotracheal tube or a surgical airway. Other respiratory causes of reduced or lost cardiac output, in particular tension pneumothorax should have been excluded with bilateral thoracostomies. These form the basis for our incision to fully open the chest. Taking this approach of 'joining up the incision that you have already made' has been established and promoted by HEMS London, as it significantly reduces the operator stress in performing this procedure.

We are not recommending lateral thoracotomies as for anyone who does not regularly perform thoracic surgery, this provides very limited exposure and access, which makes the procedure far more challenging and likely to be futile in inexperienced hands. We use the 'clamshell approach', which provides a much better view of the thoracic contents and also a better chance of control by non-cardiothoracic surgeons.

Opening the chest – 'clamshell' approach
The thoracostomies should be in the 5th Intercostal spaces and in the mid-axillary line.

Take a skin marker and draw a line on the skin, along the rib space anteriorly to the sternum, across it and up the other side to the other thoracostomy. (By doing this, it makes it easier to determine the incision when cutting and also avoids crossing ribs, which will make cutting more difficult and also leaving sharp rib end exposed.)

A scalpel is used to incise the skin and subcutaneous tissues along this line, down to the ribs. The incision is then complete, full thickness with large scissors or even Tuff-cut shears if necessary.
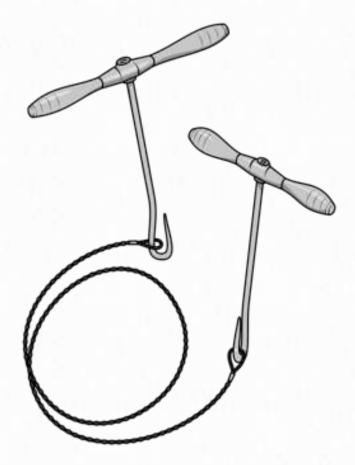
The sternum can easily be cut across with the shears, or alternatively with a Gigli saw if you have one and know how to use it safely.

Gigli safety tips

Keep your hands wide apart when sawing rather than close together in a vertical motion. Place forceps or a clip on the sternum across the cut line to stop the wire flicking upward once through the bone

Once the incision is complete, use your fingers behind the ribs to push down any tissue/adhesions behind and to guide the scissors to prevent cutting any other structures especially with the pericardium behind the sternum

We should now be able to fully open the chest superiorly and inferiorly in a 'clamshell' fashion. This should provide full access to the anterior heart, lungs and descending aorta

If you have a rib spreader, insert this on the patients RIGHT side so you do not obscure access to the heart or compress the thoracic aorta.

If more exposure is required, then simply extending the incisions more posteriorly will allow the chest to open wider and increase access.

Opening the pericardium

Once in the chest, it may be immediately obvious that the pericardial sac is full of blood, that the heart is not beating or looks empty. We must now open the pericardial sac to facilitate management of cardiac wounds or to provide effective internal cardiac massage.

The pericardium is a tough, inelastic layer, which can be 'picked' up with forceps over

the anterior surface of the heart to incise. The incision should be made with scissors, as an inverted 'T'.

The Phrenic nerves are postero-laterally located running across the pericardium and should be identified and avoided if possible.

If there is a tense pericardial tamponade, this will relieve the pressure and the patient's clinical condition may rapidly improve, but we will still need to identify the source of this blood.

Once the pericardial sac is opened, slide your hand around the heart inside the pericardial sac, to completely free the heart.
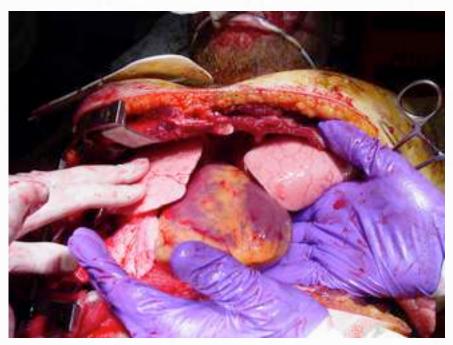
DO NOT DELIVER THE HEART OUT OF THE CHEST AT THIS STAGE

At this stage you should be able to feel how 'full' the heart is and if hypovolaemia appears to be the problem then you can gauge the response to intravenous filling. When handling the heart, try and keep it flat to avoid blocking aortic outflow.

Open/Internal cardiac massage

The pericardium must be released to do this effectively. Slide your hand behind the heart and the other across the front and with FLAT hands, gently compress the heart in a 'clapping' manner, squeezing from the apex upwards to mimic the normal output/ejection of the heart.

ALWAYS USE TWO HANDS AND KEEP THEM FLAT TO AVOID PERFORATION

(Ref: http://www.trauma.org/archive/atlas/clamshell.html)

Managing holes in the heart

One of the commonest reasons for opening the chest is to manage a penetrating cardiac injury and the associated cardiac tamponade.

Many wounds may be immediately visible when the heart is first exposed. The simplest management of this may be to simply put your finger on the hole. If control is ade-

242

quate or more experienced help is not far away, then this may be all that is required to preserve the patients life until help arrives or you get to theatre.

If a hole has not been identified, but is still suspected then we must search the back of the heart as well. To do this we must gently 'deliver' the heart but without significantly kinking the great vessels and thereby totally compromising the cardiac output.

The safest way to achieve this is to take a swab or two, opened out and then scrunched up, which can then be gently slid under the heart from the apex, almost as a mattress to elevate the heart out of the chest, without forming an acute angle at the vessels.
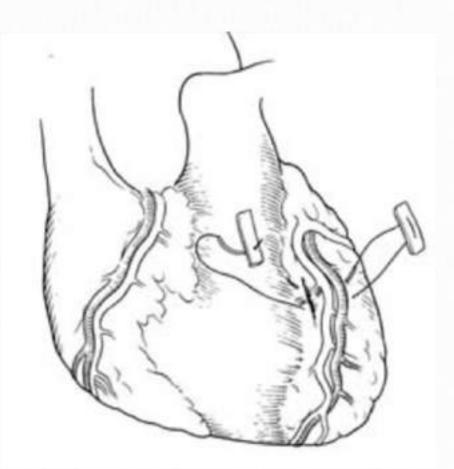
If you need to close the hole then staples or a simple stitch can be enough. Silk tends to cut out so use 4/0 Prolene is recommended, taking a good bite either side of the muscle wall.

BE CAREFUL NOT TO OCCLUDE ANY OF THE CORONARY VESSELS

If the hole is adjacent to a coronary vessel then we must use a mattress suture technique.

For large holes some recommend using a Foley catheter but this can be fraught with problems. Firstly, it can be difficult to get the catheter through the hole, when the balloon is inflated it can make the hole larger or fill the chamber, compromising cardiac output. Finally, blood will obviously come flowing down the catheter, which must be clamped. This obviously does offer a route for filling the heart rapidly and in such cases it should ideally be secured with a purse-string suture. In view of these issues, we would not recommend this approach for the inexperienced clinician.

One of the best ways to close and seal holes, if time and patient/environmental conditions allow is to use a small pledget, sutured over the hole. Surgical material can be used, but a small patch of pericardium is ideal for the purpose. This is because of the relatively lower tensile strength of myocardial muscle in compari-

son to skin, which means sutures may tear though the tissue rather than opposing the edges and make the hole bigger, due to the inflammation and weakness of the tissue. This should be sutured as shown in the diagram below.

An alternative for the non-surgically trained would be to use a surgical stapler to close the cardiac wound.

Ventricular fibrillation

If the heart starts fibrillating first ensure that it is adequately filled. Manage any holes in the heart losing blood before attempting to cardiovert the rhythm.

Check the coronary vessels as injuries may seriously compromise blood supply to the heart and inevitably cardiac function and arrest.

'Flick' the heart over the ventricles and it may start to beat effectively again.

Next consider any other reasons for irritability of the heart e.g. acidosis, low calcium levels, potassium abnormalities and hypothermia.

Internal defibrillation requires internal defib pads, which may not be available. If that is the case then simply close the chest by removing the spreader and pulling the ribs together and use external pads. If not successful then re-open the chest to perform internal massage.

Lung Injuries

The lungs can be injured in conjunction with the heart or in isolation creating major bleeding or a major air leak, both of which are potentially fatal. To manage the injured lung we must first mobilise it by releasing the inferior pulmonary ligament. Slide a hand down the inside of the chest to the base of the lung. It is often very hard to see the ligament so it is necessary to dissect it blindly. Once released the lung should lift out of the chest easily on its hilum.

Rapid Emergency Measures

It may be necessary to immediately get control of the lung injury with a rapid temporising measure. There are a number of ways that we can achieve this:

• Lung Squeeze: squash the lung down into a ball of tissue, almost like scrumpling up a piece of paper and then hold it in this position

• Hilar compression: simply get your hand around the hilum and squeeze it to compress the bronchus and its accompanying vessels.

• Lung Twist: take the lung and twist it on its hilum until the bleeding or air leak stops

• Ligature compression: a urinary catheter or tape tie can be passed around the hilum and then clipped and twisted like a windlass until it has the desired effect.

When ventilated with the chest open, holes in the lung are rarely a major problem in the emergency stage. However, if necessary they can be managed with a surgical linear stapling device (e.g. GIA, TLC, ATS). Put the anvil behind the lung, lock in the staple prong then close the stapler compressing the lung across its full width ide-

ally, isolating the hole. When the stapler is fired it will resect and seal the lung

Blunt Traumatic Cardiac Arrest

These are typically as a result of catastrophic head injuries or massive blood loss. Patients that are initially alive, with a pulse, who subsequently rapidly lose their output, may be salvageable with an emergency thoracotomy.

Once in the chest an obvious cause may be present e.g. cardiac tamponade or alternatively, it may be clear that the patient is massively hypovolaemic, but this can be tempered by compression of the aorta.

Compression of the Aorta

Compression of the thoracic aorta in the chest may provide an opportunity to restore the cardiac output to the brain, heart and lungs, while we control bleeding in the lower half of the body, restore some filling or get to operative control.

Cross-clamping the thoracic aorta is a challenging surgical skill in the emergency thoracotomy situation and therefore not the recommended technique. A far simpler option is to follow the ribs of the left chest posteriorly, behind the lung, until you reach the vertebrae. Find the soft tissue in front of the vertebrae, which will contain the aorta, oesophagus and other tissues. Use

your fingers to compress the soft tissue back against the bony vertebral bodies. This is far easier to do and likely to be more effective, without damaging other structures, than attempts at cross-clamping.

Once a degree of control is obtained, we can restore some filling and catch up the severe hypovolaemia.

Restoration of cardiac output

Once output is restored bleeding may start from numerous vessels and tissues. Much of this can be ignored but some vessels such as the internal mammary vessels on the anterior chest wall, may require ligation with either clips or ties.

Remember that the casualty may also even start to 'wake up' and now require some sedation for transfer to hospital or theatre.
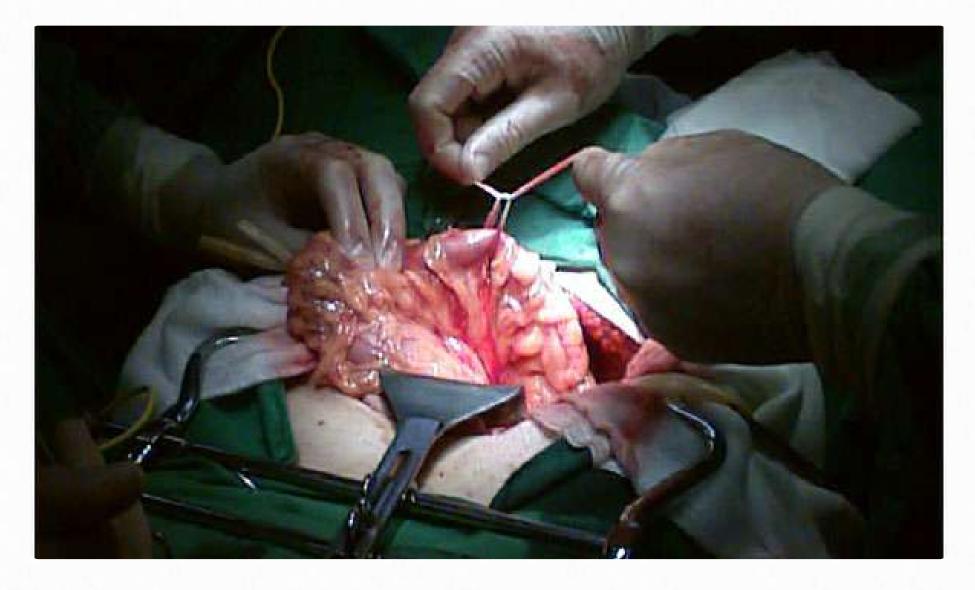
Acknowledgement

The tips and details included within this chapter are drawn from the Royal College of Surgeons PERT (Pre-hospital and Emergency Resuscitative Thoracotomy) course. The ATACC Faculty would highly recommend this course to anyone involved with the immediate resuscitation of casualties in the pre-hospital environment or resuscitation room.

5
Ongoing Trauma Care

"I would rather be kept alive in the efficient if cold altruism of a large hospital than expire in a gush of warm sympathy in a small one"

– Aneurin Bevin

Initial Approach to Damage Control Resuscitation

Life threatening major trauma is a huge insult to the body and is often described as a 'major hit' or physiological stress. In attempting to repair these injuries we must consider the impact of surgery as a further trauma or 'second hit'.

For isolated traumatic injuries or minor polytrauma then definitive repair and a standard surgical approach is still the recommended solution. However, for more extensive, life-threatening trauma the poten-

tial 'second hit' of extensive complex surgery is not the ideal plan.

Traditional Approach to Surgery

Traditionally, for all traumatic injuries it was considered best to 'stay and play' in the operating room, to manage all injuries as early as possible in a single sitting if possible. This approach aimed to minimise the number of trips to theatre and the number of further traumatic physiological insults. Wherever possible a definitive repair would

be attempted, although it was already appreciated that in cases of major abdominal contamination, with faecalent gut contents, a primary anastomosis was destined to fail and the bowel was better 'defunctioned' in some way. Traditionally this would involve formation of a stoma, however this in itself can produce problems and a more recent adaptation is a move towards temporary blind end stapling of bowel.

Further similar changes have been developed as there was an increasing realisation that in casualties with multiple serious injuries and high Injury Severity Score (ISS, >15), the traditional approach is far from ideal and badly tolerated by even the fittest of individuals as it effectively represents many hours of further ongoing trauma.

As a result, such complex or extensive trauma, that is not amenable to a simple surgical repair, will require a more extreme or radical approach with some significant compromises. This is the principle of Damage Control.

Most people will have heard of damage control and many will associate it with the Apollo 13 mission, where the astronauts on board looked unlikely to make it home

to earth following a serious of apparently catastrophic incidents on the vessel. However, through some ingenious calculation NASA Mission Control and the Crew shut down all non-essential systems on the spaceship to provide the bare minimum of power necessary to get them home. Without such extreme action they would not have survived to get back to earth.

That said the concept of damage control is not new and was first adopted by the US Navy in the 1940's during the Second World War. After losing several large vessels such as battleships and aircraft carriers whilst attempting repairs at sea, where they were vulnerable to further attack, they realised that they must take some drastic measures.

They established damage control teams on all of the ships, who would be immediately deployed to any serious damage or breach of the hull. These teams would train and be equipped and prepared to 'plug the hole' or manage the fire in whatever way they could, as quickly as possible. The results were often not be pretty and may leave parts of the ship closed off with watertight doors, but it would still be afloat and could limp back to port for definitive repairs. By taking these simple

measures [many of the damaged vessels were saved](#) and returned to full active duty.

Damage control in the US Navy

Attempts to restore normality will fail. Attempts at a definitive repair will fail. Your ship will sink!

History of Damage Control Resuscitation

In medical terms 'Damage Control' was probably first performed by Pringle in 1908 when he 'packed' a bleeding liver during surgery, but he did not actually describe it as such. However in 1993 [Rotondo and Schwab](#) described the technique and broke it down into three phases:

- Damage Control 1 – Early laparotomy

- Damage Control 2 – ICU/resuscitation

- Damage Control 3 – Definitive repair

Damage control 1 – early laparotomy

Rotondo and Schwab both identified that many patients with major polytrauma and life threatening bleeding simply need to go straight to the operating theatre for laparotomy or other live saving surgery. Any delay for further investigation or resuscitation will result in serious deterioration or death.

However they also realised that this early surgical intervention was only a part of the process and this must be supplemented with early intensive care support following a short 'damage control' procedure.

Damage Control 2 – Intensive Care and resuscitation

After the immediate life saving surgery has been performed there is a need to urgently transfer the patient to intensive care to address any ongoing state of shock or hypovolaemia. The patient is likely to be cold, under perfused and acidotic.

It is important to note a significant change in that 'resuscitation' comes after laparotomy in DC2. This is very different to the traditional approach of resuscitating all patients before inducing anaesthesia or op-

erative procedures. However, this approach is essential for patients that are actively bleeding large volumes, who will not respond to resuscitation in any sustained fashion and who will simply bleed even more with any attempt at resuscitation with volume replacement alone.

This approach is the current concept of permissive hypotension and rapid transfer to theatre for bleeding patients, whilst accepting that the patient is left in a state of shock to varying degrees until haemorrhage is controlled.

Damage Control 3 – Definitive repair

After a period of resuscitation and subsequent stability in the intensive care unit a suitable opportunity must be taken to return to theatre to perform a definitive repair, which may include removing packs, closing abdomens, anastomosing bowels etc. The timing of this stage is critical and the best time remains unclear and is dependent on many factors, including the condition of the patient

It was some years later in 2001, before Schwab published a supplemental paper on Damage Control surgery, in which he described another phase.

Damage Control Ground Zero – pre-hospital

They had realised that the principles of Damage Control Surgery must start much earlier, at the earliest possible time after the initial injury has occurred.

What had become very clear from their research and work was that many 'traditional' approaches to trauma, resuscitation and surgery were actually making casualties worse. Be it from increased bleeding, delays getting to theatre, prolonged procedures or futile resuscitation many of the problems that developed resulting in increased mortality and morbidity were in fact iatrogenic. The approach that they described was a radical departure from traditional surgical practice and would take some time to adopt and become popular.

'It's all about saving lives without making them worse!'

The opinions of Rotondo and his colleagues were further supported in more recent years by the work in Iraq and more recently Afghanistan. In 2008, Surgeon Commander John Holcomb, US Army, as he was then, made a profound statement:

'Damage control resuscitation represents the most important advance in trauma care for hospitalised civilian and military casualties from this war'

Surg Commander, J Holcomb, 2008

As you can see from this statement by Holcomb, we are not just talking about Damage Control Surgery, as there has been a realisation that there is a far bigger picture than just the operative technique. All aspects of casualty management and resuscitation from the scene through to final definitive care must be considered and as such the terms 'Damage Control Resuscitation' or 'DCR' has now been adopted to encompass all aspects of care.
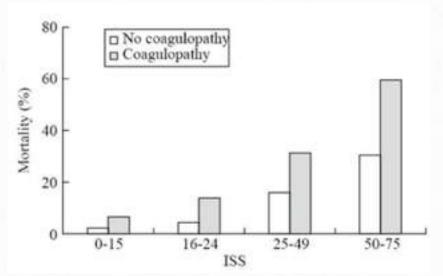
An absolutely fundamental part of this concept is a realisation that we are trying to 'restore normal physiology' as quickly as possible and 'not normal anatomy' as that can come later when the body will cope with more extensive surgery.

This is a real challenge for everyone, practically but also in changing mind-sets, especially the surgeons because as Stone & Rotondo pointed out 'it goes against normal surgical practices' - and no one likes to leave a procedure 'half done'.

However, there is now an increasing appreciation that there is no place in major trauma surgery for liver or complex bowel resections or complex corrective orthopaedics and surgery should ideally completed within one hour. Inevitably this makes the surgery as challenging as managing the resuscitation and coagulopathy etc.

When do we start Damage Control?
The process of damage control needs to start much earlier than the operating or resus room, it must start as soon after serious injury as possible and it is both a mind-set and practical techniques.

We know that patients nearly 30% of patients who arrive in the ED with major trauma will already be coagulopathic and this is associated with a poor outcome. There is a clear direct relationship between Injury Severity Score and Coagulopathy and with mortality. The worse your coagulopathy, the higher your mortality, as this graph demonstrates.

Now that we have started to identify the necessary methods in DCR, the greatest dilemma is when to adopt this approach, as it is clearly a serious compromise and for many patients it represents an unnecessary risk and even potentially a serious insult to their physiology.

For the majority of our patients who do not have major traumatic injuries with life threatening bleeding or compromise, a definitive care approach is likely to be the better option. So we need to identify the patients that might 'look good' initially but who clearly will deteriorate later and ideally will have a DCR approach adopted as early as possible. This may also allow us to identify the patients who are likely to require massive transfusion as they are often the same.

So what are the key triggers or indicators that we can use to identify patients early who are likely to require Damage Control Resuscitation?

Indicators for Damage Control Resuscitation:

Kinematics or mechanism

Simply by considering the mechanism of injury we may have sufficient concern that injuries are likely to be severe or life threatening enough to warrant DCR. Examples would include a high speed pedestrian RTC, fall from considerable height, massive crush injury, blast injuries etc.

This may all sound a little vague and it is largely based on opinion rather than hard science, but we must consider what has happened to the individual and use a degree of common sense plus clinical experience.

Hypotension

If the casualty presents with a Systolic blood pressure of less than 90mmHg, this is indicative of a 40% blood loss and also indicates a degree of failure to compensate for any further losses.

These patients are highly likely to need speedy transfer to hospital and usually theatre. They will also require a skilled anaesthetic induction with appropriately reduced doses of carefully selected agents.

Where these patients require a transfer, for example from a Trauma Unit to a Major Trauma Centre, this introduces a further challenge as unlike many other critical care transfers, this patient is potentially unstable and actively bleeding. This will introduce the challenge of performing damage control resuscitation from scene to hospital in an ambulance or helicopter.

Haemoglobin

If on the first measurement of haemoglobin, which could be taken on scene with an iStat or as a venous blood gas from the first samples drawn on arrival in ED, the level is < 11g/dl then this is due to acute blood loss until proved other wise and is a key trigger for considering DCR.
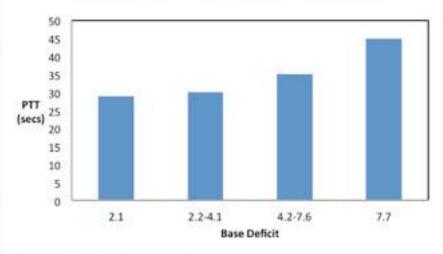
Acidosis or Base Deficit

If during the initial resuscitation phase of trauma care either pre-hospital or in the ED there is a Base deficit > 6 then this is an indication that this patient is in shock and is likely to need early transfusion.

Such a base deficit on initial assessment is also an indication that this patient is at increased risk of developing multiple organ failure, increased time in ICU and mortality. Any such acidosis is also associated with increased coagulopathy as can be seen on in the graph in the next column.

The effects of acidosis on coagulation are greater than hypothermia but both effects are commonly present in major trauma patients, which further increases the severity of the trauma induced coagulopathy.

Effects of Increasing Base Deficit (acidosis) on Coagulation

It is important to note that the based deficit will usually rise before the classic signs of hypotension and decompensation.

Temperature

A temperature of <35 degrees on initial presentation is associated with an increased mortality. As discussed above it is often also associated with under-perfusion of tissues with the inevitable acidosis.

As such, hypothermia should be considered one of the indicators to adopt a DCR approach in major trauma and should be treated aggressively if present.

We should remember that coagulation like many other body processes is an enzymatic process that slows as temperature

falls and will ultimately stop as core temperatures fall to 32-33 degrees.

Pre-hospital Damage Control Resuscitation

A key element of pre-hospital damage control must be speed. This does not mean unnecessary compromise or rushing but rather a degree of urgency and avoiding the desire to 'stay and play', perform unnecessary procedures or take too long with an extrication. Simply by instilling this sense of urgency within the team will usually be enough to facilitate the rapid transfer to hospital. At times, injuries may warrant a more rapid approach both on scene and early in the time in hospital.

All examination, procedures and time spent should be justified in your mind and we should constantly be considering, is this the time to leave or could this be done during transfer.

Circulation preservation must start early with good external haemorrhage control. All external haemorrhage should be actively and aggressively controlled and no one should die of external haemorrhage under normal circumstances.

We should also consider the possibility of internal bleeding and use gentle handling, minimal rolling and apply pelvic binders to anyone with the potential to have pelvic injury. Tranexamic acid (TXA) can wait and be delivered on the way to hospital to avoid delay. Its effect is more pronounced if given sooner, but it is still beneficial if treatment is started up to three hours post injury.

Keep the casualty as warm as possible to minimize the effects of shock and coagulopathy and avoid any unnecessary or excessive intravenous fluids (titrate to a radial pulse).

If there are obvious signs of major bleeding and hypovolaemia then we should consider blood products as early as possible. Few organisations other than HEMS London currently carry blood products pre-hospital but we can all have procedures in place for a 'CODE RED' standby, where the MTC massive haemorrhage protocol can be activated from scene to ensure that blood and blood products are ready and available at the earliest possible opportunity following the incident.

Damage Control During Transfer

Most clinicians will be used to transfers of critically ill patients, between hospital intensive care units. These patients will have been stabilised, sedated, ventilated and monitored before leaving the safety of the

base hospital. We would never consider embarking on an Intensive care transfer with an actively bleeding patient as they should be stabilised before we leave, wherever possible. ATACC believes that on a good ITU transfer with properly prepared patient, the clinician should have to do very little if anything during the journey.

Trauma transfers from scene or between Trauma Units and Major Trauma Centres are potentially very different. We need to adopt a different perspective and accept that our patients may well be unstable during the transfer and need active treatment and resuscitation en-route.

Experienced paramedics, air ambulance crews and MERT teams have all demonstrated how much can actually be performed during a transfer. In fact MERT teams in Afghanistan have taken this to an extreme level working in low light, in the confined spaces of the back of a very noisy helicopter, which maybe making evasive manoeuvres with a number of critically ill patients on board. What they manage to achieve through training, team-work and skill is truly remarkable and sets the bar for us all.

Some may criticise the levels of intervention during such transfers, but the results speak for themselves, with some remarkable outcome and survival figures being delivered by these teams in the face of some catastrophic and typically fatal injuries. Damage control, speed and an impressive transfer capability are all a part of this success story.

In civilian practice we typically take a more conservative approach and the environmental challenges may not be as great. However, we can still modify and improve our practice and learn by considering what has been achieved by others.

Many factors will affect what you can deliver on scene or en-route. Some procedures will be limited by space, others by vibration, movement, noise, your assistance, your technical skills and many other factors. It is impossible to produce an absolute list but typically we would expect the external haemorrhage and the airway to be controlled before leaving the scene. Whereas cannulation, splinting, analgesia etc may, in some circumstances, be performed en-route. Each case must be considered against your situation and a decision made and that can be challenging, but should always be based on safety or both the casualty and your team.

"A and B on scene, C and D en route"

In simple terms, do what MUST be done and what is likely to be difficult in transit and then get packaged and moving. This approach has recently been termed 'Scoop and treat'. Major trauma transfers are far more labour intensive in terms of active assessment and intervention than critical care transfers.

In the Emergency department

Once at the hospital, it is essential that damage control continues, or else it commences at the earliest opportunity. We must keep moving with the patient as the management rarely stops in the resus room, but needs to move on to CT scan, theatre or wherever else is required. We don't stay and play at the roadside so don't in the resus room either. Some schemes have a pre-arranged protocol to bypass resus altogether when arriving at a hospital if the pre-hospital team requests an immediate transfer to CT. If this is not possible on arrival in the Emergency Department (ED), ask yourself the same questions in resus as on scene:

- Do we still need to be here?

- Is this procedure essential or necessary now?

- Are we delaying something more important?

A classic example of this is the anaesthetist who insists on having an arterial line and wastes valuable minutes attempting a line in a patient who is actively bleeding and needs to get to theatre urgently. There is no doubt that an arterial line is a valuable monitoring tool, but which is more important in the actively bleeding patient needing urgent surgical control?

We need a rapid primary survey, manage the life threatening injuries and then make decision about the next course of action. This may be immediate transfer to theatre, secondary survey and investigations or transfer to CT. If in a Trauma unit we need to contact the MTC Team Leader and agree and action plan or transfer.

However, even in DCR we must ensure that the basic essentials are still done, re-assess external bleeding, ensure that the airway is patent and clear and that the patient is in the best possible condition before our next move. This does not mean physiologically normal, but just in a state that will survive to get to the next essential step. Blood must be drawn for standard tests and to get a snapshot assessment of haemoglobin and acid base status.
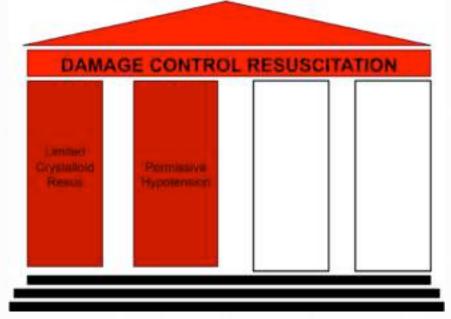
If we are not transferring immediately then we need an effective secondary survey

and evaluation of other injuries. Ensure that fractures are immobilised, analgesia, antibiotics, TXA and any other essential measures are considered.

Summary of Initial Approach to Damage Control

Damage control is a unique approach to injury. It cannot be adopted for the majority of cases because it involves a number of serious compromises, which are essential for the serious polytrauma, but would be considered an unacceptable for a less seriously injured casualty.

This is one of the greatest challenges with DCR - when to fully resuscitate a casualty and when to chose a DCR approach with all the compromises that are essential to 'get the ship home' and ensure survival. The DCR triggers in this chapter and mechanism of injury offer a useful guide as to when to adopt this radical approach.

Once we have chosen to adopt DCR we must adopt the key management pillars which form the basis of the technique. Two of these have already been described when considering the management of shock: limited crystalloid resuscitation and permissive hypotension but in conjunction with a degree of urgency, that keeps the patient moving through the trauma system

and ultimately to critical care to restore normal physiology at the earliest possible opportunity.

First 2 Pillars of Damage Control Resuscitation

References

1. Damage Control Surgery – Karim Brohi, Trauma.org, June 2000 http://www.trauma.org/archive/resus/DCSoverview.html

2. Apollo 13 mission, NASA mission log http://www.nasa.gov/mission_pages/apollo/missions/apollo13.html#.UxDw4EJ_uLM

3. USS Birmingham, Damage Control Teams – confidential report, 1943 http://www.researcheratlarge.com/Ships/CL62/BuShipsDamageReport82/

4. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR 3rd, Fruchterman TM, Kauder DR, Latenser BA, Angood PA. J Trauma. 1993 Sep;35(3):375-82; discussion 382-3
http://www.ncbi.nlm.nih.gov/pubmed/?term=rotondo+damage+control+1993

5. Evolution in damage control for exsanguinating penetrating abdominal injury. Johnson JW1, Gracias VH, Schwab CW, Reilly PM, Kauder DR, Shapiro MB, Dabrowski GP, Rotondo MF. J Trauma. 2001 Aug;51(2):261-9; discussion 269-71.
http://www.ncbi.nlm.nih.gov/pubmed?term=Johnson%20JW%255BAuthor%255D&cauthor=true&cauthor_uid=11493783

6. Increased mortality associated with the early coagulopathy of trauma in combat casualties. Niles SE1, McLaughlin DF, Perkins JG, Wade CE, Li Y, Spinella PC, Holcomb JB. J Trauma. 2008 Jun;64(6):1459-63; discussion
http://www.ncbi.nlm.nih.gov/pubmed/18545109

7. Evolution in damage control for exsanguinating penetrating abdominal injury. Johnson JW, Gracias VH, Schwab CW, Reilly PM, Kauder DR, Shapiro MB, Dabrowski GP, Rotondo MF. J Trauma 2001 Aug;51(2):261-9; discussion 269-71.
http://www.ncbi.nlm.nih.gov/pubmed/11493783

8. Resuscitative strategies to maintain homeostasis during damage control surgery Dutton R P British Journal of Surgery, 2012;99 (Suppl 1): 21-28

Trauma Team Receiving Care

In previous chapters we have discussed many of the key principles of immediate assessment and initial trauma care. New priorities have been identified with a shift towards earlier haemorrhage control. However, in the ideal world we will be working as a trauma team with a team approach, which allows a degree of simultaneous activity under the direction and coordination of the team leader.

The Trauma Team

The trauma team should ideally gather prior arrival of the patient. They should introduce themselves, put on their personal protective clothing and clear identification such as named tabards.

Some units have footmarks on the floor in the resuscitation bay, but there are not really necessary and as the patient arrives their will often need to be movement around the resuscitation area. However,

every team member should know their designated area to stand and their responsibility.

A typical trauma team will consist of:

- Team Leader (at the foot of the trolley initially)

- Anaesthetist (head end of trolley)
- ODP/Airway Assistant

- Emergency Medicine Physician (side of patient)
- Nurse 1

- Surgical doctor (opposite side of patient)
- Nurse 2

- Scribe (stood next to Team Leader)
- Orthopaedic Surgeon (stood within the trauma bay)

Other team members may be added as required e.g.

- Radiologist
- Radiographer
- Paediatrician

Only the Trauma Team should be within the red box and the Team Leader should ask anyone without a tabard to step back outside the line. A rough position guide is illustrated overleaf.
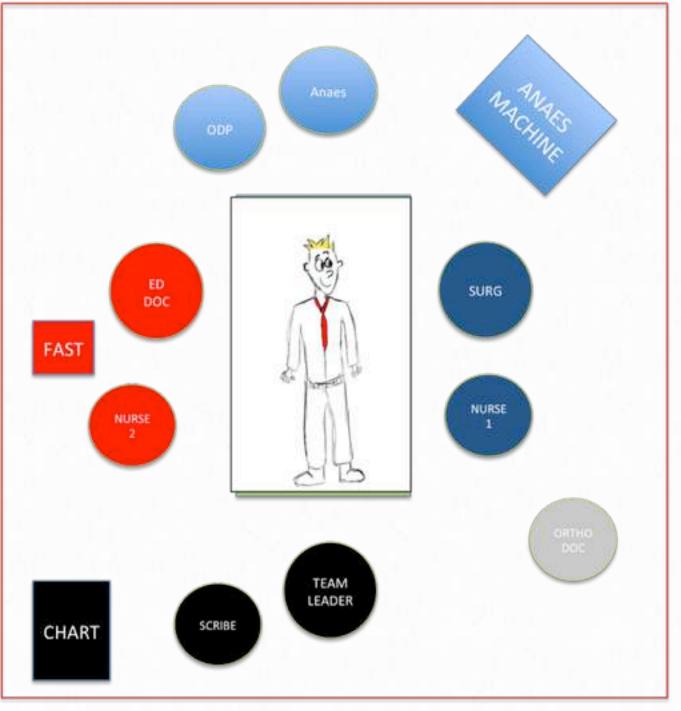
Before Patient Arrival

The team leader should ensure that everyone has arrived and then give a quick briefing of what the team are about to receive. This should also include any special details or requirements such as asking the surgeon to perform a rapid assessment of the abdomen if there is a suspected problem already reported.

Each team member should check and prepare any necessary equipment that they are likely to need and these should be open and prepared in the more major cases, with known injuries. Drugs should be drawn for analgesia, sickness or rapid sequence induction. Paediatric doses should be pre-calculated and recorded.

The Massive Haemorrhage protocol may need to be activated based upon the pre-hospital reports.

The CT scanner should be informed and the radiographer prepared

If necessary the Emergency theatre should also be given advanced notice of a poten-

Trauma Team Composition

tial immediate transfer to their department. The Team Leader should also make it clear to the team how the handover process will run, as there are a number of ways. We will describe one method, which we use on ATACC.

Patient arrival

As the patient is wheeled in on the trolley, the awaiting trauma bay should clear for the ambulance team.

1.   The Team Leader should identify himself or herself and ask who is in charge from the ambulance crew.

2. The ambulance crew will state:

   - The patients name

   - The patients age

   - What incident has occurred and when

Eg: 'This is John, who is approximately 25 years old. He was knocked of his bicycle by a heavy goods vehicle and he appears to have a significant head injury'

Photographs from scene are always useful to emphasise mechanisms

3. At this point the Team Leader will ask:

'Are there any immediately life threatening problems or concerns?'

If there are then these will be addressed immediately, if not then the team can proceed as normal

4. The patient is then moved into the bay and transferred onto the ED trolley

5. The ODP/Nurse will apply the monitoring while the rest of the team listen to the full MIST handover.

6. Once the handover is completed the Team Leader will give a short summary to the team and ask them to commence their roles, whilst highlighting any key actions required e.g.

"Anaesthetist – can you assess the airway and consider if RSI and intubation is required."

The ambulance crew should be asked to stay during the following primary survey in case there are more questions to ask them or onward transfer is required e.g. Trauma Unit to Major Trauma Centre.

Simultaneous Activity

7. We will now see a great deal of simultaneous activity whilst the team complete a Primary Survey, and evaluating anything suggested by the ambulance team:

Surgeon and nurse 1
- Responsible for Massive Haemorrhage & Internal Haemorrhage

- Will look for any signs of massive external haemorrhage then move onto a quick abdomen and pelvis examination (consider pelvic binder if not in place)

- Nurse will cut away all of the clothes covering the lower limb

Anaesthetist and ODP

• Responsible for Airway

• Will assess the airway, face & scalp and check the initial observations

  • -Oxygen sats

  • -heart rate

    • ECG

    • BP

    • Respiratory Rate

    • Temperature

    • Blood Glucose

In cases of major blood loss, the anaesthetist or ED doc, may prepare to insert a large bore infusion subclavian line e.g. a pulmonary artery catheter introducer (aka Swan sheath - use the injured side if there is obvious chest trauma)

Emergency Medicine Doctor and nurse 2

• Responsible for Respiratory Assessment and IV access

• Will assess the chest and breathing then the circulation

• Site IV cannula and draw blood

• ABG

• Move on to Full Secondary Survey

• The nurse will cut away all upper body clothing and then protect patient from getting cold or initiate active warming.

8. The venous blood gas is sent immediately to the department blood gas machine and will provide us with the necessary information to make decisions about Damage Control. This incudes as minimum:

• Acid- base status; pH

• Ventilation – pCO2

• Base deficit

• Lactate

• Blood sugar

• Haemoglobin

9. Lab bloods will also include: Urea and electrolytes, urgent x-match, full blood count, pregnancy test if female and clotting.

10. If a viscoelastic haemostatic essay (VHA) machine such as TEG or RO-TEM is available then get a small sample to test in the department, as soon as possible.

11. If during the chest assessment a thoracostomy or drain is required then the surgeon can perform this or alterna-

tively the Emergency Medicine Physician.

12. If intra-abdominal or chest problems are considered then a EFAST scan should be performed or a decision made to go straight to CT pan-scan or even theatre.

13. Analgesia, anti-emetics, Tranexamic acid and antibiotics should all be considered and administered as required

14. A second IV should be sited as soon as possible.

15. If not going immediately to CT scan then request a chest and pelvic x-ray

16. The Team Leader should make the decision to get the patient off the scoop/long board at the earliest opportunity.

There is much debate about log-rolling and the risks of dislodging pelvic or intra-abdominal blood clots, but if we don't then we will not be able to assess the back properly for wounds, spinal injuries and a PR examination. The Team Leader should make a balanced judgement on this for each case. See the spinal chapter for more information.

17. The patient should now have been fully exposed and most potential injuries identified, but ensure that they are kept as warm as possible

The Scribe

18. Throughout this period the scribe should be hanging on every word of the team leader and writing down any facts specifically directed for recording.

Fluids and resuscitation

19. If fluid is required then warmed crystalloid is given in 250ml aliquots and blood requested and started as early as possible (O-neg if in the fridge)

If necessary activate the Massive Haemorrhage Policy with the laboratory.

Sit-rep

20. All key findings, positive or negative must be fed back into the team leader who will assimilate the information and may ask more questions or define specific actions. There should only ever be one person talking if possible, or else other important information will be missed.

21. TIME OUT: at this point the Team leader should call a 'time out' and up-

date the whole team with a situation report or 'sit rep'.

22. Team Leader will now make a decision about the next steps in the pathway and this may well involve discussion with the other expert team members. This may include calls to the Major Trauma Centre if we a re in a Trauma Unit

23. Damage Control Resuscitation?– if this is major trauma then it must be clearly stated that we are following a full damage control approach to resuscitation

Scoop and run or stay and play?

24. If there are obvious life threatening injuries then the team may immediately package the patient for transfer to CT/Theatre/Angio etc

25. Alternatively we may package for immediate onward transfer to the Major Trauma Centre if we are in a Trauma Unit

26. If the casualty is more stable then we can proceed to a full head to toe assessment

27. A urinary catheter should be considered and inserted if there is no contra-

indication. Consider a supra-pubic if there is a problem.

Observations and records

28. Throughout all observations and records should be updated, at least every 5 minutes initially and more frequently if the team leader asks.

29. This should all be recorded in the Trauma Pathway documentation

Other injuries and assessment

30. This is a good time for further assessment by other team members e.g. orthopaedics, radiology

31. Orthopaedic surgeon - Any fractures with major bleeding, pulseless limbs, obvious long bone or pelvic fracture and open wounds should be reviewed by the orthopods as early as possible (usually called in by the Team Leader, when first identified) – these may require urgent splinting or dressing to reduce major blood loss and infection risk

32. More minor fractures and injuries can now be reviewed after the full head to toe assessment

33. Radiologist – if there is uncertainty about something then a departmental

ultrasound scan may be considered if the radiologist is present. More commonly, the patient will move direct to CT

Definitive Care plan

34. At this stage the Team leader and Trauma Team members should be able to make some clear decisions about where this patient is heading and for what kind of interventions

Summary

There are many ways that this can be written and directed and this is just one approach. The constitution of your team the capabilities and skills of your prehospital team and your hospital designation will all have a major impact on how this process works.

As such, what we have aimed to do here is to demonstrate that a logical process should exist that can be simulated and practiced to create and effective Trauma Team with well defined roles that work in your department.

Throughout any Trauma Team activation we should be constantly aware of the clock ticking and considering where we are going from here. This is ultimately down to the Trauma Team Leader who relies on each team member playing their

part effectively and comprehensively, avoiding distractions in other areas or parts of the casualty. Everyone plays a key part and anyone has the potential to 'drop the ball' by failing to complete their designated task.

When preparing your trauma reception and your team, remember the performance of the Formula 1 teams, with a small elements coming to together for a fast and safe 'pit stop'.

Analgesia

Whilst the the need to treat pain for humanitarian and physiological reasons is entirely obvious, the evidence is that pain is managed poorly and disjointedly at every stage of a trauma patient's journey. Who is responsible for pain management now? Who is responsible for pain management in the next few hours? Who is responsible for ongoing pain management? Is there an overriding plan and will any interventions now limit analgesic options later (e.g. anti-coagulation and regional anaesthesia)?

Assessing Pain

Pain, according to the International Association for the Study of Pain, is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. There are multiple modalities of pain relief which can be utilised, however the trauma patient poses some particular challenges that may limit the way in which we can employ them.

The dogma that analgesia should not be given lest it mask subtle surgical signs or cause gross haemodynamic instability has been debunked and is not supported by the current evidence base, however it is still a view held by some clinicians. The provision of not just adequate, but good, pain relief is not only a humane aim but also will decrease the stress response and the long term psychological sequelae of injury.

The requirement for analgesia may also vary between injuries and between individuals - there may be up to a ten-fold difference in the required doses of certain analgesics such as morphine and a reluctance to give larger doses despite having a patient that is very much awake and still in pain, as well as long term issues such as addiction. This is because of both physiological/pharmacological aspects (e.g. some patients will be ultra-rapid, rapid, slow or even non- metabolisers of some drugs such as codeine) and the psychological suppression or down-regulation of pain pathways depending on the context of the injury.

The anticipated duration of severe pain also plays a part in agent choice - e.g. For a short but painful procedure such as relocating a joint or fracture/dislocation will need very good analgesia for a short period of time, but once realignment has been achieved the amount of pain from the injury dramatically decreases. If a long acting strong analgesic and/or sedative is given (e.g. morphine and midazolam), then once the painful stimulus is removed there may be a rebound over-sedation or increased rate of side effects and complications such as respiratory depression.

The measurement of pain is essential in targeting the appropriate treatment, and as previously mentioned each individual's perception of pain will differ depending on circumstance. There may be a hyper acute suppression of what looks like a catastrophic injury (for example, the injured soldier missing an arm in the middle of a firefight but who declines analgesia) or at the other end of the spectrum is what appears to be a minor injury that requires multiple modalities of treatment, resulting in a chronic regional pain syndrome (previously known as Sudeck's atrophy or causalgia).

The time from injury and surrounding psychological issues also play an important role, as although we are primarily concerned with acute pain this may progress to being chronic and the treatment goals may change from the complete alleviation of pain where possible, to strategies aimed

at coping with pain on a day to day basis to allow a return of as much function as possible. Effective early intervention may prevent or minimise the development of chronic pain, and appropriate psychological support and non-pharmacological means of treatment have an effective role to play.

Firstly we must assess the degree of pain as this will guide the level of intervention needed. A patient with a pain score of 1/10 will not require strong opiates and conversely a patient with a 10/10 rating will require more than paracetamol! Multiple systems can be used, but a 0-10 scale is the most commonly used in the UK with 0 representing no pain and 10 being the worst pain imaginable. Other indices are available (e.g. 0-3 scale, mild/moderate/severe rating, "smiley faces", FLACC scoring) and the clinician should ultimately use whichever one they are most familiar with. Potential pitfalls include patients who cannot speak either due to injury, age, language barriers or mental disturbance, or patients who are intubated and assumed to have an adequate degree of analgesia.

With the exception of ketamine none of the standard induction agents or benzodiazepines provide any analgesia in isolation, so an intravenous opiate or opioid should be considered in these patients to offset the physiological stress response (opiates are naturally occurring compounds, i.e. morphine and diamorphine, whereas opioids refer to both the natural opiates and also the synthetically created compounds based on opiates e.g. fentanyl, alfentanil, pethidine, etc).

A reassessment of pain after intervention is always required as the clinician may be tempted to try and give repeated doses of drugs which are not indicated and run the risk of increased side effects without clinical benefit, or possibly not give more analgesia when it is needed. The likely clinical course of a patient should also be considered when selecting and implementing an analgesic strategy. An extreme example would be a patient who is trapped by their leg in a motor vehicle accident and currently complains only of 3/10 pain. If the scene becomes unsafe and there is a need for an amputation for rescue purposes (e.g. the car starts to burn and fire cannot be controlled, necessitating emergent extraction and amputation), ketamine analgesia/anaesthesia would be appropriate in this case. A less extreme example would be the same patient who is about to be extricated with a severely compound fracture of the leg. Although they may complain of 3/10 pain at the moment, moving

the car and hence the fracture fragments which may be relatively splinted by the vehicle position will be painful so either morphine, ketamine or some other analgesic reserved for severe pain may be indicated in anticipation of this.

The following segments of this chapter will be directed at specific analgesia modalities in certain kinds of trauma, but is far from comprehensive. Familiarity with drugs is paramount, as acute traumatic patients represent several management problems and are not appropriate to use a new drug or technique on unsupervised for the first time. Existing experience with certain techniques (such as regional anaesthesia) or medications (such as ketamine) is essential before being tried in the trauma arena, as liability for use of said techniques lies with the individual practitioner.

Pre-hospital practitioners may be faced with a large number of problems in the undifferentiated trauma patient, and good analgesia may make their subsequent management easier. Initial management with reassurance and simple splintage will provide a degree of relief which may be sufficient on its own, however there are several other options that can be employed.

Non-Pharmacological

Splinting

Splinting not only provides analgesia when the patient is still, but may also provide a degree of pain relief when the casualty is rolled by preventing fracture ends from rubbing together. The reduction of fractures back into their normal anatomical alignment also decreases bleeding and the risk of marrow fat embolus. Specific devices may be used (e.g. Kendrick system or Reel splint for lower limb fractures), or devices may be improvised (e.g. using a bed sheet or triangular bandage for an improvised pelvic splint if one is not available).

Reduction

The management of dislocations also relies on splintage techniques, and the dislocated limb should be splinted in the position it is found in if that is comfortable for the patient, with an assessment made of distal neuromuscular status before and after moving the limb. Following radiological assessment, limbs should be reduced into the neutral position and splinted in place.

Limbs that are ischaemic secondary to dislocation, require urgent reduction. Reduction in these instances provides significant pain relief. Be vary cautious of using long acting opioids prior to reduction, as frequently over analgesia post reduction oc-

curs, with very real risk of complications such as respiratory failure, nausea and vomiting.

Irrigating

Burns should be cooled and irrigated with normal saline, prior to dressing. In the case of chemical irritation, dry chemicals should be brushed away, followed by copious irrigation to dilute the irritated area. The inflamed area should be dried and managed as a burn.

Covering

Covering of raw exposed tissue protects from further irritation from both friction and air moving across exposed nerve endings. Plain cling film is immensely useful in the pre-hospital and early emergency department management of such pain, particularly following burns.

Routes of drug administration

A successful analgesic agent has high patient satisfaction with few adverse events and effects.

Intravenous (IV)

The commonest route of administration of drugs for rapid onset and most familiar to anaesthetists. Requires a cannula that is working and an intact vascular supply to the central circulation for onward distribution.

Interosseous (IO)

Popular historically in paediatrics and becoming more and more practiced in the adult population with the development of devices like the FAST-1, FAST-Responder, EZ-IO and BIG (Bone Injection Gun). The IO route has all the advantages of IV access, is less likely to become dislodged and has few drawbacks. It is being increasingly used in both military and civilian practice, with the caveat that the IO should be removed as soon as is reasonably practical (i.e. when reliable IV access has been secured). It should not be left in for more than 24 hours, and depending on the device used it may render some military personnel non-deployable for up to one year regardless of other injuries.

The risks associated with IO access are misplacement, joint injury and osteomyelitis, though these complications are rare if the device is used properly. There are very few contraindications to IO use, and these are all relatively self evident - overlying skin infection, fracture proximal to the site of insertion (e.g. femoral or pelvic if tibial site used, humeral if humeral head used, sternal fracture or previous sternotomy if FAST-1 used) overlying burns, inability to

locate anatomical landmarks or patient refusal.

FAST X

The insertion of various devices has not been described as unduly painful, but the initial flushing and overpressure of the marrow cavity of the bones has been reported as transiently painful but only lasts a couple of seconds. There has been suggestion of flushing the IO device with 2% lignocaine and various protocols exist regarding insertion[7-11], however in the severely obtunded patient this is probably not necessary.

Various sites of insertion have been described (medial malleolus, medial tibia, distal femur, pelvic crest, distal radius and humeral head), and whichever site is chosen must be cleaned and inspected for contra-indications prior to insertion of the needle. All drugs that are given IV (with the exception of bretyllium, a historical anti arrhythmic agent no longer commonly used) may be given through an IO needle. The speed of access has been reported to be faster than establishing IV access in in some cases, and the IO device has also been recommended in ALS guidelines for use in cardiac arrest due to ease of use and decrease in time to establish access in the arrested patient. The speed of onset of drugs given IO has been proven to be equivalent to IV access, and in the FAST-1 the time of spread to the central circulation

has been shown with dye testing to be the same as drugs given via a central line.

Intramuscular (IM)

Has been used historically, but due to unquantifiable differences in muscle perfusion in trauma has fallen out of favour. The speed of absorption of drugs from IM injections is highly variable and unreliable, and if the patient is hypovolaemic and not perfusing their muscle then the drug may not reach the circulation to have an effect. This may be further compounded by multiple doses being given to achieve an effect, and when the patient is resuscitated and normal perfusion restored then a large amount of drug may suddenly be dumped centrally causing an overdose.

Intransal (IN)

Popular route in paediatrics and becoming more popular in adults. Ketamine, diamorphine and fentanyl have been used with a mucosal atomiser. Fast onset speed and good bioavailability, few if any contraindications with the added bonus that naloxone can also be given intranasally if opiate overdose is suspected. Very good for children who are in pain and needle-phobic.

Subcutaneous (SC)

No longer used in acute care, but subcutaneous drugs can be given in palliative care via syringe drivers

Transdermal

Can be used in chronic pain for fentanyl or buprenorphine, but not of much use in the acute setting as their onset time probably precludes their use in the acute trauma. However it is important to look for patches that the patient may have been wearing at the time of their injury as they could cause either an overdose of opiate if left in position, or potentially an under dosing in acute pain if the patient is extremely opiate tolerant and a "normal" dose is ineffective in relieving acute distress.

Oral

Can be useful route of drug administration in the ICU, and in minor trauma. However acutely the absorption from the GI tract may take a prolonged period due to hypoperfusion and shunting of blood away from the gut in major trauma and hypovolaemia. Drugs which are useful in managing subacute or chronic pain (amitriptyline, gabapentin, pregabalin) may have to be given orally as there is no IV alternative.

Oral Transmucosal

Transmucosal administration of fentanyl can be achieved via either "lollipop" or lozenges that are held between the lip and gum and is of use if immediately available. As the patient is in control of their level of analgesia it is very useful, and American military practice suggests taping the stick of the lollipop to the patient's finger so if they become obtunded then they will remove the lollipop when their hand drops out of their mouth. The relatively shorter half life of fentanyl in these patients means that the duration of respiratory depression is less than with morphine, and once the drug source has been removed and the systemically absorbed fraction starts to redistribute, then the patient will wake up.

Unfortunately the case is not true with fentanyl lozenges as they do not "fail safe" and may cause continuing respiratory depression and potentially airway obstruction if they fall to the back of the airway. The availability of fentanyl lollipops in the UK is less than on deployed operations, and unfortunately they are not available in the JRCALC formulary at the time of writing so their use is restricted to independent prescribers.

Inhalational

Entonox is a gas containing 50% nitrous oxide and 50% oxygen. It is a good analgesic, with a rapid onset and offset (approximately 6-8 breaths at either end). It requires a patient who is able to cooperate and it reduces the inhaled fraction of oxygen from around 85% to 50%, and so is not suitable for patients with major shock or severe injury. It is however a useful agent for some patients and should not be forgotten, especially in pre-hospital care. It is contraindicated in chest trauma until a pneumothorax has been excluded and where the effect of nitrous oxide diffusing into air filled spaces would be deleterious for example pneumocephalus. It is also contraindicated after SCUBA diving and in decompression illness. One practical point is that the pseudo critical temperature of entonox (the temperature at which it separates out into its individual components of O_2 and N_2O) is -6°C. This means that around or below this temperature the cylinder should be repeatedly inverted to ensure an adequate mixture of the two chemicals. Failure to do this results initially in the

oxygen rising to the top of the cylinder and no N_2O, and hence no analgesia, being given. This is followed by 100% N_2O being delivered when the O_2 has been preferentially inhaled first, and thus a hypoxic mixture is delivered.

Methoxyflurane is an inhalational analgesic that is sometimes used in Australia and New Zealand by paramedics, but its use is not common elsewhere in the world. It is an anaesthetic vapour that has been discontinued in the USA, Canada and Europe due to concerns of nephrotoxicity and hepatic toxicity when used as an anaesthetic agent. It provides some analgesia at subanaesthetic doses in a similar manner to entonox, but in contrast to entonox its effects last for up to eight minutes after inhalation has ceased due to its high lipid solubility. Its inclusion here is purely illustrative.

Brief pharmacological comparison of analgesics

Opiates (oral, IV, IM, IO, transmucosal, intranasal)

Traditionally the gold standard analgesia in trauma but best in conjunction with other techniques if possible. The standard opiate in UK practice is morphine, and is given at a dose of 0.1 mg/kg as an initial bolus in severe pain. However, a more practical approach may be to give a 3 mg bolus and repeat every 5 minutes until adequate analgesia is attained, as the widespread variance in tolerance that has previously been described may lead to an inadvertent overdose in some instances. As a rule of thumb it is always easier to give more opiate than it is to take opiate out of the patient that has overdosed! The antidote to opiate overdose (naloxone) is a useful and rapid acting drug, however the half-life of naloxone s shorter than that of morphine, so in practical terms an episode of secondary respiratory depression or unconsciousness may occur after the naloxone has worn off and before the morphine has been metabolised. Traditional teaching was to give both an IM and IV dose of naloxone as it was thought that the IM dose would be absorbed more slowly and thus have a longer effect, but this has not been

born out in clinical practice. Naloxone can also be given intranasally or subcutaneously will equal efficacy,

Paracetamol (oral, IV, rectal)

Paracetamol is available in a licensed i.v. preparation for management of acute pain. It should be given regularly instead of a PRN basis as it forms the foundation of our analgesic ladder and has been shown to decrease opiate requirement. This effect has been increased when IV paracetamol has been given in anticipation of a painful stimulus (e.g. surgical incision) rather than as a reactionary medication. The benefits of IV versus oral or rectal paracetamol are a shorter time until maximum availability, a higher dose bioavailability (by definition the IV dose is 100% bioavailable in comparison to 60% oral and 40% via rectal routes), less hepatic damage (the paracetamol is given systemically rather than being absorbed and metabolised by the liver via the portal circulation) and less dependent on gut blood flow, which may be altered in trauma, or the patient may be strict nil-by-mouth in the case of some bowel injuries.

NSAIDS (oral, IV, IM)

NSAIDS work by causing inhibition of prostaglandin synthesis, typically in the arachidonic acid pathway. This suppression of prostaglandin synthesis is how it has its clinical anti-inflammatory and analgesic effects. However the inhibition of certain prostaglandins also causes a decrease in production of COX I and COX 2 which are gastroprotective, as well as decreased bicarbonate and mucus secretion. Care is needed with renal function in hypovolaemia but very effective if normovolaemic with no ongoing bleeding. Caution in asthmatics, as up to 20% of patients with susceptible asthma may experience an acute asthma attack. Typically these patient s will also suffer from allergic rhinitis and nasal polyps, and children with asthma appear to be relatively protected from this effect. Depending on other injuries (renal, orthopaedic, GI bleeding) there may be relative contraindications to NSAID use, but where possible they should be used as they are well tolerated and opiate sparing. The "kindest" NSAID in terms of GI side effect profile is ibuprofen (400mg three times a day) as this has the least anti-inflammatory effect, but not the least analgesic effect. Other NSAIDS such as diclofenac, meloxicam and ketorolac have the advantage that they are also available in an IV form, so may be used if the patient is nil by mouth for any reason.

Ketamine and S-ketamine

Ketamine is a phencyclidine derivative and a racemic mixture of two optical enanti-

omers. The R- form is responsible for approximately 30% of the analgesic activity of the mixture and has been implicated in the side effect profile more than the S- form. In Europe, the S- form is available as a purified form, however its popularity has not crossed into mainstream UK or US practice. Historical concerns about deleterious effects of ketamine have made some clinicians wary of using it, however these fears have been proven ill founded and the evidence that these concerns were based on has proven to be of poor quality. Ketamine is enjoying a resurgence in both pre- and in-hospital trauma use for analgesia and induction of general anaesthesia.

Neuropathic pain modulating agents

Gabapentin/pregabalin

Gabapentin is an anti seizure medication that has been used in the treatment of chronic pain thought to be neuropathic in origin. It is a GABA analogue and was initially thought take several days if not weeks to exert an analgesic effect, however recent data suggests that it may also have a role to play in acute pain and be opiate sparing. Some studies have concluded that gabapentin may not be any more effective than carbamazepine in neuropathic pain, is equally effective as pregabalin but cheaper and may not be effective in treating complex regional pain syndrome. Its main side effects are dizziness, drowsiness and peripheral oedema, with an increase in depression, and suicidal ideation. Gabapentin should not be stopped abruptly as it may cause a withdrawal-like syndrome, potentially resulting in seizures. Pregabalin was released as a competitor to gabapentin and the two drugs are structurally similar, however pregabalin is more potent, absorbed faster and has a greater bioavailibility. It is also marketed for treatment of neuropathic pain and post herpitic neuralgia, whereas this is an off license indication for gabapentin. The dose of gabapentin is increased over the course of a week and if

problems with insomnia or other side effects are experienced, is held at that particular level until tolerance is achieved.

Amitriptyline

Amitriptyline is a tricyclic antidepressant which has been used in the treatment of neuropathic pain, but also can be used in the treatment of post traumatic stress disorder and insomnia related to this. A typical dose for neuropathic type pain is 25-50mg at night as tolerated. The chief side effects are anticholinergic symptoms such as dry mouth, blurred vision, urinary retention, nausea, increased sweating and constipation. In relatively low doses it is well tolerated, and has a synergistic effect with gabapentin.

Regional Anaesthesia

Systemic analgesia require the administration of medications via one of the above routes and may cause unwanted side effects depending on the drugs used. One other potential method of analgesia would be a peripheral nerve block, if possible.

There have been several case reports of regional anaesthesia used in a pre-hospital environment, and depending on the indication for use and the transit time to hospital, it may be an appropriate modality to use. Certainly for secondary transfer or aeromedical evacuation, the supplementation or replacement of parenteral analgesia has many attractive advantages - it diminishes the risk of respiratory depression which may be deleterious at altitude, as well as the potential eu- or dysphoria of ketamine, it allows the patient to remain awake and promotes orientation and allows assessment of the casualty when they are awake.

It should not be employed if it delays time to definitive care, but is a useful tool in the anaesthetic armamentarium. The introduction of hand-held ultrasound machines has made regional anaesthesia pre-hospitally potentially more available than in previous years, and the Royal Flying Doctor Service of Australia has employed this technique on many occasions and some regional an-

aesthesia is taught as part of their [standard operating procedures](). Landmark techniques are possible, but in some cases the use of a nerve stimulator or preferentially an ultrasound machine is mandatory (e.g. supra- or infraclavicular blocks).

Orthopaedic surgeons are rightly concerned about the development of compartment syndrome. This is where increased pressure within a fascial compartment of a limb (classically following nailing of the tibia) increases due to muscle swelling. This swelling increases to a point where the venous drainage of the affected compartment is not possible, thus causing more swelling. The limb still has pulses as arterial pressure is much higher than venous pressure, but necrosis of the muscle begins and the patient requires a fasciotomy (an operation to cut the fibrous band that separate compartments in the limb). The hallmark of compartment syndrome is pain out of proportion to the injury, with worsening pain on passive muscular stretch.

There is currently no evidence to suggest that regional anaesthesia prevents diagnosis of compartment syndrome or delays its diagnosis if the patient is appropriately examined, though many surgeons believe this to be the case.

The diagnosis of compartment syndrome is largely clinical, and relies to a large degree on clinical suspicion and examination, as a normal compartment pressure measured by manometry does not exclude compartment syndrome completely.

There are six key blocks which theoretically may be employed in- or in some cases pre-hospitally for limb trauma. The exact details of how to perform these blocks are beyond the scope of this text, but [there are many resources]() for the interested practitioner to learn from.

It must again be reinforced that these blocks should be done in as aseptic a fashion as possible, and should not increase scene time or time to definitive care. They may be appropriate in only a small number of scenarios, usually when a prolonged transfer is anticipated or other analgesic options are not practical. Assessment of nerve function prior to any block should be attempted and recorded, as well as any block performed, the time and dose of any agent given.

The peripheral nerve blocks outlined can also be utilised as a primary anaesthetic technique in some instances for the appropriate surgery in appropriate patients, or more commonly are used to supplement

general anaesthesia for postoperative pain relief. Either a single shot injection or a continuous nerve catheter can be placed to allow for infusion of local anaesthetic for a prolonged analgesic effect. These catheters if appropriately cared for can be left in situ for over two weeks, and have the added advantage they they can be bolused for procedures such as bedside dressing changes, which may otherwise require further sedation or general anaesthesia.

The medications outlined at the end of this chapter can all be employed effectively, as can epidural analgesia/anaesthesia for lower limb injuries as outlined in the thoracic section. The only difference is that the catheter is inserted in the lumbar spine rather than at a thoracic level and the chances of nerve injury and profound hypotension are less, though the rate of post-dural puncture headache remains the same at approximately 0.2 to 1%. Primary anaesthesia for lower limb fractures can also be achieved with a spinal or subarachnoid block, though this can cause profound cardiovascular changes (hypotension and vasodilatation after injection) and is limited to operative procedures less than two hours in length. However, in the same way as adding opioids to an epidural potentiates its effects, intrathecal opiates can give up to twelve hours postoperative relief, and although surgical anaesthesia is limited to 90-120 minutes with a single shot spinal, there is a degree of postoperative analgesia that may persist for up to eight hours or beyond in some patients. Spinal anaesthesia is not appropriate in the hypovolaemic, under-resuscitated patient, the coagulopathic or the patient requiring a prolonged procedure, however is a small group of patients with longstanding respiratory disease it may be considered an alternative to general anaesthesia.

NB, hyperlinks in the following section will take you to videos demonstrating ultrasound guided regional anaesthesia

Upper limb blocks

Interscalene block
Good for proximal upper limb trauma. Commonly produces unilateral diaphragmatic palsy secondary to phrenic nerve palsy. Caution in pre-existing respiratory disease or acute chest trauma. Should never be performed bilaterally for this reason. Will give analgesia from proximal shoulder to distal humerus.

Supraclavicular Block
Performed under ultrasound, providing analgesia for upper limb, forearm and hand

as well as the shoulder. Complications are similar to those for interscalene blocks and infraclavicular blocks. More commonly performed in the UK than infraclavicular.

Infraclavicular block

Must not be performed without ultrasound. Relies on depositing local anaesthetic under the clavicle and around the subclavian/axillary artery and enveloping the lateral, posterior and medial cords of the brachial plexus where they run in close continuity with the artery. Complications include inadvertent arterial puncture, bleeding, local anaesthetic toxicity and pneumothorax due to the close proximity of the pleura. Suitable for anaesthesia distal to the mid humerus for the distal upper limb.

Axillary block

Similar coverage to the infraclavicular block, however the point of injection is the medial humerus, thus avoiding the risk of pneumothorax. The traditional technique called for a trans-arterial puncture, but with the development of ultrasound guided regional anaesthesia, it is no longer necessary to puncture the vessel as the nerves can be visualised. There is the potential to miss the musculocutaneous nerve as it is usually inferoposterior to the artery and occasionally difficult to visualise.

Lower limb blocks

Femoral block

Good for analgesia for femoral fractures. This block can be performed by a landmark technique as well as under ultrasound guidance. The femoral artery is palpated as proximally as possible in the leg and a needle is inserted 1-2cm laterally until two fascial pops are felt. The local anaesthetic is then slowly injected after a negative aspiration unless resistance or pain on injection is felt. A variant on this, the fascia iliaca block, can be used in neck of femur fractures.

Saphenous Block

Identified with ultrasound by tracing the femoral artery down the anterio-medial thigh to the point where the artery starts to disappear (typically at the lower third) Look for the fascial "corner" just above the artery and infiltrate local anaesthetic to give excellent pain relief to the knee, but without quadriceps motor block).

Sciatic block

The sciatic block innervates the knee joint and all the structures below the knee, with the exception of a small strip of skin over the medial malleolus which is innervated by the saphenous nerve, a branch of the femoral. The sciatic can be blocked high at the buttock, sub-gluteally in the anterior thigh or in the popliteal fossa.

Sciatic Bifurcation on Ultrasound

Analgesia for Thoracic Trauma

As identified in the <u>thoracic module</u>, the causes of perioperative morbidity and mortality in patients with thoracic trauma are airway obstruction, respiratory failure and haemorrhage. The anaesthetist is ideally suited and trained to deal with the problems of airway control, ventilatory and circulatory resuscitation, and adequate analgesia in all phases from pre-hospital care, through the operating room and ICU and eventually into the pain clinic in some cases.

Pain from musculoskeletal trauma to the chest is a major contributor to the failure of normal respiratory dynamics as previously highlighted. Inadequately controlled pain may cause hypoxic and/or hypercapnoeic respiratory failure. The inability to deep breath and cough adequately leads to sputum retention, atelectasis and collapse/consolidation of lung tissue, which potentially may lead to superadded infection. This exacerbates hypoxia and leads to progressive respiratory failure which may result in a need for invasive ventilation if not addressed rapidly and effectively. The best way to avoid this predictable deterioration is to provide adequate analgesia from the first presentation of the patient.

The method and magnitude of analgesia required will depend more on the amount of pain suffered than on the type and degree of injury sustained. A single lateral rib fracture in an elderly smoker with COPD may precipitate respiratory failure whereas multiple posterior rib fractures in a young fit person may be relatively well tolerated.

Effective analgesia reduces stress, helps stabilise cardiovascular function, reduces oxygen requirements and allows early mobilisation. Analgesia is best achieved with a multi-modal approach combining several different drug types rather than relying on one technique alone and minimises the potential for side effects. Suitable analgesic components include:

- **Non-pharmacological methods** (e.g. Splinting, "cough block with median sternotomy)

- Splinting by hand may offer some temporary relief but binding or strapping will result in increased respiratory complications

- Surgical fixation of ribs is currently becoming popular again

- Simple analgesia with paracetamol or NSAIDS

- Opiates

- **Regional anaesthesia** (single shot or infusion catheter)

    - Intercostal Block

    - Intrapleural/paravertebral blocks

    - Thoracic epidurals (± epidural opiates/opioids)

- **Low dose ketamine infusion**

    - 5-10 mg i.v. loading dose followed by 0.5-2 mcg/kg/min infusion in a monitored HDU environment with 10-20mg boluses as required [see reference 4]. Confusion or agitation should be managed by reducing the dose of the infusion Very good in combination with any or all of the above techniques

Regional Techniques for thoracic trauma

These can produce excellent profound analgesia with no sedation when performed well and allow almost normal chest movement with respiration. The technique chosen depends on experience and equipment available in addition to any contraindications. However, particularly high blocks may reduce the function of the intercostal muscles and cause respiratory embarrassment on their own, so caution is advised with their implementation. With certain techniques (e.g. thoracic epidural) there may also be deleterious cardiovascular effects including hypotension secondary to sympathetic blockade causing vasodilatation and bradycardia if the cardioaccelatory fibres are blocked and unopposed vagal tone predominates. If appropriately implemented and monitored then these techniques have a high success rate, but in order to avoid complications then good nursing care and appropriate monitoring are required which are best achieved in a high dependency or ITU setting.

Intercostal and intra-pleural blocks have the potential for local anaesthetic toxicity due to the relatively rapid uptake of drug from the pleural and intercostal spaces, as well as causing a pneumothorax. The insertion of these blocks may be done before or

after an operation whilst the patient is under general anaesthesia, or potentially awake in a sitting position if no operation is planned. However, the positioning required for these blocks in some patients may preclude them being done due to pain - if the patient can be positioned awake for an intercostal or intrapleural block then they probably won't require one. Intrapleural catheters can be placed under direct vision at the time of operation if a thoracotomy is performed by surgeons.

Intercostal Block

Requires multiple injections, one for each rib fracture plus one segment above and below. The needle is placed just over the lower border of the rib at the angle of the rib with injection of 5ml of bupivacaine 0.25% at each site. Limited duration of action necessitates repeated injections. Only lasts approximately four hours. Requires repeated injection at multiple levels. Not practical for upper rib or posterior rib fractures, and of limited value.
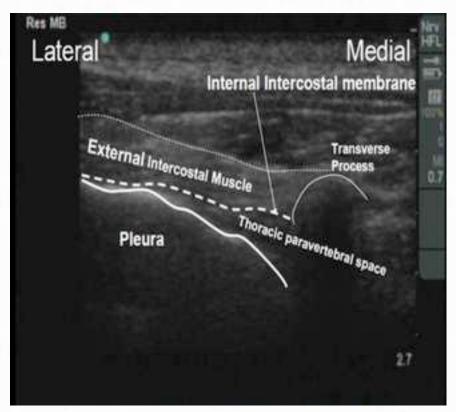
Intra/inter-pleural Block

Insertion of an epidural catheter via a 16g Touhy needle into the pleural space allows repeated injection of local anaesthetic. Various techniques have been reported for detecting the pleural cavity relying on negative pressure within the space. 20ml of

0.25% bupivacaine will produce several hours of good analgesia and may be repeated every four hours. It is effectively an intercostal block from inside and the patient should be positioned during the bolus administration to allow the local anaesthetic to pool in the paravertebral gutter on the effected side. The intercostal nerve at this point is separated from the pleural space by the thin posterior intercostal membrane through which the local anaesthetic solution diffuses rapidly. Excellent analgesia for unilateral rib fractures with bolus administration of local anaesthetic. Only requires one needle insertion into chest wall. May be used for several days. Does not work well in presence of pleural fluids or pleural adhesions. Presence of a chest drain may result in local anaesthetic being lost from pleural space.

Paravertebral Block

May be performed with continuous catheter technique to avoid repeated injections or as a single shot technique. Unfortunately there is a less definite end point detected on insertion. Drawbacks include potential for LA toxicity, potential for sympathetic blockade, risk of pneumothorax and possible injection into a dural sleeve resulting in spinal anaesthesia. Significant sympathetic blockade to the lung may predis-

pose to bronchospasm and production of tenacious secretions.

Paravertebral space seen on ultrasound

Thoracic Epidural (+/- opiate)

A midline or paramedian approach to the epidural space can be used and analgesia provided either with continuous infusion of local anaesthetic with or without opiates. Bolus administration of local anaesthetic can be very effective if continuous infusion is not a practical proposition. It is an excellent technique for unilateral and bilateral fractures of the middle and lower ribs. Epidural blockade may be used for several days but is not so good for high rib fractures, which would require very high blockade and is contraindicated with thoracic vertebral injuries and in the presence of a coagulopathy. A typical infusion regime is

using 0.125% bupivacaine with 2 mcg/ml fentanyl at 6ml/hour with a 6ml bolus infusion and a 20 minute lockout. If large numbers of boluses are demanded and the catheter is technically positioned correctly then the background infusion rate can be increased. The higher the insertion level the more potential there is for hypotension and bradycardia. Epidural solutions can be pre-made by pharmacy, or reconstituted with strict aseptic technique on a <u>volume by volume basis</u>. The addition of opiates to an epidural has the effect of covering a less than perfect block, as epidural/spinal opiate receptors can be stimulated at lower doses than would be required systemically. This also has the effect of reducing the incidence of opiate side effects, namely nausea, vomiting, decreased level of consciousness and constipation.

One other potential complication of epidural analgesia is inadvertent dural puncture (estimated at 1% of all epidurals performed). This may cause a post dural puncture headache, or in severe cases an injury to the spinal cord in the thoracic region which may result in a permanent sensory or motor defect. Even in a perfectly placed epidural there is still the risk of infection and spinal abscess. Meticulous detail to asepsis and technique is essential, and if there is any suggestion of spinal

cord abscess or infection around the site then the infusion should be stopped, the epidural discontinued and an urgent CT/MRI requested regardless of the time of day or night. Unless caught and operated on early, spinal abscesses have a very poor prognosis, so a high degree of clinical suspicion and low threshold for intervention must be maintained. This is also why an epidural is contraindicated if there is overlying skin breaks, infections or systemic sepsis as the epidural and intrathecal spaces are sterile under normal conditions, and seeding of infection into them is a purely iatrogenic complication.

Analgesia for Neuro-Trauma

There are no specific treatment modalities for neuro trauma, the only caveat that must be born in mind is that certain treatments may cause neurological symptoms. For example, high doses of opiates can decrease level of consciousness and cause pinpoint pupils via stimulation of the Edinger-Westphal nucleus, both of which may cloud a neurological exam. Another example is ketamine, which has both pro- and anti- convulsant properties, so any focal neurological examination should ideally take place with the patient as free from impediment as possible. Frequently, the initial responder may have the most reliable examination as the patient may need high doses of analgesia, or even general anaesthesia prior to getting to hospital and the attention of a neurosurgeon or critical care team. It is important therefore to examine as thoroughly as possible and document clearly the highest GCS since injury and any lateralising neurological signs in the limbs or eyes prior to induction of general anaesthesia or sedation. A focused neurological examination ("move your arms, your legs, close your eyes, where are you, what is your name, what day/week/month is it") will give a baseline to work from in hospital. It is also important to note the time of administration and dose of any neuromuscular blockers which would change a neurological examination in hospital.

It is important to adequately analgese patients who are intubated and ventilated as pain may cause or substantially contribute to a raised intracranial pressure. Also, awareness under anaesthesia and coughing can also cause raised ICP, so assessment and maintenance of an adequate plane of anaesthesia even when not in the operating room (i.e. during CT scan, transfer inter- or intra-hospitally) are of vital importance.

Summary

- Pain Score in isolation is meaningless.

- Correlate subjective pain with objective clinical findings suggestive of catecholamine surge.

    - -i.e. Raised BP, HR, RR, sweating, flushed, nausea

- Distress is distress, perhaps secondary to pain, but not always.

- Treat distress with appropriate small doses of anxiolytics and you will lower your analgesic requirement
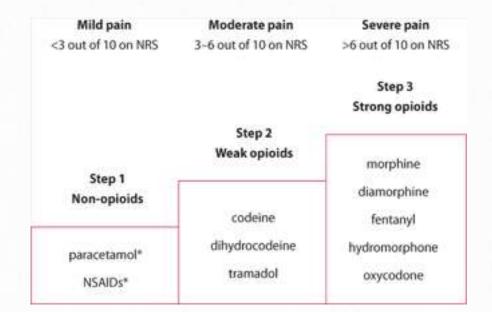
BUT… anxiolytics on their own are NOT a treatment for pain!

- Different types of pain require different approaches

    - Don't just prescribe without working out where the pain is coming from.

Decide how you are going to treat the pain (not just pain killers)!

    - Reduce

    - Splint

    - Cover

    - Irrigate

The WHO Analgesia ladder: Paracetamol + NSAID ± weak opioid in addition to strong opioid if needed

Mild pain <3 out of 10 on NRS	Moderate pain 3–6 out of 10 on NRS	Severe pain >6 out of 10 on NRS
		Step 3 Strong opioids
	Step 2 Weak opioids	morphine diamorphine
Step 1 Non-opioids	codeine	fentanyl
paracetamol* NSAIDs*	dihydrocodeine tramadol	hydromorphone oxycodone

Procedural analgesia and sedation

e.g. Short acting opiates such as Fentanyl for the procedure itself. Morphine is not appropriate yet!

- Short acting hypnotic (ketamine, midazolam or propofol)

Or

- Regional anaesthetic technique (especially useful with ultrasound).

Post Treatment Options

- Maintain analgesia base

- Consider codeine/paracetamol preparations (Solpadol 30/500 AKA co-codamol).

- Splinting/strapping/immobilisation

- Going to theatre/in-patient: Consider long acting opiate (morphine)

References

1. Joshi GP, Oggunaike BO. Consequences of inadequate postoperative pain relief and chronic persistent postoperative pain. Anesthesiol Clin N Am. 2005;23(1):21-36

2. Henning M, Pai A. Ketamine Contin Educ Anaesth Crit Care Pain (2007) 7 (2): 59-63. doi: 10.1093/bjaceaccp/mkm008

3. Arici S, Gurbet A, Türker G, Yavaşcaoğlu B, Sahin S. Preemptive analgesic effects of intravenous paracetamol in total abdominal hysterectomy, The journal of the Turkish Society of Algology (agri),2009 Apr;21(2):54-61.

4. Nelson A, Aldington D. Battlefield analgesia in Mahoney, P. F. 2011. Ryan's ballistic trauma. New York: Springer.

5. www.nysora.com

6. http://www.globalrph.com/epidurals.htm

7. http://www.vidacare.com/gfGy6S44anlz/EZ-IO-Combined-Protocol-T-445-revB.pdf

8. http://smchealth.org/sites/default/files/pictures/EMS/EMS_Proc_8_IO_3_29_2012.pdf

9. Atkins DL, Chameides L, Fallat ME, et al. Resuscitation science of pediatrics. Ann Emerg Med. Apr 2001;37(4 Suppl):S41-8

10. Waisman M, Waisman D. Bone marrow infusion in adults. J Trauma. Feb 1997;42(2):288-93

11. Lowther A. Intraosseous access and adults in the emergency department. Nurs Stand. Aug 3-9 2011;25(48):35-8

12. Byars DV, Tsuchitani SN, Erwin E, Anglemyer B, Eastman J. Evaluation of success rate and access time for an adult sternal intraosseous device deployed in the prehospital setting, Prehosp Disaster Med. 2011 Apr;26(2):127-9

13. http://healthprofessionals.flyingdoctor.org.au/IgnitionSuite/uploads/docs/Part%203%20-%20Procedures%20.pdf

14. http://www.aagbi.org/sites/default/files/Headache%20after%20an%20epidur

al%20or%20spinal%20anaesthetic.pdf

15. Wiffen, P; Collins S; McQuay H; Carroll D; Jadad A; Moore A (2005-07-20). "Anticonvulsant drugs for acute and chronic pain". Cochrane database of systematic reviews (Online) (3): CD001133

16. Finnerup, NB; Sindrup SH; Jensen TS (2010 Sep). "The evidence for pharmacological treatment of neuropathic pain". Pain 150 (3): 573–81

17. Tran de, QH; Duong S; Bertini P; Finlayson RJ (2010 Feb). "Treatment of complex regional pain syndrome: a review of the evidence". Can J Anaesth (57(2)): 149–66.

18. David F. McAuley, Pharm.D. How does pregabalin compare to gabapentin in the treatment of neuropathic pain? GlobalRPh Inc.

19. Hoskins, SL et al Pharmacokinetics of intraosseous and central venous drug delivery during cardiopulmonary resuscitation. Resuscitation. 2012 Jan;83(1):107-12. doi: 10.1016/j.resuscitation.2011.07.041. Epub 2011 Aug 25.

Further Reading

Regional anaesthesia

Www.nysora.com

www.neuraxiom.com

IO access

http://www.jems.com/article/intraosseous/pain-management-use-io

http://bestbets.org/bets/bet.php?id=2515

http://reference.medscape.com/article/80431-overview

Intransal drug administration

http://www.lmana.com/pwpcontrol.php?pwpID=6359

Compartment syndrome

http://lifeinthefastlane.com/ortho-library/compartment-syndrome/

Trauma Induced Coagulopathy

We are now going to consider another major cause of mortality in trauma related to shock and bleeding. Trauma Induced Coagulopathy is the current term adopted for a phenomenon that we have been aware of for many years. However, despite this increasing understanding of the issue, we are still well short of fully understanding the problem and how it may be effectively addressed. In this chapter we will consider the features, the causes and the best current management of this major problem.

Damage Control Resuscitation

In the assessment of our casualty we have already considered a number of key issues. We have considered the mechanism of injury and the potential traumatic effects of this mechanism in terms of specific injuries and the degree of injury. We have looked for markers and early signs of shock and how we can rapidly quantify the severity of shock and manage it, without making the casualty any worse.

These are two of the key foundations of the concept of Damage Control resuscitation and this has a number of key elements, which we have only just started to address. Any of these concepts used in isolation may delay the onset of deterioration or death but we will only see the full benefits of a damage control approach if we use all of the elements in combination.

In the Initial Approach to Damage Control Resuscitation chapter we introduced the first two 'pillars' of limited crystalloid fluid resuscitation and the adoption of permissive hypotension during resuscitation until haemorrhage is controlled. Let us now consider another of these key 'pillars'

Resuscitative Haemostasis

'Resuscitative haemostasis' refers to another concept whereby we resuscitate the casualty with fluids that will promote coagulation or minimise the effects of traumatic coagulopathy. This is a hugely complex subject and our understanding remains limited but is improving all the time as a result of work by researchers such as Karim Brohi and Rick Dutton.

Coagulopathy in trauma

In 1969 Simmons published his work on combat casualties and he identified that many of them, with major traumatic injuries, were already coagulopathic when they reached his 'MASH' field hospital. His observations went further than this as he also described that the severity of the coagulopathy was related to the degree of shock and that these were both directly related to the mortality.

Some 40 years later, we are not only starting to appreciate the significance of these observations, but we are also attempting to address the problem and also focussing on the issue of coagulopathy at a much earlier stage. By the time the casualty reaches the operating theatre or we have results back from the laboratory then it is already too late as the trauma induced coagulopathic state may already be well established.

Coagulopathy is one of the leading causes of death in major trauma and traditionally we have focused on a simple explanation for this based upon the development of a 'Lethal Triad' of hypothermia, acidosis and coagulopathy. We are now aware that the major issue is the coagulopathy and the other factors contribute to its severity. Based on this another triad can be described:

Dilution

Dilution: as a result of even moderate amounts of fluid resuscitation (1-2L). This obviously becomes far worse with the traditional larger resuscitation volumes seen in 'cyclic hyper-resuscitation techniques

Consumption

Consumption: as clot is formed and then broken down or lost there is a constant consumption of clotting factors and platelets which will inevitably result in a potential deficiency of certain essential clotting elements. This is often used as the explanation for the common heard expression "the first clot is the best clot". In other words, the first time that the body attempts to form a clot, the combination of elements required for clotting and the bodily environment is closest to perfect and after that the conditions are never quite so ideal.

Dysfunction

Dysfunction: hypothermia and acidosis result in impairment of clotting and especially of platelet function, which results in a worsening coagulopathy. As a result, we can have all of the key elements necessary for clotting and we can even supplement them (e.g. administration of Factor VIIa) but if we don't have the correct physiological environment then they will not function

as they should and may lose up to 90% of their effect. For recombinant Factor VIIa this seems to be more sensitive to hypothermia than acidosis, but both will contribute to the overall coagulopathy. This is the reason for the term 'Homeostasis for haemostasis' whereby clotting will be most effective when conditions in the body are as close to 'normality' as possible.

If we consider transfusion of units of packed red blood cells, whilst they may well carry oxygen (all be it less efficiently than warm, fresh whole blood) it is cold, devoid of clotting factors and platelets and it results in further dilution of the components within the circulation and as a result it will also contribute to the coagulopathy. Whereas, warm, fresh, whole blood will support and promote clot formation.

Optimising Coagulation

Is there an answer? How can we best manage this worsening coagulopathy?

An essential part is clearly to maintain as close to normal physiology as possible and we should go to considerable lengths to keep the casualty as warm as possible. This is often forgotten in all of the adrenaline fused management in the pre-hospital phase or the resuscitation room and allowing the patient's temperature to drop a de-

gree or two, although slower and more insidious, may be as serious as missing a pneumothorax.

Acidosis results from poor perfusion and cold which both develop in shock as the blood pressure falls and circulation is lost or diverted away from non-essential tissues. Here we have a dilemma, as much of our current teaching on haemorrhage management is based on 'permissive hypotension' whereby we accept a lower blood pressure and a degree of shock, as increasing the hydrostatic pressure in vessels would result in considerably more bleeding. However, throughout this period of low blood pressure there will be tissue underperfused resulting in a worsening acidosis. Such an acidosis will inevitably result in a worsening coagulopathy and ultimately an increased mortality.

As such we must seek a compromise and a balance between increasing bleeding with more blood pressure and worsening shock and coagulopathy with underperfusion and permissive hypotension. This balance gives us the current targets for mean arterial pressure and minimal resuscitation, but also the emphasis on speed as we want the casualty to be in this state of 'shock' and under-perfusion for the shortest possible time, to minimise the acidosis. (See Hybrid Resuscitation section).

Speed should be considered as a major part of any damage control strategy. Speed at the scene, immediate life saving measures, extrication and transport. Speed in the resus room, in terms of triage, assessment and ongoing care and then speed in getting to theatre and to achieving surgical control of bleeding and ultimately to intensive care where we can restore blood pressure and homeostasis.

We can also attempt to optimise the essential clotting agents by including them as part of our massive haemorrhage protocol.

These agents include:

- Fresh frozen plasma

- Platelets

- Fibrinogen

- Calcium

The question is, how much of these agents will be required to provide optimal clot formation at the site of injury?

Fresh Frozen Plasma

Let us consider fresh frozen plasma first as it is a rich source of clotting factors

A number of studies have demonstrated the effective use of high ratios of Fresh Frozen Plasma (FFP) to blood in managing the coagulopathy related to trauma. One such study published in 2005 looked at the problem of dilutional coagulopathy and suggested that this can be minimised or managed by adopting a ratio of 1-1.5 FFP:1 RBC unit.

A later study published in J Trauma in 2007 identified that diagnosis of coagulopathy in intensive care is too late and difficult or impossible to control. They recommend aggressive pre-ITU therapy with a high FFP to blood ratio of 1:1.

This seems like a simple and easy to adopt solution until we consider any potential negative effects of such an approach and there is considerable evidence to suggest that there is a poor risk/benefit ratio, especially in the critically ill, with little improvement in outcome. Other studies describe the increased rates of acute lung injury and ARDS associated with plasma rich blood products and platelets. This risk is further increased with high levels of packed red blood cell transfusion.

As a result we need to consider the best compromise of current evidence and this favours the adoption of high FFP: Blood ratios, with the understanding that this may not be the whole story or a complete ideal solution.

Platelets

There is a similar debate for platelets as they are obviously play an essential part in the clotting process and deficiency from consumption or dilution will potentially greatly impair clotting, but at what level does this become a problem?

Many papers and consensus documents recommend a high ratio such as 1:1:1 for blood, FFP and Platelet unit. However, others simply suggest keeping the platelet count higher than 100 (x109/L). This obviously requires a blood test and a count, which is often delayed and of limited value in a clinical major bleed situation.

Another reason for delay occur as a result of platelets being stored centrally in the Blood Transfusion Service, which will usually require an emergency transport to the hospital and a typical delay of at least half an hour. In view of this very real practical issue, many units have dropped platelets from the first Major Haemorrhage pack, unless the patient is already known or identified as thrombocytopaenic.

There is also a potential downside to platelet transfusion and many have raised concerns about the routine adoption of high ratios such as 1:1:1. Platelets are stored at room temperature which increases the potential risk of bacterial contamination. In addition, the incidence of Transfusion Related Acute Lung injury (TRALI) is significantly increased with platelet rich transfusions as a result of various inflammatory mediators and anti-leucocyte antibodies present.

In 2011, following a National Canadian consensus meeting considering these concerns, they have adopted a more conservative approach to high ratio transfusions and consider it a better balance of benefit vs risk. Their current strategy is initially 6 units of blood with 3 unit of fresh frozen plasma (2:1 but quoted as 6:3 as given as a pack). Blood counts are taken early and then results used for the next round of transfusion. Platelets are given when the count falls less than 100 or they are indicated by clot formation analysis with a thromboelastogram (TEG/ROTEM).

Fibrinogen

We must also consider all the other factors and co-factors involved in the clotting process and typically in major trauma the fibrinogen levels fall to critically low levels requiring supplementation. In the UK our main sources of Fibrinogen are currently Fresh frozen plasma (0.5-1.3g/unit) and Cryoprecipitate with one adult unit (from 10 donors and contains 3-6g) increasing the blood level by 1g/Litre.

Cryoprecipitate is also rich in other factors including Factor VII, Von Willebrand factor, fibronectin and factor XIII.

In other European countries they have freeze dried fibrinogen concentrate which can be delivered, 'off the shelf' as a 3-4g bolus and is ideal for targeted management of hypofibrinogenaemia.

Fibrinogen supplementation is currently recommended in trauma when the plasma level falls below 1.5g/L. If sample or clot analysis results are not available then typically the second massive haemorrhage pack (units 5-8 of packed red blood cells) should include additional fibrinogen, typically from the addition of cryoprecipitate to the PRBCs, FFP and platelets.

Calcium

Calcium is an essential co-factor in clotting and it also has a significant part to play in normal myocardial contractility and function.

Calcium has not been recommended during massive transfusion in recent times, but the levels are likely to be low or bound in transfused components and as such, plasma levels can fall rapidly during massive transfusion. This is because of both consumption during clot formation, dilution and loss from resuscitation fluid and binding of calcium by citrate preservatives found in packs of red cells.

Based on what current evidence is available, we would recommend that calcium supplements are given at the point of commencing the 2nd MHP, with the FFP transfusion and a further 10 ml of calcium every fifth unit of blood transfused. Alternatively, if rapidly available blood sampling can be provided, then ionised calcium levels should be kept at a level greater than 1mmol/L.

Calcium chloride offers the greatest levels of free calcium, however, Calcium gluconate is the formulation most commonly used in resuscitation and critical care areas. A typical slow IV bolus dose of calcium gluconate would be 10mls of 10% solution.

Towards a Consensus View

We can now start to see how even applying conventional and current knowledge, the management of massive transfusion in major trauma is hugely complicated. In trying to sum up what we have discussed so far we should consider the following:

- Identify and call massive haemorrhage early. This may include activation from the scene of the incident or certainly at the earliest opportunity following identification of potential or confirmed massive bleeding. Some units similar to activation of major incident plans whereby the clinicians can declare a 'Threatening' or 'Potential' massive haemorrhage and then escalate to a state of 'Massive Haemorrhage Declared'. Such a method alerts the laboratory staff earlier and informs them that this is more than just an urgent request for blood and that the request may rapidly escalate. They may then prepare staff, consider thawing FFP and prioritise their current work.

- If we are considering ordering blood in multiple units then also consider ordering fresh frozen plasma and platelets at the same time. A well produced Massive Haemorrhage policy will remove the need for such decision making or any doubts, as there is a clear process to follow, which all units should have. In terms of the amounts of each component, this should also be included in the policy but

based on current evidence and guidelines we would recommend a ratio of 1:1:1 for packed red cells, FFP and platelets, accepting that there may be delay with the FFP being thawed and in platelet delivery if off site, as they often are in UK hospitals. Further to this, consider Cryoprecipitate after the first four units of packed red blood cells have been transfused, or even better based upon some form of rapid blood analysis (see below) and consider 10-20mls of Calcium gluconate or chloride with every fifth unit transfused.

- Practically this is a real challenge in terms of identifying the need, activating the policy early, for the laboratory staff and the transfusion service providing the products in good time. However, being difficult to apply, does not necessarily mean that it isn't the best approach to adopt and the future may be more goal directed but may well be even more complicated and labour intensive.

But Does It Really Work?

In adopting these methods, we will make a difference to many of our patients but this is far from the whole story as not only is it akin to 'bucket chemistry' it is very much a simplistic approach to the bleeding and coagulopathy related. As we begin to understand more about coagulation and the importance of numerous elements, not just clotting factors, we can begin to understand why many of our patients with major trauma do not do as well as we might expect when we simply deliver high levels of blood and clotting components.

The Miami Approach

Consider a recent trial in Miami, where they delivered what could only be the current 'gold standard' of care.

- They had an effective pre-hospital care system, which would identify patients with massive haemorrhage at scene or before hospital

- The pre-hospital care did not introduce delays, but in fact facilitated more rapid transfer and transit through the ED

- They utilised a low volume of fluid approach and permissive hypotension, particularly avoiding large volumes of crystalloid

- Once on the Emergency Department the smooth and efficient process continued with a trauma team reception, rapid assessment and minimal delay before onward transfer to theatre.

- They had excellent times to theatre and were delivering a high standard of damage control surgery

- During the surgery fluids and transfusion requirements followed a high packed red cell to fresh frozen plasma ratio

This is the set up and fluid trauma pathway from the roadside to theatre and beyond that many of us would dream of having in place. They appeared to be doing everything right based on current evidence and recommended best practice and yet, they still found that their patients were arriving in the Intensive care unit and only a short time in theatre with an established coagulopathy.

Acute Traumatic Coagulopathy

This coagulopathy related to trauma has been termed Acute Traumatic Coagulopathy (ATC) or Trauma Induced Coagulopathy (TIC) and there appears to be a direct relationship between the severity of the injury and the degree of coagulopathy.

What is clear is that this is not Disseminated Intravascular Coagulopathy (DIC) with the associated consumption of clotting factors and subsequent development of impaired coagulation. This is actually a dysfunction of the normal clotting process and Karim Brohi has been leading on much of the research into this area and most of this following section is based on his work and we commend him for open-

ing our eyes to such a significant and complex problem.

He describes three things that occur in Acute Traumatic Coagulopathy:

- Reduced fibrinogen utilisation: when there appears to be adequate circulating levels of plasma fibrinogen it does not get consumed in a normal way as part of the coagulation process

- Systemic anti-coagulation: when we would expect and hope that the body is forming clot in its most efficient and reliable to control any major bleeding associated with the trauma, we do in fact find that there is considerable evidence of the systemic anti-coagulation, which is likely to further increase bleeding.

- Hyperfibrinolysis: in addition to the systemic anti-coagulation and inhibition of clot formation, there is also activation and acceleration of the bodies 'clot breakdown' process or anti-fibrinolytic system.

All three elements seem totally in conflict with what we would expect in a patient who us bleeding and has traumatic injuries and this seems to worsen with increasing levels of trauma and Injury severity score.

Brohi goes on to describe in more detail how these processes appear to occur, but

this does not explain why they occur. When the endothelium is damaged the platelets are activated with factors VIIa, IXa and Xa they produce Thrombin. This thrombin will ultimately lead to the production of fibrin and the fibrin plug, which will strengthen the clot and seal the damaged endothelium.

The thrombin formed binds to thrombomodulin on the surface of the endothelium and this forms a thrombomodulin complex (Th-TM). The thrombin also has a number of other effects including a strong positive feedback effect on the production of thrombin, which greatly accelerates the process in the typical 'thrombin burst' described in coagulation. The Th-TM complex actually inhibits this process by activating and promoting the formation of Activated protein C (APC) from Protein C. The APC slows or reduces the formation of thrombin and is effectively and anti-coagulation process.

In addition, the APC and increased levels of thrombin act on tissue plasminogen activator (TPA) to increase plasmin levels, which have a thrombolytic effect on the fibrin formed.

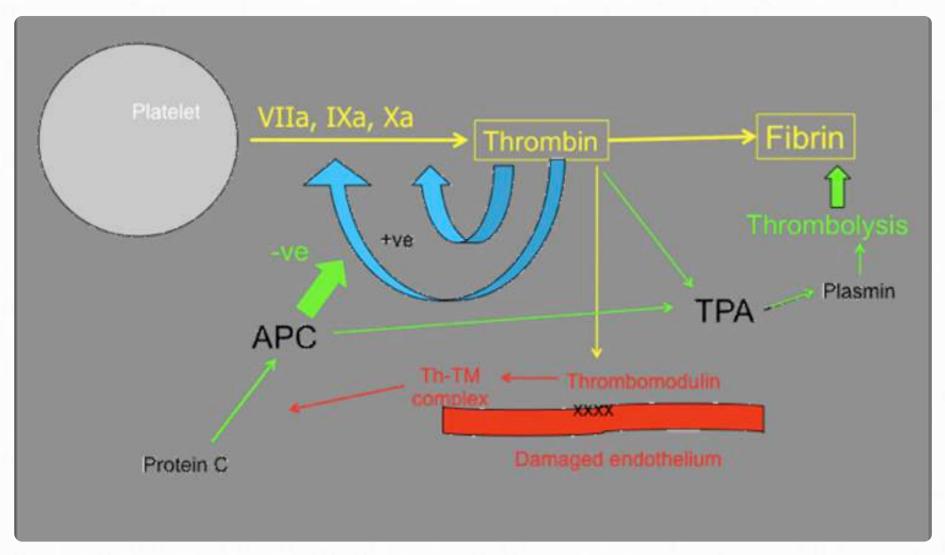In summary, as the damaged endothelium activates the normal coagulation pathway which results in the production of a fibrin plug, there is simultaneous activation of mechanisms that not only inhibit the formation of clot but that also actively break it down. Clearly as we can demonstrate a worsening coagulopathy in major trauma patients the balance swings towards the anticoagulation and fibrinolytic processes.

The coagulation processes in endothelial repair
Protective system or runaway train?

Brohi offers a credible explanation for what we are seeing in trauma. He suggests that there are two situations, minor traumatic injury and major trauma.

If we consider a minor traumatic injury such as an isolated upper limb injury with significant bleeding. This will result in activation of the clotting system to minimise the blood loss, however we do not want clot or thrombus to form in other vascular

The Coagulation process in endothelial repair

beds, especially if there is a degree of hypovolaemic and relatively low flow in some of these areas. It appears that the through our evolution the activation of our 'anti-clotting' systems through Protein C and Fibrinolysis has developed to provide the necessary degree of protection against unwanted clot in un-injured areas of the body, whilst clot can still from at the points of injury.

However, in major traumatic injury this process appears to get somewhat 'out of control' and there is a massive bias towards the anti-clotting pathways. We believe that this can be compared to the 'runaway train' effect that is seen in severe sepsis or SIRS, where a protective system of the body becomes greatly exaggerated or accelerated and actually is then the source of further harm as a protective system out of control.
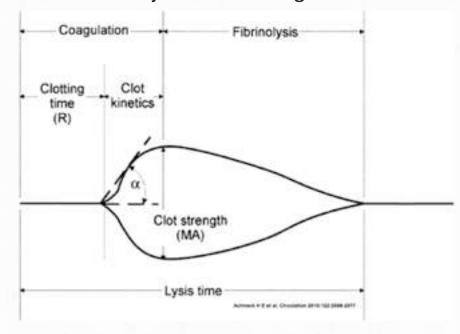
A new approach

By identifying this balance between clotting and anti-clotting systems, we can see that rather than struggling to blindly correct a worsening coagulopathy or to even prevent any form of clot inhibition, we should in fact be seeking to identify methods of measuring the current state of the balance and then to modulate and even fine tune the balance for the

benefit of the injured individual. This is very much a new concept and we do not currently have the necessary tests and tools to manage the situation, but we are already taking steps in the right direction

One such step is the increasing use of visco-elastic haemostatic assays (VHA) which look at the formation, quality and breakdown of clot in real time, in the clinical or near clinical environment. The aim must be to use such assays to identify individual factors to modulate the balance and offer a form of goal-directed therapy in coagulation in trauma. This would hopefully avoid the current 1:1:1 fixed ratio, 'bucket chemistry' approach which is ultimately likely to be inaccurate, crude, expensive and potentially not without complications.

The two VHAs being used increasingly in clinical environments are based on the thromboelastograph and the thromboelastogram and they are both produced from what was a laboratory tool. Normal coagulation tests are useless in the dynamic and rapidly changing situation of major trauma and ongoing bleeding, as the time that the results have arrived back even from the most efficient laboratory, things will have changed so much that the results are of limited or no value.

To adopt effective damage control methods, we need something that is rapid and readily accessible to those looking after the patient. Both TEG and ROTEM machines plot graphical representations of a number of key elements in the coagulation pathway.
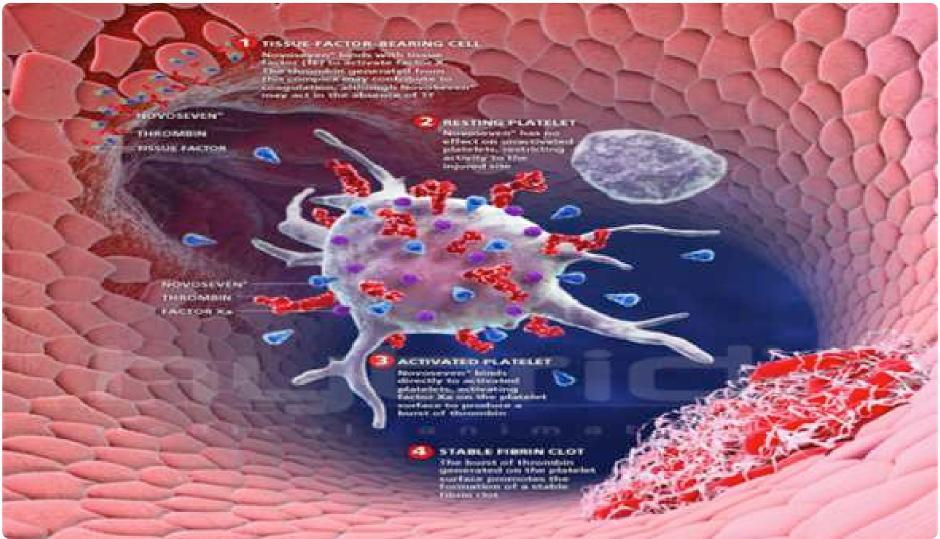
• Speed of clot initiation

• Kinetics of clot growth

• The stability or strength of the fibrin clot

• The breakdown of clot through fibrinolysis

Numerous studies have demonstrated various degrees of improved outcome or reduced mortality when these devices are utilised in major haemorrhage.

TEG tracing of hyperfibrinolysis

The curves for both thromboelastogram and thromboeleastography are the same shape but each element represents some-

The action of factor VIIa

thing slightly different. The diagram overleaf summaries what each area represents and demonstrates the coagulation and fibrinolytic phases of clot formation and breakdown.

Other clotting factors

Activated Factor VII appeared to offer so much in its early trial in trauma. Since then there have been many anecdotal reports of good effect and numerous RCTs with no clear conclusion. NICE have reviewed the evidence and they do not believe that there is currently sufficient weight of evidence to justify both the high costs and also the risks of potential thrombo-emboli.

What is very clear is that no single factor in such a complex process is going to prevent or reverse the complex process of ATC. There does seem to be a place for VIIa but as to how soon and in which patients has proved difficult to identify, although VHA has offered some potential guidance with some support in the literature. This may be the basis of a more theragnostic approach where we are delivering the necessary factors on a focused and deliberate manner in response to an indicated deficiency.

If we are going to use something as costly as VIIa then we must also be aware that if administered to casualties that are cold or acidotic then it will lose up to 90% of its effect and the dose will be wasted. Normal homeostasis plays a fundamental part in the coagulation process and VIIa is no different.

Fibrinogen

Fibrinogen levels drop rapidly during large volume transfusion, especially with crystalloids, stored blood, packed red cells and hydroxy ethyl starches. As the level falls the ability to affectively form a stable clot is impaired and replacement therapy should be considered early with blood transfusion. But how early is still under question? Traditionally it was left too late and once considered there was then also a delay in supply from the lab.

The delay in thawing fresh frozen plasma and the availability of cryoprecipitate has prompted many European countries to adopt freeze dried fibrinogen concentrate. This can be stored 'on the shelf' and reconstituted immediately as required.

An early study, which was highly supportive of fibrinogen supplementation with improved survival in combat trauma casualties, was published by Stinger et al in J Trauma, 2008. Fibrinogen concentrate is not currently available in the UK but it is recommended when measured levels are low (<1.5g/dl) and this may be a viable option that is readily available in the future.

Fibrinogen concentrate

This issue has been confused by a recent paper published in J Trauma by Wafaisade et al who have demonstrated that when fibrinogen concentrate is used, as in many parts of Europe, in exsanguinating major trauma patients they have been able to demonstrate a significant improvment in 6 hour survival to Intensive care. However, when they looked at overall survival to discharge there was a significantly increased mortality in the group that had received the fibrinogen concentrate. This was largely due to multiple organ failure (61.2% vs 49.0%) and thrombo-emboli (6.8% vs 3.4%).

This clearly supports the views of many critics that the blanket and undirected ad-

ministration of large volumes of plasma, fibrinogen and platelets is not without risk and we need to identify a far more accurate method to direct our blood product transfusions.

Prothrombin Complex Concentrate

Prothrombin complex (PCC) such as Beriplex and Octaplex contain the factors II, VII, IX and X which are essential for the formation of thrombin. The current indications for PCC are:

- The management of congenital clotting disorders such as von Willebrand's disease.

- Reversal of warfarin anticoagulation

As yet, whilst there is considerable interest, there is little supportive evidence for its use in trauma, other than in warfarinised patients. However, several studies have suggested that it may well be of value in trauma and that it certainly warrants further investigation.

Thrombin nanotechnology

As Thrombin is a key element in the coagulation process, by providing a great source of it we should promote improved coagulation and haemorrhage control. Taking this concept forward the Haemtrix dressing uses nano-particle technology to deliver thrombin into open wounds. The manufacturers are very positive about its effects but as yet there is little evidence to support its use in clinical terms.

Factor XIII

Factor XIII is rarely considered when we talk about coagulation but it may well be far more important than we have previously considered. XIII binds platelets and fibrin to strengthen the clot and it also enhances this clot firmness by resisting fibrinolysis.

In cases of poorly controlled post-operative bleeding and oozing, levels have been recorded as high as 60% of normal and clearly need to be even higher. But as yet, there is no research or evidence to guide us in this when it comes to bleeding in trauma.
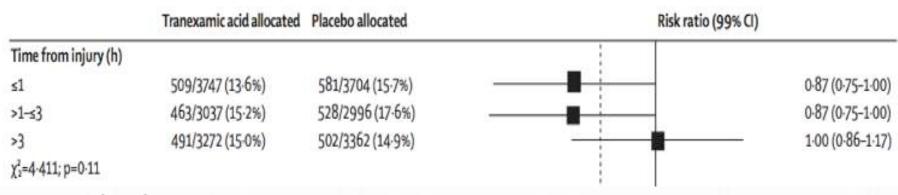
Anti-fibrinolytic agents

If we are looking for a single agent that has a proven effect on outcome then the anti-fibrinolytic agents are a good starting point. These agents inhibit the fibrinolytic process, which as we have seen, is activated as part of the 'down-regulation' of the clotting process and which can be somewhat out of control in major trauma. As such, they promote the breakdown or lysis of fibrin and clot.

Agents that inhibit this process such as aprotonin and tranexamic acid (TXA) have been used for a number of years in elective orthopaedic and liver surgery with good results in terms of reduced blood loss and postoperative transfusion. However in November 2007, Aprotonin was withdrawn from clinical use following a significant risk of thrombotic complications including stroke. Clearly it was too effective in the inhibition of clot breakdown, thereby increasing the risks of thrombosis in small vessels and low flow vascular beds. Fortunately, this rate of complication does not appear to be such an issue for tranexamic acid, but this may reflect that fact that it is not as effective as an anti-fibrinolytic agent. The CRASH-2 trial was completed to answer this question.

The 'Crash 2' – Clinical Randomisation of Antifibrinolytics in Significant Haemorrhage – 2 was a very well conducted randomised control trial in adult trauma patients. The trial was conducted in 274 hospitals in 40 countries over a 5 year period, between 2005-10.

20,000 trauma patients with a blood pressure less than 90 systolic, a heart rate greater than 110 bpm and a significant risk of a post traumatic bleed were included in the trial. They were given a dose of 1g tranexamic acid over 10 minutes, within the first 8 hours, followed by a further dose (1g) over the following 8 hours.

The results were quite astounding as they demonstrated an improved 28 day mortality, reduced from 16% to 14.5% (with a 'p' value of 0.0035). Whilst this 1.5% improvement may seem small, there are few, if any, other recent advances that have such a positive effect in major trauma cases. What was even better was the fact that there were no increased complications, including stroke or thrombosis.

Time from injury (h)	Tranexamic acid allocated	Placebo allocated	Risk ratio (99% CI)		Risk ratio (99% CI)
≤1	509/3747 (13·6%)	581/3704 (15·7%)			0·87 (0·75–1·00)
>1–≤3	463/3037 (15·2%)	528/2996 (17·6%)			0·87 (0·75–1·00)
>3	491/3272 (15·0%)	502/3362 (14·9%)			1·00 (0·86–1·17)
χ^2_1=4·411; p=0·11					

Results of CRASH-2 Trial

Following a further review of the data it became clear that to achieve the best results, without compromise or complication then the TXA must be administered within the first three hours of injury. After this, there does appear to be a reduced benefit and a potential increase in mortality. In light of this we are now considering TXA at the earliest possible opportunity, as soon as signs of significant bleeding have been identified, which may even be in the pre-hospital phase. As a result TXA has now been approved by JRCALC and has already been adopted by a number of UK ambulance services.

Theragnostic approach

The introduction of VHA machines within the resus room, theatres and critical care areas, in addition to all of the combination and selective clotting agents above, has presented the opportunity to take more of a 'goal directed' approach to coagulopathy prevention and management. This is referred to as a 'Theragnostic approach' and whilst this may well seem to be a real common sense and measured approach, rather than the 'bucket chemistry' crudity of the current 1:1:1 methods, there are as yet no successful published trials.
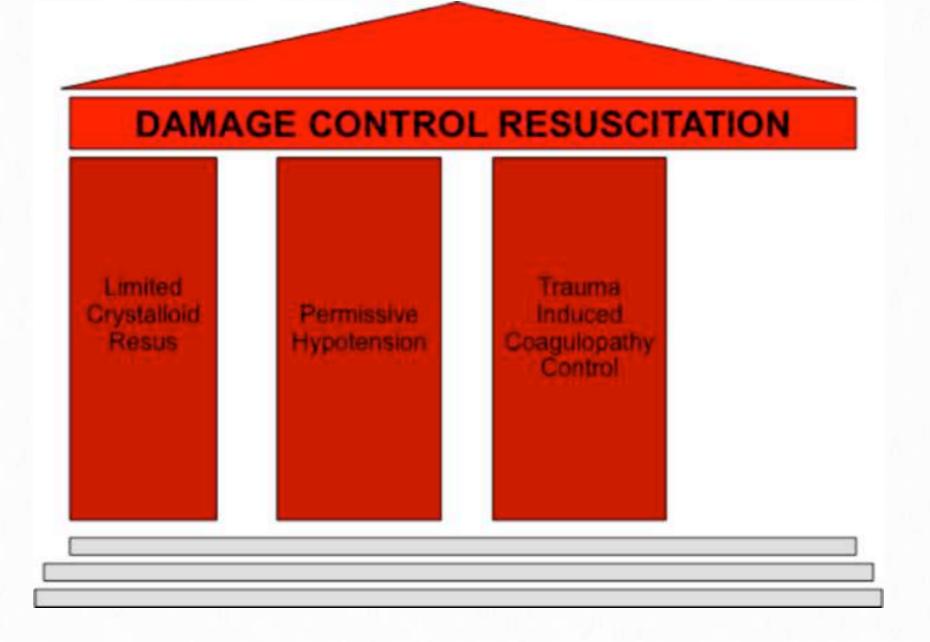
Some of the animal work has raised concerns that all of these agents that we are currently using are 'pro-coagulant' and a more balanced approach would be more physiological. Anticoagulant agents such as antithrombin 3, protein C and protein S may actually play an important part in future coagulation optimisation to control the 'runaway train effects'.

Physiological exhaustion

If the trauma induced coagulopathy is not managed effectively and the surgical and anaesthetic team do not follow the principles of damage control, then during more prolonged surgery the patient can start to bleed and ooze from everywhere, without apparent injury to a vessel. This is a state of physiological exhaustion and coagulation failure.

If this develops, it is a clear sign that surgery needs to stop as soon as possible. The wound or bleeding area must be packed effectively and the patient transferred to critical care where some degree of homeostasis can be restored

Anecdotal reports have suggested that fresh whole blood or Factor XIII may play a major beneficial role in this situation.

DAMAGE CONTROL RESUSCITATION

Limited Crystalloid Resus | Permissive Hypotension | Trauma Induced Coagulopathy Control
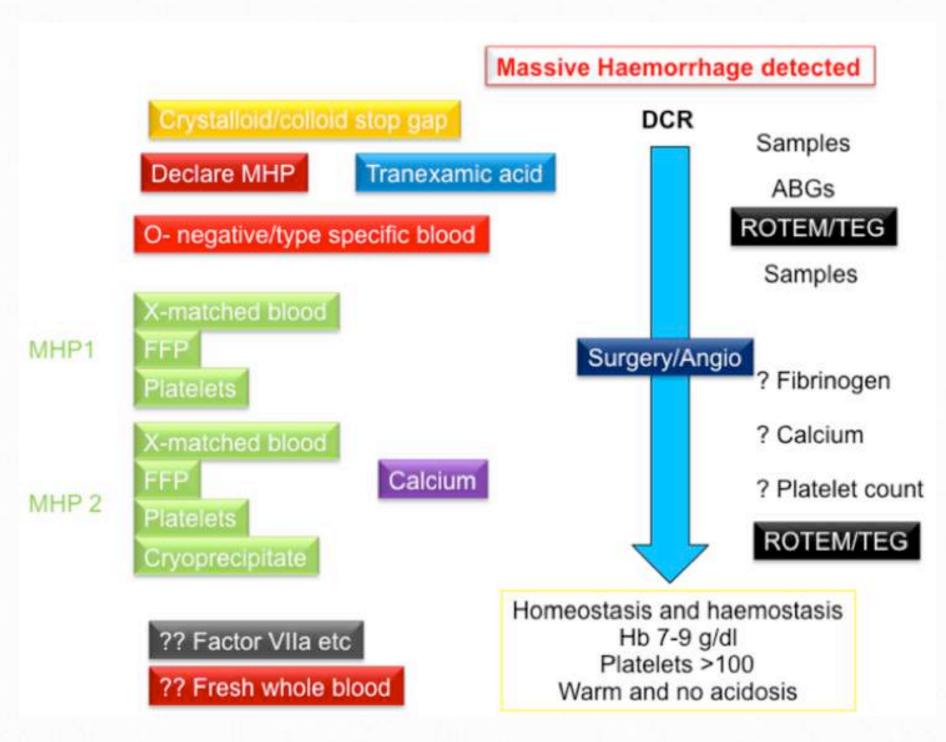
The third pillar of Damage Control Resuscitation

Considering our representation of damage control resuscitation we can now add a third pillar, which represents the control measures adopted to minimise the Trauma Induced coagulopathy. From the diagram we can see that further pillar is yet to be described in this hugely challenging process of care.

Summary

In summary, we need to identify patients with major bleeding as early as possible. We limit the crystalloid resuscitation, adopt permissive hypotension and activate the massive haemorrhage policy. Samples should be drawn early to assess acid-base status, haemoglobin and VHA samples. From there on in we should aim to either adopt a 1:1:1 approach with packed red cells, FFP and platelets or alternatively transfusion of blood products guided by the VHA analysis.

When the second MHP arrives, it should also include Cryoprecipitate and we should give calcium supplementation. Whilst this Traumatic coagulopathy is being managed, we

Massive Haemorrhage detected

Crystalloid/colloid stop gap

Declare MHP   Tranexamic acid

O- negative/type specific blood

MHP1
- X-matched blood
- FFP
- Platelets

MHP 2
- X-matched blood
- FFP
- Platelets
- Cryoprecipitate

Calcium

?? Factor VIIa etc

?? Fresh whole blood

DCR

Samples
ABGs
ROTEM/TEG
Samples

Surgery/Angio
? Fibrinogen
? Calcium
? Platelet count
ROTEM/TEG

Homeostasis and haemostasis
Hb 7-9 g/dl
Platelets >100
Warm and no acidosis

should be moving through the trauma system. Rapidly transferring from ED to CT to theatre and onto critical care as soon as possible,

This is a hugely challenging pathway and we can lose control even at some of the earliest points. From there onwards, we are playing catch up and that is inevitably going to create a greater compromise and risk to life. We certainly don't know the whole story, especially when it comes to coagulopathy, but we are learning more all the time and the increasing availability of real tests of clot formation, strength and breakdown such as VHA will hopefully allow us to modulation coagulation favourably and without complications in the future.

References:

1. Br J Anaesth. 2012 Dec;109 Suppl 1:i39-i46. doi: 10.1093/bja/aes389.Haemostatic resuscitation.Dutton RP.

2. Ann Surg. 1969 Apr;169(4):455-82.Coagulation disorders in combat casualties. I. Acute changes after wounding. II. Effects of massive transfusion. 3. Post-resuscitative changes.Simmons RL, Collins JA, Heisterkamp CA, Mills DE, Andren R, Phillips LL.

3. J Clin Pathol Suppl (R Coll Pathol). 1970; 4: 110–120.PMCID: PMC151998 R M Hardaway

4. J Trauma. 1994 Sep;37(3):426-32.Death in the operating room: an analysis of a multi-center experience.Hoyt DB, Bulger EM, Knudson MM, Morris J, Ierardi R, Sugerman HJ, Shackford SR, Landercasper J, Winchell RJ, Jurkovich G, et al.

5. J R Army Med Corps. 2009 Dec;155(4):320-2.Trauma induced coagulopathy.Brohi K.

6. DirkmannD, Hanke AA, Gorlinger k, Peters J. Hypothermia and acidosis synergistically impair coagulation in human whole blood. AnaesthAnalg 2008: 106: 1627-1632
    http://www.trauma.org/index.php/main/article/374/

7. Home>articles>permissive hypotension:Discussion archive Permissive Hypotension: Discussion Archive Permissive Hypotension for Trauma Resuscitation October 01, 2002

8. The Syndromes of disseminated intravascular coagulation. Hardaway RM, Mckay DG Rev Surg. 1963 Sep-Oct;20:297-328. Review.

9. Int J Burns Trauma. 2012;2(3):145-57. Epub 2012 Dec 5.Acidosis and correction of acidosis does not affect rFVIIa function in swine.Darlington DN, Kheirabadi BS, Scherer MR, Martini WZ, Dubick MA.

10. J Trauma. 1998 May;44(5):846-54.Hypothermic coagulopathy in trauma: effect of varying levels of hypothermia on enzyme speed, platelet function, and fibrinolytic activity.Watts DD, Trask A, Soeken K, Perdue P, Dols S, Kaufmann C.

11. J Trauma. 2011 Mar;70(3):652-63. Hypotensive resuscitation strategy re-

duces transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: preliminary results of a randomized controlled trial.Morrison CA, Carrick MM, Norman MA, Scott BG, Welsh FJ, Tsai P, Liscum KR, Wall MJ Jr, Mattox KL.

12. Targeted resuscitation improves coagulation and outcome. Doran CM1, Doran CA, Woolley T, Carter A, Male K, Midwinter MJ, Mahoney PF, Watts S, Kirkman E. J Trauma Acute Care Surg. 2012 Apr;72(4):835-43.

13. A mathematical model for fresh frozen plasma transfusion strategies during major trauma resuscitation with ongoing hemorrhage. Ho AM1, Dion PW, Cheng CA, Karmakar MK, Cheng G, Peng Z, Ng YW. Can J Surg. 2005 Dec;48(6):470-8

14. Fresh frozen plasma should be given earlier to patients requiring massive transfusion J Trauma. 2007 Jan;62(1):112-9. Gonzalez EA1, Moore FA, Holcomb JB, Miller CC, Kozar RA, Todd SR, Cocanour CS, Balldin BC, McKinley BA.

15. A randomized controlled trial of transfusion-related acute lung injury: is plasma from multiparous blood donors dangerous? Palfi M, Berg S, Ernerudh J, Berlin G: Transfusion 2001,41:317-322

16. Management of bleeding following major trauma: a European guideline Donat R Spahn, Vladimir Cerny, Timothy J Coats, Jacques Duranteau, Enrique Fernández-Mondéjar, Giovanni Gordini, Philip F Stahel, Beverley J Hunt, Radko Komadina, Edmund Neugebauer, Yves Ozier, Louis Riddez, Arthur Schultz, Jean-Louis Vincent, Rolf Rossaint Crit Care. 2007; 11(1): R17. Published online 2007 February 13. doi: 10.1186/cc5686 Correction inCrit Care. 2007; 11(2): 414.

17. Acute traumatic coagulopathy. Curr Opin Anaesthesiol. 2012 Apr;25(2):229-34. Frith D1, Davenport R, Brohi K.

18. Usefulness of thrombelastography in assessment of trauma patient coagulation. Kaufmann CR1, Dwyer KM, Crews JD, Dols SJ, Trask AL. J Trauma. 1997 Apr;42(4):716-20; discussion 720-2.

19. Recombinant activated factor VII for adjunctive hem- orrhage control in trauma. Martinowitz U, Kenet G, Segal

E, Luboshitz J, Lubetsky A, Ingerslev J, Lynn M: J Trauma 2001, 51:431-438. discus- sion 438–439
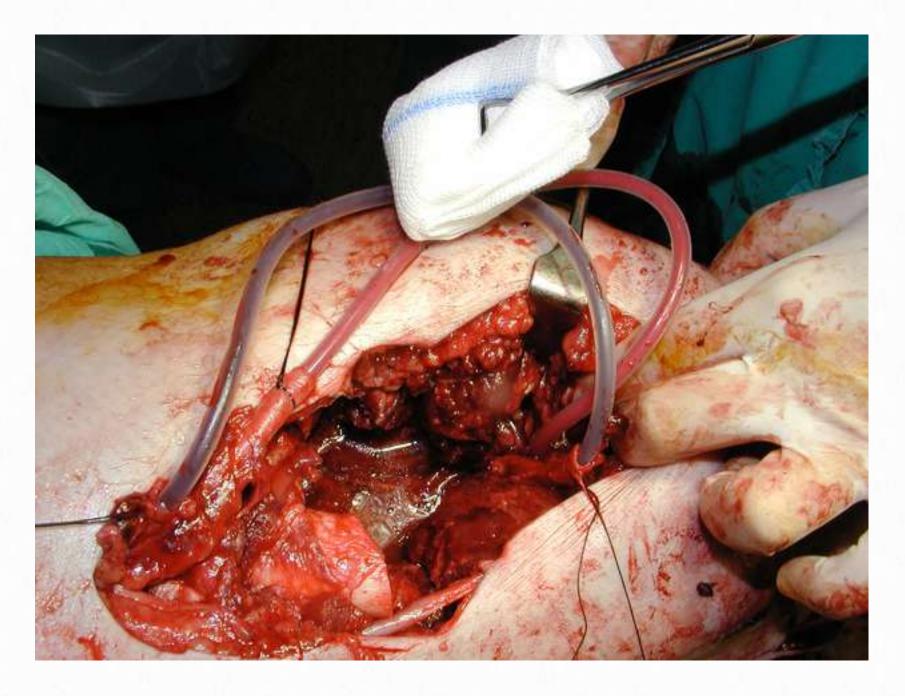
20. The effect of temperature and pH on the activity of factor VIIa: implications for the efficacy of high-dose factor VIIa in hypothermic and acidotic patients Meng ZH, Wolberg AS, Monroe DM, Hoffman M.. Journal of Trauma.2003;55:886–91.

21. Blood transfusion and the anaesthetist: management of massive haemorrhage - D Thomas, M Wee, P Clyburn, I Walker, K Brohi, P Collins, H Doughty, J Isaac, PF Mahoney, L Shewry, Association of Anaesthetists of Great Britain and Ireland Anaesthesia. 2010 November; 65(11): 1153–1161

22. The ratio of fibrinogen to red cells transfused affects survival in casualties receiving massive transfusions at an army combat support hospital.Stinger HK1, Spinella PC, Perkins JG, Grathwohl KW, Salinas J, Martini WZ, Hess JR, Dubick MA, Simon CD, Beekley AC, Wolf SE, Wade CE, Holcomb JB. J Trauma. 2008 Feb;64(2 Suppl):S79-85; discussion S85.

23. Administration of fibrinogen concentrate in exsanguinating trauma patients

is associated with improved survival at 6 hours but not at discharge. Wafaisade A1, Lefering R, Maegele M, Brockamp T, Mutschler M, Lendemans S, Banerjee M, Bouillon B, Probst C; Trauma Registry of DGU. J Trauma Acute Care Surg. 2013 Feb;74(2):387-3; discussion 393-5.

24. Tranexamic acid, Fibrinogen Concentrate and Prothrombin Complex Concentrate: Data to Support Prehospital Use?Schöchl H1, Schlimp C, Maegele M.Shock. 2013 Nov 27.

25. Decreased factor XIII availability for thrombin and early loss of clot firmness in patients with unexplained intraoperative bleeding. Wettstein P1, Haeberli A, Stutz M, Rohner M, Corbetta C, Gabi K, Schnider T, Korte W.Anesth Analg. 2004 Nov;99(5):1564-9

26. CRASH-2 trial collaborators Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. Lancet.2010;376:23–32.

27. Theragnostic performance of ex vivo testing of haemostatic intervention and

clinical effect of recombinant factor VIIa and fibrinogen concentrate. Tang M, Wierup P, Christiansen K, Greisen J, Sørensen B.Thromb Res. 2012 May;129(5):e263-5. doi: 10.1016/j.thromres.2012.03.002. Epub 2012 Apr 3.

In-hospital Damage Control Resuscitation

Lets us now consider how the 'Damage Control Resuscitation' continues within the hospital. This chapter pulls together many of the topics discussed in the previous sections and aims to get the patient in the best condition to theatre or other life-saving care. We will also touch upon the key decisions and methods adopted by the trauma surgeons in the field of damage control surgery.

At the roadside or pre-hospital we have identified the key triggers for a damage control approach, with all its necessary compromises. We will have adopted this process and aimed to rapidly stabilise the casualty as quickly as possible before rapidly transferring to the hospital.

This rapid stabilisation and transfer to what was described as 'definitive care' has been traditionally referred to as the 'Golden hour'. In that, if we take longer

than 60 minutes to get to this point, then the likelihood of survival will dramatically fall. However, if we review the literature and the potential sources of the 'Golden hour' principle, we find that there is little if any hard evidence and it is little more than a concept proposed by Cowley in the 1960's (1).

In addition, what is also clear is that many of our patients do not have anything like an hour to get to hospital, as they will die in minutes without appropriate life saving measures. As a result other terms such as the 'Platinum 10' have been adopted to highlight the need for certain life saving interventions that must occur to preserve life for transport. We are in effect producing a "Window of opportunity" to transfer our patient, with sufficient stability to survive the transfer but without unnecessary delay.

Following a rapid assessment of mechanism and injuries certain measures must be adopted immediately to manage massive external life threatening haemorrhage or airway compromise, whereas other measures such as cannulation, ongoing resuscitation, splinting etc can actually take place during transfer. This is a whole new concept, as traditionally transfers of critically ill patients are usually only conducted in a stable controlled fashion, between criti-

cal care units. During these ITU transfers, a well-packaged and prepared patient will require little, if any intervention or management beyond observation en route (2, 3).

However, we are now considering major trauma transfers, with rapidly changing states and casualties who may well be actively bleeding or deteriorating. As such we aim to simply perform the essential damage control procedures to "get the ship home" or to get the patient to a dedicated hospital facility with all its staff and resources. This is not "stay and play or just simply "scoop and run" but rather a best compromise where by we buy time for the transfer with life saving measures only and then 'scoop and manage' everything else en route.

The hospital should be pre-alerted and the Major trauma team should be gathered and briefed.

The Trauma Team

The Team Leader should ensure that members are present and assign suitable roles. These roles may vary slightly based on the pre-alert briefing, but should essentially be the same format that should have been rehearsed and practiced by the team during regular simulated sessions. Roles should be clearly identified (e.g. named tabards

under plastic aprons) and anyone not assigned a role should be stood outside the main resuscitation area or asked to leave the department.
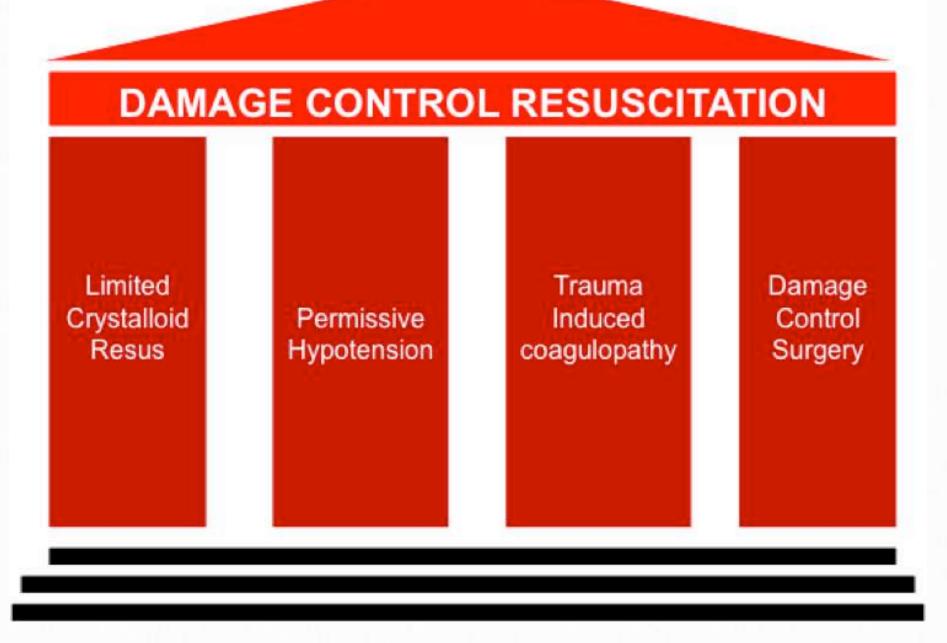
A typical team would include, from head to toe:

- Anaesthetist – airway

- ODP – supporting the anaesthetist

- ED Registrar - Left side of patient to assess chest/breathing (incl. drains and cannulation/bloods), eFAST

- Nurse – supporting ED doc

- Surgical registrar – assess circulation then abdomen and pelvis

- Orthopaedic surgeon for pelvis and extremity trauma

- Nurse to assist surgeon

- Team Leader

- Scribe

Everyone should be wearing personal protective equipment, although the Team Leader may chose to not wear gloves as ideally they do not intend to touch the patient. They are here to conduct the orchestra, not to play the instruments.

All necessary drugs & equipment should be checked and ready for advanced airway management, chest decompression, thoracotomy, access, analgesia, anaesthesia and immobilisation. CT should also be informed that a Code Red patient is expected imminently and to keep the scanner free and depending upon the case the massive haemorrhage protocol may be activated and theatre informed.

Human factors and team resource management plays a huge part in the trauma team reception and this is considered at numerous points elsewhere in the course. However, good team leadership, communication and clearly defined roles make for a far better response, An air of calm should exist across the resuscitation room and at this stage team members should prepare and mentally rehearse their roles. If more senior help is likely needed then it should be called early.

The response and stress levels of the team can be greatly improved through regular simulated practice for all sorts of eventualities and cases. Just like Formula 1, the roles of each team member can be clearly defined and whilst traumatic injuries are all different, the overall management principles and practices remain the same.

The Pillars of Damage Control Resuscitation

Challenge check-lists are considered invaluable for certain procedures such as emergency anaesthesia or rapid sequence induction (RSI). However, some actions should be considered "Bold face" and should be instinctive for any team member without need for thinking or checklists e.g. control of bleeding, ventilation in an apnoeic patient.

From the scene it may be very clear that some patients have major internal bleeding and warrant immediate theatre or angiography. We should aim to stop in resus for the shortest possible time as they continue to bleed and anything that we do will merely result in greater blood loss. For such patients, the 'shortest corridor to theatre' is the best option but this can only be agreed following a suitable discussion between the Team Leader, the surgeons and others involved. In Camp Bastion and more recently in some UK centres the system was even an arrangement called "Right Turn Resuscitation" with direct transfer into theatre as an option if necessary, to give the surgeons the most time to open the pa-

tient and to attempt some form of surgical control.

In terms of or key foundations of DCR we are currently adopting the following:

- Limited or no crystalloid resuscitation
- Permissive hypotension
- Prevention or limitation of trauma induced coagulopathy
- Moving towards theatre and damage control surgery early

A key element of this is decision making and this is where the whole trauma management process can fail or breakdown. As if these patients are not identified early and they simply enter the "conventional" management pathway then we risk increasing bleeding, losing valuable time and potentially increasing their mortality.

For some patients the mechanism of injury alone may be the only identifiable trigger for the DCR pathway in the early stages, whilst in other cases we may have still not identified the nature or severity of the injuries but our reassessment in the resus room and our immediate investigations such as bloods (including venous blood gases for lactate, base deficit and haemo-

globin), EFAST ultrasound and CT scanning may support any clinical suspicions.

For the patients that our clearly deteriorating from blood loss from one or numerous sites, then we continue our DCR management with ongoing permissive hypotension and also haemostatic hybrid resuscitation to minimise to effects of any developing trauma induced coagulopathy, which as we know, may already exist.

Permissive Hypotension – not without risk

Permissive hypotension is a necessary evil, as we know that any serious attempts to restore circulating volume or blood pressure back to normal levels will increase bleeding, further losses and mortality until haemostasis is achieved.

However, we also know that leaving the body in such a 'shocked', hypovolaemic and under-perfused state for a prolonged period will also lead to serious complications and potentially death. As a result, a number of groups have now started to consider what may be the best compromise e.g. "hybrid resuscitation". What is clear is that the shorter the period of shock the better and beyond an hour or with rapidly rising blood lactates or base deficits then the outcome will worsen.

A number of other DCR protocols under trial, focus on maintaining flow within the tissues, with anaesthetic agents such as incremental fentanyl, that maintain tissue perfusion with a degree of vasodilation, supported by sufficient blood transfusion to avoid worsening shock and under-perfusion.

Obviously, once we are confident that we have controlled the immediately life threatening bleeding then circulating volume and perfusion must be rapidly restored.

Haemostatic Resuscitation

The Haemostatic side of DCR must be as aggressive as the control of bleeding as they will both be required to stabilise and patient with severe poly-trauma. This is becoming far more than simple random allocation of clotting factors and blood products. It is also highly unlikely that any one single agent will be able to halt or immediately reverse the coagulopathy as was hoped with agents such as Factor VIIa.

The summary of our management of massive traumatic haemorrhage and trauma induced coagulopathy describes how it must commence from scene of injury with early detection, which may be easy with massive external haemorrhage or more subtle and difficult to spot with occult inter-

nal bleeds and active circulatory compensation for developing shock, especially in young and fit individuals.

Active control of bleeding and early transfer to hospital is essential, not only to minimise losses, but also to minimise the need for crystalloid resuscitation during this phase. Tranexamic acid should also be administered although the optimal time for early delivery has yet to be ascertained.

Once in hospital crystalloid fluid replacement should be stopped wherever possible and exchanged with blood and blood components.

Damage Control Surgery

Damage control surgery (DCS), also referred to as the 'Damage control laparotomy', has been adopted as a life-saving and temporary procedure for unstable patients who have sustained a major trauma which includes an abdominal emergency. DCS aims to help restore normal physiology, not anatomy, with a planned return for definitive treatment later. The concept has arisen from the realisation that a patient who has sustained a massive trauma lacks the physiological reserve to survive complex and prolonged definitive or reconstructive surgery. DCS is performed for operative control of haemorrhage followed by

vigorous resuscitation and other principles including preventing contamination and avoiding further injury. It is based on a principle that survival of the patient is the only priority, and that the potential for morbidity must be accepted. High complication rates are weighed against the preservation of life.

No randomised control trials have evaluated the concept of damage control surgery, but it has become accepted as part of trauma surgery, and is part of the damage control resuscitation process.

An alternative option to DCL is angiography and embolisation. This depends on the location of the bleeding, the ability to identify the isolated bleeding point, and the availability of interventional radiology in your hospital.

DCS is only usually applicable to the minority of trauma patients, and if used to liberally may be no better, or even worse, than immediate definitive surgery. Experience and rapid surgical assessment are key to making a positive, informed decision regarding adopting a DC strategy.

The five stages of Damage Control Surgery are as follows:

1) Decision to operate

- Once the patient has arrived within the Emergency Department, a quick decision regarding whether the patient needs theatre is needed. The decision needs to be reached early, in order to avoid the vicious triad being entered.

2) Operation

- Intra-operative priorities are haemorrhage control, limiting contamination and temporary closure. Haemorrhage control can be achieved by ligation, suture, tamponade i.e. with packing or a balloon. Definitive vascular repair with a graft or anastomosis is not considered a DCS procedure.

- Contamination control is achieved by closure of the end of the injured hollow viscus i.e. small bowel. Stapler devices are the quickest and easiest things to use in this situation. This is not the time to be performing a bowel anastomosis or fashioning a stoma, and the stapled off ends are dropped back into the abdomen, with definitive repair planned later. Abdominal closure needs to be rapid and temporary, with pre-emptive strategies employed to prevent abdominal compartment syndrome.

3) Restore normal physiology

- Once surgery is completed the patient is transferred to the ITU where attempts at correcting the patients' physiology continue. This is discussed in more detail later.

- Early return to the operating theatre is indicated if there is obvious ongoing surgical bleeding or compartment syndrome develops.

4) Return to theatre – definitive treatment

- This is dictated by improvement in the patients' physiological status. Before the decision to return to theatre is made, it's important to assemble the appropriate surgical team so the 'right surgery' can be performed in the 'right place' at the 'right time'. At this stage, bowel continuity can be restored with an anastomosis, stomas fashioned and vascular repairs performed.

5) Abdominal wall closure

- This may not be possible at stage 4 due to oedema or a risk of developing a compartment syndrome. Therefore a planned further procedure for closing the site, or covering, is made.

We will now consider the first three steps in the DCS process in more detail.

The Decision

As previously mentioned this is the first step in DCS and sometimes the hardest step. Does the patient need to go to theatre for DCS or can their injuries be managed without an operation? Does the patient need to go straight to the operating theatre to arrest life-threatening haemorrhage, or is there time to perform radiological investigations first?

Appreciating the mechanism of injury, and therefore injury pattern can help make a quick, early decision to proceed to DCS. It is important to minimise the time in the pre-hospital environment, and within the Resus department, with rapid progression through to the operating theatre.

FAST scanning (Focused abdominal sonography for trauma) is an investigation that is readily available in the Resus department with either a member of the A&E team, or the surgical registrar being trained to perform it. It is quick to perform, but will only indicate the presence of free fluid, meaning it is not truly diagnostic. It is also operator dependent and does not identify the origin of any abnormal fluid in the abdomen or chest. False negatives and posi-

tives will occur, and its benefit in trauma is poorly supported by clinical evidence.

CT scanning (FACTT – focused assessment with computed tomography in trauma vs. WBCT – whole body CT) should now be used routinely in trauma. It is the procedure of choice in evaluating the haemodynamically stable patient who has sustained blunt or penetrating trauma and has long replaced Diagnostic Peritoneal Lavage (DPL). CT will identify the presence of intra-abdominal or thoracic bleeding and will also identify the organ involved.

CT has a high sensitivity and specificity in detecting most solid organ injuries, but can miss some small bowel and mesenteric injuries (estimated at 15% in some series). In the majority of trauma situations, CT has become a useful method for selecting which patients are suitable for operative vs. non-operative management. In reality we should be performing head-to-toe CT scanning with IV contrast to increase the sensitivity of injury pick-up. Whole body CT has been shown in numerous studies to increase the probability of survival in patients with polytrauma. In some trauma centres the CT scanner is situated adjacent to the 'Trauma room' in resus and is utilized immediately after external haemorrhage, airway and breathing issues have

been addressed to help determine circulation problems. It can be used as part of the primary trauma survey to identify life-threatening injuries , determine if DCS is needed and then also to determine what definitive surgery and treatment is required later when the patient is no longer in a life-threatening condition. Implementation of this requires a CT scanner within close proximity to the trauma room, and also a well organised team of surgeons, anaesthetists and radiologists, all with the same goal in mind.

The main point is that whole body CT in the trauma situation can reveal unexpected or hidden diagnoses with a major therapeutic impact. WBCT scanning during the primary survey can in fact increase survival in major trauma.

Anaesthetic view of Trauma Radiology

The radiology department and especially the CT scanner or angiography suite is well recognised as a high-risk environment for critical care transfers.

The anaesthetist is typically remote from the patient, in a relatively unfamiliar location with low light levels and limited space. All of these things potentially compromise patient safety before we even consider the

often prolonged time that may be spent in this location with an unstable or even deteriorating patient.

In light of these risks we must ensure that we closely adhere to the well defined critical care transfer guidelines from the Intensive Care Society and the Association of Anaesthetists of Great Britain & Ireland. However, each case must be considered in its own right as some may be considered too unstable for a lengthy radiological procedure, whilst others may be considered just stable enough to justify a quick 'pan-scan' scan to establish what problems exist.

Simple things such as oxygen supplies, reliable/accessible venous access are even more important, as are reliable monitoring and skilled assistance.

The age-old description of the 'donut of death' need not apply with careful patient selection and high standards of transfer and ongoing resuscitation. Just like trauma transfers in ambulances there will be a far greater need to provide active ongoing care and resuscitation, rather than the stable support and monitoring typical of an intensive care transfer.

In addition, anyone who conducts such a transfer must communicate effectively with the radiology team and be prepared to call a halt to the proceedings to check the patient or to evacuate to theatre or critical care. As the trauma team should still be present, this should be simple and rapid decision.

Anaesthetic DCR mind-set

The major trauma patient who requires Damage Control resuscitation requires a different mind-set from the typical emergency patient. The closest equivalent, familiar to most experienced anaesthetists, is the hypotensive bleeding ruptured aortic aneurysm.

In these cases, we avoid full resuscitation and only anaesthetise the patient once everything is set up in theatre, with instruments and surgical team ready and the patient on the table, prepped and draped. Ideally blood and cell salvage will be ready, with adequate access and potentially and arterial line.

Some degree of permissive hypotension is maintained until surgical control of bleeding has been achieved.

This is closely analogous to the DCR approach to major trauma. However, whilst and arterial line may be a nice adjunct for patient monitoring and sampling, it should not delay the induction of an actively bleed-

ing patient as it is surgical control of bleeding rather than accurately knowing the blood pressure that will save the patient!

In terms of [anaesthetic agents of choice](#), whilst there is little definitive evidence in the literature to quote, Dutton in the US has recommended a cardio stable approach based on high dose synthetic opioids such as fentanyl. He, like others since has recommended that cardiovascular-depressant volatile anaesthetics are avoided, wherever possible.

Other studies recommend ketamine based anaesthesia and titration of blood pressure with fentanyl or blood transfusion for high or low pressures, whilst the patient is still bleeding. However, there is still insufficient evidence in the literature to strongly advocate anything too radical beyond the key principles of DCR and a well balanced anaesthetic.

The Operation

The patient should be placed on a heated mattress in theatre in the 'crucifix' position then prepped rapidly from neck to knees. Large abdominal packs soaked in an antiseptic skin solution can be used for this. The patient needs to be draped leaving adequate exposure for access.

A large midline incision is made from the xiphisternum to the pubis. This incision may need extending into the right chest or as a median sternotomy scar if needed, depending on the pattern of injury.

Gaining access to the peritoneal cavity should be rapid. Large clots are manually removed.   This can be achieved by pressing the sides of the abdomen together to express most the clot and blood onto the drapes and then packing any areas of ongoing haemorrhage. Plenty of large abdominal packs should be ready, along with suction, preferably via a cell-saver system.

Once in the abdomen prepare for immediate control of haemorrhage (DDIT), usually with four quadrant packing, using multiple large abdominal packs. This will provide useful information about the site and severity of bleeding. A large self-retaining retractor can then be placed into the wound to free up all surgical hands and allow for maximal exposure.

The main point of the laparotomy is to identify the main site of bleeding. Immediate control is with direct pressure using a hand or packs. All four quadrants of the abdomen are packed and left to control major haemorrhage. Packs should be removed in a rapid but orderly fashion, with the quad-

rants least likely to be the source of bleeding removed and examined first. This gives maximum room and exposure in the areas where major haemorrhage is most likely to be. The initial control of vascular injuries is with vascular clamps and suture ligatures. Definitive repair of complex arterial injuries is delayed until control is achieved. Temporary shunts can be placed (i.e. silastic catheters) to maintain vessel patency in critical vessels. These shunts are later removed and definitive repairs carried out at re-look surgery. If a vessel is identified, a clip can be placed directly on to allow it to be tied off, or a stitch used for haemostasis. Bleeding from the liver, spleen or kidney can usually be controlled with a large number of abdominal packs to apply pressure.

In patients with persistent haemorrhage from abdominal great vessel injury not amenable to packing, the priority is given to inflow and outflow control of the injured vessel. Temporary aortic control can be gained at the level of the diaphragm by the hiatus. Alternatively the aorta can be accessed via a left sided anterolateral thoracotomy if proximal control is needed. Once aortic inflow is controlled, abdominal control needs to be achieved which requires adequate exposure. The abdominal aorta and common iliacs can be visualized by a

left medial visceral rotation (Mattox maneuvre). A right medial visceral rotation (Catell-Braasch maneuver) will allow visualization of the infra-renal IVC and aorta. A Kocher maneuver then allows exposure of the IVC to sub-hepatic level as well as fully mobilizing the duodenum and head of pancreas.

Once bleeding is controlled, the next step is to prevent contamination. All solid organs need to be examined for signs of injury and then the small bowel and large bowel needs to be examined fully. A bowel walk should be performed from the DJ flexure, all the way down to the rectum, making sure to examine the duodenum separately. This should be done more than once as small enterotomies, particularly within the small bowel can be missed.

All intra-abdominal, and most retroperitoneal, haematomas require exploration and evacuation. Small haematomas arising from the bowel, pancreas or any other organ could be masking a serious vascular or enteric injury. Be sure to inform your anaesthetist as you are about to explore as a large bleed that has been previously tamponaded could be unleashed! Non-expanding peri-renal haematomas, retro-hepatic haematomas or blunt pelvic haematomas should not be explored and can

be treated with abdominal packing, with later angiography and embolization.

Packing that adequately controls a retro-hepatic haematoma should not be taken out to further explore the wound acutely.

Organ specific injuries

Spleen

Severely injured patients undergoing DCS with active haemorrhage of the spleen should undergo splenectomy. If DCS is taking place this is not the time for splenic conservation. Observational management or packing of the spleen is not feasible and prone to failure, which may require a second early return to theatre. Angiographic embolization can be a useful adjunct in the non-operative management of a haemodynamically stable patient with continued bleeding from a splenic injury.

Liver

A number of temporising techniques have evolved for hepatic injuries. These include Pringles manoeuvre, peri-hepatic packing, manual compression or plugging (using gauze, pads, gelatin sponges or balloon catheters). Definitive resection should be delayed.

Small wounds of the liver parenchyma can be managed with electrocautery or simple suturing. Direct suturing should be limited to lacerations of less than 3cm in size to reduce the risk of bile duct injury.

Topical haemostatic agents, such as fibrin sealants, can be used in conjunction with packing in larger lacerations. For deeper lacerations of the liver, packing will be successful in arresting most bleeding. Prolonged and extensive surgical procedures in the unstable patient results in poor outcome with high mortality due to the related coagulopathy, acidosis and hypothermia associated with a prolonged laparotomy.

Peri-hepatic packing is a basic damage control technique to stop haemorrhage from the liver. The procedure requires caution during the application, close observation after the operation and experience to repair the injury in the re-look operation.

Liver packing will not control an arterial bleed and any bleeding artery should be ligated before packing. An alternative is to perform peri-hepatic packing and arrange angiography and embolisation.

In cases of complex hepatic injuries, DCS should be followed by angiography which may identify intra-hepatic arterial bleeding that can be controlled with embolisation.

Peri-hepatic packing is a life-saving technique in DCS and is one of the most popular methods for controlling major liver injuries temporarily, providing enough time to correct physiological and metabolic derangements. The most common problem with this technique is determining when to perform it, and when to remove the packs. The decision to pack should be made early to give a better chance of survival. Plan to avoid complex surgical maneuvers in the first operation, but an early re-look.

Hepatic angio-embolization is recommended post-packing and can be superior over definitive surgery.

Bowel

Following the control of the vascular injuries, bowel injuries need to be controlled to prevent contamination. The whole bowel needs to be walked to examine for enterotomies, which if small, can be primarily closed with interrupted sutures. All injuries that can not be repaired by a simple suture repair are resected locally, or en-bloc if multiple injuries are within close proximity. The bowel is stapled and left in discontinuity. No attempt should be made to do a primary enteric anastomosis in the damage control setting. If resection is required, linear staplers are quick and easy to use. Now is not the time for a primary

anastomosis. Bowel continuity can be restored later when definitive surgery is planned. Stoma formation and feeding tube access should be avoided and delayed until the re-look laparotomy.

Abdominal wounds associated with colonic injuries need to be monitored closely, and serial local debridements considered as there is considerable risk of infection.

Rectal injuries may require a loop or end colostomy. This is not the time to perform extensive mobilisation and dissection of the rectum.

Pancreas and Duodenum

Pancreatic injuries should be treated with haemorrhage control, modest debridement of devitalised tissue and placement of a wide bore suction drain.

Duodenal injuries can be primarily repaired only if there is no risk of luminal compromise. It should be debrided and closed transversely if the injury involves less than 50% of the circumference of the duodenal wall. If there is complete transaction, it would be best to close both ends and perform definitive repair at a later date.

Kidney

Renal injuries often respond to compression tamponade providing Gerota's fascia

has not been damaged. Non-expanding haematomas confined to Gerota's fascia are often not explored and subsequent management is determined when the patient is stable. If the patient is unstable, with evidence of an expanding pulsatile haematoma and suspected pedicle injury, then renal exploration is required. Rather than attempt a repair which is time consuming, a nephrectomy is recommended.

Lung

If there is bleeding from the lung tissue, this is not the time for complicated lung resections. The simplest pulmonary resection should be performed by use of a linear stapling device, conserving as much pulmonary tissue as possible. Lobar resections and partial resections at this point should be avoided as they are too time consuming.

Abdominal Closure

Once all vascular and bowel injuries are controlled, the abdomen is packed. Large abdominal packs are placed over solid organs that have been injured plus over areas of dissection. Packing should be tight enough to tamponade any bleeding, but not so tight that it impedes venous return.This is the last step of the DCL, prior to transport of the patient to ITU. It needs to be rapid.

A temporary abdominal closure system can be used (TAC). The optimal TAC method would prevent evisceration of abdominal contents, allow evacuation of fluid, allow access to the abdominal cavity and allow for expansion to prevent abdominal compartment syndrome developing. It should also be quick to apply and easy to change.

Towel clips or a running suture for closure of the skin or fascia were used originally in DCL as they provided a tamponade effect when intra-abdominal packing had been used. This, however, gives an increased risk of abdominal compartment syndrome. The next generation of TAC was the Bogota bag, which acted as a non-permeable barrier. An IV bag, bowel bag or silastic drape was sutured to the skin or fascia. These were readily available, quick to apply, cheap, and allowed visualisation of the bowel and abdominal contents but they caused significant skin damage, loss of domain and didn't allow effective fluid removal. This method has also now been largely abandoned.In select cases the patient should be transferred to interventional radiology en route to ITU. This is particularly true for those patients with complex hepatic, retroperitoneal, pelvic or deep muscle injuries. If there is evidence of ongoing bleeding at this stage, embolisation

Vacuum assisted closure (VAC) devices are commonly employed today.

is indicated if possible. If this occurs, the interventional radiology suite should serve as an ITU with phase 2 of damage control commencing here.

Although the aim is to perform DCS as quickly as possible, it is important to add that all sources of surgical bleeding must be controlled. Uncontrolled surgical bleeding does not respond to packing alone and if the DCL is terminated prior to the arrest of bleeding the patient will most likely die. Speed is important, but DCS is about a series of controlled and disciplined maneuvers to control haemorrhage and contamination in minimal time.

Catching up in ITU

Whilst in theatre or the angio suite with a polytrauma patient we should constantly ask the question can we stop at this point

and transfer to ITU to restore some physiological normality. The longer that we maintain a relatively shocked state with permissive hypotension, the more complications will develop and the greater 'catch-up' will be required in critical care.

Surgical intervention must be the bare minimum to provide damage control and effectively "get the ship home". As discussed in this chapter, this largely means haemorrhage control, stopping abdominal contamination and preventing further secondary injury.

Anything more than this will result in further patient compromise, longer periods of shock, increased bleeding and greater risk of multi-organ failure. Once surgical haemostasis has been achieved in major vessels and organs, we need to rapidly restore nor-

mal physiology as persistent, hypothermia, acidosis and shock will worsen the effects of acute traumatic coagulopathy.

'Homeostasis for haemostasis'

To re-establish homeostasis, we must ensure that adequate tissue perfusion is restored using the best markers that we currently have available including: base deficit, serum lactate, urine output, ECHO, Haemoglobin/haematocrit etc. Once in critical care we can do this more aggressively under closer scrutiny and we should aim to address the permissive hypotension, shock and deficits as a matter of urgency.
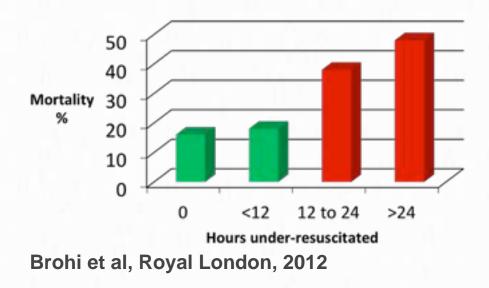
Through all of these measures we are striving to achieve another fundamental of damage control in the multiply injured casualty:

'Normal physiology, NOT normal anatomy'

To not restore normal physiology quickly will inevitably result in an increased mortality. This was demonstrated by Brohi and colleagues at the Royal London, who found that less than 12 hours of under-resuscitation could be tolerated with only an approximate 15% mortality.

However, if this continued into the 12-24hr window, then mortality rose rapidly to over

30% and beyond 24hrs this rose to nearly 50%.

Brohi et al, Royal London, 2012

If the surgery is more prolonged, extending beyond 60 minutes, then it is important to look for signs of physiological exhaustion developing. An early sign of this maybe the general ooze from tissues and contact bleeding which cannot be addressed with surgical control. This is the time for the surgery to stop, if necessary pack the wounds effectively and spend some time 'catching up' and restoring a degree of normal physiology. This may take one or many hours and should gauge when surgery should be resumed.

Another sign of such exhaustion can be a heart with signs of failure from reduced contractility with reduced cardiac output, despite apparently adequate filling. This may result from low serum calcium levels, severe acidosis, hypothermia, infarction or limited pre-existing reserves. Each case

must be managed based on the information that you have but the picture can often be confusing and difficult to interpret.

Typically, beyond the first 60 minutes of Damage Control Resuscitation, with permissive hypotension, we need to start restoring perfusion and circulating volume or else the deficits and necessary catch-up in critical care will become increasingly difficult and likely to extend beyond 12 hours.

Hybrid Resuscitation – a potential solution?

Whilst shorter operative time is the ideal solution to minimize the period of shock, it has been recognised that under certain circumstances the surgery may be unavoidably prolonged. It is beyond the remit of this text to judge on that view, but we can consider one alternative, which is being explored by a number of UK military trauma specialists. This is the concept of Hybrid resuscitation, which is based on the following:

- Target systolic BP of 80mmHg for 1 hour

- Target systolic BP of 110mmHg until haemostasis achieved

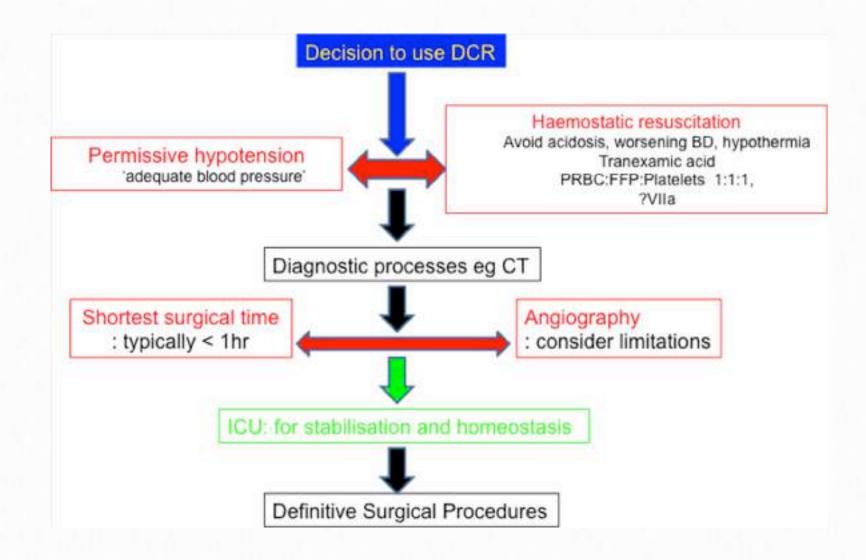- Then restore normal BP with blood products

This approach applies an aggressive approach with permissive hypotension for the first hour after injury to optimise clot formation and reduce bleeding, but beyond that point primary bleeding is not considered the greatest issue and initial haemorrhage control should have been established so the blood pressure is allowed to rise to a degree to restore some tissue perfusion.

Finally, once surgical haemostasis has been achieved the blood pressure is rapidly restored to normal levels, using blood products for any necessary filling.

When do you go back after DCR?

One of the greatest dilemmas in DCR is when is the best time to take the patient back to theatre to review the injuries, remove packs or even consider a definitive repair.

The ideal time has not yet been fully determined and there are multiple factors to consider. Inevitably, any patient with major ongoing bleeding needs to return to theatre or angiography as a matter or urgency, whereas patients who are still cold or acidotic and oozy, will not tolerate well a further physiological "hit" and it would be wise to wait until a greater degree of homeostasis has been restored.

In terms of the systemic inflammatory response to trauma and surgery, the worst time physiologically to go back is within the first 4-48hrs. Platelet function can take anything up to 4 days to recover, however, by such a late stage the risks of infection start to increase rapidly.

As a result we look for a best compromise based on these considerations and the general clinical condition of the patient. Typically this will be in the window of day 2-4. Discussions between surgeons and intensivists but take place to decide on the timing, nature and extent of the proposed surgery and limitations or triggers to abandon the procedure would be discussed wherever possible.

Summary

The principles of Damage control resuscitation cannot be applied to all trauma as it is a significant compromise to patients which can only be justified where a life threatening situation must me mediated rapidly to simply preserve life. Anything more than this would result in even great compromise and potentially death.

Once the decision is made to adopt the DCR approach, the process must be rapid and follow a clear pathway, which best identifies and manages immediately life threatening problems and then rapidly moves the patient to critical care, where normal physiology can be restored. The diagram on the previous page is a simple summary of a number of the key steps and considerations.

References

This is largely a summary chapter that attempts to pull together many of the concepts raised in previous individual sections. If you require the references then revisit the relevant section in the manual. There are a few others which are listed below.
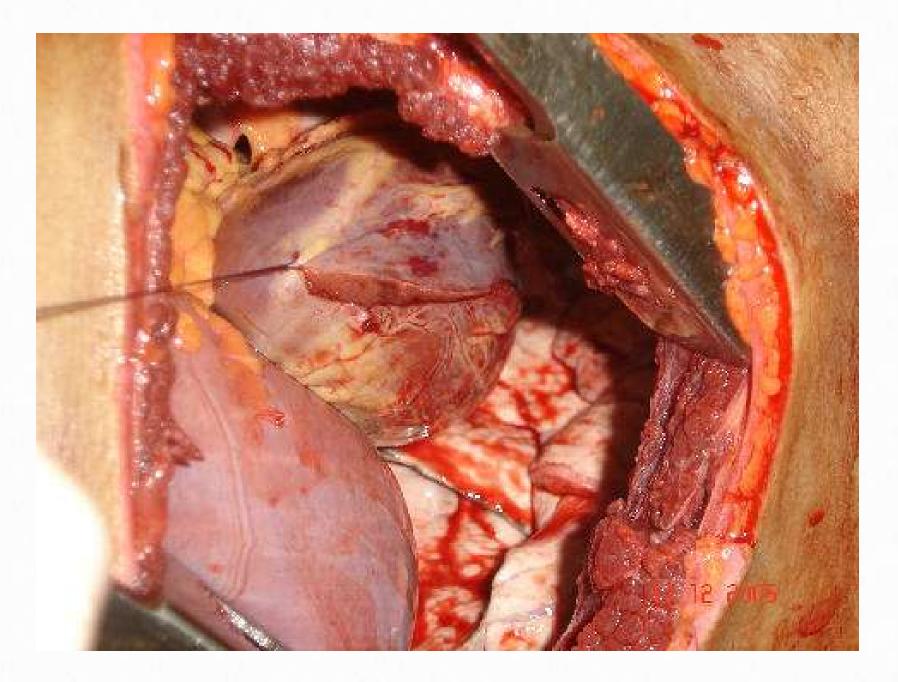
1. 'The Golden Hour'– Letter to the Governor of Maryland, USA, - Adam Cowley, 1963

2. Intensive Care Society – Transport of the Critically Ill guidelines, 2002

3. BATT Course – safe transfer principles, ATACC Faculty, 2005

4. ATACC Manual 8th edition 2014

5. Sikorski RA, Koerner AK, Fouche-Weber LY, Galvagno SM. Choice of General Anesthetics for Trauma Patients. Curr Anesthesiol Rep. 2014 Jun 10;4(3):225–32.
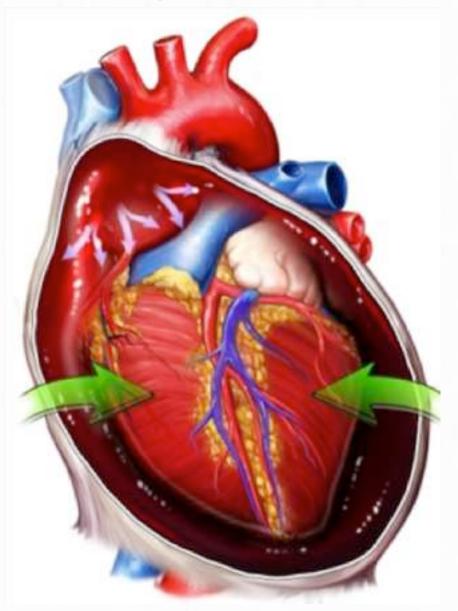
6
Specialist Care

Thoracic Trauma

Introduction

Patients presenting with major and multiple injuries requiring emergency surgery are a great challenge. It has been estimated in the United States that thoracic trauma accounts for 10% of all trauma deaths.

Patients with thoracic injuries have a mortality rate of 15.7% while those without thoracic injuries had a mortality of 12.8%. When patients present to the operating room within 24 hours of admission, the incidence of blunt thoracic trauma has been reported as high as 62.5%. About 10% of blunt and 30% of penetrating thoracic injuries need surgical intervention.

Cardiac Tamponade

Aetiology

This usually results from penetrating trauma. A very small amount of blood in the pericardial sac will impair myocardial function and cause circulatory embarrassment. Tamponade may occur from isolated chamber rupture in blunt trauma secondary to compression of the heart itself.

Diagnosis

Diagnosis is notoriously challenging. Cardiac tamponade should be suspected when there is hypotension unexplained by other findings i.e. tension pneumothorax, haemothorax, abdominal or other haemorrhage. The cervical collar may mask neck vein distension. Beck's Triad consists of increased JVP (not if hypovolaemic), decreased blood pressure (may be due to hypovolaemia) and muffled heart sounds. Additional findings may include pulsus paradoxus and PEA in the absence of hypovolaemia or tension pneumothorax. Tamponade may be picked up on E-FAST scan (Extended Focussed Assessment with Sonography in Trauma). Ultrasound is 98.1% sensitive and 99.9% specific for pericardial effusion and RV collapse.

ECG may demonstrate reduced amplitude complexes. Transoesophageal echocardiography is probably the best diagnostic tool if available, but a transthoracic is sensitive enough in the hands of a regular user

Ultrasound Image of large Cardiac effusion or tamponade

To complicate matters further a tension pneumothorax may present the same signs as a cardiac tamponade If in doubt, insert the chest drain or perform a thoracostomy. If there is no improvement in clinical condition then assume that a tamponade is present.

Management

Ideal management of cardiac tamponade is surgical exploration and decompression. Needle pericardiocentesis in trauma only works if performed early whilst the blood has not clotted. At this stage even 5-10mls aspiration may result in a dramatic improvement. However, there is the risk of lacerating a coronary vessel, lung or inducing cardiac arrhythmias.

Initial administration of a bolus of IV fluids (250mls aliquots) may improve cardiac output, by optimising filling pressures while waiting for a needle pericardiocentesis or emergency thoracotomy.

This must be followed up in a trauma situation with a theatre thoracotomy, and transfer to an appropriate facility or bring staff to the patient may be needed. A needle pericardiocentesis may be negative in the presence of cardiac tamponade if the blood has clotted. Sub-xiphoid pericardotomy or pericardial 'window 'has been

suggested as an emergency room alternative technique but may actually delay definitive management as it may not be effective and anybody with a confirmed Tamponade will require thoracotomy and exploration anyway.
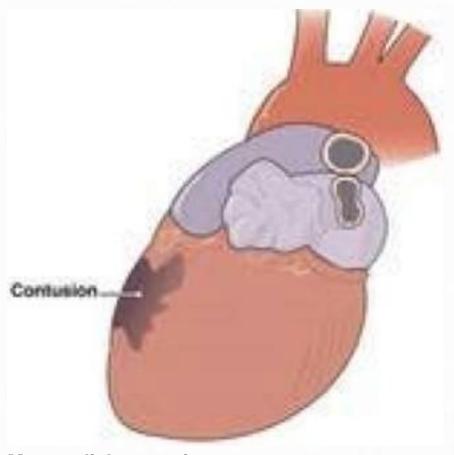
Tamponade from isolated penetrating trauma has potentially good outcome but tamponade following blunt trauma has a uniformly poor outcome. Tamponade due to single chamber rupture has 40% mortality. Mortality from two-chamber rupture is 100%. The right-sided cardiac chambers, especially the right ventricle, are most vulnerable to blunt force trauma due to their anterior position and relatively thin walls.

Myocardial Contusion

Blunt Cardiac Injury (BCI) is the most commonly missed diagnosis leading to fatality following thoracic trauma. BCI can take many forms ranging from subtle alterations in electrical conduction and cardiac function to full thickness myocardial necrosis and wall rupture. True structural injury to the heart is rare.

The present evidence is that blunt cardiac injury is common but rarely fatal. Incidence has been quoted at between 8.2 to 75% and many instances of blunt cardiac injury go unnoticed. The true incidence is un-

clear as there is no single clinical investigation available at present that provides accurate diagnosis.. Mycocardial contusion is usually a result of rapid deceleration injury with or without a direct blow to the chest. Life-threatening BCI probably occurs in 5% to 15% of patients with severe blunt chest injuries.

Myocardial contusion

Diagnosis

The best test for diagnosis remains controversial. A high index of suspicion from the mechanism of injury is useful but signs may be variable and transient and confused by concomitant injuries. Fractures to the sternum are a significant risk factor for BCI.

The ECG is unreliable unless ST elevation is present. Various other changes may be present in BCI such as: sinus tachycardia, RBBB (RV injury), AV block and atrial extrasystoles. However a normal admission ECG unfortunately does not exclude the diagnosis of myocardial contusion.

Echocardiography is extremely useful in the diagnosis of this injury, and is useful for detecting wall motion abnormalities, thrombi, valvular damage and pericardial effusions. Trans-oesophageal ECHO (TOE) is better than trans-thoracic ECHO if available.

CK-MB isoenzymes are of no diagnostic value as they will be non-specifically raised in a trauma patient. Cardiac Troponin-I is more specific for myocardial damage and it has been suggested that two levels, 6 hrs apart are better markers of acute injury. Sensitivity and specificity for Troponin-I estimations are variable in different studies and predictive value is poor.

Thallium scanning can detect areas of decreased perfusion, but cannot differentiate an acute from pre-existing lesion. This kind of investigation is not a practical proposition in the acute management of Trauma.

Management

Patients with suspected myocardial contusion are no longer routinely subjected to prolonged observation in a monitored setting. If the ECG is normal with no hypotension and no dysrhythmias the patient may go home after 12 hours if no other injuries are present. In the presence of an abnormal ECG, cardiac Troponin I should be measured and echocardiography performed if available.

Monitoring in an HDU environment is advised for at least 24hrs as arrhythmias may develop late in this condition. Management of arrhythmias and cardiogenic shock follow normal treatment guidelines. The leading cause of intraoperative death in adult patients with myocardial contusion is hypovolaemia rather than the cardiac injury itself. Pre-existing cardiac pathology carries a worse prognosis.

Myocardial Rupture

This can be an acute traumatic rupture with perforation of the ventricles, atria, intraventricular septum, papillary muscles or valves. It can also be a delayed rupture some weeks after blunt cardiac trauma with contusion or infarct.

Myocardial rupture is usually a result of high speed RTCs and they account for 15% of all thoracic injuries. The incidence in blunt chest trauma cases is 0.5-2% and typically involves the ventricles.

Interestingly some postmortem results have suggested that up to 20% of these patients survive for at least 30 minutes which further suggests that rapid diagnosis may be life saving

If the pericardium is intact then a tamponade will develop and keep the casualty alive for a short period but if it is ruptured then death from exsanguination will rapidly follow.

Diagnosis

Diagnosis is usually based on the mechanism of injury and then the findings e.g.:
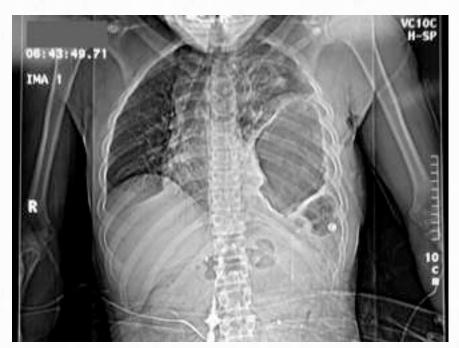
- Bruit de Moulin – harsh murmur, splashing mill wheel

- Features of tamponade

- Unexplained hypotension and poor response to resuscitation

- Persistent metabolic acidosis

- E-FAST

- CXR findings

Management

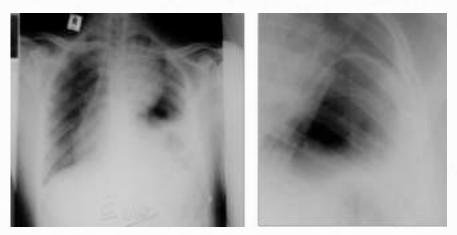- Immediate decompression of cardiac tamponade

- Emergency department thoracotomy

Diaphragmatic Injuries

Diaphragmatic injuries may result from blunt or penetrating trauma. Penetrating injuries tend to create small diaphragmatic perforations with little immediate significance but blunt trauma results in large radial tears with easy herniation of abdominal viscera. The incidence of diaphragmatic rupture in patients with blunt thoracic trauma presenting to the operating room is 9.1%.

Diaphragmatic rupture with left Gastrothorax

Diaphragmatic rupture is likely to occur as a result of a sudden and massive increase in intra-abdominal pressure. If the diaphragmatic tear is large enough, the relatively negative pleural pressure during spontaneous ventilation encourages herniation of abdominal organs into the chest compressing the adjacent lung leading to respiratory embarrassment. There is proba-

bly equal incidence of left and right diaphragmatic rupture but left sided injuries present to hospital much more frequently than right, as the right side is somewhat protected by the liver. Right sided injuries are more likely to prove fatal on scene because of the potential for massive liver disruption. Lateral impacts in motor vehicle collisions are three times more likely to cause diaphragmatic rupture than head on impacts. Diaphragmatic damage should be suspected with penetrating trauma at or below the 4th rib anteriorly, 6th rib laterally or the 8th rib posteriorly.

High Speed RTC Passenger with left diaphragmatic rupture - Note NG tube in stomach in left chest

Diagnosis

Physical examination is not very helpful in making the diagnosis of diaphragmatic injury in the patient with multiple injuries. Dyspnoea, shoulder tip pain, bowel sounds in the chest and decreased breath sounds may be present. Chest radiography may demonstrate elevation of the left

hemidiaphragm with or without an irregular outline, basal atelectasis, an arch-like curvilinear shadow above the diaphragm or evidence of a hollow viscus in the thorax. Shift of the heart and mediastinal structures to the opposite side may suggest a diaphragmatic injury. Diagnosis can be confirmed by passing a nasogastric tube as the tip of the tube may be seen on x-ray in a gastrothorax. Aspiration is a risk in the presence of intra-thoracic herniation of abdominal contents.
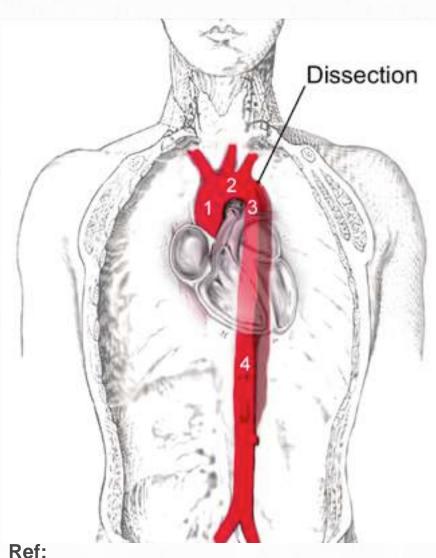
Management

Tracheal intubation and positive pressure ventilation will alleviate respiratory distress and protect the airway. Care should be taken when inserting chest drains in patients with suspected diaphragmatic injury. Surgical repair is needed and the approach will depend on associated injuries.

Thoracic Aortic Dissection (TAD)

The Aorta is the most commonly damaged vessel in blunt trauma and is particularly associated with high speed RTCs.

The incidence of traumatic rupture of the aorta in patients with blunt thoracic trauma presenting to the operating room is 4.8%. Up to 90% of patients with aortic rupture exsanguinate at the accident scene or arrive in the emergency department with ab-

sent vital signs. The remaining 10% survive to reach hospital because the aortic rupture is contained by an intact adventitia. Up to 85% of these can survive if picked up early.

Deceleration injuries due to a vertical fall of >3m and RTC at a speed >30mph especially in an unrestrained or ejected occupant or a pedestrian are risk factors for TAD. Rupture of the aorta may not be imminent in patients with aortic injury surviving long enough to reach hospital and in these there is a reasonable chance of successful surgical repair given expert resuscitative management and transfer.

Ref:
http://bionews-tx.com/wp-content/uploads/2013/09/aortic-dissection-diagram.png

Site of Rupture

The commonest site of rupture (80-90%) is in the descending thoracic aorta just distal to the left subclavian vein. But any area can be affected, although the descending aorta is more at risk than the ascending. Ascending aortic injuries have a high association (70-80%) with lethal cardiac injuries.

The aorta may be ruptured by sudden increase in hydrostatic pressure (the Water Hammer effect) but the major cause is violent deceleration. The descending thoracic aorta is relatively fixed whereas the heart and aortic arch are mobile and able to swing violently during rapid deceleration. Damage tends to occur at the transition point between the fixed and mobile portions. This is the isthmus and is just distal to the origin of the left subclavian artery. Damage to the aorta may also be caused by direct laceration from a displaced thoracic vertebral fracture.

Diagnosis

This requires a high index of suspicion as patients who may have this injury present urgently to the operating room for surgical management of other life threatening injuries before appropriate investigations can be completed. They may complain of intrascapular or retrosternal pain but there may be a great deal of other distracting injuries. The injury is therefore frequently first suspected on the plain chest radiograph. Radiographic findings suggestive of thoracic aortic injury include:

- Widening of the superior mediastinum (>8cm on AP film)

- Abnormal aortic contour (loss of the "aortic knob")

- Deviation of the nasogastric tube to the right

- Presence of a left apical pleural cap.

The best diagnostic tools are Spiral CT chest (100% sensitive and specific), Transoesophageal ECHO, but the gold standard, at present, remains Aortography with contrast.

Widened mediastinum >8cm on AP film

Management

Early repair of the aortic injury may exacerbate co-existing injuries such as pulmonary or myocardial contusions or significant head injuries. Delaying surgery required for other life or limb threatening injuries in patients with traumatic disruption of the thoracic aorta will lead to significant morbidity and mortality from the associated injuries. Definitive diagnosis is by aortography which frequently must wait until life and limb threatening injuries have been dealt with. Once the patient has achieved haemodynamic and respiratory stability, investigation and management of the aortic injury can take place. In some centres endovascular stenting of these injuries is being investigated. If aortic injury is suspected or present during an urgent surgical procedure the blood pressure should be continuously monitored. Hypertensive episodes should be avoided and mean arterial pressure should be maintained between 70 and 80 mmHg with the use of beta blockers and vasodilators. Meticulous control of blood pressure is especially important during intra-hospital transfer to definitive cardiothoracic care.

Laryngeal Injuries

Blunt laryngeal injuries are rare. A direct blow to the neck often causing hyperextension can cause fracture of the hyoid bone or thyroid cartilage or laryngeal disruption at the junction of the cricoid cartilage and trachea. Direct laryngeal trauma is also commonly caused by suspension by the neck. The classical signs of laryngeal injury include; hoarseness, subcutaneous emphysema in the neck and haemoptysis.

Signs of airway compromise must be sought on initial presentation. Even in the absence of airway compromise, examination of the upper airway by endoscopy and computed tomography is necessary to determine the degree of injury and predict whether airway obstruction could occur. Patients arriving with laryngeal injuries and an apparently normal airway may develop upper airway obstruction within hours of admission. Early tracheal intubation may be required depending on the findings at laryngoscopy. Repeated attempts to intubate the trachea via the oral route may worsen the injury and render further attempts to secure the airway via any means extremely difficult. Early involvement of a senior ENT surgeon is essential as tracheostomy is indicated in all patients requiring an artificial airway.
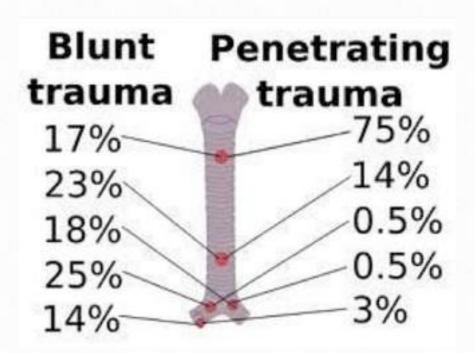

Ref: http://www.onlinejets.org

Tracheobronchial Injuries

Tracheobronchial injuries are rare but serious injuries from blunt or penetrating trauma. In blunt trauma there is a reported incidence of 0.8% and a mortality of 30% in patients with blunt thoracic trauma.

Many patients with this injury, typically have a knife wound to the neck or have sustained a rapid deceleration injury with effects on the intra-thoracic contents such as the traction on the trachea pulling it off the lungs. Many such injuries will result in death at the accident scene. Haemoptysis and subcutaneous emphysema should alert the anaesthetist to the possibility of this injury.

Risk areas for blunt and penetrating tracheal trauma

Radiographic findings of an intra-thoracic tracheobronchial injury are pneumothorax and mediastinal emphysema. A tracheobronchial injury should be seriously considered when a patient has a pneumothorax with a massive air leak that is not evacuated by insertion of a chest tube. This injury is confirmed by fibreoptic bronchoscopy. Many patients will require control of the airway with tracheal intubation because of the tracheobronchial or other associated injuries. Intubation is ideally performed with the aid of fibreoptic bronchoscopy to aid in tube placement and to properly examine and evaluate the injury. Some patients with injuries in the main bronchi may require lung isolation with a double lumen tube because of large air leaks, bronchopleural fistuale or haemoptysis. This allows ventilation of the intact lung while allowing bronchoscopy to examine the ruptured side.

Oesophageal Rupture

Rupture of the oesophagus in blunt trauma patients is relatively rare but is more frequently seen in penetrating trauma. Rapid compression of the abdomen in blunt trauma leads to a rise in intra-oesophageal pressure and bursting tear to the oesophagus. Trauma to the oesophagus is potentially lethal, in fact the mortality is nearly 100% if the diagnosis is delayed past 24

hours, as contamination of the mediastinal space with gastric content leads to florid mediastinitis and subsequent necrosis. These patients require urgent surgical assessment and repair if the otherwise high mortality is to be avoided. Surgical repair can reduce this mortality to 30%

In addition to the blunt and penetrating trauma, this can occur as a result of swallowing a foreign body, spontaneously or after caustic /chemical substances. In the latter cases agents such as household bleach can result in perforations four to fourteen days after ingestion.

Diagnosis

The patient complains of excruciating pain in the epigastic and retro-sternal region which may radiate to the chest and back. Dyspnoea, cyanosis and shock soon develop. Surgical emphysema may be present and the CXR may show pneumothorax or hydropneumothorax especially on the left side. Contrast studies of the oesophagus demonstrating a leak or oesophagoscopy demonstrating a laceration are diagnostic.

Management

Surgical repair and mediastinal lavage are usually indicated. Mortality is high.

Traumatic Asphyxia

Relatively rare but involves a severe sudden crush of the thoracic cavity by a heavy object. Typical in industrial or large animal accidents. It causes a rise in pressure in the chest and superior vena cava with the potential for retrograde blood flow into the great veins of the head and neck.

Clinically, it may kill very quickly on scene unless released/rescued. The skill of the head and neck is a blue or violet congested colour with petechial haemorrhages which can appear on the face and upper body and also in the eyes (subconjunctiva).

If survived there is a high risk of thoracic injury and CT imaging should be obtained and any injuries identified should be managed appropriately..

Typical traumatic asphyxia case: crushed lorry cab

References

1. eFAST: extended Focused Assessment with Sonography in Trauma – Liz Turner
   https://www.youtube.com/watch?v=EVQTI7ivhFM

2. Myocardial contusion. AM Ranasinghe, ME Lewis and TR Graham. Trauma 2004, vol 6, no 2,: pp 169-175(7)

3. Myocardial contusion in blunt chest trauma: a ten-year review. Macdonald RC, ONeill D, Hanning CD, et al. Intensive Care Med 1981; 7: 265.

4. Decelerational Thoracic Injury K. G. Swan, Jr., MD, Betsy C. Swan, BS, RN, and Ken- neth G. Swan, MD The Journal of TRAUMA Injury, Infection, and Critical Care. 2001; 51: 970 974

5. Diagnosis, management, and complications for nonpenetrating cardiac trauma: a perspec- tive for practicing clinicians. Voyce SJ, Becker RB. J Intensive Care Med 1993; 8:27588.

6. Improved detection of cardiac contusion with cardiac troponin I. Adams JE, Davila- Roman VG, Bessey PQ, et al. Am Heart J 1996; 131:30812.

7. Circulating cardiac troponin T in myocardial contusion. Ferjani M, Droc G, Dreux S, et al. Chest 1997; 111: 42733.

8. Rupture of right hemidiaphragm due to blunt trauma. Epstein LI, Lempke RE. J Trauma 1968;8:19

9. Blunt rupture of the diaphragm: mechanism, diagnosis, and treatment. Kearney PA, Rouhana SW, Burney RE. Ann Emerg Med 1988;18:828

10. Diagnosing Traumatic Rupture of the Thoracic Aorta in the Emergency Department. CE OConor EMJ Volume 21, Number 4, July 2004:

11. Management dilemmas in laryngeal trauma S Y Hwang; S C L Yeak The Journal of Laryngology and Otology; May 2004; 118, 5;

12. Isolated laryngotracheal separation following blunt trauma to the neck C J O±ah; D Endres The Journal of Laryngology and Otology; Nov 1997; 111, 11;

Neurosurgical Intensive Care

Further management of patients with severe head injury at this stage is dictated by the requirement for Neurosurgical intervention. Patients with focal lesions requiring surgical decompression will require transfer to a Neurosurgical unit. Local arrangements for patients with severe traumatic brain injury who do not require neurosurgical intervention vary. Many are managed on general ICUs. However, it is recommended that they are managed on specialist neurosciences critical care units.

Inter-hospital Transfer

The recommendations of the Intensive Care Society on transfer of critically ill patients with brain injuries must be followed. Special points pertaining to the transfer of patients requiring surgical decompression of clot are:

• Should there be any doubt regarding the safety of the patient's airway, or should their GCS be falling, they should be intu-

bated before transfer and their ventilation stabilised to the above guidelines.

- Sedation and muscle relaxants should ideally be given by continuous infusion.

- The patient should be prepared for transfer as rapidly as possible, as early decompression of clot saves lives and improves outcome.

- Despite the point above, during the actual transfer, the emphasis should be on smoothness rather than outright speed, as excessive acceleration and braking forces can cause surges in intracranial pressure (ICP) in supine patients.

- It is vital that hard copies of scans, all notes and any available cross-matched blood accompany the patient on transfer.

Safe fixed-wing critical care transfer

ICU Management

Patients with severe head injuries should be managed within an intensive care unit. Regional Neuroscience units accept the majority of patients with mass lesions that are surgically amenable, whereas the bulk of patients with diffuse injuries remain within District General Hospital ITU's. In either case, intensive therapy is directed towards maintenance of cerebral perfusion pressure and cerebral oxygenation. Consensus guidelines have been developed by the European Brain Injury Consortium (EBIC) to attempt standardisation of management.

Monitoring & General Care

Minimal monitoring requirements include ECG, SpO_2, invasive arterial pressure, temperature and end-tidal CO_2 if ventilated.

Ventilation Targets
- P_aO_2 >13kPa

- P_aCO_2 4 - 4.5kPa

Standard observations continue

All standard observations of ITU patients continue with particular focus on Glasgow Coma Score and Pupillary response. Any changes may warrant a further scan or intervention. However, ICP measurement is

a far more reliable methods of assessing the pressures and effects of a traumatic brain injury, as compared to a CT Head scan.

Fluids

Give IV fluids to maintain normovolaemia and blood chemistry within normal limits. There is no indication for fluid restriction as treatment of cerebral oedema. CVP/cardiac output monitoring is recommended to ensure normovolaemia

Intracranial Pressure Monitoring

There are several methods of monitoring the intracranial pressure. All are invasive and involve either a burr hole craniotomy in the skull, or a smaller hole drilled with a twist drill. Catheters may be either extradural, subdural, intra-parenchymal (i.e. within the substance of the brain) or intra-ventricular. The gold standard is an intra-ventricular catheter, which also allows drainage of CSF in cases of raised ICP. The most common technique in current use is to introduce fine catheter through a 2mm twist drill hole through the skull and dura into the brain substance. These catheters have an electronic pressure trans-ducer at the distal end and, after initial zeroing and calibration, will produce accurate results for several days.

Insertion of a Codman ICP transducer
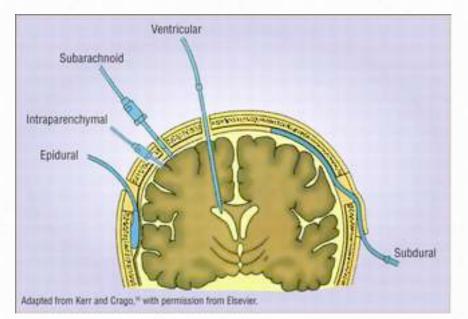
The technique has a low complication rate in experienced hands and is used both by neurosurgeons in specialist centres and by general intensivists. The EBIC guidelines do not include ICP monitoring in the minimum monitoring standards for severe head injury as not all European centres practice it routinely (it is used in around 70% of cases). This is likely to be due to the historical lack of statistically significant evidence that ICP monitoring produces an increase in the numbers of survivors with good outcome or moderate disability (as opposed to death or vegetative state).

However, more recent papers have shown improved outcome in patients managed with a cerebral perfusion pressure (CPP) of >70mmHg, and the American Brain Injury Consensus now strongly recommends ICP monitoring. Whilst we aim for a target CPP, if the ICP is persistently greater than 20mmHg then the outcome will be poor.

Cerebral perfusion pressure is the pressure gradient responsible for cerebral blood

flow. It is the difference between mean arterial pressure and intra cranial pressure. Under normal circumstances it is maintained relatively constant by auto regulation despite changes in mean arterial pressure (MAP range 60-150mmHg). After head trauma the ability of the brain to autoregulate is lost and cerebral perfusion pressure is reliant on MAP. Where ICP is 20mmHg, a MAP of 90mmHg will required to maintain a CPP of > 70mmHg.

$$CPP = MAP - ICP$$

Various ICP Monitoring Options
Ref:http://crashingpatient.com/wp-content/images/part1/icp%20monitors.jpg

Management of CPP and ICP

ICP therapy is only indicated if raised ICP has been demonstrated by monitoring, clinical signs of developing intracranial herniation, or CT evidence of raised ICP. Treatment should be aimed at restoring CPP to

appropriate levels (70mmHg in adults). ICP elevations of greater than 20-25mmHg should be treated unless local policies state otherwise.

Accepted methods of management of ICP and CPP are:

• Good levels of balanced sedation and analgesia

• Nurse 15 degrees head up

• Normocapnoea (P_aCO_2 4.5kPa)

• Volume expansion if indicated by CVP/ CO monitoring in the hypovolaemic patient

• Osmotic therapy, classically with mannitol 20% in repeat boluses of 50-100mls or with 7.5% Hypertonic saline 50-100ml boluses

• Serum osmolarity should be monitored and should not rise above 315.

• CSF drainage if a ventricular catheter is in place.

• Treatment of seizure with anticonvulsants, with a low threshold for initiating treatment.

• Paralysis

• Avoidance of hyperthermia

If the above methods fail:

- More intensive hyperventilation (P_aCO_2 <4.5kPa) preferably with monitoring of cerebral oxygenation with jugular oximetry to detect cerebral ischaemia.

- Barbiturate coma, usually with thiopentone, to reduce cerebral metabolic oxygen demands and lower ICP. Some centres monitor EEG and titrate dosage to achieve burst suppression on trace. Inotropes are usually required to maintain MAP.

- Escape or decompressive craniotomy

- Removal of brain tissue

There are currently no indications for:

- Steroids

- Calcium Antagonists

The use of induced hypothermia is controversial and complex. There is good evidence that moderate hypothermia reduces ICP and is anti-ischaemic. However, early studies showed a worse or unchanged outcome in patients treated with hypothermia and the technique was abandoned. More recent studies have shown more promise. These studies targeted hypothermia at patients with difficult to manage ICP and were conducted in experienced neuroscience centres. Management of the potential complications of hypothermia was meticulous. This technique may thus have a role in appropriate patients.

Jugular Venous Oximetry

Aggressive hyperventilation to a P_aCO_2 of less than 4kPa can cause intense cerebral vasoconstriction. Whilst this may be effective in the control of intracranial pressure, the resultant drop in cerebral blood flow may cause ischaemia to the brain. A catheter introduced into the internal jugular vein by a retrograde technique, and lying in the jugular bulb just outside the skull will be able to measure cerebral oxygen extraction by means of measuring the oxygen saturation of the venous blood. This may be done either directly, with a fibre-optic catheter, or by means of intermittent blood sampling and passing the sample through a co-oximeter.

The EBIC guidelines strongly suggest that such a technique is employed if hyperventilation is utilised to reduce ICP in order to strike a balance between intracranial pressure and cerebral oxygen supply.

Spinal Trauma

Spinal cord injury is a devastating condition, all the more so that the victims tend to be young and otherwise healthy. The incidence varies but is commonly reported to be 10-15 per million population per year, with males four times more likely to suffer the injury than females. 50% of these injuries occur in the C-spine region.

2-5.8% of all blunt polytrauma patients have an associated C spine injury with 7.7% of obtunded trauma patients having a C-spine injury compared to 2.8% of alert patients.

Mechanism of injury

Acceleration and deceleration forces are the main cause of spinal cord injuries, with Road Traffic Collisions (41-50%), falls (35-43%) and sports injuries (7-11%) the most commonly seen mechanisms.

While some authors have have questioned the validity of using the mechanism to predict injury, a history of fall, axial load, all-

terrain vehicles are predictive for the presence of cervical spine injury. Following a Road Traffic Collision the speed, mechanism e.g. head-on collision or rollover and the non-wearing of a seatbelt are all associated with increased likelihood of a cervical spine injury.

Penetrating spinal trauma is uncommon in UK practice. Unstable spinal injury is rarely seen in civilian penetrating mechanism (0.4%) and while it is more common in the military setting it is most often associated with fatal injuries.
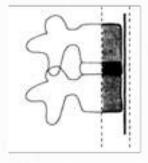
Spinal Anatomy

The spine consists of the spinal cord, arising from the medulla and terminating at the lower border of L1 (in adults). There are 31 segments, each with paired nerve roots – anterior (motor) and posterior (sensory). The cauda equina exists below the L2 level. The spinal cord contains numerous tracts, the most important of which are:
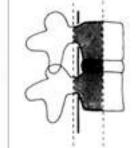
• Corticospinal tract (ipsilateral motor function)

• Spinothalamic tract (contralateral pain and temperature sensation)

• Posterior columns (ipsilateral proprioception, vibration and light touch)
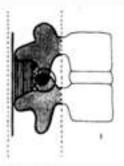
The spinal cord is contained within the 33 vertebrae of the spinal column (7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal). The cervical and lumbar regions have a greater degree of mobility than the thoracic spine, and thus are the most common sites of injury. Neck flexion/extension is greatest at the atlanto-occipital junction, while most rotation of the neck occurs at the atlanto-axial (C1- 2) level.

Vertebrae are held in alignment by the anterior longitudinal ligament, the posterior longitudinal ligament, the ligamentum flavum and the interspinous ligaments as well as the bony support from the facet joints.

Holdsworth originally described two notional "columns" within the spine. and this was later refined to a three column model. The anterior column comprises of the anterior part of the vertebral body and the anterior longitudinal ligament, the middle column (posterior part of the vertebral body and posterior longitudinal ligament) and the posterior column (posterior bony arch and ligamentous complex).

Injury may occur to any of these bony columns, and may include primary injury to the spinal cord. Secondary spinal cord injury may result from haematoma, cord oedema, hypoxia, hypotension or movement of unstable bony structures.

Not all spinal fractures are 'unstable'. Instability is defined as disruption of two or more of Holdsworth's columns. It is impossible to ascertain the extent of damage to these columns prior to imaging, therefore the basic aim of pre-hospital spinal care has traditionally been immobilisation.

Assessment

Initial assessment of the trauma patient should follow <C>ABCD or MARCH, as described in Section 3. During the primary survey particular attention should be paid to the respiratory pattern – diaphragmatic breathing indicates possible airway obstruction or a high cervical injury. When assessing the level of consciousness painful stimuli should be applied in the cranial nerve distribution i.e. above the clavicles.

Potential for spinal injury should be suspected from a combination of the mechanism of injury and any back or neck pain described by the patient, as well as appropriate targeted clinical examination. The presence of a moderate or severe Trau-matic Brain Injury is associated with concurrent spinal injury. The presence of severe facial fractures and other painful distracting injuries should raise the index of suspicion of spinal injury.

The focus of the pre-hospital team must remain with the priorities of massive haemorrhage control, appropriate airway management and maintenance of effective oxygenation and ventilation. Further detailed clinical assessment of neurological injury (e.g. the level of injury based on assessment of dermatomes and myotomes) should be undertaken when appropriate, which may be during, and not necessarily prior to transport.

Many blunt trauma patients will require log roll prior to packaging, and the opportunity should be taken to examine the back at this time to visualise any wounds as well as to palpate the spine to assess for physical deformities, or tenderness in the awake patient. This approach is now being questioned by some units that simply scoop at scene and then pan-scan without log-roll. While there is no definitive evidence that a log roll could displace a pelvic haematoma, a potential risk of this remains. A significant wound to the patient's back may be missed if a log roll is not performed, and a decision to log roll should

be made on a case by case basis. Among others, a blunt vs penetrating mechanism, a need to roll the patient for packaging/extrication and the time to CT scan will be factors in this decision.

Digital rectal examination adds little reliable clinical information, particularly in the initial stages of resuscitation and should not be done in the pre-hospital phase. In male patients, priapism may indicate unopposed parasympathetic stimulation, but may also be as a result of perineal trauma.

With penetrating trauma it is vital to examine the entire body to find all wounds, and with a low likelihood of an unstable spinal injury from this mechanism the patient can be safely moved to accomplish this. In the case of blunt polytrauma, early extensive CT imaging will take place and in unstable blunt trauma patients full cataloguing of injuries (as described in Section 4.2) should take place in the hospital phase of treatment. To minimise scene time, further patient examination can be done en route to hospital if appropriate.

Patients with clinical indications should undergo appropriate imaging. The presence of a cervical spine fracture has been shown to be associated with another spinal fracture, and in this context the rest of the spine should also be imaged.

Clearing the Cervical Spine

The ability to exclude spinal injury on scene can significantly decrease the resources required to successfully manage a multi-patient scene, as well as eliminate the complications associated with spinal immobilisation.

Two validated algorithms - the Canadian Cervical Spine rule (CCR) and the National Emergency X-Radiography Utilization Study (NEXUS) exist to enable clinicians to reliably exclude cervical spinal injury, and many prehospital services utilise protocols for 'clearing' the cervical spine.

The CCR has been shown to have a higher sensitivity and specificity, and to be more "accurate" than the NEXUS guidelines, however this applies only to adult patients, as the CCR study did not include any children. The NEXUS database included few children, and none under the age of two years, however a subsequent analysis suggests that the NEXUS criteria can be safely applied to paediatric patients despite the small numbers (n=30).

In patients with a GCS of 15 with no neurological deficit, who are not under the influence of alcohol or prehospital analgesia,

the absence of pain and tenderness on palpation has been shown to reliably exclude clinically significant fractures of the thoracolumbar spine. This remains unvalidated in patients with a 'distracting injury', and excluding spinal injury in the polytrauma patient usually requires a combination of radiology and clinical assessment.

Cervical Collars

While cervical collars reduce neck movement by approximately 30%, head and neck movement can still occur when a collar is in situ, especially if it is incorrectly fitted, and collars are more effective when combined with head blocks and tape.

Some trauma centres (e.g. The Princess Alexandra Hospital, Brisbane, Australia) routinely use soft collars on this cohort of patients, not to 'immobilise', but to the remind the trauma team a c-spine injury has not yet been ruled out. Queensland Ambulance Service plans to trial this protocol prehospital in 2014.

ATACC currently recommends that cervical collars should be correctly applied and fitted to patients with potential spinal trauma. In patients with Traumatic Brain Injury who have been intubated the collar should be loosened, sufficiently to avoid compression of the neck veins, as part of the neuroprotective anaesthesia regime.
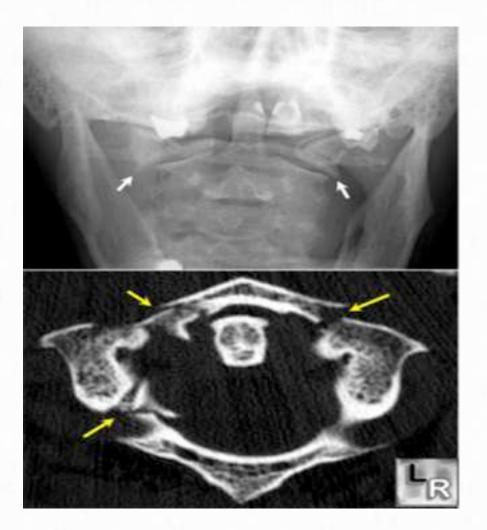
We endorse the recent Faculty of Prehospital Care consensus statement that Clinical decision rules should be used to select those patients who require immobilisation, and eliminate the need for immobilisation in those patients who do not. This has benefits for the individual patient, and preserves prehospital resources as well as reducing downstream workload in the Emergency Department.

Attempts at spinal immobilisation in children and agitated, awake patients may result in increased spinal movement, and these patients may be best managed by foregoing immobilisation, or using MILS without a collar.

Similarly, immobilising a casualty who has been out of a crashed vehicle and walking around normally does not require full spinal immobilisation.
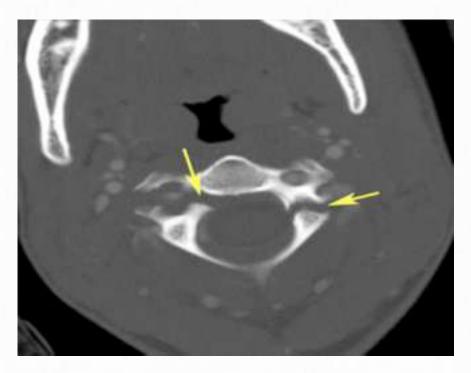
Spinal fractures

Atlas (C1) fracture

This commonly results from an axial load mechanism. A Jefferson fracture consists of fracture of the anterior and posterior rings of C1, with lateral displacement.

Axis (C2) fracture

The odontoid process of C2 fits into a foramen of C1, and this allows for rotation of the head. A hangman's fracture of the posterior part of C2 may result from a hyperextension mechanism.

C3-C7 fractures

The greatest range of flexion/extension of the C-spine occurs at the C5-C6 level, and this is the most common site for cervical spine injury. Typically flexion risks more traction and injury to the spinal cord than extension.
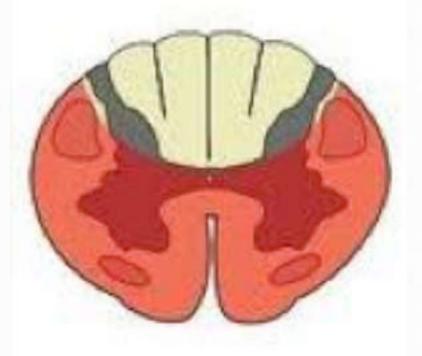
Thoracic fractures

The thoracic spine is relatively stable, but wedge fractures may result from hyperflexion or axial loading, and burst fractures may be seen with high-energy axial loading.

Lumbar fractures
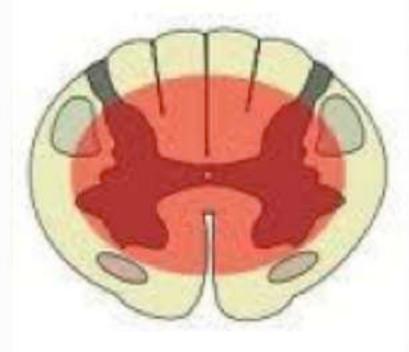
The thoracolumbar junction is a common site of injury, and chance fractures of the upper lumbar vertebrae may result from hyperflexion, sometimes seen with lap-belt injuries. This may be associated with retroperitoneal or bowel injuries.

Cord Syndromes

Anterior Cord Syndrome
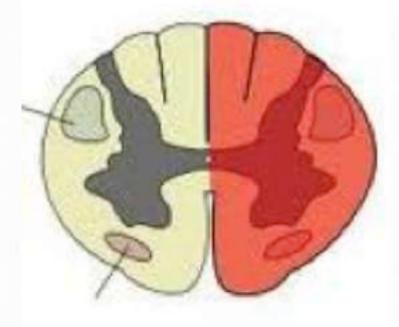
Anterior cord syndrome is characterised by paralysis and loss of temperature and pain sensation due to disruption of spinothalamic and corticospinal tracts. This is often associated with hyperflexion mechanism, and the posterior column may be unaffected, leading to preservation of proprioception, vibration sense and crude touch. Vascular disruption of the anterior spinal artery may also cause anterior cord syndrome.

Central Cord Syndrome

Central cord syndrome causes asymmetrically reduced power, with upper limbs more affected than lower limbs. It is associated with hyperextension injury leading to damage of the centrally located fibres of the corticospinal and spinothalamic tracts.

Brown-Sequard syndrome

Brown-Sequard syndrome results from hemi-section of the cord, usually from a penetrating mechanism. There is motor pa-

ralysis and loss of proprioception and vibratory sense, with contralateral pain and temperature sensory loss.

Paediatric Spinal Trauma

5% of all spinal injuries happen to children, with road traffic collisions and sports injuries the most common mechanisms. Some underlying medical conditions such as Down's Syndrome, Klippel-Feil Syndrome and achondroplasia predispose to spinal injury.

Their size and developing skeleton leave children vulnerable to particular injury patterns, particularly lower cervical injuries as well as ligamentous and spinal cord injuries without radiographic abnormality (SCIWORA). SCIWORA represents about 6% of all paediatric spinal injuries. Clinical examination can be difficult in young and non-verbal children, but is particularly important to out-rule SCIWORA in the paediatric trauma patient.

Assessment and management of paediatric patients is largely similar to adult patients, but strict immobilisation may be counter-productive if the child becomes agitated, and a clinical decision may be made to omit this, including cervical collar. If immobilising children, the anatomical differences (e.g. large occiput) must be

taken into account to maintain neutral position.

There is no evidence to support the use of hypothermia or steroids as having any neuroprotective benefit in paediatric spinal cord injury.

A SCIWORA Injury in an Adult

Management of Spinal Trauma

Airway Considerations with Spinal Trauma

When managing a trauma patient, life-threatening conditions must be identified during the primary survey, and management of these must take priority over a potential spinal cord injury. Airway management is vital, and basic techniques such as trauma jaw thrust/tongue-jaw lift and the placement of oropharyngeal and/or nasopharyngeal airways should be used as necessary.

Manual in-line stabilisation (MILS) of the head should be maintained if personnel and access allow for this. To obtain this it may be necessary to place a pad under the head to obtain 'neutral alignment', particularly if the patient is wearing thick clothing or has increased body mass index, both of which can result in hyper-extension when lying supine. This also aids with optimising conditions for laryngoscopy if intubation is necessary.

Small head movements necessary to return the head and neck to neutral alignment are likely to be clinically insignificant compared to the movement that occurred at the time of injury.

While the correct placement of an endotracheal tube remains the preferred method of airway management, advanced airway options include the placement of a supraglottic airway (SGA). In the hospital phase of management fibreoptic intubation via the SGA post muscle relaxation may be an option depending on the skillset and experience of the treating clinician. Due to the risk of coughing we do not recommend awake fibreoptic intubation. Early elective tracheostomy may also be a worthwhile option.

Video laryngoscopy may have some theoretical advantages over direct laryngoscopy in spinal injury, however the ATACC consensus is that the best device for intubation is the one with which the clinician is most familiar.

Respiratory Considerations with Spinal Trauma

A high level spinal injury may impair the activity of the diaphragm (innervated by spinal nerves from C3-5) and result in various levels of Type II respiratory failure. This may require assisted ventilation in the form of Bag-Valve-Mask (or more ideally with a C-circuit) although in reality this is difficult to perform well in the awake patient or intubation and ventilation. If the equipment is

available, [non-invasive ventilation is also effective](#) in the awake, cooperative patient.

In the ventilated patient, SpO2 >94, with a low-normal EtCO2 (30-40mmHg; 4-4.5 kPa) should be targeted.

Circulation Considerations with Spinal Trauma

Little direct evidence exists for a target blood pressure in spinal cord injury, however hypotension should be avoided in order to minimise secondary neurological injury. ATACC recommends maintenance of Systolic Blood Pressure >100mmHg in patients with isolated spinal cord injury, but a lower systolic pressure (i.e. permissive hypotension, SBP >80mmHg) in the presence of multi system trauma or non-compressible haemorrhage. In isolated SCI a mean arterial pressure [(MAP) of >85mmHg for the first seven days](#) may be the optimal ICU management.

Neurogenic shock

Sympathetic nervous stimulation is provided by the sympathetic chain, which is a plexus of nerves exiting the spinal cord from the T1 to L2 levels. In particular, branches from T1 to T5 levels provide cardiac and aortic innervation. Any spinal cord injury above the level of T6 may lead to unopposed parasympathetic (mediated by the Vagus (CN X) nerve) stimulation and result in neurogenic shock. This is characterised by bradycardia with hypotension, as well as hypothermia. Neurogenic shock should not be confused with spinal shock, which can cause temporary paralysis and loss of reflexes but no haemodynamic compromise.

Clinical neurogenic shock is present in 20% of cervical spinal cord injuries, and the [incidence is lower in thoracic injuries](#). In the injured patient shock must be presumed to be haemorrhagic in origin until proven otherwise.

Hypotension can be treated with vasopressors such as metaraminol or noradrenaline, however further bradycardia may result from the baroreceptor reflex. Peripheral adrenaline may be administered, either as intermittent boluses (20-50mcg at 1 minute intervals) or preferably as an infusion (1mg in 1L crystalloid, or 1mg in 50ml via syringe driver) titrated to effect.

Extrication of Patients with Potential Spinal Injuries

Extrication of potentially spinal injured patients can be challenging. While care must be taken to minimise movement during extrication some movement will inevitably occur, however this is unlikely to be clinically

significant compared to the violent movement that caused the initial injury.

Several devices such as the Kendrick Extrication Device (KED), the Res-Q-Roll and the Speedboard are available, and the individual practitioner must be familiar with the device(s) used in his/her system. There are also some newer narrow, carbon-fibre short boards produced by companies such as Ferno, but none have been definitively shown to be any better than any other and considering this, some are very expensive. Future developments may include allowing the patient to self extricate, and this has been shown to cause the least cervical movement in several proof of concept studies. This method has recently been adopted by the Mexican Fire & Rescue Service and under consideration by others.

Do refer back to previous Chapter on Road Traffic Collision Extrication.

IMMOBILISATION

Suspected or Confirmed Spinal Injury

Traditional teaching has been that suspected or confirmed spinal cord injury should be treated with whole body immobilisation, including cervical collar and head blocks. Recently the benefit, and indeed the safety of this regime has been questioned., The pertinent questions for the pre-hospital practitioner relating to spinal immobilisation are:

• Is it indicated?

• If it is, what is the best method of immobilisation?

Spinal immobilisation has not been shown to reduce the incidence of spinal cord injury, and is associated with several complications, including patient discomfort, pres-

sure ulceration, respiratory compromise, aspiration, raised intracranial pressure, and can compromise airway management.

A Cochrane review in 2001 concluded that spinal immobilisation might increase mortality and morbidity, particularly due to airway compromise and it recommends further randomised controlled trials to obtain a definitive answer to the question. Given that immobilisation is considered standard of care in the developed world it is unlikely that ethical approval to conduct such a trial would ever be granted.

Current methods are described and discussed further in the extrication chapter, including the long-board vs scoop vs vac-mat debate.

It has been suggested that in the alert and co-operative, haemodynamically stable patient allowing the patient to adopt a position of comfort may be optimal, however it remains sensible to fully immobile the spine of unconscious patients.

There are many myths in trauma and another is to always approach the occupants of a crashed car from the front, avoiding head rotation, in case of spinal injury. This is an outdated technique and not necessary as anyone with a neck injury will not be looking around and turning their head

as they will have pain in their neck or on movement, if that badly disrupted.

For details of Immobilisation, see previous Patient Transfer Chapter.

Immobilising Children

Attempts at spinal immobilisation in children and agitated, awake patients may result in increased spinal movement, and these patients may be best managed by foregoing immobilisation.

Penetrating Spinal Trauma

Penetrating trauma involving or potentially involving the spine may present with life threatening airway compromise, potentially fatal thoracic injuries or catastrophic haemorrhage, however it is unlikely to result in spinal column instability. Particularly in the context of penetrating neck trauma the focus should be on managing the immediate life threatening issues. By distracting the prehospital practitioner from effective management of the immediate life threatening issues, spinal immobilisation may have a negative effect on mortality in this group of patients. The ATACC consensus is that in penetrating trauma the absence of any neurological deficit negates the need for immobilisation.

Steroids

The NASCIS trials in several decades ago showed some initial encouraging results from the administration of steroids to patients with spinal cord injuries, however further studies failed to show benefit, and the CRASH trial was stopped early after an increase in mortality was found. Another recent Japanese study has also shown no benefit from high-dose methylprednisolone, but it was associated with an increase in major complications. ATACC considers the use of steroids in management of spinal cord injury to be obsolete.

ICU management/clearing the spine

The patient with spinal trauma may be admitted to the Intensive Care Unit due to a requirement for ventilation, or in the pre/post operative phase of damage control resuscitation. Caring for the spinal injured patient presents several unique challenges.

Prevention of pressure ulcers is vital, and clearing the spine is a priority once immediate life threatening problems have been managed. Until this is accomplished, log rolls should be performed each time the patient is moved, and the hard collar should be replaced as soon as possible. The Miami J® collar is the most effective type, and has the least potential for causing ulceration

Plain films of the cervical spine are inadequate to rule out spinal injury in the obtunded patient, and CT is the preferred modality. MRI is more sensitive and specific for ligamentous injury and SCIWORA, but transferring a critically ill patient to the MRI scanner can be technically complex, and should be deferred in the unstable patient. MRI scanning should be performed when the CT is inconclusive, or when there is unexplained neurology.

Nursing these patients flat, but slightly head up is often best. If sat up, they can often struggle with their abnormal breathing pattern.

Transfer of haemodynamically stable patients with acute traumatic SCI to definitive centre should occur within 24h of initial admission.

References

1. Grundy D, Swain A: ABC Of Spinal Cord Injury 4th edition. London: BMJ Books; 2002.

2. McDonald JW, Sadowsky C: Spinal-cord injury. The Lancet 2002, 359(9304):417-425.

3. Oliver M, Inaba K, Tang A, Branco BC, Barmparas G, Schnüriger B, Lustenberger T, Demetriades D.The changing epidemiology of spinal trauma: a 13-year review from a Level I trauma centre. Injury. 2012 Aug;43(8):1296-300.

4. B. Lenehan S. Boran J. Street T. Higgins D. McCormack Æ A. R. Poynton. Demographics of acute admissions to a National Spinal Injuries Unit Eur Spine J (2009) 18:938–942

5. Aung T, El Masry W. Audit of a British Centre for spinal injury. Spinal Cord 1997;35:147–50.

6. Burstein JL, Henry MC, Alicandro JM, et al. Evidence for and impact of selective reporting of trauma triage mechanism criteria. Acad Emerg Med 1996;3:1011-5.

7. Cooper ME, Yarbrough DR, Zone-Smith L, et al. Application for field triage guidelines by pre-hospital personnel: Is mechanism of injury a valid guideline for patient triage? Am Surg 1995;61:363-7.

8. Thompson WL, Stiell IG, Clement CM, Brison RJ; Canadian C-Spine Rule Study Group.Association of injury mechanism with the risk of cervical spine fractures. CJEM. 2009 Jan;11(1):14-22.

9. Lustenberger T, Talving P, Lam L, Kobayashi L, Inaba K, Plurad D, Branco BC, Demetriades D.Unstable cervical spine fracture after penetrating neck injury: a rare entity in an analysis of 1,069 patients. J Trauma. 2011 Apr;70(4):870-2.

10. Ramasamy A, Midwinter M, Mahoney P, Clasper J.Learning the lessons from conflict: pre-hospital cervical spine stabilisation following ballistic neck trauma. Injury. 2009 Dec;40(12):1342-5

11. Holdsworth. F. W.: Fractures, dislocations and fracture-dislocations of the spine. J. Bone Joint Surg. 52A(R):1534. 1970

12. Denis.F.:The three column spine and its significance in the classification of

acute thoracolurnbar spinal injuries. Spine 8317. 1983

13. PHTLS/ATLS

14. Holly LT, Kelly DF, Counelis GJ, Blinman T, McArthur DL, Cryer HG.Cervical spine trauma associated with moderate and severe head injury: incidence, risk factors, and injury characteristics. J Neurosurg. 2002 Apr;96(3 Suppl):285-91

15. Hasler RM, Exadaktylos AK, Bouamra O, Benneker LM, Clancy M, Sieber R, Zimmermann H, Lecky F.Epidemiology and predictors of cervical spine injury in adult major trauma patients: a multi-center cohort study. J Trauma Acute Care Surg. 2012 Apr;72(4):975-81

16. Hankin AD, Baren JM Should the digital rectal examination be a part of the trauma secondary survey? Ann Emerg Med. 2009 Feb;53(2):208-12

17. J Ul Islam, R Browne, J Thornhill Mountain Bikers Priapism: A Rare phenomenon Irish Med J 2014 (107)1

18. Bryan P. Conrad, Gianluca Del Rossi, Mary Beth Horodyski, Mark L. Prasarn, Yara Alemi, Glenn R. Rechtine. Eliminating log rolling as a spine trauma order.

Surg Neurol Int. 2012; 3(Suppl 3): S188–S197

19. Singh R, Taylor DM, D'Souza D et al. Injuries significantly associated with thoracic spine fractures: a case control study. EMA 2009;21:419-23

20. Viccellio P1, Simon H, Pressman BD, Shah MN, Mower WR, Hoffman JR; NEXUS Group. A prospective multicenter study of cervical spine injury in children. Pediatrics. 2001 Aug;108(2):E20

21. Apple D.F. Anson C.A. Hunter J.D. Bell R.B. Spinal cord injury in youth. Clinical Pediatrics. 1995;34:90–95.

22. Bilston LE, Brown J. Pediatric spinal injury type and severity are age and mechanism dependent. Spine (Phila Pa 1976). 2007 Oct 1;32(21):2339-47

23. Dickman C, Rekate H, Volker S, Zabramski J. Pediatric spinal trauma: vertebrae column and spinal cord injuries in children. Pedriatr Neurosci. 1989;15:237–256

24. Tilt L, Babineau J, Fenster D, Ahmad F, Roskind CG. Blunt cervical spine injury in children. Curr Opin Pediatr. 2012 Jun;24(3):301-6.

25. Cirak, B., Ziegfeld, S., Knight, V.M., Chang, D., Avellino, A.M., Paidas, C.N. (2004). Spinal injuries in children. J Pediatr Surg. 39, 607–612.

26. Parent S, Mac-Thiong JM, Roy-Beaudry M, Sosa JF, Labelle H. Spinal cord injury in the pediatric population: a systematic review of the literature. J Neurotrauma. 2011 Aug;28(8):1515-24.

27. Hirabayashi Y, Fujita A, Seo N, Sugimoto H.Cervical spine movement during laryngoscopy using the Airway Scope compared with the Macintosh laryngoscope. Anaesthesia. 2007 Oct;62(10):1050-5.

28. Malik MA, Subramaniam R, Churasia S, Maharaj CH, Harte BH, Laffey JG.Tracheal intubation in patients with cervical spine immobilization: a comparison of the Airwayscope, LMA CTrach, and the Macintosh laryngoscopes. Br J Anaesth. 2009 May;102(5):654-61.

29. Bach JR.Noninvasive respiratory management of high level spinal cord injury. J Spinal Cord Med. 2012 Mar;35(2):72-80.

30. Markandaya M, Stein DM, Menaker J.Acute Treatment Options for Spinal Cord Injury. Curr Treat Options Neurol. 2012 Feb 3.

31. Guly HR, Bouamra O, Lecky FE; Trauma Audit and Research Network.The incidence of neurogenic shock in patients with isolated spinal cord injury in the emergency department. Resuscitation. 2008 Jan;76(1):57-62. Epub 2007 Aug 3.

32. Engsberg JR, Standeven JW, Shurtleff TL, Eggars JL, Shafer JS, Naunheim RS. Cervical spine motion during extrication. J Emerg Med. 2013 Jan;44(1):122-7.

33. Dixon M, O'Halloran J, Cummins NM. Biomechanical analysis of spinal immobilisation during prehospital extrication: a proof of concept study. Emerg Med J. 2013 Jun 28

34. Deasy C, Cameron P. Routine application of cervical collars--what is the evidence? Injury. 2011 Sep;42(9):841-2

35. Hauswald M.A re-conceptualisation of acute spinal care. Emerg Med J. 2013 Sep;30(9):720-3.

36. Hauswald M, Ong G, Tandberg D, et al. Out-of-hospital spinal immobiliza-

tion: its effect on neurologic injury. Acad Emerg Med. 1998; 5(3): 214-219.

37. Chan D, Goldberg R, Tascone A, Harmon S, Chan L. The effect of spinal immobilization on healthy volunteers. Ann Emerg Med 1994;23(1):48–51.

38. Hewitt S. Skin necrosis caused by a semi-rigid cervical collar in a ventilated patient with multiple injuries. Injury 1994;25(5):323–4.

39. Totten VY, Sugarman DB. Respiratory effects of spinal immobilization. Prehosp Emerg Care 1999;3(4):347–52.

40. Lockey DJ, Coats T, Parr MJ. Aspiration in severe trauma: a prospective study. Anaesthesia 1999;54(11):1097–8.

41. Ho AM, Fung KY, Joynt GM, Karmakar MK, Peng Z: Rigid cervical collar and intracranial pressure of patients with severe head injury. Journal of Trauma, Injury Infection and Critical Care 2002, 53(6):1185-8.

42. Davies G, Deakin C, Wilson A. The effect of a rigid collar on intracranial pressure. Injury 1996;27(9):647–9.

43. Goutcher CM, Lochhead V. Reduction in mouth opening with semi-rigid cervical collars. Br J Anaesth 2005;95(3):344–8.

44. Kwan I, Bunn F, Roberts I. Cochrane Database Syst Rev. 2001;(2):CD002803. Spinal immobilisation for trauma patients.

45. Jonathan Benger, Julian Blackham. Why Do We Put Cervical Collars On Conscious Trauma Patients? Scand J Trauma Resusc Emerg Med. 2009; 17: 44.

46. Haut ER, Kalish BT, Efron DT, Haider AH, Stevens KA, Kieninger AN, Cornwell EE 3rd, Chang DC. Spine immobilization in penetrating trauma: more harm than good? J Trauma. 2010 Jan;68(1):115-20;

47. NASCIS

48. Sayer FT, Kronvall E, Nilsson OG. Methylprednisolone treatment in acute spinal cord injury: the myth challenged through a structured analysis of published literature. Spine J. 2006 May-Jun;6(3):335-43

49. Roberts I, Yates D, Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head

injury (MRC CRASH trial): randomised placebo-controlled trial. Lancet 2004; 364:1321–1328.

50. Chikuda H, Yasunaga H, Takeshita K, Horiguchi H, Kawaguchi H, Ohe K, Fushimi K, Tanaka S.Mortality and morbidity after high-dose methylprednisolone treatment in patients with acute cervical spinal cord injury: a propensity-matched analysis using a nationwide administrative database. Emerg Med J. 2014;31:201-206

51. Tescher AN, Rindflesch AB, Youdas JW et al. Range-of-motion restriction and craniofacial tissue-interface pressure from four cervical collars. J Trauma 2007;63:1120-26.

52. Scott PE, Coates PJB, Davies SR, Gay DAT. Clearing the spine in the unconscious trauma patient: an update. JICS 2012 13(3):227-231

53. Ahn H, Singh J, Nathens A, MacDonald RD, Travers A, Tallon J, Fehlings MG, Yee A. Pre-hospital care management of a potential spinal cord injured patient: a systematic review of the literature and evidence-based guidelines. J Neurotrauma. 2011 Aug;28(8):1341-61.

Musculoskeletal Trauma

Pelvic Fractures

Fractures of the pelvis account for <5% of all skeletal injuries, but are particularly important because of the high incidence of associated soft tissue injuries and risk of massive haemorrhage. Open pelvic fractures have a >50% mortality rate. Pelvic fractures are generally high-energy injuries, primarily following pedestrian RTCs and falls from a height.

In simple terms, pelvic trauma can be considered as:

- Pelvic ring fractures

    - High risk life-threatening haemorrhage

- Acetabular fractures

    - May bleed, greatest issue in surgical reconstruction/long term outcome. Beware associated hip dislocation

- Pubic rami fractures

- Generally stable, occur in the elderly but can be a cause of mobility limited by pain.
- Sacro-coccygeal fractures
  - Generally stable

This chapter will concentrate on pelvic ring fractures due to their significant mortality.

Pelvic fractures are life-threatening injuries
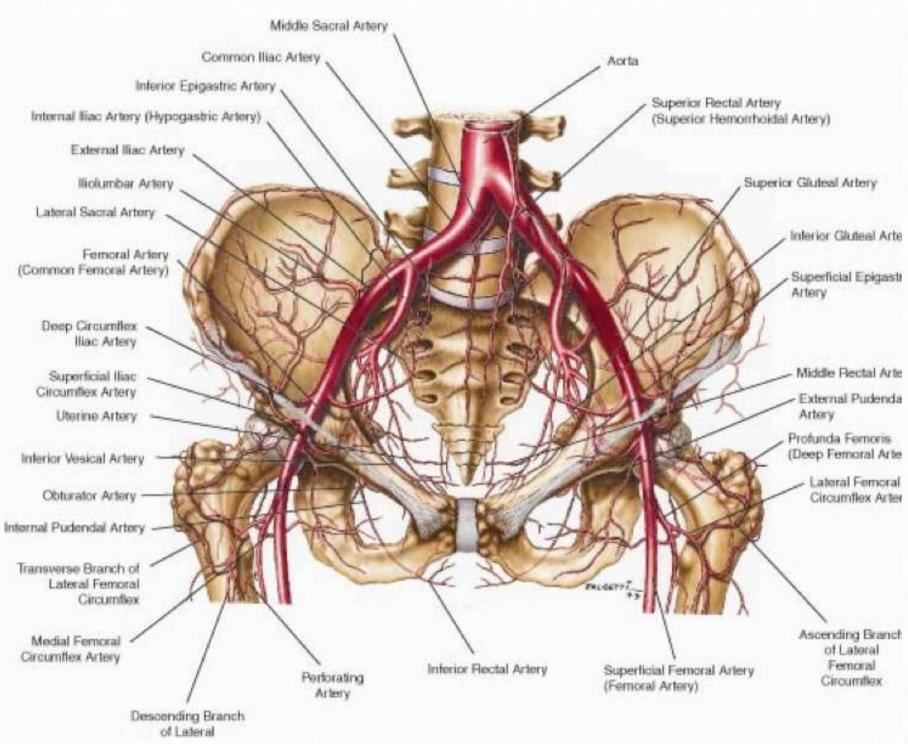
Pelvic Anatomy

The pelvic ring is made up of the two innominate bones and the sacrum, articulating anteriorly at the symphysis pubis and posteriorly at the sacroiliac joints. This ring contains the pelvic viscera, vessels and nerves. The stability of the pelvic ring depends upon the rigidity of the bony parts and the integrity of the strong ligaments that bind the three segments together. The strongest of these ligaments are the sacroiliac and iliolumbar, supplemented by the sacrotuberous, sacrospinous and the ligaments of the symphysis pubis.

The major branches of the common iliac vessels arise within the pelvis and are particularly vulnerable to posterior pelvic injuries. The nerves of the lumbar and sacral plexuses are also at risk with these injuries. The bladder lies behind the symphysis pubis and is held in position by the lateral ligaments, and in the male, by the prostate. The prostate is held laterally by the medial fibres of levator ani, and anteriorly is firmly attached to the pubic bones by the puboprostatic ligament. In females the urethra is much more mobile and less prone to injury.

In severe pelvic injuries the membranous urethra is damaged when the prostate is forced backwards whilst the urethra

Middle Sacral Artery

Common Iliac Artery

Inferior Epigastric Artery

Internal Iliac Artery (Hypogastric Artery)

External Iliac Artery

Iliolumbar Artery

Lateral Sacral Artery

Femoral Artery
(Common Femoral Artery)

Deep Circumflex
Iliac Artery

Superficial Iliac
Circumflex Artery

Uterine Artery

Inferior Vesical Artery

Obturator Artery

Internal Pudendal Artery

Transverse Branch of
Lateral Femoral
Circumflex

Medial Femoral
Circumflex Artery

Descending Branch
of Lateral

Perforating
Artery

Inferior Rectal Artery

Superficial Femoral Artery
(Femoral Artery)

Aorta

Superior Rectal Artery
(Superior Hemorrhoidal Artery)

Superior Gluteal Artery

Inferior Gluteal Arte

Superficial Epigastr
Artery

Middle Rectal Arte

External Pudenda
Artery

Profunda Femoris
(Deep Femoral Arte

Lateral Femoral
Circumflex Arter

Ascending Branch
of Lateral
Femoral
Circumflex

Pelvic Arterial Anatomy
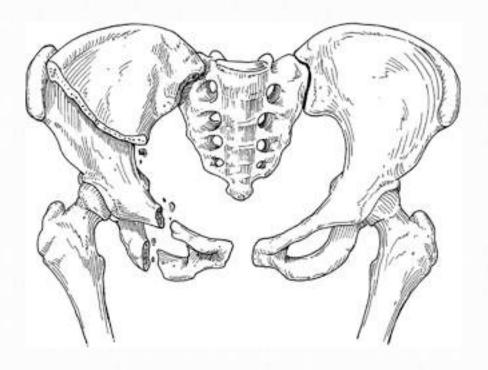
remains static. When the puboprostatic ligament is torn, the prostate and base of the bladder can become grossly displaced from the membranous urethra. The pelvic colon and mesentry is mobile and hence not readily injured. However, the rectum and anal canal are more firmly tethered and vulnerable in pelvic fractures.

When an anterior force is applied to the pelvis, the symphysis pubis is forced apart – an 'open book fracture'. The degree of separation depends on the force applied. A force which continues after the pubic symphysis has separated, will sequentially disrupt the anterior then posterior sacroiliac ligaments. If only the anterior SI ligaments are torn there will be rotational, but not vertical instability. If, however, the posterior ligaments are also in-

jured there will be both rotational and vertical instability with posterior translation of the injured hemipelvis. Vertical instability is therefore an ominous sign suggesting complete loss of ligamentous support.

Pelvic Ring Fractures

The major concern with pelvic ring fractures, is that the pelvic volume can be greatly increased allowing significant hidden haemorrhage from the damaged vessels. Stabilising the ring and reducing this potential increased volume ('closing the book') is the most important management issue. Venous bleeding can generally be controlled by the tamponade effect of reducing the pelvic volume. Arterial bleeding may not be controlled by this simple measure and these patients may continue to be haemodynamically unstable. Management options in this situation, when other bleeding sites have been excluded, include arteriography and embolisation, pelvic packing or aortic cross compression/clamping or REBOA in severe cases.

Mechanism of Pelvic Injury
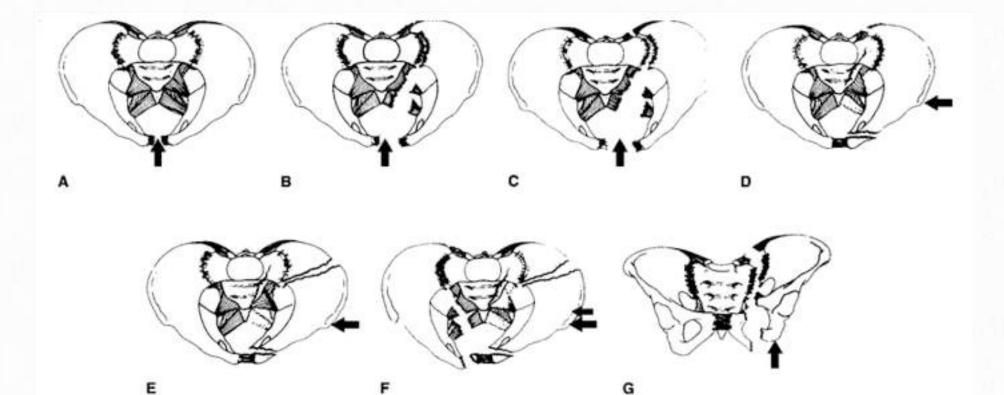
Certain mechanisms of injury are classically associated with particular types of pelvic fracture. The key message is to maintain a high index of suspicion for any type of pelvic injury:

Anteroposterior compression (A, B & C below)

• Frontal collision

• AP compression injuries may be stable/unstable depending on the degree of force and the involvement of posterior ligaments, as previously mentioned.

Lateral compression (D, E & F below)

• Side on impact

Young-Burgess Classification of Pelvic Fractures

- Lateral compression injuries are generally stable as one side of the pelvis is crushed in against the other and actually decrease the pelvic volume.

Vertical shear

- Fall from height onto one leg (G below)

- Vertical shear injuries are unstable.

Assessment of Suspected Pelvic Injury

A fractured pelvis can be difficult to diagnose clinically, so a high index of suspicion is required in every patient with serious abdominal or lower limb injuries and patients involved in RTCs and falls.

The patient must be assessed as a whole, following MARCH principles. In specific relation to pelvic injuries, the abdomen and perineum must be examined for signs of bruising or swelling. Classic teaching involves 'springing the pelvis', which essentially involves gripping a hemipelvis in each hand and applying a separating force to see if there is any movement or crepitus indicating pelvic ring disruption. This can be a potentially devastating manoeuvre by dislodging clots and creating further bleeding. Think - if you suspected a femoral fracture, would you try and disrupt it to assess stability or immobilise it on index of suspicion and x-ray it? Not only is springing the pelvis a clinically useless test if a pelvic x-ray is to be ordered as part of the standard trauma series, but it is both painful and dangerous.

Diagnosis of a pelvic ring fracture can often be made by on suspicion from mechanism, inspection or viewing radiographs, leaving this examination unnecessary and anything more then gentle palpation should be avoided. A Trauma CT Pan-scan or at very least a plain AP radiograph of the pelvis must be performed.

Do not 'spring' the pelvis

A rectal examination must be performed, feeling for location of the prostate and any fractures or tenderness. Blood at the external urethral meatus is a classic sign of a ruptured urethra. Neurological examination is important as there may be damage to the lumbar/sacral plexus, but as we saw in the spinal chapter this can be unreliable.

Other x-rays, such as inlet/outlet and right/left obliques (Judet views) can be helpful and may be requested following consultation with the orthopaedic surgeons. However CT is now by far an away the commonest and most reliable investigation in suspected pelvic trauma, especially where reconstructive surgery is likely to be required.

In assessment of the urological tract, the patient can be encouraged to void urine. If a urethral injury is suspected due to clinical findings, early urological referral is imperative. A trial of passing a urethral catheter may be attempted by the urologist, but can convert a partial to a complete tear and so is not recommended in inexperienced hands. Further investigations may include an intravenous urethrogram or retrograde urethrogram.

Management of Pelvic Injury

Early management must keep a sense of priorities with consideration given to the possibility of a pelvic fracture.

If a pelvic ring fracture is suspected, early intervention to address a potential increase in pelvic volume can reduce blood loss. A pelvic strap/binder is a quick, safe, easy method of doing this. A circumferential strap can be applied in the pre-hospital environment and should be at the level of the greater trochanters. There are a number on the market that we would recommend including the T-pod, SAM-sling and Prometheus Pelvic splint (see below). If you do not have one of these devices, then a triangular bandage can serve the same purpose.

A selection of Pelvic binders: T-pod, SAM sling, Prometheus

Once the pelvic strap is in place, ideally we should also turn the feet into a neutral position and fasten them together to reduce 'turnout' of the legs and stress on the pelvis.

Pelvic straps can be used for prolonged periods (up to two weeks post injury), but in this situation it is advisable to alternate the anatomical level (greater trochanter / proximal thighs) of application every three hours to prevent pressure areas developing. It is often important to control external rotation of the feet by strapping together. This also reduces pelvic volume.

Pelvic ring fractures can be definitively managed with external fixators or open reduction and internal fixation, depending on patient and fracture factors. External fixators can be applied anteriorly into the iliac crests or inferior iliac spines, or posteriorly through the posterior iliac wing/SI joint.

They can be used to temporarily stabilise an unstable ring fracture and are probably best applied in an operating theatre by experienced hands. There are very few, if any, indications for putting an external fixator on a haemodynamically unstable patient in the emergency department. Always beware of the overly-optimistic orthopaedic surgeon who claims that they can be applied in 15 minutes or less! A pelvic binder takes less than a minute and is just as good at least in the initial stages of care. A pelvic strap can be applied to control the pelvic volume temporarily whilst patient assessment continues and will not de-

lay patient transfer to theatre if indicated. That said, the C-clamp can be applied rapidly by a suitably trained and experienced surgeon.

A pelvic 'anti-shock' C-clamp

The complications of emergency ex-fix include:
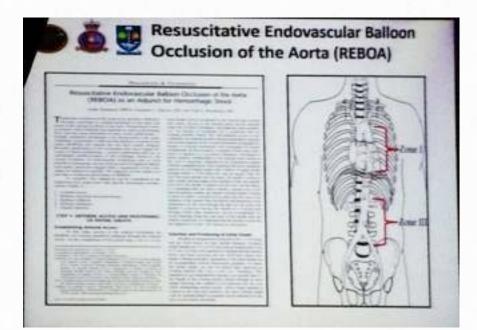
- Incorrect pin site placement

- Risk of closing the book anteriorly, only to open up posteriorly if the fracture pattern not properly assessed

- Delays to definitive management

- Stay and play in ED when patient needs life-saving laparotomy

- Pin site infections

- Pelvic osteomyelitis

ORIF is the gold standard, but can generally wait until the patient is stable and appropriate investigations have been undertaken.

Management of patients with signs of persistent bleeding with a stabilised pelvic fracture is difficult. There may be arterial bleeding related to the pelvic injury, as previously discussed, and in this instance spiral CT with contrast, followed by 'directed' angiography and embolisation is indicated and should be the gold standard. Don't forget Tranexamic acid within the first three hours of injury (CRASH 2)!

If the patient is too unstable to transport to CT then we can either consider immediate transfer to theatre for pelvic packing or alternatively we can use REBOA – Resuscitative endovascular balloon occlusion of the aorta, which can be performed by anyone who has experience of inserting large bore femoral lines. This is a 1.4ml embolectomy balloon catheter, inserted through the femoral artery to the point above where the aorta splits right and left (Zone III). This inevitably stops all blood flow to the lower half of the body, below this point and the clock is ticking as soon as the balloon is inflated.

Transfer may then be possible to the angiography suite, although identifying the bleeding point may then be difficult with the balloon inflated. A short deflation may be necessary to identify this point for embolisation.

REBOA is increasing in use but as yet there is little published in support of its use but watch this space!

2012

A simpler alternative may be the Abdominal tourniquet, which has shown some promise in simple trials. ATACC have limited experience with this device, and remain unable to actively recommend it, especially with our concerns about venous congestion in the pelvis during application.

If things are still not improving then other bleeding sources must be excluded, think chest, abdomen and long bone fractures. Suspect, examine, E-FAST, CT.

Caution when considering a laparotomy in a patient with a pelvic fracture, as this procedure can release any abdominal tamponade effect resulting in catastrophic pelvic haemorrhage. Experienced surgeons must be available with the ability to pack the pelvis or cross clamp the aorta if required.

Pelvic ring fracture suspected? Apply pelvic strap

Extremity Trauma

Extremity trauma is common, often dramatic, but rarely life threatening. It is important to ensure that even apparently minor injuries are identified, as later when the patient has recovered from the initially more significant injuries, the potential limb dysfunction can have the greatest impact on returning to normal activities.

Extremity trauma is a huge subject and this chapter aims to give guidance on the safe assessment and initial management of such injuries. One must not just consider fractures, but also dislocations, traumatic amputations, crush injury, degloving and burns. The skin, musculo-tendinous units, neuro-vascular structures, bone and joints can be affected and must be appropriately examined.

Assessment of Injured Extremities

Initial patient assessment should follow MARCH Principles. Early fracture stabilisation is important in haemorrhage control.

Limb Examination

Further limb assessment often occurs during the secondary survey following adequate exposure.

- Look
    - Wounds
    - Deformity
    - Colour / perfusion
- Feel
    - Tenderness
    - Crepitus
    - Temperature
    - Pulses
- Move
    - Joint stability

Following the above limb examination and appropriate documentation, it is necessary to perform a neurological assessment. The following table should act as a quick aide memoire.

NERVE	MOTOR	SENSATION
Ulnar	Finger abduction	Little finger
Median	Thumb abduction	Index finger
Radial	Wrist extension	Dorsal 1st web space
Axillary	Deltoid	Regimental badge
Femoral	Knee extension	Anterior knee
Sciatic	Ankle movements	Foot
Posterior Tibial	Toe flexion	Sole of foot

Classification of Closed Fractures with Soft Tissue Injuries (Tscherne)	
Type 0	Minimal soft tissue damage. Indirect violence. Simple fracture patterns
Type I	Superficial abrasion or contusion caused by pressure from within. Mild to moderately severe fracture configuration
Type II	Deep, contaminated abrasion associated with localised skin or muscle contusion. Impending compartment syndrome. Severe fracture configuration.
Type III	Extensive skin contusion or crush. Underlying muscle damage may be severe. Subcutaneous avulsion. Decompensated compartment syndrome. Associated major vascular injury. Severe or comminuted fracture configuration.

Classification of Open Fractures (Gustilo)	
Type I	Skin opening of 1cm or less, quite clean. Most likely from inside to outside. Minimal muscle contusion. Simple transverse or short oblique fractures.
Type II	Laceration more than 1cm long, with extensive soft tissue damage, flaps, or avulsion. Minimal to moderate crushing component. Simple transverse or short oblique fractures with minimal comminution.
Type III	Extensive soft tissue damage including muscles, skin and neuro-vascular structures. Often a high velocity injury with severe crushing component.
A	Extensive soft tissue injury. Adequate bone coverage. Segmental fractures. Gunshot wounds
B	Extensive soft tissue injury with periosteal stripping and bone exposure. Usually associated with massive contamination.
C	Vascular injury requiring repair.

Scoring Systems

There are various scoring systems in use for grading soft tissue injuries following fractures and an awareness of them is important although they are not commonly used in clinical practice. More important is an awareness of the mechanism of injury and potential for significant soft tissue involvement.

A fracture is simply a broken bone within a traumatised soft tissue envelope

These classifications of open fractures are often quoted, but are clinically of limited value. An accurate grade can probably only be applied after the initial debridement, when assessment of soft tissue injury/loss can be made. they can be confusing classifications to use, because the various severity types are multifactorial (address mechanism of injury, energy involved, length of laceration, fracture pattern) and therefore, are not mutually exclusive. Recent studies have shown poor interobserver concurrence when using these systems.

A break in the skin with an underlying fracture is an open fracture and should be treated as such. The significance of the severity of the soft tissue component and

contamination can be addressed at the time of debridement. The adequacy of the debridement is arguably the most crucial factor in whether deep infection/osteomyelitis occurs as a late complication.

Limb Salvageability Scoring

With continuous improvements in pre-hospital care, more patients with severe extremity injuries involving either vascular compromise or partial amputation are surviving. The dilemma between sacrificing a potentially useful limb segment and the time-consuming and resource-intensive salvage of a functionally useless extremity has been addressed by several investigators. Johansen et al scored using four variables.

In the initial study, limbs with a MESS score of 7 or higher predicted, with a high degree of confidence, the need for initial or delayed amputation, whereas, all limbs with a score of 6 or less remained viable. This scoring system needs further validation in larger studies. It is only designed for use in the lower leg and its greatest weakness is for false-negative predictions (in young patients with intact vessels, minimal blood loss and major muscle or skeletal destruction may score less than 7, but primary

MESS Mangled Extremity Severity Score		
COMPONENT		POINTS
Skeletal/soft tissue injury	Low energy stab Medium energy open # High energy shot gun Very high energy - contaminated	1 2 3 4
Limb Ischaemia	Perfusion normal ±pulse Pulseless, diminished cap refill Cool, paralysed, insensate	1 2 3
Shock	Systolic BP> 90mmHg Hypotensive transiently Persistent hypotension	0 1 2
Age(yr)	<30 30-50 >50	0 1 2

amputation may be the treatment of choice). In general terms, the MESS score and other similar scoring systems are not routinely used in clinical practice in the UK, and the decision to amputate is one based on experience and individual factors.

Management of Injured Extremities

Initial management must concentrate on haemorrhage control, early fracture realignment and analgesia.

Please revisit the chapter on Massive Haemorrhage Control. Haemorrhage from a limb is normally controllable by the application of direct pressure. Simple bandaging may be insufficient initially, and some one may need to apply firm manual pressure through a dressing, as accurately onto the major bleeding point as possible. Fracture realignment, frequently stems seemingly uncontrollable haemorrhage.

Wounds:

- Polaroid/digital photograph of wounds if possible
- Remove gross contamination
- Wound dressing saline soak
- Analgesia
- Tetanus
- IV antibiotics

Fractures and Dislocations:
- Assessment distal neuro-vascular status

    - If compromised, one attempt at reduction by experienced hands

- Splint in position limb found

- AP/lateral radiograph

- Reduce joint and splint

- Re-xray and reassess neurovascular status

Amputations
- Haemorrhage control

- Chlorhexadine/saline soaked dressing to stump (betadine interferes with CT imaging due to iodine molecules mimicking contrast)

- Amputated part saline irrigation / wrap in gauze / plastic bag / ice

- DO NOT FREEZE / AVOID DIRECT CONTACT WITH ICE

Surgical Issues
Timing Of Surgery
Often patients with significant limb trauma have injuries to other anatomical areas, including head, chest and abdomen. Limb injuries can be surgically treated by two main principles early definitive care and

damage control orthopaedics (DCO). For early definitive care, a patient needs to be otherwise stable to undergo necessary investigation (for example CT) and surgery, which may take several hours and provide further insult to other organ systems (for example the lungs with intra-medullary reaming for nails).

The concept of DCO exists for patients who are not deemed fit for early definitive care. Wounds must be adequately debrided and the long bones immobilised, often by the use of external fixators. Experienced surgeons must carefully plan their strategy for DCO with the anaesthetic team, as the use of external fixators can hamper ideal definitive care. If definitive care can not be provided immediately, a window of opportunity exists up to 48 hours post injury, beyond that we must move to 5-10 days or >3 weeks, due to the inflammatory response and second hit effect of trauma and surgery.

Open Fractures

Management of open fractures involves debridement, washout, bony stabilisation (not necessarily definitive fixation, as above), soft tissue coverage and antibiotic therapy. The most reliable strategy in reducing infection rates is adequate debridement, by an experienced surgeon as soon as possible and early antibiotic therapy.

Any wound overlying a fracture makes that fracture open and susceptible to contamination with significant risk of infection

Compartment Syndrome

A compartment syndrome may occur in any site in which muscle is contained within a closed fascial space. Common areas are the lower leg, forearm, foot, hand, gluteal region and the thigh. Compartment syndrome develops when the pressure within the closed compartment rises, causing muscle ischaemia and subsequent necrosis leading to fibrotic contractures. The pressure can rise due to an increase in contents, such as from fracture bleeding, or decrease in size, such as from tight constricting dressings.

Signs And Symptoms Of Compartment Syndrome:

*   HIGH INDEX OF SUSPICION

*   Pain – greater than expected

*   Pain on passive muscle stretch

    *   Paraesthesia in peripheral nerve distribution

    *   Functional loss of nerves traversing compartment

*   Tense swelling in involved region

*   Palpable pulse – only lose pulse late

Intra-compartmental pressure measurements can sometimes be helpful in diagnosis, but are not 100% reliable. Tissue pressures in the affected compartment within 30mmHg of diastolic blood pressure are an indication for immediate surgical decompression by fasciotomy. In the majority of cases, however, it is often a case of using clinical suspicion then, proceed directly to fasciotomy.

It is useful to note that individual nerve blocks are unlikely to affect the ability to detect a developing compartment syndrome, although a dense epidural block may well mask all signs from the patient.

Emergency Amputation in the Field

Amputation can be traumatic or clinically planned. It can also be partial or complete.

Many traumatically amputated limbs can now be re-implanted and should be clearly labelled, with the patient (as they may become separated) and then stored clean an cool, ideally in an iced environment, but not in direct contact with the ice or frozen.

Rapid transfer to a dedicated plastics centre is recommended if other injuries have been excluded or to a MTC with plastics support.

Other amputations, as frequently seen in Afghanistan caused by IED explosions, cause massive tissue loss and life threaten-

ing injuries. In these cases, life takes complete priority over limb and there is often little time wasted in attempting to salvage such badly injured limbs. Life-saving rather than limb-saving interventions are the priority and there are highly effective rehab programmes, which have demonstrated unbelievable recovery results.

Amputation itself can also be performed to save life. This can be late as in relief of gangrene, as first performed by Hippocrates and described in De Articularis or it can be acute and life saving during an entrapment.

A Traumatic amputation of an upper limb – note priority is haemorrhage control

Modern rescue equipment and enhanced level pre-hospital medical care can usually effectively release or maintain a casualty during a complex release without amputation. However, if the casualty is deteriorating or the limb is mangled beyond repair or impossibly trapped (e.g. in heavy machinery) then a field amputation should be performed.

Typically this should be agreed by all present as the only option and if possible the patient should be informed, but this may not be realistic given the situation.

Assuming the scene is safe, control of bleeding and analgesia are likely to be the initial priorities. From a pre-hospital and emergency medicine perspective, opioids and NSAIDS are ineffective in managing entrapment or tourniquet-associated pain. NMDA receptor antagonist such as Nitrous Oxide and Ketamine (at analgesic doses of 0.2mg/kg), are the agents of choice. Ketamine will also provide the best agent for deepening analgesia to anaesthetic levels for the actual amputation.

Local anaesthetic or a regional block may also reduce the need for such high doses of ketamine, but this should not delay the process, which should be as quick as possible.

This procedure can be performed very quickly and does not require an orthopaedic surgeon. Any PHEM doctor, should be capable of this technique.

Decide on the level of amputation, which should be as distal as possible. Through joint amputations are often much quicker. We are not considering any kind of skin flaps or closure at this stage. This is a strictly no frills rescue procedure.

If possible a CAT or similar tourniquet should be applied just proximal to the planned incision (not too close to impair the cut), this is not essential and there may be insufficient room for this to be applied. If possible, a second tourniquet should be positioned which can be rapidly tightened if the first is inadequate.

Importantly, there should be as much anaesthetic pre-planning as possible before commencing the procedure. A plan for airway management (including what to do if the patient vomits on induction or mid procedure, as well as post-procedure anaesthesia or completion of RSI if not intubated prior to amputation) should be agreed and prepared for as best as possible.

Anaesthesia is commenced and once ready the skin is incised circumferentially, proceeding through all layers and tissues down to periosteum.
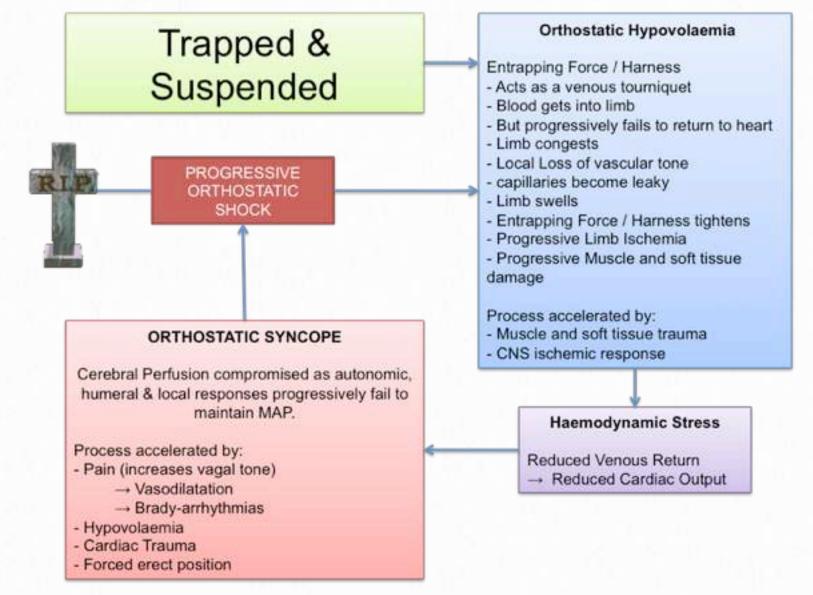
At bone, this can be sawn clean through with a bone or hack saw. An alternative is to use a Gigli saw, which can even cut through the skin if space is very limited around the limb. This type of amputation can performed in a matter of a few minutes. Spencer wells clips are occasionally necessary, but the tourniquet is normally all that is required.

Don't forget, Tranexamic acid and antibiotics if there is a delay to hospital care

In an extreme situation, such as an underwater entrapment where there is a serious risk to life and surgical procedures would be impossible, the fires service hydraulic cutters can be safely used, with good and rapid effect. This has been trialled and produces an adequately clean cut for closure.

Trapped & Suspended

Orthostatic Hypovolaemia

Entrapping Force / Harness
- Acts as a venous tourniquet
- Blood gets into limb
- But progressively fails to return to heart
- Limb congests
- Local Loss of vascular tone
- capillaries become leaky
- Limb swells
- Entrapping Force / Harness tightens
- Progressive Limb Ischemia
- Progressive Muscle and soft tissue damage

Process accelerated by:
- Muscle and soft tissue trauma
- CNS ischemic response

PROGRESSIVE ORTHOSTATIC SHOCK

ORTHOSTATIC SYNCOPE

Cerebral Perfusion compromised as autonomic, humeral & local responses progressively fail to maintain MAP.

Process accelerated by:
- Pain (increases vagal tone)
    → Vasodilatation
    → Brady-arrhythmias
- Hypovolaemia
- Cardiac Trauma
- Forced erect position

Haemodynamic Stress

Reduced Venous Return
→ Reduced Cardiac Output

Mechanism of suspension syncope

Crush Syndrome & Suspension Trauma

Crush injury occurs when a prolonged static compressive force sufficient to interfere with normal tissue metabolic function is applied to a body part. The extremities are most commonly affected, with the lower limbs being more frequently involved than the upper limbs. Significant crush injuries to the head and torso often result in early death from other causes (e.g. traumatic brain injury, asphyxia, exsanguination).

When a crushed limb is released a predictable sequence of pathophysiological events occurs, known collectively as Crush Syndrome. These events include hypovolemia, rescue cardioplegia, electrolyte and acid-base abnormalities, rhabdomyolysis, and acute renal failure.

Suspension Syncope occurs due to motionless vertical suspension and orthostatic pooling of blood in the lower extremities. The inability to regain a horizontal position and the resultant hypo-perfusion of the brain leads to orthostatic syncope and if uncorrected, death. The term Suspension Trauma defines a 'crush syndrome' resulting from compressive forces applied by a harness to the lower extremities during prolonged vertical suspension.

Rescue Cardioplegia

Heart Damaging Delivery
Rapid Bolus
into a Shocked System
with reduced Cardiac Output
Ensuring Maximal Atrial Stretch

Cardioplegic Solution
Hypertonic
Cold
↑ KCl
↑ Mg
↑ Ca
↑ Phos
↑ Amino Acids
↓ pH

Rescue Cardioplegia – The problem with uncontrolled limb release

Rescue Cardioplegia describes the myocardial stunning that can occur on uncontrolled release of a compressing force, harness or tourniquet. There are a number of mechanisms that can combine to cause fatal cardioplegia on limb reperfusion. It is unlikely that any one mechanism in isolation is responsible for such rapid pathophysiology in an otherwise fit individual. However, the combined changes in systemic haemodynamics and biochemistry during limb isolation and then subsequent limb reperfusion, in the presence of generalised systemic insult can be fatal.

As cold, pooled blood under pressure in congested limbs is rapidly released back into the systemic circulation, a sudden, transient and considerable increase in preload to the right heart occurs. Lying a casualty flat or placing them in the Trendelenburg position, as is common place during motor vehicle extrication from the seated position, could conceivably further increase this venous return. Increasing preload by so much and so rapidly results in sudden atrial stretch, which shortens the effective atrial refractory period and causes anisotropic alterations in conduction properties, potentially stunning the myocardium into asystole or initiating atrial fibrillation.

⇧CO2
-ve inotropic effect
Vasodilates
Worsens Shock

⇧Adenosine
Heart Block

⇧Free Ca²⁺
-ve inotropic effect
Arrythmogenic
Triggers Apoptosis

⇧Lactic Acid
-ve inotropic effect

Reperfusion Injury

⇧K⁺
Arrythmogenic

⇧Phosphate
Microvascular impairment
3rd Space Fluid Shift

⇧ Thromboembolic Material
Micro & Macro Vascular impairment
Pulmonary Embolism
Fat Embolism
DIC

This occurs at the same time as afterload and systemic vascular resistance (SVR) is rapidly reduced, by allowing blood to flow again into the previously isolated limb. Hypotension is further compounded by a post-ischaemic reactive hyperaemia in the released limbs as ischaemic metabolites such as adenosine and lactic acid directly effect a generalised systemic vasodilatation. This causes a decrease in venous return and a further rapid reduction in cardiac output. The effect can be reliably reproduced and thus studied during reperfusion following lower limb orthopaedic surgery of a single limb, with multiple reports of drops in cardiac output by an average of 18% or 26mmHg in a healthy adult.

Taking the weight off a harness or releasing two limbs trapped by a dashboard, may result in both limbs being suddenly released, and as such the reduction in cardiac output can in itself be fatal, particularly in the presence of a relatively fixed cardiac output from heart disease, drugs or pre-existing valvular heart defects.

Mechanism of rescue cardioplegia

Cardiac electrical activity functions in both a very narrow pH range and concentrations of intra and extra-cellular ions, in particular calcium and potassium. There are a number of mechanisms that can illicit fatal cardioplegia. Damaged, congested, ischemic limbs can quiet literally brew an "ideal cardioplegic solution" which when released into circulation can precipitate a fatal arrhythmia, especially in combination with the hemodynamic effects described. It is well recognised that these electrolyte abnormalities may develop over time as limb reperfusion is not an all or nothing effect, but rather a 'bolus' then tapering off 'infusion' or 'reperfusion' phenomena.
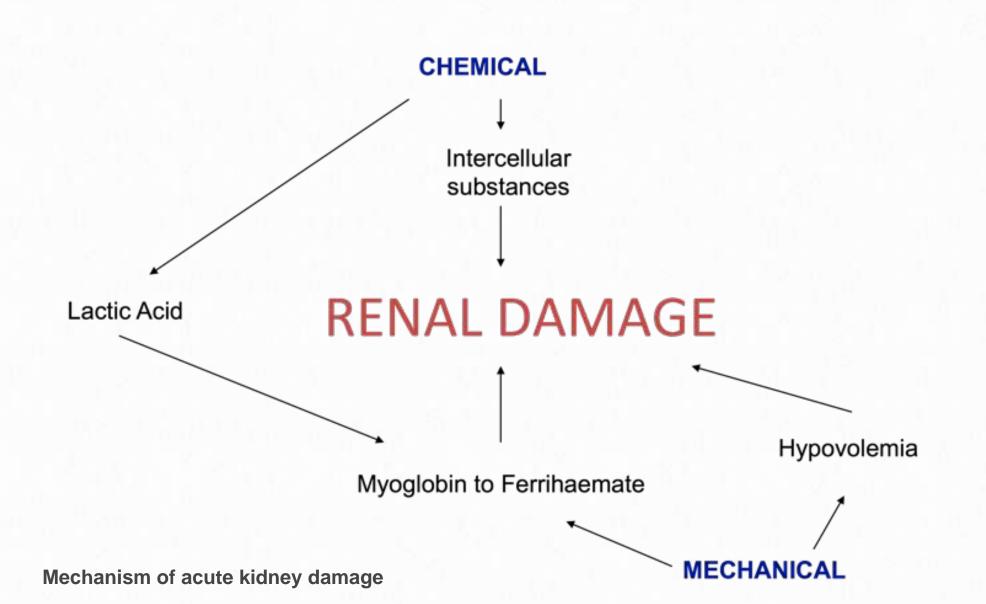
Muscle and Soft Tissue Trauma – Whole System Effects

Even if the initial trauma is insufficient to damage muscle and soft tissues, the combination of pressure, stasis and ischemia underneath an entrapping force, harness or tourniquet will usually initiate some irreversible muscle death. A constant external mechanical pressure prevents muscle from adequately maintaining cell wall integrity by literally forcing extracellular cations and fluid against their normal electrochemical and osmotic gradients. Cell wall extrusion pumps eventually become overwhelmed, allowing water with dissociated sodium, chloride and calcium ions to enter the cell. Progressive tissue oedema ultimately leads to cell death.

Compartment syndrome occurs where intramuscular compartment forces act continually above the diastolic blood pressure resulting in compression and ultimately death of nerves, blood vessels and muscle inside their normal anatomical spaces within the body. An external entrapping force will nearly always compress more than one compartment.

With so many uncontrollable variables in the prehospital environment, it is impossible to accurately predict muscle viability against ischemia time. Lengthy entrapments will obviously have a detrimental effect, but it important to appreciate that there is absolutely no 'minimum' universally agreed time for 'safe' suspension or entrapment.

When the integrity of muscle cell walls are breeched by an external force, intracellular components leak extracellularly whilst water and extracellular ions will flow into the damaged tissue, so called third space fluid loss. Cell contents will literally be forced into the vascular compartment and as

Mechanism of acute kidney damage

such, an isolated insult has the potential to initiate a distal systemic effect, the principle effect being direct and indirect renal damage.

Acute Kidney Injury – an indirect effect of Muscle Damage

Direct renal damage result from the nephrotoxic properties of a variety of leaked intracellular substances such as proteases and purines. However, damage principally occurs indirectly as the kidneys attempt to filter acidotic plasma and the muscle protein, myoglobin.

It is very important, from a therapeutic perspective, to appreciate that myoglobin itself causes no renal damage. It is a small protein that is freely filtered and eliminated by the kidneys with no nephrotoxic properties. Myoglobin release unfortunately does not occur in isolation. Organic acids are released into the circulation during cell death, whilst progressive muscle ischemia results in lactic acid production. This by-product of anaerobic metabolism, together with other organic acids being released from cells, lowers the pH of urine. As the filtered myoglobin combines with urine below a pH of 5.6, it is converted to a larger protein, ferrihaemate. Ferrihaemate is both directly nephrotoxic to renal tubules and

causes mechanical obstruction by precipitating within the lumen of nephrons. Inadequate circulating volume due to hypovolaemia and third space fluid shift will contribute significantly to the development of an acute kidney injury and subsequent renal failure as it reduces renal perfusion, just as the kidneys are seeking additional filtrate to dilute urine and wash away rapidly accumulating ferrihaemate and other mechanical obstructions.

Management of Crush and Suspension Trauma

Isolate and move to a place of safety. By applying arterial tourniquets just proximal to a harness or entrapping force, one can prevent the massive haemorrhage or rescue cardioplegia frequently encountered with sudden release of an entrapment on scene; transferring the problem to a safer, controlled environment. This ethos fits well within the established philosophy of "scoop and run." In the case of a suspended casualty, they should be rescued as soon as is safely possible and placed in the horizontal recovery position if consciousness is impaired. There is no evidence to support rescue in the semi-recumbent position.

Tourniquets:

- Halt the cyclical progression of orthostatic shock, avoiding further 'relative' hypovolaemia.
- Allows the safe, rapid transport of a potentially complex critical care patient, via conventional means, to an appropriate facility.
- Allows the casualty to lie flat, thus improving cerebral perfusion and airway management
- Enable emergency service staff to treat the casualty within their normal guidelines and scope of practice en-route to hospital, without being distracted by the complexities of harness pathology and crush.
- Allows for resuscitation, monitoring and systemic preparation of the casualty prior to release and reperfusion.
- Allows for 'controlled' or 'staged' reperfusion.

Tourniquets

Tourniquets must be purposefully designed for prehospital use; and ideally provide a broad, evenly distributed force, sufficient to isolate arterial supply to the limb. They will inevitably cause a degree of ischemic reperfusion injury themselves, but the benefits greatly outweigh this risk, especially where ambulance transit times are short. If rescue has been completed without the application of a tourniquet and the patient remains stable then delayed application of a tourniquet is not required as 'washout' will have already occurred.

Studies have shown us that on tourniquet application, without exsanguination of the lower limb, mean arterial blood pressure (MAP) increases by 26% . This immediate

haemodynamic effect has been attributed primarily to the 20% increase in the SVR caused by tourniquet isolation of a single lower limb, a sizable percentage of the body's vascular bed, together with pain from physical compression.

Where tourniquets have been applied, they should remain in place until the patient is fully resuscitated, potential haemorrhage points addressed and in a safe environment. Ideally this will be in the hospital resuscitation room or operating theatre, with full cardiovascular monitoring and support. There may be cases where there is a long delay to definitive care and in these cases 'staged release' should be employed.

In the prehospital setting, the balance of risk pivots between uncontrollable major haemorrhage and unsalvageable critical limb ischemia. Anecdotal reports exist of fit and healthy soldiers safely sustaining tourniquet times of 4-6 hours without loss of limb. The Israeli Defence Force published 90 cases of tourniquet application, finding complications only after 150 minutes, none of which resulted in limb loss.

Limb amputation may be indicated if the limb is deemed non-viable or if the patient's clinical condition deteriorates during rescue. Amputation prior to release will

also prevent the sequelae of the reperfusion syndrome by removing the source of the problem.

Resuscitate the System

A haemodynamically stable system will handle a reperfusion injury better than a collapsed, shocked system. A great deal of thought needs to be applied to preparing the circulation prior to entrapment release. There is a wealth of data from disaster medicine literature to support early circulatory resuscitation prior to reperfusion. Spending time optimising an entrapped person poses significant health and safety risks, the obvious being the stability of the entrapping structure and the potential for injury to personnel. Medical staff must work in close collaboration with rescue personnel, ideally as an integrated team, to understand differing roles and needs.

Systemic resuscitation prior to extrication in earthquake entrapment has been shown to significantly improve outcome. An initial 20ml/kg bolus (10ml/kg in the elderly) of 0.9% Saline should be administered prior to release in patients trapped for over one hour. Ongoing fluid administration should continue at a rate of 5ml/kg/hr with additional fluid boluses titrated against clinical response. Administration of potassium-containing solutions (e.g. Hartmans) must

be strictly avoided in the field to avoid hyperkalaemia. When the patient is collapsed in a confined space, intravenous access maybe challenging and intraosseous infusion should be considered.

For prolonged transfers the patient should have a urinary catheter placed to monitor urine output. Improving urine output is a good indication of end organ perfusion and that preventative management is starting to become effective. When this is not possible simple verification of urine output may be an acceptable compromise.
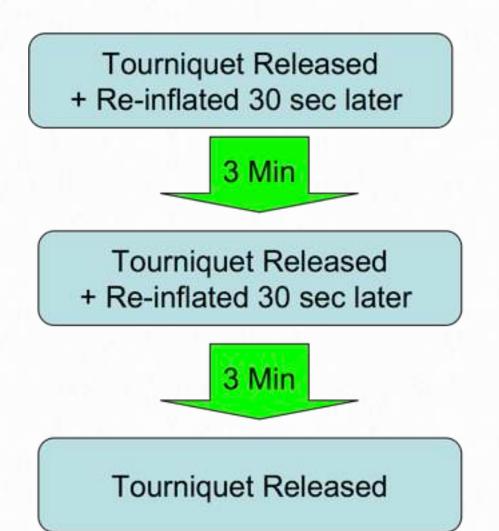
Analgesia

Pain is often minimal in the early post-crush phase due to circulating endorphins and pressure neuropraxia. As limbs become progressively more swollen and the intrinsic analgesic effects of endorphins wear off, pain will become more problematic.

45 to 60 minutes after initial tourniquet application, tourniquet associated hypertension usually begins to become apparent in approximately 11% of patients. This increase in MAP is independent of SVRI and is largely initiated and sustained by stimulation of C fibres which then activate N-methyl-D-aspartic acid (NMDA) receptors. NMDA receptor activation increases blood pressure, and blocks vagal attempts to restore normotension. From a pre-hospital and emergency medicine perspective, opioids and NSAIDS are thus ineffective in managing entrapment or tourniquet-associated pain. NMDA receptor antagonist (Nitrous Oxide and Ketamine) are the agents of choice as previously discussed. Regional local anaesthetic blocks may also be useful in providing additional analgesia for the trapped limb.

Staged Tourniquet Release Strategy

Whilst metabolic changes are time dependent and are more pronounced the longer the period of ischemia, in order to ameliorate false reassurances that limb ischaemic time is the only variable involved in Rescue Cardioplegia, "Safe Tourniquet Time" needs to remain a variable with an upper limit set for limb salvage only and not be factored into systemic risk stratification for Rescue Cardioplegia, which can occur much earlier.

Tourniquet application causes further rhabdomyolysis, skin necrosis, and neurovascular damage. A staged or staggered tourniquet release strategy may therefore be considered when evacuation to hospital is likely to be prolonged (e.g. >1 hour).

Tourniquet Released
+ Re-inflated 30 sec later

3 Min

Tourniquet Released
+ Re-inflated 30 sec later

3 Min

Tourniquet Released

This release process, conducted three times, for thirty seconds with three minutes recovery between each release, allows for controlled washout and systemic redistribution of ischemic metabolites during reperfusion. It should be employed on one limb at a time and the patient must be fully monitored closely. If at any point the patient becomes unstable or deteriorates then the tourniquet should be immediately retightened and the patient's cardiovascular state managed prior to re-instituting the release strategy.

Note that the tourniquet should NOT be gradually released, as this risk arterial inflow whilst venous outflow is obstructed.

This will produce limb congestion, oedema and further compromise.

The tourniquets should be fully on or fully off.

Once optimal volume resuscitation has been achieved further hypotensive episodes may be treated with inotropic or vasopressive agents.

Management of Hyperkalaemia

Post-release hyperkalaemic ECG changes (QRS widening) should be treated emergently with 10-30ml of intravenous 10% calcium gluconate and administration of 10 units of rapidly acting insulin in 50ml of 50% dextrose to encourage potassium uptake into cells. This is the only time when Calcium should only be considered and should be done mindful of the very real risk of precipitating metastatic calcification and further muscle damage. The use of calcium resonium exchange resins has been a traditional teaching point, however multiple reviews have said that they are not useful in the treatment of acute hyperkalaemia, and do not increase potassium excretion any more than using laxatives alone.

Standard medical management strategies for hyperkalaemia, tends to be ineffective, as hyperkalaemia in a crush injury results

from muscle wall damage, and not ionic or osmotic shifts. Patients must therefore be immediately transferred to an intensive care environment capable of haemofiltration.

In the event that prehospital anaesthesia is required as part of the resuscitative process, non-depolarising muscle relaxants (e.g. rocuronium) should be used instead of suxamethonium to minimise avoidable elevations in serum potassium.

Alkaline Diuresis

When evacuation times are prolonged (>4hrs) the use of alkaline diuresis may be considered. Alkaline diuresis will prevent the precipitation of toxic myoglobin metabolites in nephrons and help ameliorate acidosis and hyperkalaemia. 50ml of 8.4% sodium bicarbonate can be added to each alternate litre of fluid administered, titrated to a urinary pH ≥6.5. If prolonged alkaline diuresis is planned then 0.9% saline should be alternated with 5% Dextrose to reduce the sodium load and risk of pulmonary oedema (particularly in the presence of pre-existing renal or heart failure). The risk of iatrogenic metabolic alkalosis and sodium overload is greater in the unmonitored prehospital environment and where possible alkaline diuresis should be left for the hospital environment where it can be

titrated to urine output, urine pH and serum pH. Bicarbonate will also aid in potassium excretion via renal mechanisms (if still operating).

Triage

Where possible, all patients with significant crush injury or suspension trauma should be triaged to a facility capable of providing renal replacement therapy (haemofiltration).

Future Considerations

Strong evidence is emerging that calcium antagonists, given either before the onset of ischemia or at the time of reperfusion, ameliorate myocardial 'stunning' by preventing excessive calcium uptake. Calcium mediated mechanisms undoubtedly play a role in several aspects of rescue cardioplegia. Calcium is considered an important positive inotrope with unique vasodilatory properties. Its role is pivotal in the normal cardiac action potential. A release of calcium from the sarcoplasmic reticulum is induced by calcium influx into the cell through voltage-gated calcium channels on the sarcolemma. This phenomenon is called calcium-induced calcium release and is the key event that couples electrical excitation with contraction in the heart.

During even brief periods of ischemia, in response to a fall in pH, calcium binding to albumin decreases, thereby increasing plasma calcium concentration. Upon release of a tourniquet, this increase in ionized calcium may further reduce SVR. Furthermore, if this relative hypercalcaemia is prolonged, a cascade of events has been described which ultimately results in myocardial ischaemic injury. Whereas excess cytosolic calcium can permanently damage organelles that regulate the contractile cycle, it is proposed that an excess oscillation of this cytosolic calcium may be highly significant in contributing to reperfusion ventricular arrhythmias or cardioplegia. Reperfusing a limb, as opposed to a single organ, results in a sudden release and then re-uptake of free Calcium ions. Further investigation into this process is warranted. A staggered tourniquet release interestingly demonstrated significantly less oscillation of ionized calcium levels.

References & Further Reading

1. Stannard A1, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. J Trauma. 2011 Dec;71(6):1869-72. doi: 10.1097/TA.0b013e31823fe90c.

2. Scalea, TM, Optimal timing of fracture fixation: have we learned anything in the past 20 years? J Trauma. 2008 Aug;65(2):253-60. doi: 10.1097/TA.0b013e31817fa475

3. McNicholas MJ1, Robinson SJ, Polyzois I, Dunbar I, Payne AP, Forrest M. 'Time critical' rapid amputation using fire service hydraulic cutting equipment.Injury. 2011 Nov;42(11):1333-5. doi: 10.1016/j.injury.2011.05.002. Epub 2011 Jun 1

4. Adisesh A, et al. Harness suspension and first aid management: development of an evidence-based guideline. Emerg Med J 2011;28:265-268

5. Ashkenazi I, et al. Prehospital management of earthquake casualties buried under rubble. Prehosp Disast Med 2005;20(2):122–133.

6. Sever MS,et al. Management of crush-related injuries after disasters. N Engl J Med.2006 Mar 9;354(10):1052-63.

7. Townsend HS,et al. Tourniquet release: systemic and metabolic effects. Acta Anaesthesiologica Scandanavica 1996; 40: 1234– 7.

8. van der Velde JJ, Iohom G, Serfontein L. Reducing the Potential for Tourniquet Associated Reperfusion Injury. Accepted for Publication, European Journal of Emergency Medicine Aug 2012

Trauma in Pregnancy

When trauma occurs in pregnancy we immediately have two casualties to manage, but as a simple principle, if we manage the mother well, then she will look after the baby, unless there is direct injury to the materno-foetal unit.

Trauma is now the most common cause (46%) of maternal death in the USA, with accidental injury occurring in line with the baseline population. There is a however a disproportionate incidence (8%-17%) of domestic violence in the pregnant population and a 7% increased risk of suicide. Although the initial assessment and management priorities for the resuscitation of the injured pregnant patient are the same as those for other traumatised patients there are anatomical and physical changes that alter the response to injury. The main guiding principle however is that resuscitation of the mother will simultaneously resuscitate the foetus.

Top 10 Causes of Foetal Demise from ANY pathology
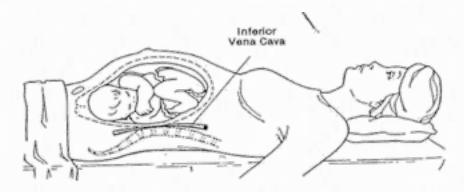
1. Maternal Hypotension
2. High Maternal ISS
3. Motor Vehicle Ejection
4. Maternal Pelvic Fracture
5. Pedestrian Accident
6. Maternal Alcohol Use
7. Young Maternal Age
8. Motorcycle Accident
9. Maternal Smoking
10. Uterine Rupture

Primary Survey

Primary survey should be exactly the same as all trauma patients but in addition:

- Supplemental oxygen is vital
- Aortocaval compression must be avoided at > 24/40 gestation
- up to a 30mm Hg drop in systolic BP and 30% drop in stroke volume.
    - I.e. Don't lie flat !!
- Ensure left lateral tilt or manual uterine displacement to the left
- Hypovolaemia may be present before overt clinical signs of shock develop.
- An assessment of foetal wellbeing and viability is essential.
    - Foetal distress can be an early sign of blood loss prior to any cardiovascular change in the mother
- Involve an obstetrician as soon as possible

Secondary Survey

The secondary survey should include:

- Full obstetric history

- Examination for signs of PV bleeding, ruptured membranes or presence of uterine contractions

- There is a 30% incidence of foeto-maternal haemorrhage (transfusion of foetal blood into maternal circulation) in trauma. All Rh-ve patients should therefore be given Anti-D.

Foetal Assessment

Check for foetal heart rate (FHR) and variability (Normal FHR is 120-160 bpm) and signs of foetal movement. Continuous monitoring of FHR is preferable. The foetus is considered viable when it has a 50% chance of extra-uterine survival. This is unlikely at <24/40 weeks gestation. As a rough guide it is recommended that if uterus is above umbilicus then the foetus should be considered viable.

Trauma in 1st Trimester

Key messages:

- Treat exactly the same as any trauma patient

- Kleihauer's test to estimate foetal leak

- If significant abdominal trauma AND If Rhesus –ve

    - Px Rhesus anti-d immunoglobulin 250 iu if positive (many units now advocating full 500 iu dose).

    - Once stable – Obstetric review

Trauma in 2nd Trimester onwards

Key messages:

- Treat exactly the same as any trauma patient

- Maintain left lateral position, tilt or uterine displacement

- Damage to abdominal organs may be difficult to detect

- Kleihauer's Test to estimate foetal blood leak If significant trauma.

    - Give Rhesus anti-d immunoglobulin 500iu if positive

- Once stable – Obstetric review

- Increased risk of aspiration due to decreased gastric emptying and lax lower

oesophageal sphincter with higher incidence of difficult intubations.

- Avoid over stimulating airway
- Good Cricoid Pressure
- Low threshold for RSI by experienced anaesthetist.

Physiological Changes from 2nd Trimester

The placenta does not possess the ability to auto-regulate its blood flow. Therefore, falls in maternal BP or cardiac output directly affect placental blood supply.

Maternal cardiac output and blood volume increases to 40% above non-pregnant levels by 28 weeks gestation. This hypervolaemic, haemodiluted state is designed to protect the mother during haemorrhage. As a consequence, up to 40% of maternal circulating volume may be lost before the clinical signs of shock become manifest. There is a physiologically low Hb, but this is only due to dilution. Aortocaval compression by the gravid uterus occurs in the supine position and can result in up to a 30% reduction in cardiac output. Note too that woman will be in a relatively hyper-coagulable state, so trauma induced coagulopathy can initially be masked.

Be wary that a raised diaphragm pushes intra-abdominal organs upwards. "Chest Trauma" could be abdominal trauma. (Remember to also place intercostal drains higher than usual). In the same light, be wary of pelvic injuries because of the massively increased placental, uterine blood supply.

There is a steady decline in functional residual capacity (FRC) such that by the second trimester the FRC is reduced by 20%. This results in airway closure during normal tidal volume ventilation in the supine position. This coupled with a 20% increase in resting oxygen requirements means that the parturient patient rapidly desaturates during apnoea. There is an increased risk of acid aspiration in pregnancy secondary to decreased lower oesophageal tone and decreased gastrointestinal motility. For this reason, 'No-desat' high flow nasal prong oxygen is recommended in the major trauma anaesthetic induction, wherever possible.

Uterine Rupture

- 0.6% of all injuries during pregnancy
- Various degrees ranging from serosal hemorrhage to complete avulsion
- 75% of cases involve the fundus
- Maternal mortality 10%

- Usually due to other injuries

- Higher incidence in previous C-Sections

- Foetal mortality approaches 100%

Maternal – Foetal Haemorrhage

- 5x more common in trauma

- Predictor for Intrauterine death

- Risk of Rhesus Disease later pregnancy

- Kleihauer–Betke stain used in diagnosis

    - Maternal blood test

    - Measures amount of foetal Hb transferred into mothers circulation

    - MUST put gestational age on blood form

Amniotic Fluid Emboli

- Profound respiratory failure

- Cardiovascular collapse and arrhythmia

    - Presents like massive PE

- Rapidly develop disseminated intravascular coagulopathy with haemorrhage

- 60% maternal mortality

Preterm Labour

The most common obstetric complication of trauma is the onset of contractions. This results from prostaglandin release from damaged myometrial cells. Most stop spontaneously but may require treatment with tocolytics.

Consider administering slow-released progesterone for all woman with contractions after trauma.

Placental Abruption

Some degree of placental abruption occurs in up to 50% of cases of major trauma and may occur with little or no signs of external injury. Foetal mortality is high at 20-35%. The placenta is devoid of elastic tissue while the myometrium is very elastic predisposing to shearing. Blunt trauma will deform the elastic and flexible myometrium which gets sheared from the relatively inflexible placenta.
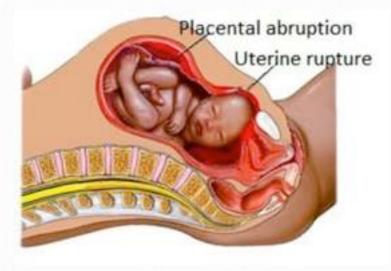
Clinical signs to watch for are PV bleeding, ruptured membranes, uterine tenderness or uterus larger than dates. Diagnosis is usually confirmed by USS. In this it is particularly vital to monitor foetal status.

Direct Abdominal Trauma

The main diagnostic aide is ultrasound scan (USS) followed by CT scan. Diagnos-

tic peritoneal lavage is relatively contra-indicated due to the displacement of the abdominal organs and the risk of damaging the gravid uterus. Blunt abdominal trauma is likely to produce placental abruption whereas penetrating trauma is likely to involve the uterus. If the uterus is penetrated then risk of foetal injury is over 90%.

Electrocution carries >90% mortality risk to the foetus due to its low electrical resistance.

Placental abruption
Uterine rupture

X-rays and Pregnancy

The risk to the foetus from ionising radiation is almost always outweighed by the benefits to the mother. Non-urgent imaging should involve a discussion between a radiologist, a senior member of an appropriate speciality, and the mother if possible.

NORMAL Risk of spontaneous abortion, major malformations, mental retardation and childhood malignancy is 286 per 1,000 deliveries. The foetus is at greatest risk

from radiation at 10-17 weeks of gestation as this is key in neurodevelopment.

Exposure of:

- 0.5 rads adds only 0.17 Malignancy cases per 1,000 deliveries (1 in 6,000)

- Exposure to 1-2 rad increases Leukemia risk from 3.6/1000 to 5/1000

- It takes 50-100 rads to double the baseline mutation rate

The American College of Obstetricians and Gynecologist have stated that exposure to x-rays during a pregnancy is not an indication for therapeutic abortion.

In simple terms the number of studies for dangerous levels of radiation for the developing foetus, assuming all normal lead screening measures, is:

- C-spine   2500

- CXR   70 000

- Pelvis   13

- PFA   20

- L-Spine   125

- CT Brain   100

- CT Chest   50

- CT Abdo   1

Perimortem Cesarean Section

- Over 100 successful cases reported in the literature

- Indicated within 5 minutes of CPR where gestational age >20 weeks

- May improve maternal resuscitation

- 23 weeks gestation survival chance is 0%

- Maternal CPR <5 minutes, foetal survival excellent

- Maternal CPR >20 minutes, foetal survival unlikely but still possible

- One recent review

  - 94 cases from extensive literature search

  - Mean gestation 33/40 (median 35, 10-42)

  - 1/3 occurred pre-hospital

  - ROSC - 60%

  - Mean time to delivery 16 mins (range 1-60)

  - 4/94 cases delivery was in less than 4 minutes

  - Maternal survival 51/94 (54.3%)

    - 40/51 Cerebral performance category 1 or 2 (i.e. Normal or slight deficit)

- Timing relates to outcome (p<0.001)

- Survivors: 10 minutes (1-37, median 9)

- Deaths: 23 minutes (4-60, median 20)

- Foetal survival 42/66 (63.6%)

- Timing relates to outcome (p=0.016)

- Survivors: 14 minutes (1-47, median 10)

- Deaths: 22 minutes (4-60, median 20)

A quick decision is needed. If in doubt JUST DO IT!

7
Environmental Trauma

"A good plan executed now, is better than a perfect plan executed next week"
— *George Patton*

Tactical Medicine

Gun crime in the UK is thankfully rare with the reported rates of gun related homicide reported as 0.04 per 100,000 population in 2010, with 2.4% of all intentional homicides being committed with firearms of any description - amongst the lowest in the world. Despite this however, there have been incidents in living memory where an individual has caused multiple injuries and deaths with firearms that have been legally owned. For example, incidents in Northumbria, Cumbria[and Dunblane to name a few have meant that although rare, the potential still exists for pre- and in-hospital providers in the UK may be called on to deal with shootings as part of their practice.
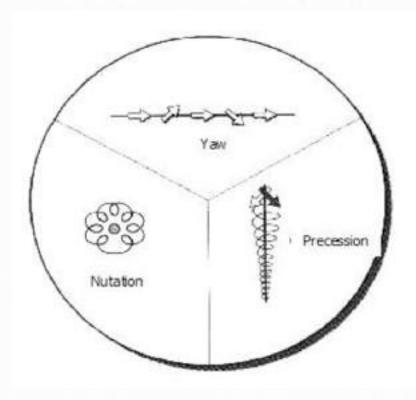
Globally there have been many incidents that have come to press attention such as in Mumbai, Sandy Hook, and Columbine, all of which has emphasised that ballistic trauma can occur in both rich and poor areas, to any age of victim and with seem-

ingly little discrimination. It is for this reason that civilian medical staff must have an understanding of the fundamentals of looking after the ballistically injured as well as their military counterparts.

During times of conflict military personnel may be injured and transported to civilian hospitals as part of their ongoing care and rehabilitation Due to the speed of aeromedical evacuation it is not uncommon for severely injured soldiers to be in an NHS hospital in the UK within 24 hours of being injured and being looked after by a combination of military and civilian staff. The understanding of conflict medicine and ballistics in warfare is not therefore, the exclusive purview of the military doctor, but increasingly is becoming a part of NHS work.

Ballistics – Mechanism of Injury

Injuries from gunshot or blast have always been encountered in peacetime practice as well as during times of conflict. But there is increased potential now that usage of firearms in criminal activity is on the rise, as is the risk of terrorist activity on the British mainland. Body armour is routinely used by the Police forces throughout the UK and in some areas by members of the Ambulance service.

The motion of a bullet in flight after leaving the gun (external ballistics) and behaviour on entering tissues or target (terminal ballistics) depends on:

- Size, shape stability, composition & velocity.

- Density and elasticity of tissues it encounters.

- Degree of deformation of the bullet.

Bullets unlike arrows are aerodynamically unstable and oscillate around their long axis. This is known as Yaw. If allowed to increase it reaches the stage where the bullet will tumble end-over-end. Yaw is reduced by spin stabilisation of the bullet by rifling of the gun barrel.

With a spin stabilised bullet any irregular movements in flight are rapidly damped by the gyroscopic action of spin. This keeps the projectile at the correct attitude and greatly increases range and accuracy. Spin may be as high as 3500 rpm but this is not sufficient to stabilise the missile in any medium other than air. Soft tissues are 800-900 times as dense as air so when a spin stabilised bullet hits tissue the stabilisation will be overcome and it will become acutely unstable. Any yaw present will be increased frequently to the point of end-over-end tumbling.

Energy of Wounding

What really matters is Energy of Wounding! Energy of wounding is dependent on energy deposition during transit of the tissues. This results in a spectrum of injury ranging from low energy transfer wounds to high-energy transfer wounds.

Energy deposition

$$E_d = \frac{1}{2}M[(V_{Encountering})^2 - (V_{Exiting})^2]$$

$$Retardation \propto PresentingArea$$

$$ViscousDrag \propto V^2$$

A stable perforating bullet may only use up 10% of its kinetic energy in wounding as it transits the tissues, whereas a more unstable bullet of the same mass and velocity which tumbles on entering the tissues is subjected to much greater retardation forces and will give up much more of its energy and subsequently create a much more severe wound. The actual wounding effect achieved depends on the total energy that is given up when it strikes tissues and the rate at which the energy transfer takes place. Retardation is directly proportional to the presenting area of the bullet. Retardation is also a function of viscous drag, which is proportional to the square of the velocity. Fragmenting rounds increase their surface area and result in high-energy transfer. All military rounds should have a full metal jacket and should not fragment. Semi-jacketed and hollow point ammunition is officially illegal for military use according to the Hague convention of 1899. These mushroom on impact increasing the cross-sectional area of the missile and the resultant retardation forces. If a bullet is stopped completely by tissue the energy liberated must by definition be equal to the total kinetic energy of the bullet. If it passes out of the tissue with a residual velocity the energy transfer depends on the degree of retardation or slowing of the bullet.

Weapon	Projectile Velocity (metres/second)	Mass (g)	Kinetic Energy (Joules)
Typical Rifle (e.g. AK-47)	710	7.5	1993
Pistol (e.g. 0.357 Magnum)	436	10	970
12 Bore Shotgun	500	9.7 - 28	2-3000 (dependent on number of pellet impacts)
Air Rifle	200-300	0.5 - 1.3	10

Type of Firearm

Most handguns fire relatively heavy bullets at low muzzle velocities. This produces relatively low available kinetic energy for wounding. Modern military rifles have small bullets fired at very high velocity. This may result in either low energy transfer or high-energy transfer depending on factors explained below.

Velocity

Why velocity matters – but not that much! Kinetic energy possessed by a body in motion is proportional to the square of the velocity

$$KE = \frac{1}{2}MV^2$$

Gunshot Injuries

Tissue damage is caused by:

- Simple laceration and crushing

- Shock waves

- Temporary cavitation

Simple laceration and crushing

This is the same mechanism of injury caused by a knife or pointed stick. Tissue is cut, crushed and forced apart by the direct impact of the penetrating bullet. Tissue damage is by direct contact and confined to the bullet track itself. This is unlikely to be lethal unless vital organs or major blood vessel lies in the path of the bullet and is directly injured.

Shock waves

More common with high velocity rounds exceeding the speed of sound in air (approx. 350 m/s). Penetration results in instant compression of tissues and this zone of transient compression moves away through the tissues as a shock wave or stress wave. The velocity of the shock wave in tissue is much the same as the speed of sound in water, about 1500 m/s. Shock wave pressure changes may only last for about a micro second but with peak overpressures in excess of

1500psi they can cause damage at a considerable distance from the permanent wound track. This may cause fractures without direct contact or even spinal cord damage.

Cavitation

Cavitation is confined to high-energy missiles. Energy is transferred from the bullet to the local tissues causing acceleration of tissue away from the bullet track. Tissue moves forwards and outwards leaving a large cavity, which may be fourty times the diameter of the bullet. For examples of cavitation and different bullet impacts, many videos are available on youtube.
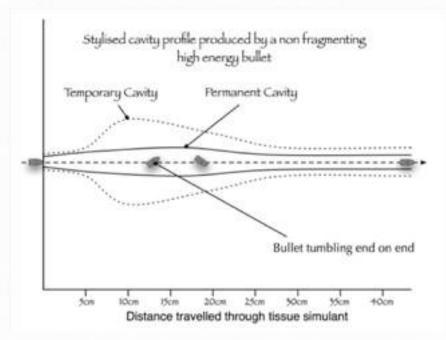
The maximum size of the cavity is reached only after the bullet has passed through. The transfer of energy imparts velocity and momentum to the tissues so the cavity keeps expanding despite containing a sub-atmospheric pressure. The low pressure sucks air and debris into the wound through the entry and exit wounds created by the bullet. This is why knowing if the victim was shot whilst on the ground, in a field or standing etc as it is important to assess the degree of potential contamination. This has been incorporated into treatment algorithms in the UK military of when to prescribe certain antifungal medications for patients involved in IED incidents.

After reaching its maximum size the temporary cavity collapses down to leave the resulting permanent cavity which depending on tissue characteristics may be lined by devitalised tissue or fragments of detached muscle.
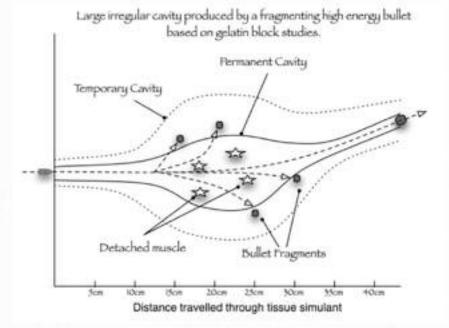
The resulting shape of the cavity depends on the point of maximum energy dissipation and how soon the bullet starts to tumble. There may be small entry and exit wounds with a massive cavity between or cavitation may result in a small entry wound and massive blow out exit wound. There is very little clinical importance of determining which of the two wounds is the entrance and which is the exit - the tract between the two is much more important.

External appearances may be very deceptive

Spheres and most irregular fragments present their maximum area where they strike so the maximum retarding forces (proportional to the presenting area and the square of the velocity) come into play immediately. The maximum rate of energy dump is at the beginning of the wound track so any cavity produced by high velocity fragments reaches its maximum diameter immediately under the skin and decreases in diameter as it goes deeper so the overall cavity has a conical shape.

Mechanism of injury produced by a non-fragmenting bullet

Injuries produced by bullet fragmentation or multiple shotgun pellets

Vulnerability of different tissues

In general damage is directly proportional to density of the tissue. Dense tissue like liver, spleen and brain are very sensitive. Spongy elastic tissue like lung and elastic mobile skin are highly resistant to damage.

Skeletal muscle is intermediate. Bone is dense, inelastic and relatively brittle and therefore very sensitive to cavitation.

Solid Visci – Liver and other solid intra-abdominal viscus

Low velocity, low energy wounds may be serious if they hit blood vessels or bile ducts. In practice bullets from low powered handguns are seen to simply punch holes through liver tissue and may lead to very little leakage of blood or bile and cause no significant shock wave or cavitation.

A High velocity, high-energy missile is likely to be associated with cavity formation and catastrophic disruption of liver substance. This leads to pulping of liver tissue and haemorrhage in most cases is rapidly fatal. Rapid excision of affected lobe may provide slim chance of survival. The liver may be injured indirectly by cavitation or shock wave when a bullet passes through the chest.

Brain

Low powered handgun bullets at close range will commonly penetrate the skull but are usually arrested within. A high velocity rifle bullet creates a significant cavity in the brain and the skull is extensively fractured from within. Brain matter is liquified

and prognosis is appalling. The importance of assessing the best GCS post injury cannot be overstated, as it allows an accurate estimation of the primary brain injury. If a patient is GCS 13-14/15 after being shot, then they need urgent neurological assessment and treatment as any subsequent damage is potentially preventable. Injury due to hypoxia, hypotension and raised intracranial pressure should be avoided at all costs. Bullets that cross the midline of the brain (regardless of calibre) are almost uniformly unrecoverable, inoperable and fatal. Much of the time, neurosurgical options are not targeted at foreign body (i.e. bullet) removal, but on mitigating the effects of being shot. These may include the surgical treatment of bleeding, debridement or in some cases decompression of the skull vault to allow room for swelling without causing a deleterious rise in the ICP. This will be discussed further in the chapter and lectures on head injury.

Thorax

The thorax contains tissues that contrast in vulnerability to missile wounds. It contains air-filled, spongy elastic lungs that do not lend themselves to temporary cavity formation. They are therefore remarkably resistant to damage from high velocity bullets. However, bleeding and subsequent pulmonary contusion can be a cause of morbidity and mortality if not appropriately managed early with good pulmonary toilet and recruitment strategies on ventilation. The heart and great vessels however are filled with fluid and are extremely susceptible to damage from cavitation. Such injuries are usually instantly fatal.

Muscle

Vulnerability of muscle to cavitation is midway between liver and lung. Presence of a cavity may be inferred at surgery by a zone of bruising around the permanent track of the wound. Muscle may be devitalised or actually detached in the volume of the temporary cavity. However depending on the location of the tract, very little in the way of intervention other than a bedside washout may be required. For example for superficial calf wounds, an ankle-brachial pressure index is performed and if found to be >0.8 then no angiographic studies are required. If distal sensation is intact and there are no concerning features for super contamination (e.g. the patient was shot whilst lying on the floor in a dirty field) then simple bedside irrigation may be all that is needed with primary closure. This also depends on the duration of time that has elapsed since the shooting, as delayed closure is rarely indicated. Extensive wounds may need operative intervention, but for simple, low calibre wounds without any

neurovascular or orthopaedic involvement and no retained bullet fragments, expectant management (in some cases on an outpatient basis) may be appropriate.

Gut

Effects can be very variable. Low energy transfer wounds to the gut may cause uncomplicated perforations that are potentially a cause of death from peritonitis but amenable to simple surgical repair. Increasing the velocity of the bullet results in increasing levels of damage to the gut wall with increasing margins of bruising and devitalised tissue. High velocity bullets result in extensive tearing of gut with a wide margin of cell death extending from the edges of the gut wound. The state of the gut at the time of injury i.e. fluid content and gas content modifies cavity formation and shock wave progression. It may be appropriate to leave the abdomen open in heavily contaminated cases that will require multiple washouts. Damage control surgery for haemostasis with or without initial soiling and placement of an abdominal vac dressing are accepted practice in US trauma centres and the UK military. Bowel may be resected and left in discontinuity at the time of the initial operation if the patient is physiologically unstable, and either delayed primary anastomosis or diversion ileostomy or colostomy may be performed at a later date if the patient will not tolerate the procedure at the time of initial operation.

Vessels and nerves

Vessels are relatively elastic and mobile but are filled with fluid. They may be transected or merely displaced. Shock waves may cause intimal disruption and thrombosis in vessels which are externally intact.
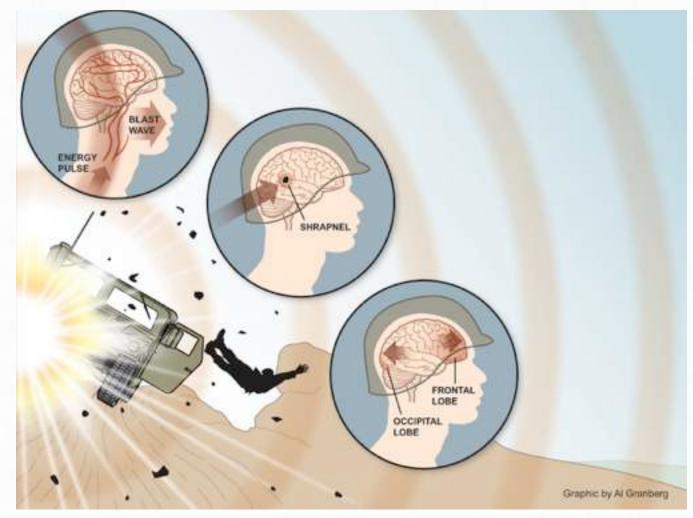
Large nerves are often grossly intact even if they traverse the area in which a large cavity formed containing disrupted tissue. Function may be retained but transient displacement of the nerves may cause stretching sufficient to cause temporary dysfunction or even axonal death.

Blast Injury

Detonation of an explosive device can result in a wide spectrum of injuries to personnel and damage to objects.

Injuries caused by explosions fall into the following categories:

- Primary - shock wave induced
- Secondary - fragmentation particle induced
- Tertiary - blast wind induced
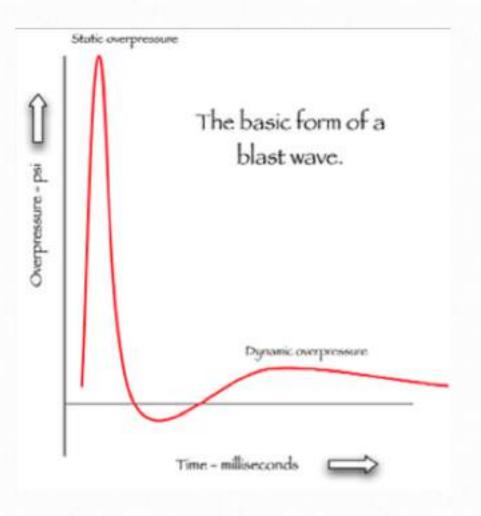
There is also the possibility of burn, crush or psychological injuries distant from the time of the explosion.

Characteristics of an Explosion
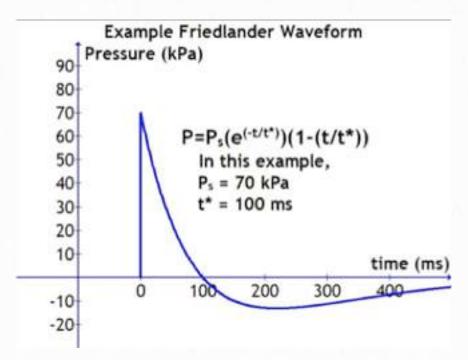
An explosion is essentially a rapidly expanding sphere of high-pressure, high-temperature gaseous products resulting from the rapid chemical decomposition of an explosive compound.

- Blast wave consists of a shock wave and the following blast wind

- Peak overpressure $1/\propto$ distance^3 (i.e. as distance from the blast point doubles, the pressure reduces to 1/8th)

- Has an initial positive overpressure phase followed by a transient negative pressure

Static overpressure

Overpressure - psi

The basic form of a blast wave.

Dynamic overpressure

Time - milliseconds

powerful and transient change in air pressure as it passes. This 'static overpressure' may be damaging or lethal. The outward mass movement of air displaced by the expanding gaseous products of the explosion results in 'blast winds' which follow the shock wave and are responsible for the 'dynamic overpressure' which is exerted on people or objects. The shock wave and dynamic overpressure are frequently described collectively as a 'blast wave' as for most explosions they arrive at a given point nearby at the same time.

The very high initial pressure within this sphere — the 'overpressure', is reached almost instantaneously. This very high initial pressure and the consequent velocity of expansion of this ball of incandescent gas result in the production of a shock wave which travels outward through the surrounding (and as yet still) air at more than the speed of sound. As the sphere expands, the pressure within it falls and the expansion slows and eventually stops. This results in breakaway of the shock wave as it continues through the air at a velocity gradually dropping towards the speed of sound, leaving the expanding fireball and products of combustion and smoke cloud behind. The shock wave is a

Example Friedlander Waveform
Pressure (kPa)

$$P = P_s(e^{(-t/t^*)})(1-(t/t^*))$$

In this example,
$P_s = 70$ kPa
$t^* = 100$ ms

time (ms)

Primary damage

Primary damage from the shock wave depends on the peak pressure generated and the duration of the positive pressure part of the pulse. The higher the peak pressure

and the longer the duration of the positive overpressure produced, the greater its potential for damage. The shock wave may cause distortion of the body or may couple into the body to produce shear and stress waves.

As a shock wave interacts with the substance of the object it strikes internal shock waves are created. These internal shock waves set up differential stresses as they reach interfaces between tissues of different densities with part of the shock wave being reflected and part carrying on. The body is full of such interfaces and these pressure differentials may have destructive effects.

- Ruptured Tympanic membrane

- Primary lung injury

- Gut & Abdominal Visceral Injury

- Traumatic Amputation

- Burns

- Death

Blast wind/blast wave causes bomb casings and nearby objects to become projectiles. In an open area detonation, this is the greatest threat to life. An individual close

enough to the detonation to sustain a primary blast injury (e.g. to the lungs) would be highly likely to have suffered fatal secondary blast injuries. However improved combat body armour design has meant that some people are surviving the secondary injuries, so in certain military populations primary blast injuries are on the increase. The design of new munitions now is aimed at enhancing the primary blast injury and reducing fragmentation for this reason. Also, if explosives are detonated in an enclosed environment such as a building, bus or train then the duration of the peak overpressure is much longer as the shock wave is reflected and amplified by solid structures such as walls. This has been confirmed by data from civilian terrorist attacks where bombings have occurred in confined spaces, which indicate that blast lung is a common feature in this circumstance.

Blast Lung injury

This arises from direct transfer of the shock wave from the surrounding air into the thoracic cavity. Stress waves are set up concentrating within the lung substance. Pressure gradients are set up between capillaries and alveolar air spaces with subsequent disruption of the alveolar/capillary membrane and haemorrhage into the alveolar spaces. Shear waves may

cause shearing of lung tissue off the more rigid bronchi and bronchioles. Further bleeding may occur resulting in a spectrum of damage from minor areas of haemorrhage to major gross haemorrhage causing obstruction of the airways. Widespread haemorrhage and oedema may lead to progressive development of solidified or hepatised (looks like liver) areas of lung in which no gas exchange can take place. Tears in lung tissue and vessels may result in pneumothorax or air embolism. Blast lung may be unilateral or bilateral, and is usually but not exclusively confined to the lung facing the blast. The contusions sustained may progress over 72 hours, so a casualty who may score low on an initial triage sieve/sort may progress to requiring rapid intubation and ventilation for fulminant ARDS. The damage cause by the shock wave (capillary rupture) is worsened by the presence of extravasated blood and free haemoglobin which leads to the formation of free radicals which cause further oxidative damage. This may initiate or amplify a pro-inflammatory response. This combination of physiological shunting due to pulmonary haemorrhage and oedema (primary bast injury) plus an increased metabolic demand (MODS/SIRS) can be fatal.

Abdominal Injury

Blast injuries may occur to gut or solid viscera but are comparatively rare in casualties surviving a blast in a non-confined environment. It is more common in those surviving underwater explosions due to the more powerful transmission of shockwaves in water. The usual picture is one of contusion of air filled segments of small bowel but may in severe cases result in the bowel being perforated. Coupling of the shockwave into the abdomen creates a stress wave, which dissipates energy at the air/fluid interfaces common in the gut. Damage to the more fixed large bowel is by gross displacement of the abdominal wall by blast waves. This creates internal shear waves which result in forcible stretching of tissues.

Shearing effects can cause mesenteric and peritoneal injuries causing degloving and devascularisation including gross avulsion injuries. Characteristically, multifocal haematomas are seen intramurally and extend from the mucosa progressively more externally to involve the serosa, mesentery and vascular supply in confluent haematomas in more severe injuries. Serosal injury at laparotomy should always be considered indicative of transmural injury, and in a series by Cripps in experimental pigs, serosal lesions greater than 15mm in the

small intestine and 20mm in the large bowel were at greatest risk of perforation and should be resected. Delayed perforation up to two weeks post injury is possible and may be indicative of a primary injury worsened by progressive ischaemia. Certainly the use of vasopressors in the ICU setting which will preferentially divert blood away from the gut may worsen an already tenuous blood supply, causing not only mesenteric ischaemia but a potential translocation of gut flora and worsening sepsis.

Perforation of the gut leads to peritonitis secondary to leakage of bowel contents with the presence of severe pain. If contusions only are present the presentation may be more insidious with colicky abdominal pain and possibly bloody stools for several weeks before spontaneous healing occurs. However, late perforation of contused areas may occur up to fourteen days post injury with little in the way of preceding symptoms.

Injuries to solid viscera may be caused by shear waves, but they are more likely to be as a result of secondary or tertiary effects. In patients who survive the primary blast wave in an enclosed environment, bowel injuries are more common than blast lung if the patient survives to hospital admission. This is presumably due to the fact that although the duration of exposure to blast overpressure is longer, the mean pressure is lesser.

Traumatic Amputation

There are differences in the pattern of limb amputation caused by the effects of the shock wave and the blast wind. The shock wave acts at regions of high tissue density and tends to fracture long bones and causes mid shaft amputation of limbs. The blast wind in contrast causes gross displacement of the limbs and avulses limbs at points of weakness tending to cause amputation of limbs at joints. The effects of blast may propagate along tissue planes of least resistance and cause significant tissue damage at a distance from the level of the amputation. Amputations may also be worsened by the effects of dirt and debris being driven up tissue plains causing foci of infection and sepsis. The initial debridement of these wounds should be thorough and cut back to bleeding tissue if the patient's physiological reserve allows in the operating room. Multiple subsequent trips may be required as tissue hypoperfusion and necrosis may progress over the course of the first week post injury, and delivery of oxygen (DO_2) and maximal tissue oxygen extraction (VO_2) may not be complementary.

Secondary injury

This is the result of impact of fragments and debris on the human body. Secondary damage is caused by fragmentation of bomb casing or deliberate inclusion of nails, ball bearings and other debris within or around the device. In addition objects in the vicinity of the device may be accelerated by the blast striking the body and causing serious injury. Deliberate inclusion of faeces and rotting organic matter has also become commonplace in IED's encountered in Afghanistan, so aggressive wound debridement and appropriate antibiotic (and in some cases, antifungal) coverage should be encouraged.

Tertiary Injury

This involves gross displacement of the body by blast winds, which may throw the casualty against solid objects causing injury. Demolition of surrounding structures may also result in crush injury and entrapment.

Thermal Injuries

Caused by hot gases and products of combustion or flash burns close to incendiary type devices. Close proximity to high explosive devices is usually not survivable. Secondary fires started by explosion are the usual cause of significant burns.

Psychological Injury

Post Traumatic Stress is common after incidents involving explosions and may exist in the absence of obvious physical injury as well as being associated with major polytrauma.

Approach

When working with police firearms units or in with the military in relatively austere environments, the acute management of ballistic trauma in the pre-hospital phase is definitely influenced by the tactical environment, and the "gold standard" of medical care may not be possible if there is an active shooter in the vicinity. Good medicine may make for poor tactics and vice versa, so a degree of compromise is needed between appropriate casualty care and tactical/operational objectives, and ensuring the safety of not only the casualty but the rest of the medical team too.

Tactical medicine within the UK is predominantly concerned with working with police firearms units, who operate a three pyramid model of tactical care (see overleaf).

In cases of severe injury, all that may be achievable is to extricate the casualty as quickly as possible from the environment - occasionally the rescue is the medicine and the medicine is the rescue. This is

| Casualty location | Level of care achievable | Most likely provider |

Delivery of tactical level medical care by UK police

highlighted by the limited level of care at the top of the central pyramid. It may be possible to perform a quick intervention, or for the casualty to help themselves (e.g. by self application of a tourniquet or rolling the patient onto their side to maintain airway drainage). This is comparable to battlefield first aid teaching in the military, which stresses that if the injured soldier's patrol is under effective enemy fire, unless the casualty is immediately accessible when the two above interventions may be possible, the priority is to win the firefight before rendering aid. A similar approach is employed by UK firearms units, with the mnemonic TARDIS being used to dictate the initial sequence of events: Tactics, Access the casualty Rapid Diagnosis and InterventionS. Forward care consists of basic airway management and c-spine control if necessary, though if a patient has a significant ballistic injury the neck according to a study by the British military, the likelihood of a significant c-spine injury in survivors is very small due to the immediately catastrophic nature of the injury. The risk to the rescuer of putting the collar on in a hostile environment and also masking other pathology favours not using a collar.

The principles of ballistic kinematics are considered elsewhere in this chapter, and there are other weapons systems that may be used by the police or security services. A brief description of each system and the practical implications of which are discussed below.

Tasers

Tasers have been called electroshock devices that work by causing a current to pass through muscles and cause neuromuscular incapacitation or electromuscular disruption[9]. They are non-lethal weapons which use barbed projectiles connected to a battery

to deliver an electric shock and cause both muscular and sensory stimulation. This can cause both an involuntary muscular spasm which incapacitates the victim, or a painful stimulus which can be used as a compliance aid. The "Drive Stun" mode is a feature which allows a painful shock to be given without a muscular component, and can be used by holding the taser against the target without firing the barbs.

In the initial model, the barbs were gunpowder propelled, whereas current models use compressed nitrogen to fire the projectiles. The effective range of the taser used by US police is 35 feet/10.6 metres[10], though the civilian model has an effective range of 15 feet/4.5 metres[11]. Although designated non-lethal, this refers to the fact that the taser is not designed to kill although there have been some fatalities. Furthermore, a study by the UK Home Office revealed that the electrical current employed by the taser can be a source of ignition in certain circumstances, so should not be used in a flammable environment. This study also showed that the solvent used in CS gas canisters (methyl isobutyl ketone) could also be ignited by tasers when it had been sprayed on clothing[12]. This causes a problem if the Police need to use a taser after CS spray has proved ineffective.

Provided that the barbs themselves do not pierce at-risk tissue (eyes/face, genitals, through and through digital injury), removal of the barbs is simple - they are pulled out in one piece in a swift motion without twisting. There is no indication for a routine 12 lead ECG unless the patient complains of chest pain or has a significant cardiac history.

CS Spray

CS gas otherwise known as pepper spray or tear gas, is another non-lethal agent that can be used to control individuals or disperse a large crowd. The active ingredient is 2-chlorobenzalmalononitrile ($C_{10}H_5ClN_2$) and was discovered in 1928 by Ben Corson and Roger Stoughton (hence the name), and further refined at Porton Down, Wiltshire in the 1950's and 60's and is not technically a gas, but an aerosol. The clinical effects depend on the method of delivery and the concentration and can range from mild tearing and sneezing to vomiting, incapacitation, copious mucus discharge from the nose and bronchospasm. CS spray can cause a burn on the skin where it has been contacted if the patient is sweating, and irrigating the area with hot water can actually worsen this. If a patient has been exposed to CS spray, the correct treatment is to dab or wipe as much as it off as possible in a dry fashion,

and to tepid sponge the eyes with small amounts of cold water. If irrigation is required, a weak alkaline solution of cold water and 5% sodium bisulfite should be used. People exposed to CS spray should be warned not to have a hot shower for up to four hours after exposure as it may reactivate the compound and worsen symptoms.

One interesting point is that the use of CS gas by the military in war is prohibited by the 1997 Chemical Weapons Convention, but its use by civilians remains entirely legal. CS gas is being rapidly replaced with PAVA (pelargonic acid vanillylamide) spray which is a synthetic capsacinoid, the same family of chemicals which make chilies hot. PAVA is significantly more potent than CS spray, but after 15 minutes exposure to warm air the effects are negligible.

Firearms

A useful mnemonic for handing over information about a casualty who has been injured is "ARCTIC"

- Area of hit
- Range
- Cavity affected (chest/abdomen/pelvis/extremity/head/neck)
- Type of weapon used
- Impact protection (none/kevlar/plates etc)
- Condition of the patient.

In the UK, most firearms injuries occur at relatively close range with one study finding that the majority of confrontations occur within a 7 metre range, with 11% of assailant shots hitting their intended targets compared with 25% of rounds fired by police. Shot placement and bullet characteristics are more important than weapon type in dictating their effectiveness.

References

1. Gunpolicy.org. (2014). United Kingdom — Gun Facts, Figures and the Law. Available: http://www.gunpolicy.org/firearms/region/united-kingdom. Last accessed 1st February 2014

2. Wikipedia. (2013). Cumbria shootings. Available: http://en.wikipedia.org/wiki/Cumbria_shootings. Last accessed 1st February 2014.

3. Wikipedia. (2011). 2010 Northumbria Police Manhunt. Available: http://en.wikipedia.org/wiki/2010_Northumbria_Police_manhunt. Last accessed 1st February 2014.

4. Wikipedia. (2014). Dunblane Massacre. Available: http://en.wikipedia.org/wiki/Dunblane_massacre. Last accessed 1st February 2014.

5. Wikipedia. (2013). 2008 Mumbai attacks. Available: http://en.wikipedia.org/wiki/2008_Mumbai_attacks. Last accessed 1st February 2014.

6. Wikipedia. (2014). Sandy Hook Elementary School Shooting. Available: http://en.wikipedia.org/wiki/Sandy_Hook_Elementary_School_shooting. Last accessed 1st February 2014.

7. Wikipedia. (2014). Columbine High School massacre. Available: http://en.wikipedia.org/wiki/Columbine_High_School_massacre. Last accessed 1st February 2014.

8. NHS. (2007). Airlifting the wounded. Available: http://www.nhs.uk/Livewell/Militarymedicine/Pages/Aeromed.aspx. Last accessed 1st February 2014.

9. " Neuromuscular Incapacitation (NMI)", Taser International, published March 12, 2007, accessed May 19, 2007

10. TASER Cartridges (Law Enforcement & Corrections), TASER site. Last accessed 3rd February 2014

11. http://www.taser.com/products/consumers/Pages/TASERCartridges.aspx Retrieved 3rd February 2014

12. http://www.guardian.co.uk/Archive/Article/0,4273,4316213,00.html, last accessed 6th February 2014

13. McPherson JJ (2006) Prevalence of tension pneumothorax in fatally wounded combat casualties J Trauma. 2006 Mar;60(3):573-8

14. Ramasamy A, Midwinter M, Mahoney P, Clasper J. Learning the lessons from

conflict: Pre-hospital cervical spine sta-
bilisation following ballistic neck
trauma. Injury. 2009 Dec;40(12):1342–5

Decompression Illness

Decompression illness is a range of pathologies that result from breathing gases under pressure. The usual victims are therefore SCUBA divers, but occasionally industrial or tunnel (Caisson) workers may suffer the condition. Hyperbaric medicine is a relatively specialised aspect of practice and much of it is beyond the scope of this chapter. There are two key pathologies.

Firstly the absorption of inert gases (usually nitrogen in compressed air diving) under pressure into the bodies tissues and cavities whilst at depth. On ascent to surface if this is too rapid these gases are rapidly released from the tissues and can cause bubbles to form in the tissues and circulation. These bubbles either cause symptoms locally or embolise. If the patient has a patent foramen ovale then the bubbles can cross to the left heart and result in everything from minor skin bends with a torso rash, to the potentially fatal cerebral bends.

Gas bubbles in the tissues and especially the joints produce the classical symptoms of joints pains described as 'the bends.'

Spinal bends are caused by bubbles effecting the blood supply to the spinal cord, pro-

ducing paralysis. Post mortem large areas of bruising have been seen in the cord.

The second pathology is the adverse effect of pressure on air filled cavities such as the lung or middle ear. The most serious is pulmonary barotrauma with gas embolism. If a diver breath-holds while ascending rapidly to the surface, the air within the lungs will expand until the alveoli potentially burst. This causes air emboli, mediastinal emphysema, pneumothorax, and is often fatal.

Managing Decompression Illness

Any patient who becomes unwell within 24hrs of SCUBA diving may have decompression illness and should be assessed by an appropriately qualified doctor – this usually involves contacting a recompression chamber.

Initial treatment is supportive therapy for the ABCs. The patient should be laid flat and as close to 100% oxygen administered. Divers often have their own oxygen therapy equipment with demand valves, and these should be used in preference to standard medical kits until the cylinder is exhausted. This may provide some relief from symptoms, but should not mean that an urgent referral to a hyperbaric unit is not made. There are 24hr emergency contact numbers at all UK medical chambers.

Keep the casualty well hydrated - if necessary intravenous fluids should be administered. Simple analgesics can be given and the patient should be transferred ideally with their cylinder, dive computer and dive log. Urgent contact should be made with a hyperbaric facility. If you do not have the contact details for such a chamber, dial 999 and ask for the coastguard, who will advise.

The treatment involves recompressing the patient back to depth in a hyperbaric chamber, then slowly bleeding them back to surface breathing 100% oxygen, or in severe cases Oxygen-helium mixtures. Symptoms will typically disappear once at pressure, which is a good diagnostic test, unless the bend presents late.

Treatments are usually 90 minutes but can extend significantly if symptoms re-appear on ascent in the chamber. During this time, the Nitrogen gas bubbles are compressed back into solution and are then slowly released and exhaled

NEVER try to re-compress a patient in water at a dive site.

Immersion and Submersion
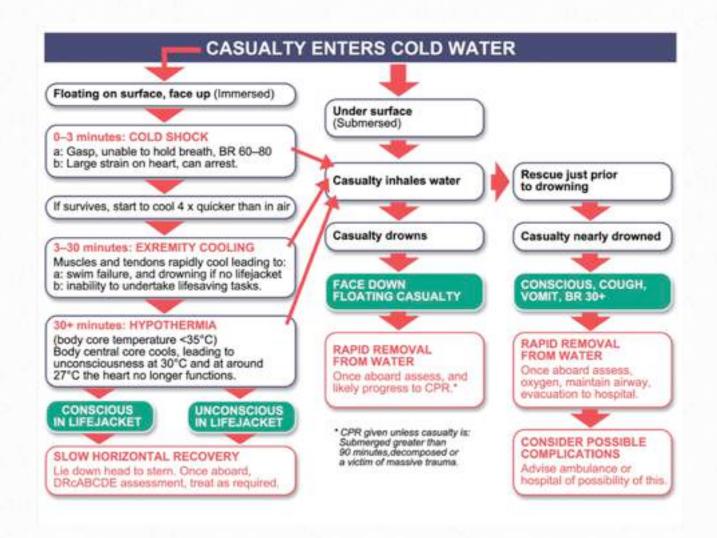
In recent years the International Liaison Committee on Resuscitation (ILCOR) and the World Congress on Drowning have re-defined drowning in order to prevent confusion from such terms as near drowning, dry drowning, secondary drowning etc. and all these terms are now obsolete. It can be seen from this that the major issue is a respiratory one and thus all treatment should be aimed at the alleviation of hypoxia and its sequelae.

Drowning is the process of experiencing respiratory impairment following submersion / immersion in liquid.

Epidemiology

Drowning accounts for > 500,000 deaths worldwide annually. Mortality from drowning in Europe shows two peaks:

- Children <5 years old a

- Adult males >14 years old

CASUALTY ENTERS COLD WATER

Floating on surface, face up (Immersed)

0–3 minutes: COLD SHOCK
a: Gasp, unable to hold breath, BR 60–80
b: Large strain on heart, can arrest.

If survives, start to cool 4 x quicker than in air

3–30 minutes: EXREMITY COOLING
Muscles and tendons rapidly cool leading to:
a: swim failure, and drowning if no lifejacket
b: inability to undertake lifesaving tasks.

30+ minutes: HYPOTHERMIA
(body core temperature <35°C)
Body central core cools, leading to
unconsciousness at 30°C and at around
27°C the heart no longer functions.

CONSCIOUS IN LIFEJACKET   UNCONSCIOUS IN LIFEJACKET

SLOW HORIZONTAL RECOVERY
Lie down head to stern. Once aboard,
DRcABCDE assessment, treat as required.

Under surface (Submersed)

Casualty inhales water

Casualty drowns

FACE DOWN FLOATING CASUALTY

RAPID REMOVAL FROM WATER
Once aboard assess, and
likely progress to CPR.*

* CPR given unless casualty is:
Submerged greater than
90 minutes,decomposed or
a victim of massive trauma.

Rescue just prior to drowning

Casualty nearly drowned

CONSCIOUS, COUGH, VOMIT, BR 30+

RAPID REMOVAL FROM WATER
Once aboard assess,
oxygen, maintain airway,
evacuation to hospital.

CONSIDER POSSIBLE COMPLICATIONS
Advise ambulance or
hospital of possibility of this.

Alcohol and drugs clearly play a significant role in incidents involving the latter. Access to water is one of the biggest risk factors especially among children; unfenced swimming pools, ponds, rivers and ditches being the biggest killers worldwide.

There is an unfortunate association with child abuse in drownings amongst the under-fives. Attending clinicians in these cases should think about intentional drowning or severe neglect.

Pathophysiology

The process of drowning is a less simple one than is imagined. Drowning can be the primary pathology in strait forward submersion, but is often secondary to other physiological processes surrounding cold water immersion.

A fundamental misunderstanding exists around drowning. We know that the whole process usually occurs in seconds to minutes – yet what is puzzling is that:

- 67% of victims were deemed to be good swimmers

- 55% of drownings occur within 3 metres of safety.

To understand these figures we need to understand the physiology associated with cold water immersion.

Immersion vs Submersion

Immersion in this context is defined as the body entering cold water, but then floating on the surface with a clear airway, such as would be achieved wearing a correctly fitted lifejacket. Submersion is defined by the body being kept under the surface of the water, in which case the inability to breathe is obvious.

Simple Immersion

Simply being immersed in water for a prolonged length of time will cause cardiovascular instability:

External hydrostatic pressure causes redistribution of the circulating volume towards the thorax and intracellularly.

- Hormonal responses causes a forced diuresis (up to 2 litres in the first two hours) and profound relative hypovolaemia.

- Water, having a very high specific heat capacity, causes rapid loss of body heat

Rapid Immersion: Cold Shock 0-3 Minutes

Cold shock results from a sudden cooling of a large area of skin following sudden contact with cold water. It occurs in water below 25°C, reaching a maximum effect in water at 10°C or less. It predominantly affects the respiratory and cardiovascular systems via a huge sympathetic nervous system response.

Respiratory system effects are:

- Initial large gasp, which can lead to drowning (the reason a helmet visor is worn by lifeboat crews)

- Rapid respiration (up to an eightfold increase in respiratory rate)

  - as it is uncontrolled it can also lead to drowning if there is any wave splash (another reason for a helmet visor to be worn).

- Inability to hold breath voluntarily

  - if trapped in rising water, unable to hold a breath to effect escape.

- Impaired ability to coordinate breathing with swim stroke leading to even experienced swimmers drowning if not wearing a lifejacket.

Circulatory system effects are:

- Generalised peripheral vasoconstriction, weakening skeletal muscle in limbs

- Increase in venous return leading to pulmonary artery hypertension and significant increase in heart rate.

- Autonomic Conflict resulting in arrhythmias, potentially precipitating cardiac arrest.

- Cerebral hypertension, impairing cerebral function and potentially precipitating CVA.

These cold shock effects account for 56% of open water deaths in the UK within the first three minutes of Immersion. If cold shock leads to cardiac arrest via a stroke, arrhythmia or drowning, the casualty may present as drowned and should be treated as per submersed. However you can expect these patients to have dry lungs with minimal aspiration if the route to arrest was arrhythmic.

Extremity cooling: 3-30 minutes

If a casualty survives the cold shock effects, their extremities (not core) cool rapidly. As extremities cool to the high 20s°C muscle and superficial nerve function deteriorate resulting in swim failure. If no flotation aids are worn, the casualty will quickly be unable to support themselves in water and they will inhale water and then drown.

The small muscles of the hands are also affected quickly so any simple lifesaving tasks are impossible (for example, using lifejacket buckles, firing flares). If extremity cooling leads to drowning and subsequent cardiac arrest the casualty will present as drowned and should be treated as submersed.
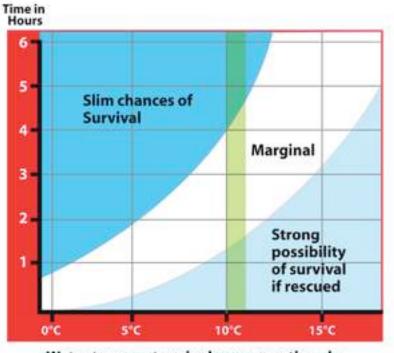
Note, at this stage core temperature is still above 35°C and the casualty is not yet hypothermic.

Hypothermia in Water: 30+ Minutes

Hypothermia starts when the bodies core temperature drops from the norm of 37°C to below 35°C. This is very different to limb temperature, which will have cooled much quicker to well below 30°C by that time through extremity cooling.

In the coldest of coastal UK and ROI waters, due to protective body responses, core hypothermia still takes around 30 minutes to occur. As the core temperature reduces from 35°C, shivering increases in intensity until the body adopts an almost rigid foetal position. Conscious level drops steadily with a fall in temperature resulting in confusion and an inability speak or give a history due to muscle spasm, and as the

core reaches 30°C the casualty will become unconscious.

At this stage if they are not wearing a lifejacket their face will enter the water and they will drown. If they are in a lifejacket and continue to cool, then, as core temperature reaches 28°C the heart enters ventricular fibrillation (VF) and the casualty goes into cardiac arrest.

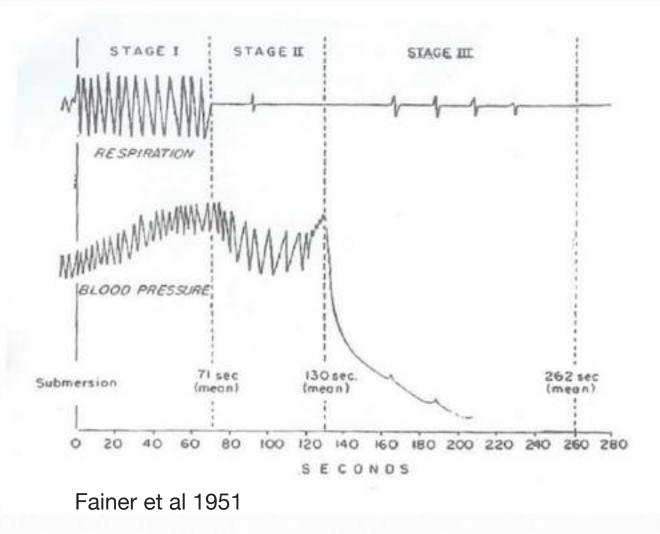The above graph shows the realistic upper limit of survival time for people in the water wearing normal clothing, from time of entry into the water. Water temperatures around the British Isles typically range from 4° - 15° Centigrade (shaded green). The graph indicates the expected survival time of casualties in different water temperatures. This is used by the coast guard as an aid in determining appropriate search durations.

Submersion

If a casualty is not wearing a lifejacket, is a non-swimmer, or is held under the surface for a variety of reasons (for example trapped, full stomach, heavy clothes), initial breath hold will give way to swallowing, coughing then around seventy seconds of uncontrolled respiration leading to mass aspiration.

Salt water will then fill the alveolar space and prevent oxygen transport, whereas fresh water will cross the lung membrane, entering the blood stream and washing out surfactant causing alveolar collapse. Either route will cause hypoxemia rapidly leading to loss of consciousness and apnea. The casualty proceeds to hypoxic cardiac arrest with rhythm disturbance - usually tachycardia followed by bradycardia then PEA and finally asystole. At this point the casualty will usually be found floating face down.

A submersed, drowned casualty should be rapidly removed from the water by any means, assessed and given CPR in line with standard ALS. No CPR is given in extreme trauma, decomposition or proven submersion time of more than 90 minutes.

Fainer et al 1951

Prolonged Submersion and Survival

New evidence has clarified the reasons behind prolonged submersion survival:

- Patients entering freezing (<5°C) water inspire freezing water for seventy seconds prior to drowning.

- Central core temp drops by 7°C during these seventy seconds as respiratory cooling leads to cardiac cooling and selective brain cooling.

- At 30°C body temp, brain survival time doubles to twenty minutes.

- Further cooling occurs due to radiation of heat from the body.

Children and very slight adults cool at a rate of 2.5°C every 10 mins. Larger adults cool much slower due to body fat. Therefore a child or slight adult drops 5°C in the first 20 mins of brain survival time, resulting in a core temp of 25°C by the end of the first 20 minutes. This buys another 20 minutes of brain survival at this point, and that allows further cooling of 5°C. The process continues until about a maximum survival time of 65 minutes.

Adults do not radiate heat well to water, and therefore never really pass the initial 20 minute survival that the first seventy seconds of respiration bring.

It should be noted that coastal waters of the UK never fall below 5°C, and therefore it is now strongly believed that survival times in excess of 30 mins of true submersion at sea are virtually impossible.

Still lakes and lochs do have the potential to drop below 5°C, and therefore survival times for children and slight adults may reach as high as sixty minutes.

Autonomic Conflict

We have already explained that the cold shock response is driven by a huge sympathetic nervous system response triggered by large areas of bare skin in contact with cold water. There is however another autonomic driven response to cold water immersion – this time of the parasympathetic nervous system, called mammalian dive reflex.
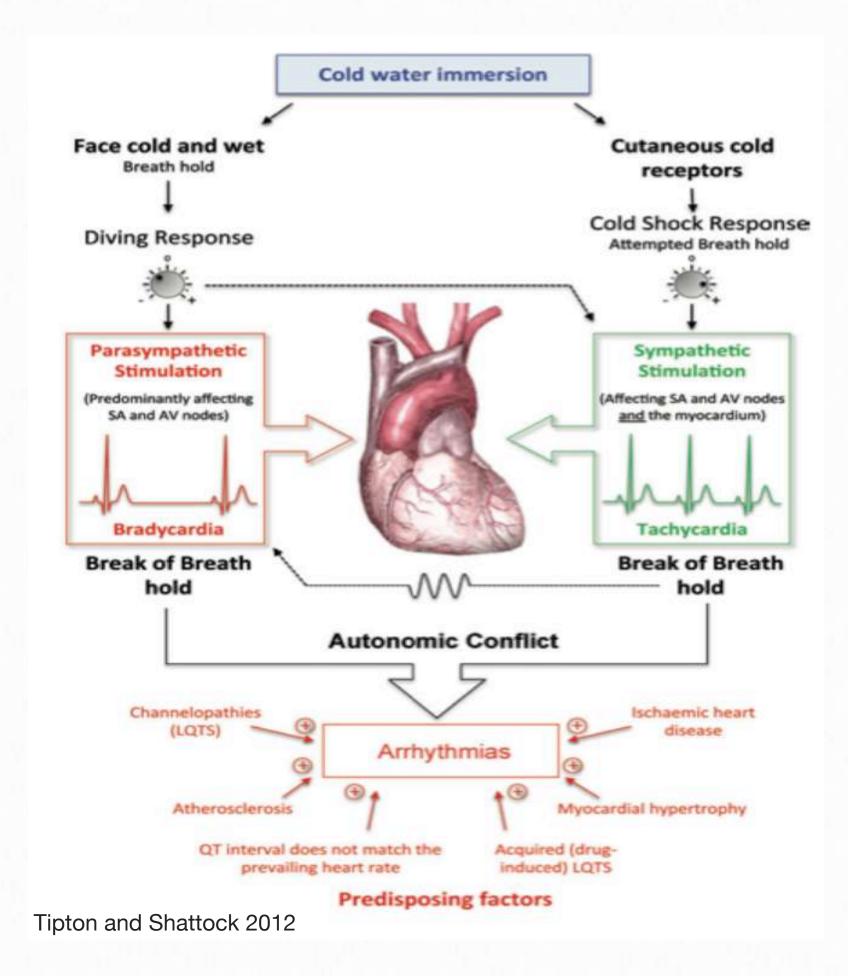
Mammalian Dive Reflex

The mammalian dive reflex is evoked by stimulation of the cold receptors of the face, innervated by Cranial nerves X and XII, driving a large parasympathetic response causing:

- Profound sinus bradycardia driven by excitation of cardiac vagal motor neurones

- Expiratory apnoea ("breath hold") by reflex inhibition of central respiratory neurones

- Excitation of sympathetic vasoconstrictors

- Stimulation of vagal receptors in pharynx and larynx can give rise to similar responses

Very rarely would a situation occur where either cold shock, or the mammalian dive reflex happened in pure isolation, and therefore Prof. Mike Tipton at Portsmouth University has been studying what happens if both reflexes are stimulated together causing an "autonomic conflict".

What has been proven from this work is that when there is both the parasympathetic and sympathetic triggered, a significant arrhythmia is caused at the break of breath hold. Whilst this may be not too catastrophic in young fit individuals, if the casualty happened to have any cardiac predisposing factors, the arrhythmia produced could be fatal.

Cold water immersion

Face cold and wet
Breath hold

Cutaneous cold
receptors

Diving Response

Cold Shock Response
Attempted Breath hold

Parasympathetic
Stimulation

(Predominantly affecting
SA and AV nodes)

Sympathetic
Stimulation

(Affecting SA and AV nodes
and the myocardium)

Bradycardia

Tachycardia

Break of Breath
hold

Break of Breath
hold

Autonomic Conflict

Channelopathies
(LQTS)

Ischaemic heart
disease

Arrhythmias

Atherosclerosis

Myocardial hypertrophy

QT interval does not match the
prevailing heart rate

Acquired (drug-
induced) LQTS

Predisposing factors

Tipton and Shattock 2012

This evolving concept could explain the high death rates in the first three minutes of immersion, previously thought to be purely accountable to cold water shock. However, as arrhythmias are not detectable at post mortem it is hard to prove, but certainly accounts for a significant number of casualties having dry lungs and no signs of drowning.

The Conscious Drowned Patient
(Previous nomenclature for this has included "dry drowning" and "near drowning".)

This describes the casualty that is just on the surface of the water, but may have aspirated wave splash or had a period of brief submersion.

The aspiration of salt or fresh water may cause coughing, spluttering with associated wheezy or crackling noises on auscultation. They may complain of chest pain, vomiting and in serious cases have difficulty breathing with rapid shallow respirations and an associated rapid thread pulse. This can then lead to a reduction in conscious levels.

Pre-hospital management will need to focus on oxygenation and care of the unconscious airway.

Any patient who has been at risk of aspiration must be seen in hospital by a doctor, as the latent effects of this aspiration can cause serious later complications.

Complications of Drowning
(Previous nomenclature for this has included "secondary drowning" or "late onset near drowning".)

Water (especially salt water) irritates the lung tissue causes a localised inflammatory response, causing inflammatory damage to lung membrane leading to pulmonary oedema. The victim may initially appear fine, but may rapidly deteriorate. This usually occurs in the first 12 hours, but can take up to 72 hours. Look out for excessive coughing, chest pain, crackling or wheezy chest sounds.

2-4ml/kg of water in the lungs will impair gas exchange directly and remove surfactant. Water can block the small airways due to surface tension and cause bronchospasm in others. Pulmonary oedema is common from the irritant and osmotic effects of the water (disrupting the basement membrane) and ARDS can often be precipitated.

Casualties who aspirate will develop a large ventilation-perfusion mismatch with as much as 75% of pulmonary blood reported to flow through unventilated lungs (increased shunt). This coupled with impaired gas exchange in ventilated alveoli from pulmonary oedema and inhaled fluid will result in a profound hypoxia.

Much has been made of the differences between fresh and seawater in animal models but it seems to have no appreciable ef-

fect on outcome in humans. There is a reported excess of DIC in cases of freshwater immersion however. Contaminated water can cause either microbiological or chemical injury to the lung parenchyma. Serious thought should be given to any specific complications from immersion in these media.

BiPAP and CPAP have been used to good effect in awake patients with worsening hypoxia following drowning and mortality seems to follow that of other forms of non-cardiogenic pulmonary oedema. If evidence of ARDS is established then the usual protocols and lung protective ventilation strategies should be followed.

Infection

Prophylactic antibiotics are not recommended in all cases though in the case of contaminated water inhalation they do seem to be of some use. Clearly the possibility of an unusual organism being involved is high and early involvement of microbiologists would be advisable. Other interventions such as steroids or have not been evaluated specifically in drowning, but have roles defined in other related conditions.

Rescue from Water

Hydrostatic squeeze is the pressure the sea exerts on the casualty when they are vertical in the water. This pressure maintains a blood pressure within the core. If the casualty is removed vertically from the water the rapid loss of supporting hydrostatic pressure and reintroduction of gravity creates significant effects in the body and instant cardiac arrest can occur.

It is important to remove the casualty from the water horizontally to minimise the potential for collapse. There are a number of ways to achieve this.

Mud rescue

Casualties trapped in mud are subject to the same physiology as casualties immersed in water for a period of time. Therefore once released from the mud they should, ideally, be transported lying down, where practical, especially if trapped to a depth greater than the knees. Concepts based on either crush injuries or suspension trauma are not relevant to the mud environment.

Concomitant injury or medical conditions

Remember that drowning is usually the result of an underlying event or condition that contributes to unintentional immersion or immersion with incapacitation, such as trauma, intoxication, seizure, hypothermia, dysrhythmia, shallow-water blackout, and, for divers, a lost or improper breathing source. It is important to search for and treat any underlying triggering factors or complications.

Thermal Injuries
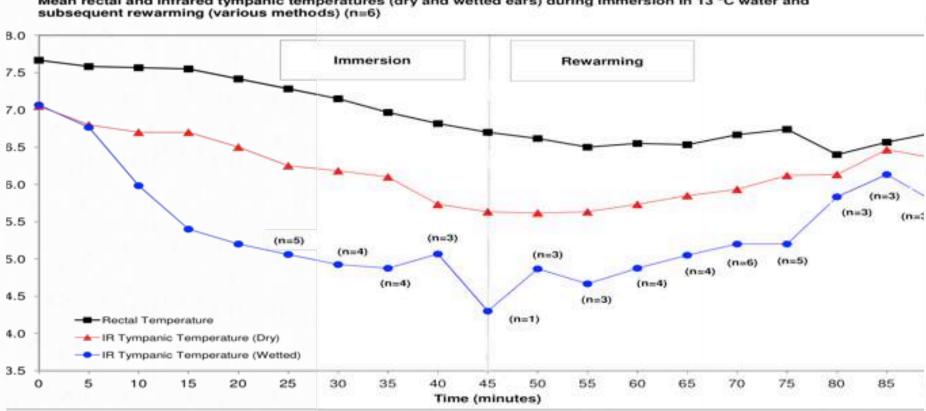
Hypothermia

Hypothermia is defined as a decrease in core body temperature below 35°C. Several factors predispose to accidental hypothermia:

- metabolic disease

- CNS disease

- drug therapy

- psychiatric disorders

- trauma/surgery

- extremes of age and

- alcohol or illicit drug misuse.

Alcohol abuse is the most commonly associated factor. It depresses the sensorium, depresses shivering and promotes vasodilatation. Neonates have a high body surface area to weight ratio and therefore cool quickly. At the other extreme of age, the elderly have a reduced ability to generate heat from metabolism and a higher incidence of concomitant medical and psychiatric illness.

Mean rectal and infrared tympanic temperatures (dry and wetted ears) during immersion in 13 °C water and subsequent rewarming (various methods) (n=6)

Reliability of Thermometers

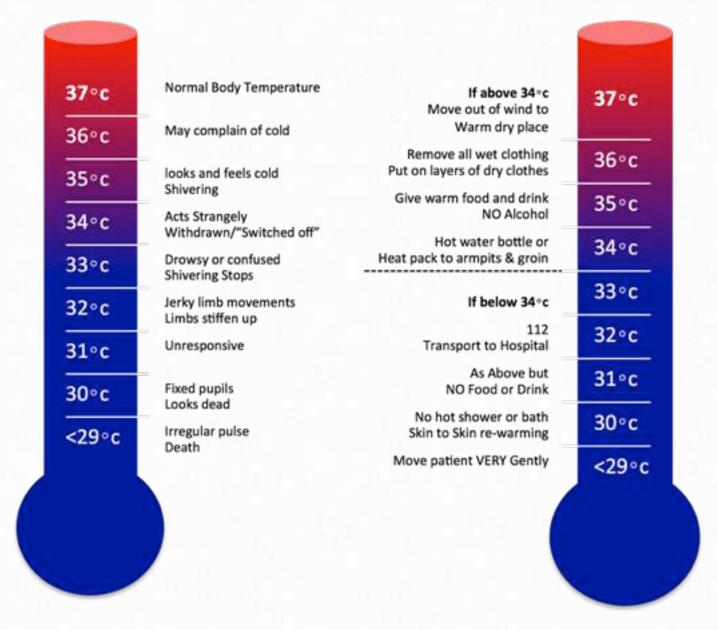Temperature homeostasis is controlled by the hypothalamus. As core body temperature falls below 35°C, the hypothalamus stimulates a sympathetic and catecholamine response resulting in increased cardiac output, increased basal metabolic rate, and peripheral vasoconstriction. Shivering also begins.

As the temperature falls below 32°C, basal metabolic activity falls, shivering stops and cardiac output falls as a result of a fall in myocardial contractility. Myocardial irritability increases and AF is common. Pathognomonic J-waves appear. Peripheral vasoconstriction results in central pooling of blood, which coupled with a temperature induced renal resistance to antidiuretic hormone (ADH) results in a diuresis. Respiratory rate falls (mainly due to decreased metabolic demand) and conscious level begins to deteriorate. The oxygen dissociation curve shifts to the left, further inhibiting oxygen release. Cerebral oxygen demand decreases by 6% for every 1°C fall in temperature. Temperatures below 20°C allow total arrest of brain perfusion for periods of 1 hour without detrimental effect. Consciousness is reliably lost at around 28°C. There is a cold induced depression of platelet function. Ileus is common, as is pancreatitis.

Left thermometer:

Temp	Symptoms
37°c	Normal Body Temperature
36°c	May complain of cold
35°c	looks and feels cold / Shivering
34°c	Acts Strangely / Withdrawn/"Switched off"
33°c	Drowsy or confused / Shivering Stops
32°c	Jerky limb movements / Limbs stiffen up
31°c	Unresponsive
30°c	Fixed pupils / Looks dead
<29°c	Irregular pulse / Death

Right thermometer:

If above 34°c
Move out of wind to Warm dry place

Remove all wet clothing
Put on layers of dry clothes

Give warm food and drink
NO Alcohol

Hot water bottle or
Heat pack to armpits & groin

If below 34°c

112
Transport to Hospital

As Above but
NO Food or Drink

No hot shower or bath
Skin to Skin re-warming

Move patient VERY Gently

Temp
37°c
36°c
35°c
34°c
33°c
32°c
31°c
30°c
<29°c

Ear (tympanic) thermometers are significantly unreliable in cold, wet casualties. Dry cold ears can read 2-3 degrees lower than actual body temperature, and wet cold ears can be more than 6 degrees lower. Therefore these devices should not justify any treatment decisions.
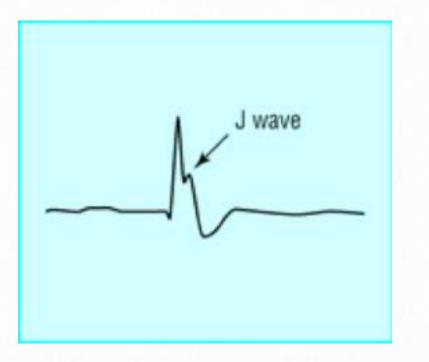
Pre-hospital Care

The neuro- and cardio-protective effects of hypothermia are well demonstrated and active re-warming on scene is not advised. Patients should be moved carefully so as to avoid precipitating life-threatening arrhythmias, as ventricular fibrillation in a grossly hypothermic patient is likely to be resistant to DC or chemical cardioversion.

Insulation is crucial to stop further heat loss. 60% of heat loss is via convection, 40% is via evaporation. Before wrapping in blanket, strip some outer layers of wet clothing off of the hypothermic casualty, but again take care to avoid rough handling. Always transport head down to maximise the amount of blood in the core of the body. Space blankets stop radiation but do nothing for evaporation or convection and have been withdrawn from use in the coast guard. Therefore wrapping in thick blanket and outer windproof cover

and balaclava, whilst looking primitive stops all convection and evaporation and is still our best option. If one is available, place the casualty inside a survival bag.

Investigations in hypothermia
ECG:

- J waves

- Bradycardia and AF (physiological arrhythmias of hypothermia)

- Systole prolonged > diastole

- Increased conduction time > relative refractory period predisposes to re-entrant arrhythmias

- Prolonged PQ, QRS and QT intervals Osborne wave (J wave), most common in II and V3-V6

J wave

Biochemistry:
- Hypo- or hyperkalaemia

- rhabdomyolysis may occur.

- Raised urea and creatinine

- Hypo- or hyperglycaemia

- Raised serum amylase (pancreatitis is common complication)

- Elevated CK

Haematology:
- Increased haematocrit due to intra-vascular volume depletion

- Thrombocytopaenia

- Coagulopathy including DIC is common

Blood Gas Analysis
Hypothermia is protective against hypoxia by shifting the oxyhaemoglobin dissociation curve to the left. Initial respiratory alkalosis is followed by a respiratory and metabolic acidosis. Complex changes related to gas solubility and acid-base physiology occur with hypothermia. When assessing ABGs taken from hypothermic patients, do not temperature correct PCO_2 or pH but aim for normal values in the uncorrected results. Temperature correction is impor-

tant however for PO_2 and SaO_2 evaluation. Thus when examining ABGs from a hypothermic patient, both temperature corrected and uncorrected values should be requested.

General Management
- ABC

    - Intubation as required, as gently as possible

- Warmed, humidified oxygen at 40-46°C

- Remove wet clothing and insulate to prevent further heat loss

- Gentle handling at all times to avoid precipitating dysrhythmias

- Consider co-existent pathology

- IV, Urinary and gastric catheters if necessary for warming.

- Temperature and cardiac monitoring

    - Bladder or rectal temperature probe

- Warmed fluid resuscitation (dehydration is frequently present)

    - Dextrose containing fluids will also provide energy substrate

    - Avoid Ringer's lactate

    - Avoid central lines and Swan-Ganz catheters that may irritate the myocardium

Endogenous Rewarming
- Ideal for mild hypothermia

- A part of all rewarming protocols

- Requires some endogenous thermogenesis

- Warm environment, warm clothing and insulation

Virtually all patients above 32°C will rewarm spontaneously as long as further heat loss is minimised. Blankets, warm air blankets, warmed IV fluids and warmed humidified gasses may be provided. Patients will re-warm at a rate of 1-3°C per hour. Shivering should only be controlled in those patients in whom the extra cardiovascular stress may be contra-indicated (e.g. patients with ischaemic heart disease).

External Exogenous Warming (External application of heat)
- Required at temperatures <32 ° C

- Purported link with peripheral vasodilatation, hypotension and core temperature after-drop

- Immersion is not recommended

- Forced air warming blanket (Bair Hugger)

- Should achieve maximum warming rate of 2°C per hour

Core Exogenous Rewarming

- Application of heat to the core

- Warmed humidified inhaled oxygen

- Warmed IV fluids provide little heat but prevent ongoing loss

- Insulate IV lines

- Blood warmer for blood and IV fluids

- Cardiopulmonary bypass is life saving in arrested hypothermic patients.

- Most other methods are less effective

Arrhythmias

- VF may occur spontaneously or be precipitated by rough handling with temperatures <29° C

- Sinus bradycardia and AF with slow ventricular response are common and should be regarded as physiological arrhythmias of hypothermia

- AF usually reverts spontaneously during rewarming (other atrial arrhythmias should be regarded as innocent)

- Transient ventricular arrhythmias should be ignored

- Magnesium may be effective   (lignocaine appears to be less effective, procainamide should be avoided)

CPR in hypothermia

- Misdiagnosis of cardiac arrest is a hazard in the pre-hospital setting

- Chest wall elasticity and myocardial compliance are reduced and may make chest compression difficult

- CPR rate as for normothermic victims

- Drug metabolism is reduced and accumulation is a big risk

- Adrenaline and other drugs are often withheld until core body temperature is >30°C

- Above 30°C intervals between doses should be doubled and the lowest doses recommended used

- Ventricular fibrillation may not respond to defibrillation if the core temperature is less than 30°C

- If no response to 3 initial shocks, subsequent shocks should be delayed until the core temperature >30°C

- Bypass is better that external rewarming in hypothermic cardiac arrest.

Prognostic Factors in Hypothermia

- There are no strong indicators to predict death or permanent neurological dysfunction in patients with significant hypothermia

- There are no definitive indicators to suggest which patients can or cannot be resuscitated successfully

Parameters that may identify the non-salvageable patient

- Elevated serum potassium >10 mmol/l

- Core temperature <6-7°C

- Core temperature <15°C if there has been no circulation for >2 hours

- Venous pH <6.5

- blood gas measurements should not be corrected for temperature

- Severe coagulopathy

- Clots within the heart on thoracotomy

- Failure to obtain venous return during cardiopulmonary bypass

Cold Extremity Injuries

Injury from cold may be either local or systemic. Local damage includes frost bite and non-freezing cold injury. In both these conditions, the principles of management are to protect the injured part from further damage and to evacuate to hospital. Strong opioid analgesics and ketamine are likely to be required to manage pain.

Passive rewarming should be allowed to occur in the context of definitive rewarming. Rewarming, followed by re-freezing is particularly damaging. This is followed by a surgical assessment of viability. Debridement may be required, but often not immediately. Surprisingly good functional results can occur after months of rehabilitation.

Use passive rewarming. Do not allow to re-freeze.

Heat Exhaustion

A casualty suffering with heat exhaustion will be sweating, thirsty, feeling weak and possibly have a headache, nausea and cramps. Their body temperature will usually be around 38-39°C. There may be a history of some form of over-heating.

Once assessed, the casualty should remain inactive and be told to rest in a cool area and to remove any excess or tight clothing. They should also be encouraged to drink water to rehydrate. Paracetamol may also be offered as this will help reduce some of their symptoms. The casualty should be laid down with their legs raised where they can be assisted to cool down by wetting and fanning. The casualty should improve within 30 minutes and should be advised to see their doctor if at all unsure.

Heat stroke can be fatal so it must be treated seriously and treated rapidly; this is where the body temperature is above 40°C. Casualties may be confused or unconsciousness, with hot but dry skin. Seizures are common. Aggressive active cooling measures needs to be initiated.

Thermal Burns

Thermal burns result from hot fluids, flame or radiated heat. Most fatal burns occur in house fires. Burns are strongly associated with low socioeconomic status, poor housing, drug and alcohol abuse, and mental illness. Incidence of burn injuries has fallen dramatically in the last 30 years, thanks largely to effective public health measures.

Severity is determined by temperature and duration of contact. The time taken for damage to occur decreases exponentially with temperature. Scalds generally involve contact for only a few seconds unless the victim is unable to respond and limit the time of contact. Flame burns involve very high temperatures, and therefore produce the most serious injuries.

Temperature	Time for cellular destruction
44 C	6 hours
54 C	30 seconds
60 C	5 seconds
70 C	1 second

Large burns result in a major systemic inflammatory insult to the body as well as localised injury and scarring. A significant burn (> 10% Total Body Surface Area), leads to a hypermetabolic response, increasing energy and protein requirements

to double their basal level. This leads to weight loss, and a challenge to provide a sufficient calorific requirement for the recovery process.

Associated injuries and problems depend on the circumstances of the burn and may result from:

• Explosion

• Jumping from height to escape

• Carbon Monoxide

• Cyanide and other toxic gases

• Airway or facial burns

• Inhalational injury.

Rescue and Resuscitation are the first phases in a long process of care in major burns. This chapter addresses only these initial steps. The subsequent treatment objectives are the remainder of the so-called 7 R's. Each of which is vital to a good outcome for the patient:

• Rescue

• Resuscitate

• Retrieve

• Resurface

• Rehabilitate

• Reconstruct

• Review.

Assessment

Again, the mechanism of injury - the history of the incident will give clues to likely associated injuries. There should be special reference to the timing and type of burn, any history of being trapped, and unconsciousness at any time. Patient age and co-morbid disease are important parts of past medical history.

The key aspects to determine the severity of a burn are:

• Depth

• Extent – as percentage body surface area (BSA)

• Special areas

• Inhalational Injury

Depth

Superficial (previously 1st degree)

Epidermis only. Erythema. No blistering. Do NOT include this superficial burn in the assessment of a burn area.

Partial thickness (previously 2nd degree)

Dermis damaged. Pink or mottled skin. Blistering. Sensation retained.

Full Thickness (previously 3rd degree)

Epidermis, dermis and perhaps deeper structures damaged. White or charred skin. Painless and leathery.

Extent

As well as appraising severity, assessment of the area of a burn is important in guiding resuscitation. Fluid resuscitation algorithms are based on the patient's weight and the percentage BSA affected.

A quick method, suitable for initial use is serial halving. The principle is that it is easy to visual halves and work from there. For example, a patient with around 75% burns has all of one half (50%) and half of the other half (25%) burnt. A patient with approximately 25% burns has half of one half burnt. This method allows quick assessment and triage.

The rule of nines is another technique. The body is divided into 11 areas of 9% plus the perineum as 1% (Total 100%).
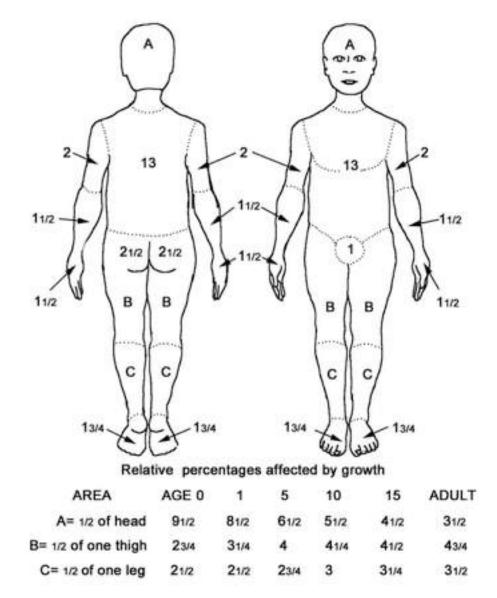
The 11 areas are:

- Head
- Front of chest
- Back of chest
- Front of abdomen
- Back of abdomen
- Left arm
- Left thigh
- Left lower leg
- Right arm
- Right thigh
- Right lower leg

Small areas of burn can be calculated using the patient's palm as a guide approximating 1% BSA. This can be useful also for large areas, by counting unburnt skin and subtracting. Intermediate sized burns will be miscalculated using this method.

The currently accepted best method of estimation is the use of Lund and Browder charts. These charts allow accurate assessment of body areas and have weighting to compensate for the different relative body

TBSA (Total Body Surface Area) chart

Relative percentages affected by growth

AREA	AGE 0	1	5	10	15	ADULT
A= 1/2 of head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2	3 1/2
B= 1/2 of one thigh	2 3/4	3 1/4	4	4 1/4	4 1/2	4 3/4
C= 1/2 of one leg	2 1/2	2 1/2	2 3/4	3	3 1/4	3 1/2

Adapted from Lund and Browder chart approved by Care of Burns in Scotland.

proportions of children. The assessor must not include simple erythema when calculating the area of the burn.

Special areas are the hands, face, feet, perineum as well as the major joints. They may require special management if burnt to obtain the best cosmetic or functional outcome. Some are at high risk of infection and require intensive nursing care.

Do not include simple erythema in estimates of burn area.

Rescue

Scene safety is paramount. Follow the advice of the fire and rescue services. Any burning clothing should be extinguished, and removed unless stuck to the skin. Cooling with water remains appropriate first aid for burns less than 10% BSA. This is to stop the burn and limit continuing damage to tissues.

Victims of major burns should NOT be cooled with water. This can cause hypothermia and worsen shock. Remove clothing and cover with cling film as an analgesic and to retain moisture and heat.

Burn Management

As always, stick to MARCH principles, a number of important considerations unique to burns management.

Airway Considerations in Burns

Facial burns are a marker of possible airway burn, but can result in airway obstruction in their own right from facial or perioral swelling. Hot fat injuries classically can compromise the airway, whereas scalds are rarely severe enough.

The rate of oedema formation is maximal 8 – 12 hours after injury. A patient may have a perfectly adequate airway early on, unless the patient is unconscious, and deteriorate precipitously hours after the injury.

Upper Airway Burns

Usually results from inhaling hot smoke, steam or gases. Being trapped in a burning building is a major risk factor.

Warning signs of airway burns are:

• Drooling

• Stridor

• Hoarseness

• Face or neck burn

• Increased work of breathing

• Carbonaceous sputum or deposits around the nose and mouth

In airway burns, early elective intubation should be performed. Be prepared for a narrow and swollen glottis. Use a bougie on the first attempt, as even gentle attempts to intubate can rapidly obstruct the fragile burnt airway. Have a range of small, uncut tubes available.

Suxamethonium should be avoided later than 24 hours after the initial injury, but could be used early on if necessary. Site a nasogastric feeding tube at the same time to enable early enteral feeding.

Do not cut the endotracheal tube.

Cervical Spine in Burns

The cervical spine may be at risk of injury secondary to the patient jumping to safety or from an explosion. If in doubt, immobilise the neck.

Respiratory consideration in burns

Problems range from carbon monoxide poisoning, wheeze from smoke inhalation and circumferential burns to the chest that may impair breathing. Following explosion, there may be a pneumothorax or pneumomediastinum.

Administer high flow oxygen. Don't be falsely reassured by a high SpO_2 as this reading could be representative of carboxyhaemoglobin or methaemaglobin in some cases. High flow oxygen is the treatment for carbon monoxide poisoning as a higher FiO2 will displace and eliminate carbon monoxide quicker than the 21% O_2 found in room air. Always get a chest X-ray after intubation or if the patient has been in an explosion and is not yet intubated. Circumferential burns may necessitate escharotomies.

An escharotomy is a procedure to release tight burnt skin to allow either swelling of tissue or expansion of the chest to allow ventilation. They are performed by making large cuts through the injured tissue. Escharotomies should be performed by an experienced surgeon in an operating theatre. Bleeding can be extensive and they may need to use diathermy.

Inhalation Injury

Smoke inhalation is the most common cause of death within the first hour following burn injuries. It is injury to the lung parenchyma, usually due to deposition of acidic smoke residues rather than thermal burn, unless super-heated steam or gases are involved. 48 hours after injury, the lungs suffer bronchospasm, increased permeability of the microvasculature and oedema. Surfactant dysfunction, epithelial damage and decreased ciliary clearance leads to airway casts. Secretions from the upper airways move down into the lower airways. ARDS ensues.

These patients often require intubation and ventilation to facilitate pulmonary toilet via a fibre-optic bronchoscope. An adult bronchoscope may fit down a size 7.0mm endotracheal tube, but there will be no room for ventilation during the procedure.

Endeavour to use at least a 7.5mm tube.

Circulatory Considerations in Burns

Burns shock does not occur in the first two hours. If shock is present, look for a source of bleeding. Check the distal circulation in all limbs as circumferential burns may compromise circulation, resulting in compartment syndrome.

Secure good IV access, taking care to insert lines as aseptically as possible. Only if necessary, insert them through burnt skin. Take bloods including group and save. Commence WARMED Hartmann's solution and insert a urinary catheter, aiming for a urine output >= 0.5ml/kg.

An estimate of the extent of burn is now required as a percentage BSA. The Parkland formula for fluid resuscitation is the most commonly used in the UK, and helped reverse a trend of under-resuscitation.

Fluids

The Parkland formula for fluid resuscitation shows that large volumes of fluids are initially required:

First 24 hours fluid requirement (ml) = 4 x %BSA x weight (kg)

So for example, an 80kg man with 20% burns would need 4 x 20 x 80 = 6400ml within the first 24 hours. This would be administered ideally as half the requirement (3.2 litres) within the first 8 hours post injury, and the remainder over the subsequent 16 hours.

In paediatric practice, the fluid calculated by the Parkland Formula is given IN ADDITION to the maintenance fluid requirement.

The use of colloids in resuscitation of the critically ill during the initial phase shows no mortality benefit. Hydroxyethyl starch may increase mortality. Many starches have been withdrawn from the market due largely to adverse renal effects. Physiologically it makes sense not to use colloids within the first 24 hours, because the disproportionate inflammatory response causes leaky capillaries even in healthy tissue, so manipulation of fluid shifts using oncotic pressure is ineffective. After 24 hours, capillary integrity may be adequate in non-burned tissue, but colloids won't reduce oedema formation in the burn itself.

The principle of titrating fluid administration to avoid over or under resuscitation has been upheld since at least 1978. Formulae aside, assessment of the urine output as a marker of end-organ perfusion is a wise course. Although oedema, including airway and pulmonary oedema will be worsened by necessary fluid resuscitation,

you should avoid excessive unnecessary fluid resuscitation. Modern practice is showing a tendency to over-resuscitate the burns patient. This risks abdominal and extremity compartment syndrome as well as causing difficulty with ventilation and weaning, and worsening ileus. Aggressively correcting pre-load in the treatment of burns does not offer a benefit. Obviously each patient should be considered within the context of their comorbidities.

In adults, titrate fluid resuscitation to 0.5ml/kg/hr urine output. In young children, urine output of 1 or 2ml/kg/hr is an appropriate target.

Head Injury/Disability Considerations in Burns

With a reduced level of consciousness, causes to consider are:

- carbon monoxide
- cyanide poisoning
- hypoglycaemia
- post-ictal state
- drug ingestion
- head injury

Exposing Burns Patients

Burnt patients lose the thermal barrier effect of the skin. They cool rapidly and the process is worsened if active cooling with water has been performed. A cooled patient has an additional metabolic load which they may not be able to meet.

Pay meticulous attention to maintaining normothermia. Cover burns in a simple non-adherent dressing (Clingfilm is suitable). Wrap the patient fully in blankets or use active warming devices. Use warmed fluids. Monitor core temperature continuously or at least every 15 minutes.

Do not cool major burns patients.

Carbon Monoxide

A product of incomplete combustion, CO production is a risk of fires in enclosed spaces. Carbon monoxide combines more readily with haemoglobin than oxygen does, thereby dramatically decreasing the oxygen delivering capacity of the blood.

Symptoms include: Headache, nausea, dizziness, fatigue, visual disturbance, convulsions and loss of consciousness.

Have a high index of suspicion from the history. Signs include tachycardia, pyrexia and pallor. Cherry red complexion is rare.

The SpO_2 will read 100% regardless of the level of carbon monoxide. A full co-

oximeter blood gas will include a value for carboxyhaemoglobin (COHb) and should be checked as soon as possible. Normal COHb is up to 3%. Levels above 10% begin to produce symptoms and seizures occur at around 50%.

Treatment is with 100% oxygen, which decreases the half-life of CO from 4 hours to about 90minutes. Hyperbaric oxygen at 2.5 atmospheres further reduces the half-life to 23 minutes. A particular indication for hyperbaric oxygen therapy is significant carbon monoxide poisoning in pregnancy. The foetus will experience carbon monoxide levels up to 15% higher than the mother. However a compression chamber is not always accessible within the acute period.

CO poisoning may be found in conjunction with cyanide poisoning. The symptoms overlap.

Give 100% oxygen

Cyanide

More common in fires at industrial sites, hydrogen cyanide can also be found in residential fires from burning plastics, rubber, wool or silk. It is readily absorbed and can be lethal within seconds. Cyanide prevents oxygen being utilised at the mitochondrial

level by halting the electron transport chain. So despite adequate oxygen, the tissues behave as if they have none at all.

Symptoms include:

- Headache
- Nausea
- Confusion
- chest tightness
- Breathlessness
- Convulsions and
- Loss of consciousness.

Signs of Cyanide poisoning include a low A-V oxygen difference, and a Lactate which remains high despite adequate resuscitation. Cyanide levels are not a routine blood test.

Echo will show severe global myocardial dysfunction.

The specific treatment for cyanide poisoning - hydroxocobalamin, is also known as vitamin B12. It is marketed as Cyanokit. Even high doses are very safe, so treatment should be given early on the basis of suspicion from the history of smoke inhalation particularly in a confined space. The dose is 5g IV over 15 minutes, and can be repeated once if necessary. There are rare

case reports of anaphylaxis to vitamin B12.

Treat with Hydroxocobalamin

Feeding

Early Enteral feeding appears in guidelines for treatment of the critically ill burned patient across the world despite the fact that better mortality outcomes have not yet been proven. Although the hyper-metabolic and catabolic response remains inevitable, early enteral feeding helps reduce ileus and stress ulceration and may attenuate the effects of hyper-metabolism. Parenteral nutrition increases mortality, possibly due to over-feeding. Initiation of Enteral Nutrition within 24 hours makes sense, is safe, and reduces ITU length of stay. Feeding should be high carbohydrate, low fat to decrease the risk of pneumonia. Children particularly benefit from avoiding prolonged fasting. Gastric protection from stress ulceration with omeprazole or ranitidine is mandatory in these patients even if full feeding has been established due to the massive catabolism they experience.

Site a nasogastric feeding tube and feed early

Referral to Burns Centre

Criteria for transfer to a regional burns centre include:

- 10% partial thickness burns in the under 12s or over 60s

- 15% partial thickness burns in 12 – 60 year olds

- 5% full-thickness burns

- Burns to special areas mentioned above

- Circumferential burn

- Inhalational burn

- Chemical, radiation, or high-voltage electrical burns

Chemical Burns

The management of chemical burns is somewhat specialist. There are few chemicals for which adding water will worsen the problem. For example, Lime makes a burning liquid when water is added. Some metal compounds should be diluted using mineral oil. The basic management is:

- Ensure scene safety

  - Fire Service assistance may be required for decontamination.

  - Consider whether you have adequate personal protective equipment.

- Dry powder chemicals should be brushed off first

- Irrigate with COPIOUS amounts of water

  - Use more water than you think.

  - "The solution to pollution is dilution."

- Support ABC

- Give analgesia

- Refer to a burns centre for advice

Electrocution

Electrocution occurs when an electric current passes through the body. The amount of current that flows depends on Ohm's law: $I = \dfrac{V}{R}$

Where:

I = Current
V = Potential difference (voltage) across the body
R = Resistance (or impedance) of the body

As there is a wide variation in the resistance of the human body, there is a wide variation in current flow for any given voltage. The paths of least resistance are often along nerves and blood vessels. Sadly, the human body is most susceptible to frequencies of 50-60Hz, which happens to be the usual commercial frequency. At this frequency, the threshold for perception of current passed hand to hand is around 1 milliampere (mA). If the current rises above 15mA, then the muscles in the forearm contract, and the victim cannot let go of the source of electricity.

Prolonged exposure of 15mA across the chest causes diaphragmatic spasm so the victim cannot breathe. A current of 60mA across the chest for 30 seconds will induce ventricular fibrillation in an adult, whereas a current as low as 30mA can induce VF in a child. Humans can withstand over 10 times more current flow at DC rather than AC. Frequencies above 100Hz are relatively safe. Operating theatre diathermy works at a frequency of 100,000 Hz.

Immediate death can therefore occur from VF or asphyxia. Survivors may have massive soft tissue damage from the passage of current, or may have sustained multiple injuries from being thrown by the shock.

Electrocution - Initial Management

The first priority is to ensure scene safety. High voltage electricity can arc or travel for several feet through the soil.

Use an ABC approach. The passage of electricity though muscle beds can cause

paralysis for up to 30 minutes, so resuscitation may need to be prolonged. VF is commonest after AC shocks, whereas asystole is more common after DC shocks such as lightning strike.

The secondary survey should include a full history from a witness and careful physical examination for electrical entry and exit points, as this may give a clue to the likely path of the current and thereby which internal tissues may be damaged. The amount of soft tissue damage is often much greater than the skin burns would suggest, so large fluid volumes may be required during the initial resuscitation.

Cardiac

Apart from VF and asystole, up to 50% of electrocution victims exhibit ECG changes. These are most commonly non-specific ST segment changes. Most are transient but need to be differentiated from true myocardial damage. Serial ECGs and cardiac troponins should be obtained.

Renal

The massive soft tissue destruction leads to myoglobinuria and potential severe hypovolaemia. Both put the kidneys at risk. The prevention of acute renal failure lies in promoting a good urine output of 1-2ml/kg/min. This requires initial vigorous fluid

resuscitation supplemented by IV mannitol if required (12.5g bolus followed by 12.5g/hour infusion). See crush section.

Neurological

Neurological deficits may occur in either the CNS or peripheral nerves. Immediate effects include loss of consciousness or apparent complete spinal paralysis. In the absence of superimposed mechanical spinal trauma, this invariably resolves over hours or days. Peripheral nerve injuries are the most common permanent sequelae.

Extremities

There may be full thickness burns at the contact points. All non-viable skin should be debrided. The passage of the current through the muscle beds can cause massive soft tissue destruction. The resulting swelling may result in a compartment syndrome requiring urgent fasciotomy. Pain on passive movement is an early sign but the diagnosis should be confirmed by measuring compartmental pressures. A pressure within 30mmHg of the diastolic requires urgent treatment.

See section on Compartment Syndrome.

Thermal Injuries Further Reading

1. Group ALS. The burned or scalded child. In: Samuels M, Wieteska S, editors. Advanced Paediatric Life Support: The Practical Approach. 5th ed: Blackwell Publishing Ltd; 2011. p. 188-98.

2. Brusselaers N, Monstrey S, Vogelaers D, Hoste E, Blot S. Severe burn injury in europe: a systematic review of the incidence, etiology, morbidity, and mortality. Critical Care. 2010;14.

3. Hettiaratchy S, Papini R, Dziewulski P. ABC of Burns (ABC Series): BMJ; 2004.

4. Moritz A, Henriques F. The relative importance of time and surface temperature in the causation of cutaneous burns. Am J Pathol. 1947(23):915 - 41.

5. Miminas D. A critical evaluation of the Lund and Browder chart. Wounds. 2007;3(3):58 - 68.

6. COBIS. Total Body Surface Area Chart. In: COBIS TCAbtpso, editor.: Care of Burns In Scotland; 2009.

7. Keller B, Schub T. Burns, Chemical: Inhalation Injuries -- Smoke and Cyanide. In: Pravikoff D, editor. Ipswich, Massachusetts: EBSCO Publishing; 2012.

8. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database of Systematic Reviews. 2013(2).

9. Bechir M, Puhan M, Neff S, Guggenheim M, Wedler V, Stover J, et al. Early fluid resuscitation with hyperoncotic hydroxyethyl starch 200/0.5 (10%) in severe burn injury. Critical Care. 2010;14(3):r123.

10. Rioux J, Lessard M, De Bortoli B, Roy P, Albert M, Verdant C, et al. Pentastarch 10% (250 kDa/0.45) is an independent risk factor of acute kidney injury following cardiac surgery. Critical Care Medicine. 2009;37:1293-8.

11. Pham TC, L. Gibran, N. American Burn Association Practice Guidelines Burn Shock Resuscitation. Journal of Burn Care and Research. 2008;29(1):257-66.

12. Rousseau A, Losser M, Ichai C, Berger M. ESPEN endorsed recommendations: Nutritional therapy in major burns. Clinical Nutrition. 2013;32(4):497-502.

13. Wasiak J, Cleland H, Jeffery R. Early versus delayed enteral nutrition support for burn injuries. Cochrane Database of Systematic Reviews. 2006(3).

14. Williams F, Branksi L, Jeschke M, Herndon D. What, How, and How Much Should Patients with Burns be Fed? Surgical Clinics of North America. 2011;91(3):609-29.

15. Herndon D, Barrow R, Stein M, Linares H, Rutan T, R. R, et al. Increased mortality with intravenous supplemental feeding in severely burned patients. Journal of Burn Care and Rehabilitation. 1989;10(4):309-13.

16. Jeejebhoy K. Total parenteral nutrition: potion or poison? Am J Clin Nutr. 2001;74(2):160-3.

17. Mosier M, Pham T, Klein M, Gibran N, Arnoldo B, Gamelli R, et al. Early Enteral Nutrition in Burns: Compliance with Guidelines and Associated Outcomes in a Multicenter Study. Journal of Burn Care and Research. 2011;32(1):104-9.

18. Masters B, Aarabi S, Sidhwa F, Wood F. High-carbohydrate, high-protein, low-fat versus low-carbohydrate, high-protein, high-fat enteral feeds for burns. Cochrane Database of Systematic Reviews. 2012(1).

19. NAEMT. Pre-hospital Trauma Life Support. 7th ed: Jones and Bartlett Learning; 2010.