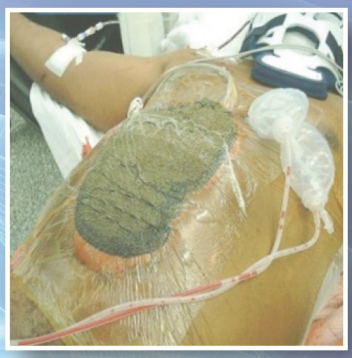
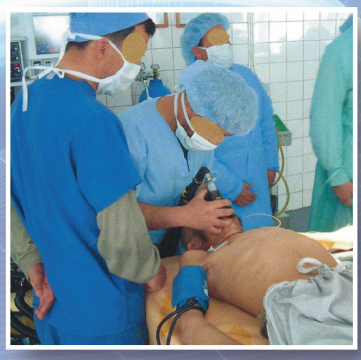
FIFTH EDITION

Manual of Definitive Surgical Trauma Care

incorporating

Definitive Anaesthetic Trauma Care





Kenneth D Boffard

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Manual of Definitive Surgical Trauma Care: Incorporating Definitive Anaesthetic Trauma Care

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Manual of Definitive Surgical Trauma Care: Incorporating Definitive Anaesthetic Trauma Care

FIFTH EDITION

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This manual is dedicated to the six surgeons who, in 1993, saw the need for a course in operative trauma surgery and surgical decision-making for those surgeons who would not routinely be involved in the care of the trauma patient, and from whose foresight the course has been developed.

Howard Champion, Bethesda, Maryland, United States

Stephen Deane, Sydney, Australia

Abe Fingerhut, Poissy, France

Stenn Lennquist, Linkoping, Sweden

David Mulder, Montreal, Canada

Donald Trunkey, Portland, Oregon, United States

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Video Contents

A companion card is included with this copy of *Manual of Definitive Surgical Trauma Care, Fifth Edition*.

To use the card:

1. Insert.
2. Open from My Computer.
3. Click on Index.
4. If asked, 'allow blocked content.'
5. The welcome page will appear, together with a human skeleton.
6. Click on the '+' sign of each area to open files for procedures in that area.

Included on the card:

- Access to the neck
- Access to the anterior mediastinum
- Aorta
- Access to the axilla
- Bleeding control
- Craniotomy
- Fasciotomy
- Heart
- Heart and lung
- Iliac shunting
- Kidney
- Laparotomy
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- Pancreas
- Pelvic packing
- Small bowel
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- Stomach
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- Excision and grafting of major burns
- Application of the Vacuum Dressing

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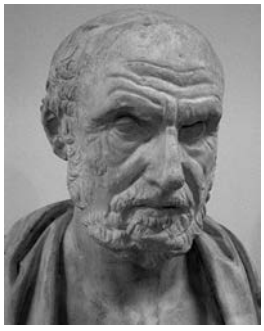
Preface

'He who desires to practice Surgery must go to war'
Corpus Hippocraticum
Hippocrates (460–377 BCE)



'Related to this is the surgery of wounds arising in military service, which concerns the extraction of missiles. In city practice experience of these is but little, for very rarely even in a whole lifetime are there civil or military combats. In fact such things occur most frequently and continuously in armies abroad. Thus, the person intending to practice this kind of surgery must serve in the army and accompany it on expeditions abroad; for in this way he would become experienced in this practice'.

Hippocrates – The Physician, 14, trans. by Paul Potter
Loeb Classical Library, *Hippocrates, Vol. VIII*

Unless dealing with major trauma on a frequent basis, few surgeons, anaesthesiologists or intensive care specialists can attain and sustain the level of skill necessary for decision-making in the care of a patient with multiple injuries. This includes both the intellectual decisions (mind-set), and the manual dexterity (skill-set) required to perform all the manoeuvres for surgical care. These can be particularly challenging, may be infrequently required, yet rapid access to, and control of sites of haemorrhage following trauma can be a life-saving intervention. The correct sequence of the decisions required is critical, and many situations require specialist trauma expertise, but often this is simply not available within the time frame or situation in which it is required.

In years past, many surgeons honed their skills in war, and translated them into the techniques required in peace. In the 21st century, this has changed, so that most surgeons work in an environment of peace, while a few serve in lower key conflicts. In many countries, the incidence of injury, particularly from vehicle-related trauma, has fallen below the numbers recorded since records were first kept. Many injuries are now treated non-operatively, so operative exposure and the skills required are reduced. Occasionally, for this reason, the decision *not* to operate is based on inexperience or insecurity, rather than on good clinical judgement.

It is not enough to be a good operator. The effective practitioner is part of a multidisciplinary team that plans for and is trained to provide the essential medical and surgical response required in the management of the injured patient.

Planning the response requires a clear understanding of:

- The causation including mechanism of injuries occurring within the local population.
- The initial, pre-hospital and emergency department care of the patient.
- The condition in which the patient is delivered to the hospital and subsequently to the operating theatre will be determined by the initial response, which itself may determine outcome.
- The resources, both physical and intellectual within the hospital, and the ability to anticipate and identify the specific problems associated with patients with multiple injuries.
- The limitations in providing specialist expertise within the time frame required.

In 1993, five surgeons (Don Trunkey and Howard Champion, USA; Stephen Deane, Australia; Abe Fingerhut, France; and David Mulder, Canada), all

members of the International Society of Surgery – Société Internationale de Chirugie (ISS–SIC) and the International Association for Trauma Surgery and Intensive Care (IATSIC), met in San Francisco during the meeting of the American College of Surgeons. It was apparent that there was a specific need for further training in the technical aspects of surgical care of the trauma patient, and that routine surgical training was too organ specific or area specific to allow the development of appropriate judgement and decision-making skills in traumatized patients with multiple injuries.

They suggested that a short course focusing on the life-saving surgical techniques and surgical decision-making was required for surgeons, in order to further train the surgeon who dealt with major surgical trauma on an infrequent basis. This course would meet a world-wide need, and would supplement the well-recognized and accepted American College of Surgeon Advanced Trauma Life Support (ATLS®) course. The experience that Sten Lennquist, who joined the group, had gained offering five-day courses for surgeons in Sweden was integrated into the programme development, and prototype courses were offered in Paris, Washington, and Sydney.

At International Surgical Week in Vienna in 1999, IATSIC's members approved a core curriculum and a manual that forms the basis of the Definitive Surgical Trauma Care (DSTC™) course. The manual was first published in 2003 and subsequently in 2007, 2011, 2015, and this fifth edition in 2019. The manual is updated approximately every four years.

Initial Definitive Surgical Trauma Care (DSTC™) courses were then launched in Austria (Graz), Australia (Melbourne and Sydney), and South Africa (Johannesburg). The material presented in these courses has been refined, a system of training developed using professional education expertise, and the result forms the basis of the standardized DSTC™ course that now takes place. The course uses a mixture of education (to modify the 'mind-set' of the participating learners), and training (to modify the 'skill-set' of those learners). A unique feature of the course is that while the principles are standardized, once the course has been established nationally in a country, it can then be modified to suit the needs and circumstances of the environment in which the care takes place. The Education Committee of IATSIC oversees the quality and content of the courses. In addition to the initial 'founding' countries (Australia, Austria, and South Africa), courses have been delivered in more than 32 countries across the world, with the new participants joining the IATSIC programme each year. The course and its manuals are presented in Japanese, French, Hebrew, Portuguese, and Spanish, as well as English. The requirements for the programme can be found in Appendix C of this manual.

By 2014, it was recognized that the indispensable contribution also made by anaesthetic and critical care colleagues has enhanced the approach to trauma, as has the concept of a fully multidisciplinary trauma team. Anaesthesiology, through the enthusiastic inputs of the Anaesthetic Faculty in the Netherlands, Scandinavia, Switzerland, the United Kingdom, and many other countries has in parallel with this course, developed the Definitive Anaesthetic Trauma Care (DATC™) course. We are delighted to incorporate these aspects of care into this manual, and many countries are now presenting a fully integrated course.

This fifth edition had been revised and updated, considering new evidence-based information. The increasing (and occasionally harmful) role of non-operative management (NOM) has been recognized. With the increased need for humanitarian intervention, as well as military peacekeeping, and modern asymmetrical conflicts, each carrying their own spectra of injury, the military module has been substantially updated and broadened to reflect recent conflict experience, and a new expanded section highlighting trauma management under austere conditions has been added.

The Board of Contributors, responsible for this manual, is made up of those who have contributed to global trauma care and the DSTC™ and DATC™ programme, and continues to support and update this manual. I would like to thank them for their very great efforts put into the preparation, editing, dissection, redissection, and assembly of the manual and the course. The keynote chapter (Chapter 1), written by Nigel Tai and Joe Dawson, sets the tone for the manual, and for trauma surgery today. Their efforts, and those of the entire board are greatly appreciated.

The book is divided into sections:

- Trauma system and crew resource management (CRM) communication principles.
- Physiology and the body's response to trauma:
  - Resuscitation physiology.
  - Transfusion.
  - Damage control.
- Chapters on each anatomical area or organ system, divided into both an overview of the problems and pitfalls specific to that system, and the surgical

techniques required to deal with major injury in that area including burns, brain injury, and extremes of age.
- Chapters on modern diagnostic and therapeutic technology:
  - The role of minimally invasive surgery.
  - Imaging.
- Additional modules which cover specific aspects of specialized care:
  - Trauma anaesthesia.
  - Trauma support services.
  - Austere and military conditions.
- Critical care.
- A separate appendix for the use of operating room scrub nurses is included.
- As before, the manual contains all the resources for trauma scoring and injury assessment.

This manual is dedicated to those who care for the injured patient and whose passion is to do it well.

Kenneth D Boffard

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Introduction

In both developed and developing countries, trauma continues to be a major public health problem and financial burden, both in the pre-hospital setting and within the hospital system, claiming 6 million lives every year. In addition to increasing political and social unrest in many countries, and an increasing use of firearms for interpersonal violence, the motor vehicle has become a substantial cause of trauma worldwide. These socioeconomic determinants result in large numbers of injured patients. Injury prevention is a key element in limiting the societal impact of trauma but once injured, effective acute care and rehabilitation are essential for optimal patient outcomes. Improving all aspects of emergency care is important, however improved surgical and resuscitation skills have a particular role in saving lives and minimizing disability.

In most developed countries, there is a limited exposure to the full range of trauma presentations. This means it is difficult to develop and maintain experience in trauma resuscitation and trauma surgery. Standard surgical training is increasingly organ-specific, reducing even further the broad skills required for trauma management. Laparoscopic surgery, microsurgery, robotics, interventional radiographic procedures, and other sophisticated operating techniques may improve outcomes pertaining to elective surgery, but have a negative impact on acquisition of the complex skill-set needed to manage a severely injured trauma patient.

INJURY PREVENTION

Injury prevention can be divided into three parts:

- *Primary prevention*: Education and legislation are used to reduce the incidence of injury, for example, driving under the influence of alcohol.
- *Secondary prevention*: Minimizing the incidence of injury through design, for example, seatbelts, helmets, etc.
- *Tertiary prevention*: Once the injury has occurred, minimizing the effects of that injury by better and earlier care, preferably evidence-based.

Although primary and secondary prevention of injury will undoubtedly play the major role in reducing the incidence of trauma, it will not be eliminated, and therefore there is a need to maintain effective tertiary prevention. This requires training within complex multidisciplinary teams, and a focus on both the decision-making, and the medical and surgical procedures required, for the advanced management of patients with multiple injuries, and correction of any associated deranged physiology.

TRAINING IN THE INITIAL MANAGEMENT OF SEVERE TRAUMA

The Advanced Trauma Life Support® (ATLS®) Course

Promulgated by the American College of Surgeons, this is the most widely accepted trauma programme in the world. It has been in use for nearly 40 years, and with over 60 national programmes involved, more than 1 million physicians have been trained. Its focus is on the initial management of the severely injured patient and addresses those injuries and their consequences that can cause death within the first hour after injury.

The National Trauma Management Course (NTMC™)

IATSIC has developed the NTMC™ as part of an initiative towards improving trauma care in resource-challenged countries, where medical expertise may be available, but the area is resource poor. Based on the ATLS® course, modified for local conditions, and very

cost effective, the course can be offered either as an isolated course organized by IATSIC, or under the banner of a local trauma organization. To date, some 7000 physicians have been trained under the aegis of organizations such as the Academy of Traumatology of India (www.indiatrauma.org), The College of Surgeons of Sri Lanka (www.lankasurgeons.org), and IATSIC itself, in some 12 countries worldwide.

Surgical Trauma Training Beyond Initial Care

Globally, injury is the third leading cause of death for all ages, and the leading cause of death from age 1 to 44 years. More than 50% of all deaths occur minutes after injury, and most immediate deaths are due to massive haemorrhage or neurological injury. Autopsy data demonstrate that central nervous system injuries account for 50%–70% of all injury deaths, and haemorrhage accounts for 15%–30%. It is within this latter group of haemorrhage-related deaths where prompt decision-making and effective use of surgical techniques has the greatest opportunity to save lives.

With improving pre-hospital care across the world, patients who would previously have died are reaching the hospital alive. In many situations, their airway and ventilation are controlled, but the deaths occur in hospital from uncontrollable bleeding. While there are surgical techniques for the control of bleeding, the timing and appropriateness of their use and a clear understanding of the physiology of trauma are essential for a successful outcome.

There is a further problem in caring for the injured in a military context. Modern conflicts are in general asymmetric (with only one side in uniform), are generally local and well-contained, and do not produce casualties in large numbers nor on a frequent basis. For this reason, it is difficult to maintain the number of military specialists required, who can be deployed immediately to perform highly technical surgical or resuscitative procedures in the battlefield arena or under austere conditions. It is also difficult for career military surgeons to gain adequate exposure to battlefield casualties, or indeed penetrating trauma in general, and many military training programmes are now looking to their civilian counterparts for assistance.

The statistics mandate that surgical teams responsible for the management of injured patients, whether military or civilian, are skilled in the assessment, diagnosis and operative and resuscitative management of life-threatening injuries. There remains a poorly developed appreciation of the potential impact that timely and appropriate surgical intervention can have on the outcome of a severely injured patient. Partly through lack of exposure, difficulty in time availability or release from hospital duties, and partly because of other interests, many specialists quite simply no longer have the expertise to deal with such life-threatening situations.

There is thus an increasing need to provide training in the skills and techniques necessary to resuscitate and manage seriously injured patients surgically, not only in the emergency department, but also during the period *after* initial care is complete. A course is needed and must be flexible so that it meets the local needs of the country in which it is being taught.

Surgical Training Courses in Trauma

THE ADVANCED TRAUMA OPERATIVE MANAGEMENT (ATOM®) COURSE

This American College of Surgeons course was originally developed by Lenworth M. Jacobs about 15 years ago and is a one-day course comprising a didactic lecture series followed by exercises on live, tissue models. It is an effective method of increasing surgical competence and confidence in the operative management of penetrating injuries to the chest and abdomen.

THE ADVANCED SURGICAL SKILLS FOR EXPOSURE IN TRAUMA (ASSET®) COURSE

Another programme developed by the American College of Surgeons. It is a one-day cadaver-based course designed to teach the anatomical exposures necessary for control of haemorrhage in the trunk, neck, extremities, and junctional areas.

THE DEFINITIVE SURGICAL TRAUMA CARE™ (DSTC) COURSE

This course was developed in 1993, through an international collaboration of six surgeons, and is controlled by the International Association for Trauma Surgery and Intensive Care (IATSIC), the Integrated Society of the International Society of Surgery – Société Internationale de Chirugie (ISS–SIC) in Zurich, Switzerland. It comprises a three-day course with short interactive presentations, group discussions, case discussions, and operative exercises on a live tissue model. The emphasis teaches learners both the critical decision-making processes

required through advanced education (modification of mind-set), and training in the surgical techniques required (modification of skill-set) to choose the best method of management. Currently, the course has taken place in 32 countries, and in several languages (English, French, Hebrew, Japanese, Portuguese, and Spanish).

The Definitive Anaesthetic Trauma Care (DATC™) course was established in 2006 as pre-deployment training for military anaesthesiologists. It developed as an add-on to the DSTC™ course to enhance understanding of trauma management. In 2015, the DSTC™ was introduced into IATSIC as a subgroup of DSTC™. Cooperation between the two specialities allows the complex teamwork required in the management of a major trauma patient to be simulated and practised. The integration of DATC™ into the DSTC™ course programme and including the aspects of Critical Care required highlights the importance of modern-day trauma management techniques with the focus on the multidisciplinary nature of trauma care by a trauma team. The current DATC™/DSTC™ courses having participants from interventional radiology, medical and surgical specialities as well as nursing scrub staff, thus making the course unique in its team approach. Details of the course appear in Appendix C of this manual.

THE DSTC™ COURSE

Course Objectives

By the end of the course, the participant has:

- Enhanced knowledge of the surgical physiology of the trauma patient
- Enhanced resuscitation and surgical decision-making capabilities in trauma
- Enhanced surgical expertise in the techniques for the management of major trauma
- An improved awareness of the treatment possibilities in major trauma and their evidence base.

THE DATC™ COURSE

Course Objectives

By the end of the course, the participant has:

- An enhanced knowledge of trauma surgery decision-making and the procedures involved.
- An enhanced knowledge of the physiological abnormalities associate with trauma and the management before, during, and after surgery.
- An enhanced knowledge of trauma induced coagulopathy and its management.
- Enhanced technical skills needed to expedite the surgical and critical care process.

Description of the Course

A prerequisite of the DSTC™ and DATC™ courses is a complete understanding of all the principles outlined in a general surgical training, and the ATLS® course. For this reason, there are no presentations on the basic principles of trauma surgery, nor the initial resuscitation of the patient with major injuries.

The course consists of a core curriculum, designed to be an activity lasting at least two and one-half days. In addition to the core curriculum there are a variety of add-on modules that can be used to enhance the course, thereby adapting to local needs.

The course consists of several core components:

- Interactive presentations – designed to introduce and cover the key concepts of surgical resuscitation, the end points and an overview of the best access to organ systems.
- Cadaver sessions (optional session) – in which use is made of fresh or preserved human cadavers and dissected tissue. These are used to reinforce the vital knowledge of human anatomy related to access in major trauma. Other alternatives are available if local custom or legislation does not permit the use of such laboratories.
- Skills laboratories with use of live tissue. The instructor introduces various injuries. The objects of the exercise are to both improve psychomotor skills and teach new techniques for the preservation of organs and the control of haemorrhage. This creates the real-world scenario of managing a severely injured patient in the operating room.
- Case presentations – this component is a strategic thinking session illustrated by case presentations. Different cases are presented that allow free discussion between the students and the instructors. These cases are designed to put the didactic and psychomotor skills that have been learned into the context of real patient management scenarios.

SUMMARY

The course is designed to prepare the relatively fully-trained surgeon to manage difficult injuries that might present to a major trauma centre. The combined DSTC™/DATC™ courses provide a higher level of trauma understanding by focusing on the multidisciplinary nature of the decision-making processes and the core concepts of teamwork in managing patients with severely compromised physiology. The course fulfils the educational, cognitive, and psychomotor needs for surgeons and anaesthetists, be they mature or trainee, civilian or military, all of whom need to be comfortable in dealing with life-threatening penetrating and blunt injury, irrespective of whether it is in the military or the civilian arena.

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Professor Kenneth D Boffard is Professor of Surgery and Trauma Director at Milpark Hospital, Johannesburg, and until recently, Head of the Department of Surgery at Johannesburg Hospital and the University of the Witwatersrand. He was previously Head of the Johannesburg Hospital Trauma Unit. He qualified in Johannesburg, and trained in surgery at the Birmingham Accident Hospital and Guy's Hospital.

He is the Secretary-General, and a previous President of the International Society of Surgery (ISS), President of the International Association for Trauma Surgery and Intensive Care (IATSIC), and Chair of the IATSIC Education Committee. He is a fellow of five surgical colleges, and has received honorary fellowships from the American College of Surgeons, Royal College of Surgeons of Thailand, College of Surgeons of Sri Lanka, the Japanese Association for the Surgery of Trauma, and the Association of Surgeons of Great Britain and Ireland.

His passion is surgical education, and various aspects of trauma resuscitation, intensive care, and regional planning of trauma systems. His interests include flying (he is a licensed fixed wing and helicopter pilot), scuba diving, and aeromedical care. His research interests include coagulation, haemostasis, and critical bleeding.

He is a colonel in the South African Military Health Service.

He is a Freeman of the City of London by redemption, and an elected Liveryman of the Guild of Air Pilots of London.

He is married with two children.

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Part 1

Trauma system and communication principles

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Safe and Sustainable Trauma Care 1

1.1 INTRODUCTION

In terms of hazard, the injured patient faces *'double jeopardy'*: the risks to their health owing to the traumatic insult to tissue and physiology; and the risk posed by the therapy required to restore health. Minimizing the potential for iatrogenic harm through the provision of safe care is especially challenging in trauma owing to the complexity and urgency of major trauma as a disease. Factors such as injury severity, acute physiological derangement, temporal urgency limiting diagnosis, or physiological stabilization, a multitude of definitive treatment options and interdisciplinary specialist interactions, can combine to compound the risk of something going wrong.

Despite this, safer and better trauma care is certainly achievable by focusing on reducing the risk of harm to an already injured patient from both the *injury* and the *treatment*. Reducing the risk that the original injury poses to the patient involves both technical and systematic elements. The technical aspects include pre-hospital and emergency care, imaging, surgery, interventional radiology, and post-operative critical care, and are considered in detail elsewhere in this manual.

Treatment of major trauma is a multifaceted endeavour, set within a complicated system that comprises an almost infinite range of interconnecting 'moving parts'. The difficult question of how to improve safety in trauma care may be simplified by taking a hierarchical approach, focusing initially on the individuals and teams that provide trauma care, and then adopting a more strategic perspective concerning hospital institutions, regional and national trauma networks and governance, and finally a consideration of trauma care on an international level.

The question of safe trauma care also requires an examination of the sustainability of trauma care within the workforce and training, and the role of innovative simulation models, research and innovation, translation from military experience as means to ensure that healthcare professionals working with trauma patients can continue to offer the very best care available, and the use of reliable existing data. The aim of this chapter is to iterate the minimal essential components of individual, hospital, and system practice in order to deliver safe care.

1.2 SAFE TRAUMA CARE

1.2.1 Individual Factors

It is accepted that in order to practise safe surgery in the trauma setting, the trauma surgeon and trauma anaesthesiologist must have undergone a validated general training pathway culminating in exposure to a period of specific trauma training. Domain knowledge and technical skills represent the foundational aspects, but by themselves are insufficient. Professionalism is also characterized by rigorous adherence to personal safety (personal protection, sharps, needle-stick, vaccinations), consistent use of the World Health Organisation Safe Surgery Checklist (see Section 1.2.6), and compliance with continuing medical education imperatives. However, the reality is that a significant amount of trauma care in the world is delivered by individuals who may not have had the requisite training nor resources required.

Over the past decade, the importance of non-technical skills (see also Chapter 2) has become increasingly well recognized. The nomenclature for such skills differs from sector to sector (e.g. medicine = *non-technical skills*; aviation = *crew resource management (CRM) skills;* social science = *interpersonal skills*; psychology = *emotional intelligence*; US Army = *soft skills*), but the competencies are broadly the same: teamwork, communication, leadership, decision-making, conflict resolution, assertiveness, management of stress and fatigue, workload management, prioritization of tasks, and situational awareness.[1]

Consistent delivery of non-technical skills is very important in minimizing error, as very few preventable trauma deaths are attributable to purely technical mistakes.

1.2.1.1 HEURISTICS AND COGNITIVE BIASES

Inherent bias in cognition affects perception; such biases are a universal feature of human decision-making and result from default to heuristics; numerous, unconscious mental shortcuts that allow the brain to arrive at quick, but approximate, conclusions with limited information. Such heuristics are advantageous (and thus highly conserved in evolutionary terms) when time or resource pressure demands a quick solution or judgment under conditions of uncertainty or limited knowledge in dynamic, complex or dangerous situations.[2] These cognitive short-cuts can help us manage:

- Information overload – quickly filter and skim data for importance.
- Lack of meaning – fill in the gaps if data is lacking and map it to existing mental model.
- The need for swift action – survival and success can depend on decisions without time for deep analysis.
- The need to remember – helps decide what new information to remember and what can be forgotten.[2]

However, heuristic processes are a double-edged sword. Filtering out information regarded as useless may also discard information that is important. Assessment without all available information may cause false assumptions to be made and false narratives to be laid down. Rapidity of decision-making increases risk of error and relying on experience does not always map on to the present, particularly if post-hoc processing leads to overly-optimistic interpretation of the success of previous strategy and thus reinforcement error.

There are numerous classes of cognitive bias, and some are highly relevant for trauma decision-makers to understand:

- *Confirmation bias*: This is the tendency to interpret data in a way that confirms pre-existing belief. It can act to combat information overload and fit data to pre-existing diagnoses. However, aspects of the case that contradict pre-existing beliefs are not acknowledged as meaningful or are dismissed.
- *Confabulation bias*: In attempting to deal with scarcity of data in order to combat lack of meaning, generalizations lead to false narratives and inappropriate assumptions.
- *Overconfidence effect*: Unreflective practice without objective review of performance leads to overly-optimistic assessment of capability.

1.2.1.2 INDIVIDUAL – LEADERSHIP

The leadership function within a trauma team encompasses prioritization of competing injuries; swift planning, coordination, and orchestration of clinical assessment and intervention mediated by effective communication; assessment of patient trajectory and dynamic appraisal of response to treatment and motivation/supervision of team members (such that individual/team performance is lifted). However, investing all these functions within one individual (i.e. The classical model of 'Leader-Follower') may not be ideal for trauma practice settings. Expertise is distributed among various members of the trauma team and the leadership function may flux between different team members according to the phase of trauma resuscitation and the need to respond dynamically to changes in patient condition (the 'Hierarchical-but-Fluid' Model).[3] Leadership demands accurate assessment of the situation and the ability to continually monitor progress and reappraise the array of options for each decision-node – a characteristic formulated as the '3D trauma surgeon' by Hirshberg and Mattox.[4] Elements that the 3D surgeon should be able to deliver include:

- Tactics – technical aspects of the operation.
- Strategy – the 'big picture' appreciation of the risks that the patient faces immediately and in the near- and medium-term elements of operation.
- Team – clear communication to coordinate efforts to ensure working towards same goals.

Mishandling the team dimension during a trauma operation is one of the worst mistakes you can make

1.2.1.3 TRAUMA TEAM

Trauma[3] and theatre teams[5] face several challenges in performing to a consistent level. Perhaps the greatest of these is that it is rarely the same team that manages the patient: shift work introduces different leaders, different specialists and different levels of expertise. Furthermore,

beyond the effect of staff rotas there is staff turnover, with new team members assigned only while their rotation lasts. Given the fact that they may face the requirement to deliver critical resuscitative functions within moments of meeting each other, there is ample opportunity for error owing to inappropriate skill mix, unfamiliarity with each other's style or even names, with significant potential for excess morbidity and mortality.[5]

Dysfunctional teams are usually obvious to external observers but not necessarily so from within, when poor behaviours (lack of communication leading to failure to establish shared mental model) may become habitual and normalized. Sharing clinical information amongst trauma team members has been shown in two test scenarios to be as low as 27%,[6] and two-thirds of serious medical errors result from lapses in communication.[7]

Conversely, high performing teams talk to each other and are safer. They demonstrate the following behaviours:[5,7]

- Situational awareness (SA) – seeking behaviour.
- Clear leadership + followership with the facility to model adaptive behaviours when needed with appropriate distribution of workload and monitoring/support of team members.
- Closed-loop communication (seeking confirmation that intended messages are understood by recipient) with clear means of escalating urgency (standardized prompts), and facilitation of calm assertiveness.
- Low gradient or flat hierarchy of communication such that team members are empowered to speak up and relay concerns as they see fit.
- Readiness to participate in an open team debrief in order to review performance, learn why things went well or less well, and adopt change if required.

1.2.1.4 TEAM – TRAINING

Simulation training has been shown to improve teamwork behaviour and performance[5] including performance in the operating theatre[8,9] and that of Emergency Department (ED) trauma teams,[10] ATLS courses,[11] and amongst physicians and nurses rotating through a trauma centre,[12] with evidence supporting a significant reduction in surgical mortality following such team training.[13,14] Specific courses designed to deliver non-technical competencies (situational awareness, decision-making, communication, teamwork, and leadership) are increasingly available (e.g. The Non-Technical Skills for Surgeons (NOTSS) course[15] – see also Chapter 2), and contain lessons derived from other safety-critical industries such as in aviation,[16] where failure may lead to catastrophe. These courses tend to provide maps of how errors occur (e.g. the well understood 'Swiss Cheese' Model[5]) whilst teaching communication skills, assertiveness, decision-making under pressure, and appropriate leadership. The Interpersonal Competence Training – run by the German Society for Orthopaedics and Trauma in conjunction with Lufthansa Flight Training[16] – teaches the edicts of crew resource management (CRM), as does the US Department of Defense 'TeamSTEPPS' programme.[17]

1.2.2 Institutional Factors

1.2.2.1 DEDICATED TRAUMA SERVICE

A dedicated trauma admitting team is responsible for the poly-trauma patient from admission to discharge. This involves acute and ongoing in-patient trauma care, daily ward rounds, and identification of ongoing care needs, liaison with other surgical and non-surgical services, safe discharge, and follow-up.

The extent to which a consultant's role is explicitly defined within this framework tends to be ill-defined with variable degrees of formal training and definition of the competencies required to lead such a team. Ongoing care does not automatically map to an individual surgical specialty, and could be any appropriately trained surgeon, emergency medicine consultant, or clinician from another acute specialty.[18] Where such roles and responsibilities are formalized, mortality and preventable death rates can be positively impacted. At the Royal London Hospital in the UK, the implementation of a dedicated trauma service (in-patient) team was associated with a reduction in mortality in severely injured patients by 48% and a reduction in preventable deaths from 9% to 2%.[19] At the same time, St George Hospital in Sydney Australia reported similar results with a reduction in deaths from 20% to 12%.[20]

1.2.3 Performance Improvement Activities

Institutional performance improvement or quality improvement activity encompasses:

i. The identification of preventable death and contributory factors.[1] (Such peer-review endeavour may be more effective than use of Trauma and Injury Severity Score (TRISS) for the identification of potentially-preventable death.[21])

ii. Tracking of trends via long-term mortality monitoring. (Which allows for institution of corrective action plans and is associated with improvement in patient outcome in level 1 trauma centres.[22,23])
iii. Improvement in patient pathways, development of evidence-based standard operating procedures designed to reduce variation of care, teamworking, decision-making, and inter-professional dynamics.[21]

One of the most effective methods to improve patient safety at a hospital level is a robust, respectful, and constructive mortality and morbidity meeting. These meetings should **never** be used to humiliate or apportion blame! The purpose of these meetings is to review and discuss all trauma management errors and to peer review all trauma deaths. Stratification of cause of death or severe complications into 'anticipated' (not-preventable), and 'unanticipated but without room for improvement' (potentially-preventable), and 'unanticipated with room for improvement' (preventable), allows further in-depth discussion where issues are discussed, action plans made, and most importantly, implemented and audited. Areas for improvement are often related to resuscitation issues,[1,21,22] airway management,[1] massive transfusion,[22] pelvic fracture management,[22] venous thrombo-embolism (VTE) prophylaxis[1], and missed injuries.[1] Other common themes included timely interventional radiology and surgical intervention, prompt spine clearance, reduced time to computerised tomography (CT) scan, reduced dwell time in the ED, and neurosurgery management.[21] Within this framework 'near-misses' are just as important to discuss, as deaths.[1,21,22] In the USA, this process is required for trauma centre verification, and in the English major trauma system such activity is required to be formally resourced and evidenced during major trauma centre (MTC) peer review in order to retain designated status.

1.2.4 Regional Activities

Safety propagates through, and is marked by, a collaborative approach between providers of trauma care within a set geographical area. Whilst severely injured patients are 15% to 20% less likely to die if admitted to a designated trauma centre,[24] not all trauma systems are the same. An 'inclusive' trauma system is one in which all trauma-care facilities (pre-hospital, local district hospitals, trauma centres, and rehabilitation hospitals) are incorporated at a regional level to provide continuity of care to the entire population by matching injured patients to appropriate facilities, thereby ensuring system-wide efficient use of available resources.[25] Inclusive trauma systems have been shown to reduce mortality across the world as the concentration of experience and expertise improves outcomes.

This contrasts with 'exclusive' trauma systems that are institution-based, focusing exclusively on designated trauma centres.

As previously discussed at an institutional level, ongoing audit and governance of processes ensures the highest possible outcomes and this is no different on the larger scale of trauma networks. Most trauma networks have in place governance frameworks such as those described within the NHS in London,[25] and Australian states such as South Australia.[26]

Remote and rural communities are particularly vulnerable as the risk of death from trauma in remote areas is four times higher than that in a major city.[27] Strong regional trauma systems can thus have additional benefits over and above expected impacts in rural communities. The unique healthcare requirements that these areas need has driven the introduction of sub-specialty surgical training in some countries that are characterized by vast areas and sparse populations, such as Australia (rural surgery) and Scotland (remote and rural surgery). Although such training is characteristically general, a significant component includes the initial management of trauma.

1.2.5 National Activities

The benefit of audit and quality improvement initiatives can be followed from the team level all the way up to the national level, for example, the NHS Major Trauma Review in which several recommendations were made at each level of the trauma system, including network improvements, pre-hospital care, reception and resuscitation, definitive care, and rehabilitation.[28]

National trauma registries such as the National Trauma Database (NTDB) of the American College of Surgeons, TARN (Trauma Audit and Research Network) in the UK, and the Australian Trauma Registry as part of AusTQIP (Australian Trauma Quality Improvement Program[29]) involve the collection of data of trauma patients including mechanism of injury, injuries sustained, including injury severity scores, treatments received, and outcomes. Such national registries are

valuable for research, audit and peer-comparison, and institution of national quality improvement initiatives.

Surgical colleges have a vital role in trauma care by raising awareness, community advocacy, injury prevention, providing position papers, and delivering education. The three-point lap belt was invented in Sweden in 1958, by Nils Bohlin, a Swedish engineer and inventor working for Volvo, and a year later was fitted in all Volvo cars from. The 1970s saw the first car seat belt legislation introduced in Victoria, Australia and Sweden. Other initiatives include cycle helmet and drink-driving counter measures. Courses such as ATLS®, and DSTC™ enable healthcare workers involved in trauma care to develop and practice skills in a safe environment.

Several non-government, non-profit organizations exist to audit, research, educate, and implement national initiatives in injury prevention. As road traffic collisions comprise the majority of trauma in Australasia and Western Europe, the majority are based on road safety. These include the Australasian College of Road Safety, the Australian Road Safety Conference, and Sweden's impressive 'Vision Zero' campaign, which led to a drop in traffic fatalities of 30% since its inception in 1997, despite a significant increase in traffic volume during the same period.[30] The Vision Zero initiative has since spread globally, including the USA and Canada.

1.2.6 Global Activities

A massive disparity exists in the likelihood of survival of a person sustaining a life-threatening but salvageable injury in a low-income country (36% mortality) compared to a high-income country (6%).[31] Consequently, 90% of trauma deaths occur in low- and middle-income countries and are the leading cause of death globally, killing more people than HIV, malaria, and TB combined. Road traffic accidents are the eighth leading cause of death globally, and international meetings such as the World Innovation Summit for Health (WISH) Forum for Road Traffic and Trauma Care focus on the global impact of such trauma, a large majority of which occur in low- and middle-income countries.[32] The excess burden of trauma mortality shouldered by the developing world is a stark fact that has been recognized for decades; in 2004, following the Essential Trauma Care (EsTC) Project, the world Health Organisation (WHO) produced *Guidelines for Essential Trauma Care*, a set of minimum standards for worldwide trauma care.[31] These were based on low-cost improvements that are achievable in virtually every setting across the globe. In 2008, WHO produced the Safe Surgery Checklist, and whilst not specific to trauma, certainly has application and utility. Initial uncontrolled studies demonstrated reduced mortality associated with its use in both low- and high-income countries.[33] Further rigorous studies (randomized with controls) replicated these initial findings with mortality odds ratios dropping from 1.16 without the checklist to 0.4 with the checklist.[33] In addition, there is evidence that this low-fidelity process can also reduce complications with a relative risk reduction of 0.42 associated with the checklist and a number needed to treat of 12 to prevent major morbidity.[33]

With the success of the standard WHO checklist now firmly embedded into routine surgical practice, WHO produced a Trauma Care Checklist which has subsequently been tested in 11 centres around the world, nine of these in low- and middle-income countries.[34] The 18-point checklist covers history, examination, investigations, and monitoring, and improved the processes measured and may improve outcomes.[34]

Charitable organizations can play an important global role, particularly in trauma education. 'ATLS-like' courses in developing countries. Examples include the University of Maryland running the Sequential Trauma Education Programme (STEPS) course in Egypt,[35] and the largest programme for developing countries is the IATSIC National Trauma Management Course (NTMC™), across India, Sri Lanka, and 12 other developing countries (see also Introduction: Management of severe Trauma). Safe Surgery 2020 is a collaboration of foundations, non-profit organizations, educational institutions, and local governments with the aim of making surgery safe, affordable, and accessible across the world. Currently working in Ethiopia and Tanzania they are developing local surgical leaders and scaling programs that directly address local challenges.[36]

Delivery of safe trauma care in the model generated by high-income countries is typified by complexity and expense. In developing countries with more limited resources, the focus needs to be on strengthening local processes preferentially, by using existing resources to ensure solutions are locally relevant. For example, one study in the most landmine-infested province of Northwest Cambodia looked at the result of systemically training local, non-graduate care providers (non-doctors) in trauma care. They found that 150-hours of training over a 4-year training period improved the quality of trauma surgery in rural hospitals.

1.3 SUSTAINABLE TRAUMA CARE

1.3.1 Workforce Development

Safe delivery of trauma care requires a trained healthcare workforce, but in many countries, trauma surgery is not recognized as a surgical specialty within its own right, with a lack of formalized training pathways to produce trauma consultants. As such, the delivery of trauma surgery in many countries relies on small groups of enthusiasts. In addition, in many high-income countries, there is a reduction in trauma surgery exposure due in part to increasing elective sub-specialization, increasing non-operative management, and interventional radiology management, associated with a reduction in working hours. In this environment, where previously the general surgeon had usually acted as a holistic provider of trauma care, this role is eroding, again in part due to a rise in super-specialist training in elective disciplines.[37,38] Recently the 'acute care surgeon' has emerged, a general surgeon dedicated to the management of acute surgical illness, including trauma. This role has developed for different reasons in different countries, including the need to recover a generalist approach to emergency surgical care, dispossession of such territory by super specialists, and the move to increase surgical operating time by trauma surgeons deprived of suitable case loads owing to the rise of non-operative management. Advocates of the acute care surgeon model argue that such training programmes can deliver the knowledge base, technical skills, and scope of practice required to deliver major trauma care,[39] and evidence from the USA supports the model.[40]

For trainee surgeons there are several opportunities that can be pursued in order to address deficiencies in standard training programmes. These include informal fellowships at high volume centres (typically USA and South Africa), dedicated Trauma/Critical Care Fellowships in the USA and the Resuscitative Trauma Surgeon pilot scheme in the UK,[38] military deployment, simulation training, and specific trauma courses. Development of specific technical competence can be facilitated through simulation and advanced human patient simulators have been developed to allow simulation training in trauma skills ranging from low-fidelity rigs to highly sophisticated mannequins. Examples include 'Trauma Man' and 'Synman' (intercostal drain insertion, pericardiocentesis, peritoneal lavage, cricothyroidotomy, and tracheostomy), 'Air-Man' (airway complications), 'VIRGIL' (chest trauma), and 'UltraSim' (FAST scan training).[7] The novel use of cadavers, such as a pulsatile cadaveric model (perfused cadaver), allows very realistic training in cardiac penetrating injuries, lung lacerations, liver and retrohepatic vena cava injuries, cricothyroidotomy, tracheostomy, open fractures, and carotid and extremity vessel injuries.[41] Simulation is a key component of the burgeoning suite of courses that exist to develop trauma surgery skills and teamwork utilizing mannequins, simulators, cadavers, and animals. Simulation can also be used to evaluate competency, to refine leadership and teamwork skills, and may enhance patient safety through facilitation of insight and rapid feedback and reinforcement of correct skills. Simulation training appears effective, although the long-term retention of skills acquired through simulation is unproven.[7] However, simulation is still inadequate when it comes to mimicking physiological dynamic changes, coagulation, haemostasis, packing, and anatomical reconstruction. The mere fact that it is a simulation removes some credibility. The more sophisticated a simulator, often the more expensive it is, making expensive courses even more expensive, and sometimes unaffordable.

1.4 CONCLUSION

There are numerous opportunities for safer trauma care at every level. At the individual and team level, this is predominantly in the reduction of human error by understanding cognitive biases and improving communication and teamwork. At the institutional level, scrupulous audit and peer review identifies not only individual and team errors, but more importantly institutional systemic failures and ensures constant performance improvement. At a regional level, the implementation and governance of trauma networks ensures the best trauma care to a whole population or geographical area, taking into consideration its individual requirements and needs. Injury prevention initiatives at a national level have been shown to have tremendous impact on mortality, and finally, to redress the imbalance of trauma outcomes between low- and high-income countries, numerous initiatives are in place.

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2 Communication and Non-Technical Skills for Surgeons (NOTSS) in Major Trauma: The Role of Crew Resource Management (CRM)

2.1 OVERVIEW

The recognition that human factors, and not mechanical failure, were a recurring theme in many aviation disasters led to an increased focus on non-technical skill training in the training programme of pilots, referred to in aviation as crew resource management (CRM).

Other high-risk industries subsequently followed suit and 30 years ago the medical community adopted CRM as a component in the training of critical clinical situations. The subsequent years have seen implementation of CRM in numerous surgical and medical subspecialties, of which the setting of traumatology has been no exception. Trauma team training based upon the principles of CRM has become an integral part of the daily practice in trauma centres all over the world.

The principles of CRM are built upon a concept of training in human factors with the aim of optimizing communication dynamics in the setting of the multidisciplinary team. The goal is to reduce medical errors, and improve decision-making and outcomes in trauma care.

The most important aspects of CRM include:

- Situational awareness.
- Preparation and planning.
- Calling for help early.
- Effective leadership.
- Allocating attention wisely and use of all available resources.
- Prioritizing and distributing the workload.
- Communicating effectively.

The complex task of managing a severely injured trauma patient requires both highly specialized surgical and medical skills, as well as an ability to master the core concepts of CRM. It is mandatory for the entire trauma team to participate in training on the CRM principles.

2.1.1 The 'Swiss Cheese' Theory

Described initially in 2001, by James T. Reason, a British psychologist at the University of Manchester, the model has become the standard for assessing patient security in order to expose the failure of system such as medical mishap. and has been used by the healthcare industry, emergency services organizations, aviation industry, and safety industry since it was developed. It is also known as the cumulative act effect.[1,2] Reason's Swiss cheese model has become the dominant paradigm for analyzing medical errors and patient safety incidents.

In a complicated system, prevention of hazards is done by analysis of a chain of barriers. All barriers contain unplanned holes or weaknesses; hence the likeness to Swiss cheese. The holes in the Swiss cheese model randomly close and open owing to inconsistent weaknesses. The defence of an organization against the failure are represented like barriers in slices of Swiss cheese, and individual weaknesses are shown by the holes in the slices as part of the system; all holes are different in position and size in those slices. The hazard reaches the patient only when all the holes simultaneously align.

In most of the cases, there can be four levels of failure for an accident:

- Unsafe supervision.
- Unsafe act.
- Organizational influence.
- Preconditions for unsafe acts.

The failure of the system occurs when holes in slices simultaneously align in aggregate, giving permission, as James Reason called 'a trajectory of accident opportunity', so that in all the defences, jeopardy passes through all the holes, which causes failure.

2.2 COMMUNICATION IN THE TRAUMA SETTING

For trauma care to be effective, there needs to be open communication between all members of the trauma team. At the centre of this process are the surgeon and the anaesthetist, who in collaboration have the responsibility of prioritizing interventions and management. The team leader, be it the surgeon or anaesthetist, must keep the entire team constantly orientated regarding the management of a patient whose physiology and subsequent treatment plans are under constant evolution. The delivery of optimal care in a trauma patient undergoing damage control surgery is a complex process whereby a group of individuals need to function as one unit with all members being heard (listened to) and their individual skills utilized to deliver the most appropriate medical care.

Human factors are vital to the timely assessment and treatment of the complex trauma patient.[3] In the stressful and sometimes highly charged environment of the trauma bay or in the theatre, surgeons and anaesthetists can be tempted to focus on immediate individual tasks with the potential of developing what is commonly termed 'tunnel vision'. This can lead to a situation where management and focus on single problems is prioritized instead of addressing other life-threatening issues, thereby leading to loss of control of the situation. The loss of situational awareness described and the evolution of fixation errors may prove fatal to the outcome of the severely injured trauma patient. In trauma care, as in other disciplines, inadequate communication, poor teamwork, and lack of leadership have been shown to have a profound impact on patient outcomes.

2.2.1 Initial Handover

The handover is a critical phase during patient management; many medical errors arise here and thus it has been the focus of intense evaluation as to which strategies should be optimized. It is important that the handover is conducted in silence, unless there is an imminent threat to the patient's survival. The most commonly-used acronym, MIST, is used to describe in a short and concise manner the salient aspects of a traumatized patient:

- **M**echanism of injury.
- **I**njuries sustained.
- **S**igns and symptoms.
- **T**reatment.

2.2.2 Resuscitation and Ongoing Management

Communication during the resuscitation and ongoing management of the unstable trauma patient, from an operational perspective, can be described in four phases:

- Initial decision-making process prior to resuscitation in the emergency department.
- Before commencement of surgery.
- Re-evaluation during surgery.
- Completion of surgery, and transfer to the critical care environment.

Prior to patient arrival, it is pertinent to establish what has been termed 'zero-point survey' (Cliff Reid, unpublished 2017). This enables the team to survey and optimize their clinical environment and assign roles prior to engaging in clinical/non-clinical care. The information gleaned from the incoming pre-hospital services form the backbone for this survey.

The critical decision-making process is typically started in the emergency department/trauma bay and the surgeon together with the anaesthetist agree on a management plan for resuscitation, damage control surgery, and critical care.[4] At this early stage, evaluation of futility of care is also a legitimate consideration.

In the operating room, or shortly before the commencement of surgery, the surgeon states the surgical plan based on the clinical, laboratory, and imaging findings (if present). The anaesthesiologist summarizes the physiological status of the patient including blood volume/transfusion status, the presence of coagulopathy

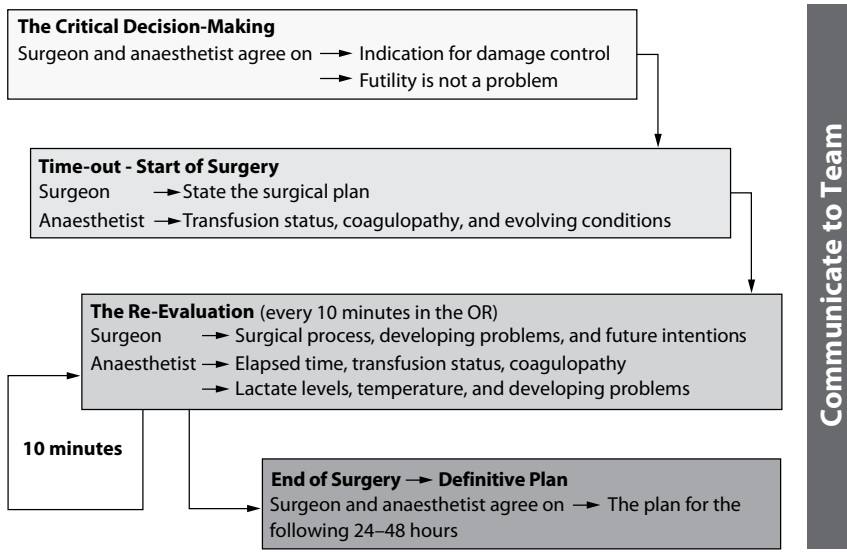

Figure 2.1 Communication in trauma.

and any other evolving conditions that will affect immediate management.

During damage control surgery and resuscitation, re-evaluation is performed around every 10 minutes in order to maintain team situational awareness and allow effective anticipation and planning of all aspects of the care of the patient. During the re-evaluation, the surgeon states the surgical progress, developing problems, and future intentions while the anaesthetist reports the elapsed time, transfusion status, developing coagulopathy, lactate levels, temperature, and other relevant developing problems.

Upon completion of surgery, the anaesthetist and surgeon recapitulate the patient's condition and the procedures performed and agree on the plan for the following 24–48 hours. They are responsible for ensuring the transfer of relevant information internally in the team and externally with collaborators such as the ICU, the blood bank, etc. The trauma leader plans the tertiary survey within the following 24 hours. The process is summarised in Figure 2.1.

2.3 LEADERSHIP IN TRAUMA CARE

Leadership is key to the management of trauma. A good team leader should possess the ability to make quick decisions under pressure based on all available information, maintain an overview, plan and execute treatment strategies in cooperation with team members, and perform regular reviews of the patient's response to treatment. Communication should be performed concisely and be unambiguous, but at the same time the team leader should be open and responsive to input from all team members. There are several ways of describing successful leadership; one is the Non-Technical Skills for

Table 2.1 Non-Technical Skills for Surgeons (NOTSS) Taxonomy

Category	Element
Situation awareness	Gathering information Understanding information Projecting and anticipating future state
Decision-making	Considering options Selecting and communicating options Implementing and reviewing decisions
Task management	Planning and preparation Flexibility/responding to change
Leadership	Setting and maintaining standards Supporting others Coping with pressure
Communication and teamwork	Exchanging information Establishing a shared understanding Coordinating team activities

Table 2.2 Potential Errors Related to Each Behavioural Theme

Themes	Potential Errors
Cognitive Skills – Situational Awareness	
Mechanism	• Failure to obtain data from the pre-hospital setting • Failure to incorporate knowledge of mechanism into understanding of potential forces patient has been subjected to, or which structures may potentially have been injured
Physiological burden	• Failure to obtain vital signs from pre-hospital setting • Failure to evaluate for neurological status • Failure to assess need for airway control • Failure to evaluate for respiratory status • Failure to evaluate for haemodynamic status • Failure to take into consideration trend in physiological parameters • Failure to take into consideration timing of physiological parameters
Injury and pattern recognition	• Failure to recognize a critical/unstable patient and lacking awareness of the overall trauma burden • Failure to pick up on the subtle cues that suggest severe injury
Active and confirmatory reconciliation	• Failure to evaluate response to treatment • Failure to consistently reassess diagnoses and management plans
Data processing and metacognition	• Losing sight of the bigger picture by focusing too much on irrelevant details (inattentional blindness/tunnel vision) • Allowing non-empirical data and biases to influence judgment
Environmental limitations	• Lacking awareness of the resources of the institution, thereby leading to delays • Failure to call for additional resources and personnel when necessary • Transferring the patient without rendering the patient safe for transfer
Self-limitations	• Failure to recognize when a given situation surpasses one's abilities (e.g. skill-set, experience, comfort level, and fatigue) • Failure to call for additional personnel when help is needed
Cognitive Skills – Decision-Making	
Forward planning	• Minimizing severity of injuries • Failure to mobilize the proper resources in a timely fashion • Failure to plan for worst-case scenarios and potential patient deterioration
Managing the injury	• Failure to follow ATLS protocols • Failure to follow best-practice guidelines and established management algorithms • Failure to investigate all body cavities to determine site of injury
Prioritizing	• Under-triaging patients • Failure to address life-threatening injuries before non-urgent injuries
Escalation of aggressiveness	• Devising and implementing a management plan that is not aggressive enough • Devising and implementing a management plan that is inappropriately aggressive

(*Continued*)

Table 2.2 (*Continued*) Potential Errors Related to Each Behavioural Theme	
Themes	**Potential Errors**
Interpersonal Skills – Leadership	
	• Failure to introduce yourself as team leader • Ineffective coordination of team members (e.g. losing control of team members) • Overly micromanaging specific tasks instead of acting as the leader • Inability to cope under pressure in a chaotic environment • Failure to provide feedback to team members on performance
Interpersonal Skills – Teamwork and Communication	
	• Failure to establish team member roles ahead of time • Failure to listen to other team members • Failure to obtain confirmation of task delegation (closed loop communication) • Failure to effectively share management plan with other team members • Failure to limit the physical environment to necessary personnel only
Source: Reproduced with permission: Madani A et al. *J Surg Educ.* 2018;75(2):365.	

Surgeons (NOTSS) taxonomy identified by the University of Aberdeen Industrial Psychology Research, Scotland, and the Royal College of Surgeons of Edinburgh, UK.[5] NOTSS can also be used for behavioural scoring and research. A multilevel assessment, which is simulation based, has been described (Table 2.1).[6–9]

These non-technical skills are not learnt from a textbook; they are acquired and reinforced through training in simulated environments with professional instructors who have expertise in both the principles of CRM as well as briefing and debriefing. These skills should then be used in daily practice.

2.4 POTENTIAL ERRORS RELATED TO EACH BEHAVIOURAL THEME

Errors will occur. By understanding the potential pitfalls, and with a mind-set like that in the aviation environment, they can be minimized (Table 2.2).[10]

Error is normal.
Therefore do not blame error:
Anticipate and prevent it.

2.5 SUMMARY

Trauma care is particularly challenging, in that the decision-making processes and interventions must be executed in a very condensed time period, for example, minutes or even seconds compared to hours and even days/weeks in other less urgent forms of medical practice. Factors such as injury severity, physiology, and interdisciplinary specialist interactions, combine to compound the risk of error in high-stress situations, and elicits the same responses as in the aviation environment. Advance planning, good leadership and teamwork, and the timely anticipation of problems where possible will minimize risks to the patient.

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Recommended Reading

NOTSS Handbook

The Non-Technical Skills for Surgeons (NOTSS) System Handbook V1.2. University of Aberdeen and Royal College of Surgeons of Edinburgh, Scotland. 2012. Available from https://www.iscp.ac.uk/static/help/NOTSS_Handbook_2012.pdf (accessed online Dec 2018).

Pre-Hospital and Emergency Trauma Care 3

3.1 RESUSCITATION IN THE EMERGENCY DEPARTMENT AND PRE-HOSPITAL SETTING

Patients with life-threatening injuries represent approximately 10%–15% of all patients hospitalized for injuries.[1] Some authors have defined severe trauma as a patient who has an Injury Severity Score (ISS) greater than 15.[2–4] For triage purposes, information available in the pre-hospital phase and primary survey should be used.

A standardized approach, utilizing the 'MIST' (also known as the '((AT)MIST') handover, should be used (Table 3.1).

3.2 MANAGEMENT OF MAJOR TRAUMA

The principles of management for patients suffering major trauma are:

- Simultaneous assessment and resuscitation.
- Life-saving surgery.
- A complete physical examination.
- Diagnostic studies if the patient becomes haemodynamically stable.

The first physician to treat a severely injured patient must start the resuscitation immediately and collect as much information as possible. In addition to patient symptoms, necessary information includes mechanism of injury and the presence of pre-existing medical conditions that may influence the critical decisions to be made. Time is working against the patient: 62% of all trauma patients who die in hospital do so within the first 4 hours of hospitalization.[5] The majority either bleed to death or die from primary or secondary injuries to the central nervous system. In order to reduce this mortality, prompt restoration of adequate tissue oxygenation and perfusion, and control of haemorrhage is critical; however this requires time, which is usually not available, and the work-up of the critically injured patient often must be rushed. To maximize resuscitative efforts and to avoid missing life-threatening injuries, various protocols for resuscitation have been developed, of which the Advanced Trauma Life Support Course® (ATLS)[6] is a model.

Guideline times for the length of stay in the emergency department (ED) should be as follows:

- **For the unstable patient, time in the ED should be no longer than 30 minutes (unless surgery is performed in the ED), and the unstable patient should either be in the operating room or the intensive care unit (ICU) within 30 minutes.**
- **For the stable patient, time in the ED should be no longer than 30–60 minutes.**
- **The *stable* patient should be in the computed tomography (CT) scanner or ICU within 60 minutes.**

3.2.1 Resuscitation

Resuscitation is traditionally performed in the (C) ABCDE format, where (C) is for controlling bleeding. Where there is extremity bleeding including traumatic amputation, the use of pre-hospital tourniquets has found a place, initially used in the military setting, and subsequently in the civilian setting.

Table 3.1 The MIST Handover

Trauma/Medical Handover		
(AT)	**Age**	Name, age, sex
(AT)	**Time**	Time of incident
M	**Mechanism of injury** **Medical complaint**	Speed, mass, height, restraints, number and type of collisions, helmet use and damage, weapon type. Medical onset, duration, history.
I	**Injuries sustained** **Illness**	Pain, deformity, injuries, injury patterns STEMI/stroke
S	**Signs and symptoms**	Vitals: Initial/current/worst RR, SPO_2, $ETCO_2$, blood gases HR, BP GCS: Eyes ____ Motor ____ Verbal ____ Total ____/15
T	**Treatment**	Tubes, lines (location and size), fluids Medications and response Immobilization and dressings

3.2.1.1 CIVILIAN PRE-HOSPITAL TOURNIQUET USE

Although underused, civilian pre-hospital tourniquet application was independently associated with a six-fold mortality reduction in patients with peripheral vascular injuries. More aggressive pre-hospital application of extremity tourniquets in civilian trauma patients with extremity haemorrhage and traumatic amputation is warranted.[7]

Pitfall

It is essential to document the time of application of the tourniquet (there is usually a tag on the tourniquet itself to write this down). It is very easy to miss this and then ischaemic damage to tissue occurs instead of exsanguination!

Resuscitation itself is divided into two components:

- The primary survey and initial resuscitation.
- The secondary survey and continuing resuscitation.

All patients undergo the primary survey of airway, breathing, and circulation. Only those patients who become haemodynamically stable will progress to the secondary survey, which focuses on a complete physical examination that directs further diagnostic studies. The great majority of patients who remain haemodynamically unstable require immediate operative intervention.

3.2.1.2 PRIMARY SURVEY

The priorities of the primary survey are:

- Establishing a patent airway with cervical spine control.
- Adequate ventilation.
- Maintaining circulation (including intravascular volume and cardiac function).
- Assessing the global neurological status.

3.2.1.2.1 Airway

Patients with extensive trauma who are unconscious or in shock benefit from immediate endotracheal intubation,[8,9] which may often happen pre-hospital. To prevent spinal cord injury, the cervical spine must be protected during intubation. Intubation via the oral route is successful in most injured patients. On rare occasions, bleeding, deformity, or oedema from maxillofacial injury will require emergency cricothyroidotomy or planned tracheostomy. Patients who may require a surgical airway include those with a laryngeal fracture and those with a penetrating injury of the neck or throat. The airway priorities are to clear the upper airway, to establish high-flow oxygen initially with a bag mask, and to proceed immediately to a definitive airway (cuffed tube in the trachea) – an endotracheal tube in most cases and to a surgical airway on a few occasions.

3.2.1.2.2 Breathing

Patients with respiratory compromise are not always easy to detect. Simple parameters such as the respiratory rate (RR) and adequacy of breathing should be examined within the first minute after arrival. One of the most important things is to detect a tension pneumothorax, necessitating direct drainage by needle thoracostomy followed by the insertion of a chest tube. Intubated patients are usually on positive-pressure ventilation, and in time critical conditions such as in the pre-hospital setting, a thoracostomy incision alone with an occlusive dressing sealed in three of the four sides can often be enough. The chest tube could be inserted upon arrival at the hospital. Other major threats to life, for example, massive haemothorax, flail chest and pulmonary contusion, cardiac tamponade, and tracheal-bronchial injury must be identified and treatment instituted urgently.

3.2.1.2.3 Circulation

Simultaneous with airway management, a quick assessment of the patient will determine the degree of shock present. Shock is a clinical diagnosis and should be apparent. A quick first step is to feel an extremity. If shock is present, the extremities will be cool and pale, lack venous filling, and have poor capillary refill. The pulse will be thready and consciousness will be diminished. As a guideline, major clinical shock results from bleeding into only five sites:

'Blood on the floor, and four more...'

- External bleeding ('blood on the floor').

... and four more:

- Bleeding into the chest (exclude by chest x-ray).
- Bleeding into the abdomen.
- Bleeding into the pelvis (exclude by clinical examination and pelvic x-ray).
- Bleeding into the extremities (exclude by clinical examination and long bone x-ray).

At the same time, the status of the neck veins must be noted. A patient who is in shock with flat neck veins is assumed to have hypovolaemic shock until proven otherwise. If the neck veins are distended, the most likely possibilities are:

- Tension pneumothorax.
- Pericardial tamponade.
- Myocardial contusion (cardiogenic shock).
- Myocardial infarction (cardiogenic shock).
- Air embolism.

Pitfall

Note that the absence of distended neck veins does not exclude these diagnoses because the circulating volume may be so depleted that the circulation is empty.

Tension pneumothorax should always be the number one diagnosis in the physician's differential diagnosis of shock since it is the life-threatening injury that is easiest to treat in the ED. A simple tube thoracostomy is the definitive management.

Pericardial tamponade is most commonly encountered in patients with penetrating injuries to the torso. Approximately 25% of all patients with cardiac injuries will reach the ED alive. The diagnosis is often obvious. The patient has distended neck veins and poor peripheral perfusion, and a few will have pulsus paradoxus. Ultrasonography may establish the diagnosis in those few patients with equivocal findings. Pericardiocentesis is of doubtful diagnostic or therapeutic use; ultrasound is a more reliable diagnostic modality, and a subxiphoid pericardial window is preferable therapeutically. However, proper treatment is immediate thoracotomy, preferably in the operating room, although ED thoracotomy can be life-saving.[10]

Myocardial contusion is a rare cause of cardiac failure in the trauma patient.

Myocardial infarction from coronary occlusion is not uncommon in the elderly. It may be the cause of the initial crash.

Air embolism[11,12] is a syndrome that has relatively recently been appreciated as important in injured patients; it represents air in the systemic circulation caused by a bronchopulmonary venous fistula. Air embolism occurs in 4% of all major thoracic injuries. Thirty-five per cent of the time it is due to blunt trauma, usually a laceration of the pulmonary parenchyma by a fractured rib. In 65% of patients, it is due to gunshot wounds or stab wounds. The surgeon must be vigilant when pulmonary injury has occurred. Any patient who has no obvious head injury but has focal or lateralizing neurological signs may have air bubbles occluding the cerebral circulation. The observation of air in the retinal vessels on fundoscopic examination confirms cerebral air embolism. Any intubated patient on positive-pressure ventilation who has a sudden cardiovascular collapse is presumed

to have either tension pneumothorax or air embolism to the coronary circulation. Doppler monitoring of an artery can be a useful aid in detecting air embolism. Definitive treatment requires immediate thoracotomy followed by clamping of the hilum of the injured lung to prevent further embolism, followed by expansion of the intravascular volume. Open cardiac massage, intravenous adrenaline (epinephrine) and venting the left heart and aorta with a needle to remove residual air may be required. The pulmonary injury is treated definitively by oversewing the laceration or resecting a lobe.

If the patient's primary problem in shock is blood loss, the intention is to stop the bleeding. If this is not possible, the priorities are:

- To gain venous access to the circulation.
- To obtain a blood sample from the patient.
- To determine where the volume loss is occurring.
- To give appropriate resuscitation fluids.
- To prevent and treat coagulopathy.
- To prevent hypothermia.

Access is preferably central, via the subclavian route, and an 8.5 French Gauge (FG) introducer, more commonly used for passing a pulmonary artery catheter, can be used. Alternative routes are the jugular or femoral veins, or venous cut-down.

As soon as the first intravenous line has been established, baseline blood work is obtained that includes haematocrit, toxicology, blood type and cross match, and a screening battery of laboratory tests if the patient is older and has premorbid conditions. Blood gas determinations should be obtained early during resuscitation.

The third priority is to determine where the patient may have occult blood loss. Three sources for hidden blood loss are the pleural cavities, which can be eliminated as a diagnosis by rapid chest x-ray or ultrasound, the thigh and the abdomen, inclusive of the retroperitoneum and pelvis. A fractured femur should be clinically obvious. However, assessment of the abdomen by physical findings can be extremely misleading. Fifty per cent of patients with significant haemoperitoneum have no clinical signs.[13] Common sense dictates that if the patient's chest x-ray is normal, the femur is not fractured, and there is no external bleeding, the patient who remains in shock must be suspected of having ongoing haemorrhage in the abdomen or pelvis. Most of these unstable patients require immediate laparotomy to avoid death from haemorrhage. An important caveat is not to delay mandated therapeutic interventions to obtain non-critical diagnostic tests.

The fourth priority for the resuscitating physician is to consider activation of the massive bleeding protocol and order resuscitation fluids, starting with crystalloids and adding type-specific whole blood or blood components as soon as possible. Although whole blood is preferred, especially in the military situation, it is commonly difficult to obtain whole blood from modern blood banks, forcing the use of blood components. Loss of more than 2 units of blood and ongoing bleeding that requires blood transfusion should invoke a predefined massive bleeding protocol (most current massive transfusion protocols – MTPs, aim at predefined ratios of packed red blood cells:fresh frozen plasma:platelets, mimicking whole blood), and monitored by frequent coagulation tests, conventional laboratory tests, clotting studies, and more functional goal-directed haemostasis using thrombo-elastography (TEG) or rotary thrombo-elastomerography (RoTEM) when available. Use pharmacological haemostatic adjuncts (topical and systemic) such as tranexamic acid when indicated (see also Chapter 5). Blood components such as red blood cells, liquid plasma, or cryoprecipitate are now used in several systems, including Helicopter Emergency Medical Systems.

The use of crystalloids should be restrictive, and permissive hypotension is preferable in unstable bleeding patients both pre-hospitally and initially in the ED until haemorrhage control (see also Chapter 6).

The criteria for adequate resuscitation are simple and straightforward:

- Keep the atrial filling pressure at normal levels.
- Give enough fluid to achieve adequate urinary output (0.5 mL/kg per hour in the adult, 1.0 mL/kg per hour in the child).
- Maintain peripheral perfusion.

In elderly patients with extensive traumatic injuries, utilizing a cardiac computer may be prudent because it will be used to direct a sophisticated multifactorial resuscitation in the operating room or ICU. Resuscitation should be directed to achieve adequate oxygen delivery and oxygen consumption. An important caveat is not to delay mandated therapeutic interventions to obtain non-critical diagnostic test results.

3.2.1.2.4 Neurological Status (Disability)

The next priority during the primary survey is to quickly assess neurological status and to initiate diagnostic and

treatment priorities. The key components of a rapid neurological evaluation are:

- Determine the level of consciousness.
- Observe the size and reactivity of the pupils.
- Check eye movements and oculovestibular responses.
- Document skeletal muscle motor responses and spontaneous movement of extremities.
- Determine the pattern of breathing.
- Perform a peripheral sensory examination.

3.2.1.2.5 Neurological Status is Often Described Using the Glasgow Coma Scale (3–15/15)

A decreasing level of consciousness is the single most reliable indication that the patient may have a serious head injury or secondary insult (usually hypoxic or hypotensive) to the brain. Consciousness has two components: awareness and arousal. Awareness is manifested by goal-directed or purposeful behaviour. The use of language is an indication of functioning cerebral hemispheres. If the patient attempts to protect himself from a painful insult, this also implies cortical function. Arousal is a crude function that is simple wakefulness. Eye-opening, either spontaneous or in response to stimuli, is indicative of arousal and is a brainstem function. Coma is a pathological state in which both awareness and arousal are absent. Eye-opening does not occur, there is no comprehensible speech detected, and the extremities move neither to command nor appropriately to noxious stimuli. By assessing all components and making sure the primary reflexes (pupillary, ankle, knee, biceps, and triceps) are assessed, and repeating this examination at frequent intervals, it is possible to both diagnose and monitor the neurological status in the ED. An improving neurological status reassures the physician that resuscitation is improving cerebral blood flow. Neurological deterioration is strong presumptive evidence of either a mass lesion or significant neurological injury. A CT scan (including the cervical spine) should be done as soon as possible.

3.2.1.2.6 Environment

The clothes are to be removed in order to examine the whole patient. A logroll must be performed, especially after penetrating injuries, in order to identify all wounds. The patient is at risk of hypothermia, and warming measures should be promptly instituted.

The body temperature of trauma patients decreases rapidly, and if the 'on-scene time' has been prolonged, for example by entrapment, patients arrive in the resuscitation room hypothermic. This is aggravated by the administration of cold fluid, the presence of abdominal or chest wounds, and the removal of clothing.

Patients can be expected to drop their core temperature by 1°–2°C per hour.

All fluids need to be at body temperature or above, and there are rapid infusor devices available that will warm fluids at high flow rates prior to infusion. Patients can be placed on warming mattresses and their environment kept warm using warm air blankets. Early measurement of the core temperature is important to prevent heat loss that will predispose to problems with coagulation. Hypothermia will shift the oxygen dissociation curve to the left, reduce oxygen delivery, reduce the liver's ability to metabolize citrate and lactic acid, and may produce arrhythmias.

The minimum diagnostic studies that should be considered in the haemodynamically unstable patient as part of the primary survey include:

- FAST ultrasound.
- Chest x-ray.
- Plain film of the pelvis.

Focused assessment with sonography in trauma (FAST) examination may be helpful:

- To assess whether there is blood in the abdomen or chest (extended FAST).
- To exclude cardiac tamponade.
- Extended FAST will assess for pneumothorax as well.

It must be emphasized that resuscitation should not cease during these films, and the resuscitating team must wear protective lead aprons. Optimally, the x-ray facilities, and especially the CT scanner, are juxtaposed to the ED, but the essential x-rays can all be obtained with a portable machine.

3.2.1.3 SECONDARY SURVEY

Finally, if the patient stabilizes, a secondary survey and diagnostic studies are carried out. However, if the patient remains unstable, he or she should be taken immediately to the operating room in order to achieve surgical haemostasis, or to the surgical ICU.

The patient must have a full 'top-to-toe' and 'front-to-back' examination. If the patient has been haemodynamically unstable, the site of the bleeding is traditionally:

'Blood on the floor, and four more'.

3.2.1.3.1 The Haemodynamically Normal Patient

There is ample time for a full evaluation of the patient, and a decision can be made regarding surgery or non-operative management. CT scanning is currently the modality of choice.

3.2.1.3.2 The Haemodynamically Stable Patient

The stable patient, who is not haemodynamically normal, but who is maintaining blood pressure, and other parameters with resuscitation, will benefit from investigations aimed at establishing:

- Whether the patient has bled into the abdomen?
- Whether the bleeding has stopped?

Thus, serial investigations of a quantitative nature will allow the best assessment of these patients. CT scan is the modality of choice, provided awareness of the fact that the patient may decompensate.

3.2.1.3.3 The Haemodynamically Unstable Patient

Efforts must be made to try to define the cavity where bleeding is taking place, for example, chest, pelvis, or abdominal cavity. Negative chest and pelvic x-rays leave the abdomen as the most likely source. Diagnostic modalities are of necessity limited. FAST is effective for detecting free fluid in the abdomen and pericardium, but is operator dependent – haemodynamic instability caused by intraperitoneal haemorrhage is likely to be readily found, but a negative FAST does not exclude intra-abdominal bleeding. FAST can be performed without moving the patient from the resuscitation area, since an unstable patient should not have a CT scan, even if it were to be readily available. Diagnostic peritoneal lavage (DPL) can also be used in mass casualty incidents when there is a lack of CT scanners due to a larger number of patients.

3.2.2 Management of Penetrating Trauma

Many forces can act on the torso to cause injury to the outer protective layers or the contained viscera. Penetrating trauma is most often due to knives, missiles, and impalement. Knife wounds and impalement usually involve low-velocity penetration, and mortality is directly related to the organ injured. Secondary effects such as infection are due to the nature of the weapon and the material (i.e. clothing and other foreign material) that the missile carries into the body tissue. Infection is also influenced by spillage of contents from an injury to a hollow viscus organ.

An equally important component of the physical examination is to describe the penetrating wound. It is imperative that surgeons do not label the entrance or exit wounds unless common sense dictates it – an example is a patient with a single penetrating missile injury with no exit. However, in general, it is best to describe whether the wound is circular or ovoid and whether there is surrounding stippling (powder burn) or bruising from the muzzle of a weapon. Similarly, stab wounds should be described as longitudinal, triangular-shaped (hunting knives), or circular depending on the instrument used. Experience has shown that surgeons who describe wounds as entrance or exit may be wrong as often as 50% of the time. Experience with forensic pathology is required to be more accurate.

It is good practice to place metallic objects such as paper clips on the skin pointing to the various wounds on the chest wall, which aid in determining the missile track. It is recommended that an 'unfolded' paper clip be placed on any anterior penetrating injury, and a 'folded' one on any posterior injury (Figure 3.1).[14] This also can be useful for stab wounds. Tracking the missile helps to determine which visceral organs may be injured and

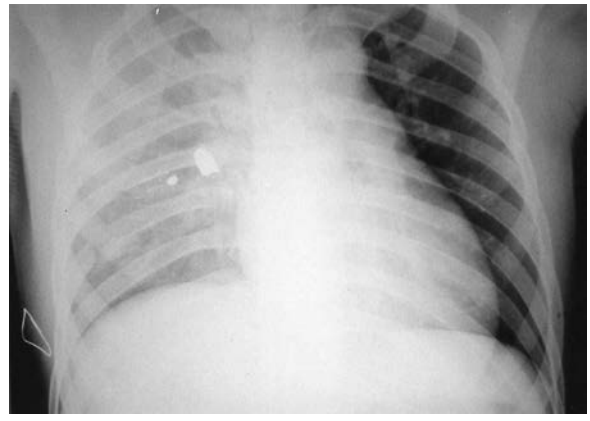

Figure 3.1 Chest x-ray showing the use of markers to show the wound track.

whether there is potential transgression of the diaphragm and/or mediastinum.

In the pre-hospital setting patients with penetrating torso trauma should be treated with a 'scoop and run' modality where on-scene time is minimized. Procedures should rather be performed en route to the hospital instead of at the scene of the incident.

3.3 EMERGENCY DEPARTMENT SURGERY

The emergency management of a critically injured trauma patient continues to be a substantial challenge. It is essential to have a very simple, effective plan that can be put into place to meet the challenges presented by resuscitating the moribund patient. ATLS® principles apply throughout.

As a basic consideration, for all major trauma victims with a systolic blood pressure of less than 90 mm Hg, there is a 50% likelihood of death, which, in one-third of cases, will occur within the next 30 minutes if the bleeding has not been controlled. If death is likely to occur in the next 5 minutes, it is essential to determine in which body cavity the lethal event will occur, as the only chance of survival will be the immediate control of haemorrhage.

If death is likely to occur in the next hour, there is time to proceed with an orderly series of investigations and, time permitting, radiographic or other diagnostic aids, to determine precisely what is injured, and to effect an operative plan for the management for this life-threatening event.

3.3.1 Head Trauma

In the event of severe facial, (and often associated severe neck injuries) surgical control of the airway may be necessary, using ATLS® described techniques.

It is unusual but possible to exsanguinate from a massive scalp laceration – ('Blood on the floor...'). For this reason, it is essential to gain control of the vascular scalp laceration with rapidly placed surgical clips or primary pressure and immediate suturing, using deep sutures rather than staples, and a pressure dressing.

The more common cause of death is from intracranial mass lesions. Extradural haematomas and subdural haematomas can be rapidly lethal. A rapid diagnosis of an ipsilateral dilated pupil with contralateral hemiplegia is diagnostic of mass lesion with significant enough intracranial pressure to induce coning. This requires immediate decompression. Moderate hyperventilation to produce mild hypocapnia, and vasoconstriction is only used immediately prior to neurosurgical intervention. The use of mannitol or hypertonic saline is useful as a temporizing adjunct only.

Attention should be paid to monitoring the end-tidal carbon dioxide, as a proxy for $PaCO_2$, which should not be allowed to fall below 30 mm Hg (4 kPa). This should decrease intracranial volume, and therefore intracranial pressure. There should be an immediate positive effect that usually lasts long enough to obtain a three-cut CT scan to determine a specific site of the mass lesion and the type of haematoma. This will direct the surgeon specifically to the location of the craniotomy for removal of the haematoma.

Intravenous mannitol should be administered as a bolus injection in a dose of 0.5–1.0 g/kg or hypertonic (7.5%) saline as a dosage of 1 mmol/kg. This should not delay any other diagnostic or therapeutic procedures.

3.3.2 Chest Trauma

Lethal injuries to the chest include tension pneumothorax, cardiac tamponade, and transected aorta.

Tension pneumothorax is diagnosed clinically with deviation of the trachea away from the lesion (a late sign), hypertympany on the side of the lesion, and decreased breath sounds on the affected side. There is usually associated elevated jugular venous pressure in the neck veins, unless the patient is hypovolaemic. This is a clinical diagnosis, and once made, an immediate needle thoracostomy or tube thoracostomy should be performed to relieve the tension pneumothorax. The tube should then be placed to underwater seal.

It is far better to perform a thoracotomy in the operating room, either through an anterolateral approach or a median sternotomy, with good light and assistance and the potential for autotransfusion and potential bypass, than it is to attempt heroic emergency surgery in the resuscitation suite. However, if the patient is in extremis with blood pressure in the 40 mm Hg or lower range despite volume resuscitation, there is no choice but to proceed immediately with a left anterior thoracotomy to relieve the tamponade and control the penetrating injury to the heart. If there is an obvious penetrating injury to either the left or the right ventricle, a Foley catheter can be introduced into the hole and the balloon distended

to create a tamponade. The end of the Foley should be clamped.

Pitfalls

Great care should be taken to apply minimal traction on the Foley – just enough to allow sealing. Excessive traction will pull the catheter out and extend the wound by tearing the muscle. Once the bleeding is controlled, the wound can be easily sutured with pledgetted sutures.

- *The chance of survival after emergency thoracotomy is better after a penetrating rather than a blunt trauma mechanism.*

Massive haemorrhage from intercostal vessels secondary to multiple rib fractures will frequently stop without operative intervention. This is also true for most bleeding from the lung. It is helpful to collect the shed blood from the hemithorax into an autotransfusion collecting device and return it to the patient.

Aortic transection is usually diagnosed with a widened mediastinum and confirmed with an arteriogram or a CT scan (see also Chapter 8). Once the diagnosis has been made, it is useful to maintain control of hypotension in the 100 mm Hg range so as not to precipitate free rupture from the transection, until stenting or operative repair can take place.

NB: Abdominal injury generally takes priority over thoracic aortic injury.

3.3.3 Abdominal Trauma

Significant intra-abdominal or retroperitoneal haemorrhage can be a reason to go rapidly to the operating room. The abdomen may be distended and dull to percussion. Ultrasound (FAST) is a useful tool as it is specific for blood in the peritoneum, but it is operator-dependent. A positive FAST result in an unstable patient is an indication for laparotomy. Conversely, a negative FAST result does not exclude intra-abdominal bleeding and repeat FAST or other investigations need to be considered. A definitive diagnosis can be made with FAST, a grossly positive DPL, or CT scan. The decision to operate for bleeding should be based on the haemodynamic status.

Non-operative management has become the treatment of choice in haemodynamically stable patients with liver and spleen injuries regardless of injury grade. (See Sections 9.4: Liver and 9.5: Spleen.)

The CT scanner is highly sensitive and very specific for the type, character and severity of injury to a specific organ. However, patients whose condition is unstable should NOT be considered.

3.3.4 Pelvic Trauma

Pelvic fractures can be a significant cause of haemorrhage and death. It is essential to return the pelvis to its original configuration as swiftly as possible. As an emergency procedure, a compressing sheet, or commercially available pelvic binders can be used. There are also external fixation devices such as the C-clamp and the external fixator, which can be placed in the resuscitation suite, and return the pelvis to its normal anatomy. However, their fixation may be time-consuming, requires skill, and may not present advantages over the non-invasive binders for initial management. As the pelvis is realigned, it helps to compress the haematoma in the pelvis. Since approximately 85% of pelvic bleeding is venous, compressing the haematoma usually stops most pelvic bleeding.

If the patient continues to be hypotensive, resuscitation should continue, and an angiogram should be considered. This will identify the presence of significant arterial bleeding in the pelvis, which then can be embolized immediately. If the patient is exsanguinating from the pelvic injury or is haemodynamically unstable, damage control surgery should be performed with extraperitoneal packing of the pelvis combined with a laparotomy, on occasion surgical central vascular control, before angiography. Resuscitative endovascular balloon occlusion of the aorta (REBOA) (see also Section 15.3) has recently been introduced as an alternative to surgery in several EDs (and even a few pre-hospital settings), but the benefit over surgery remains largely unproven.

3.3.5 Long Bone Fractures

Lone bone fractures, particularly of the femur, can bleed significantly. The damage control approach to fractures is external fixation. The immediate treatment for a patient who is hypotensive from haemorrhage from a femoral fracture is to put traction on the distal limb, pulling the femur into alignment. This not only realigns

the bones but also reconfigures the cylindrical nature of the thigh. This has an immediate tamponading effect on the bleeding in the muscles of the thigh. It is frequently necessary to maintain traction with a Thomas or Hare traction splint. Attention should be paid to the distal pulses to be sure that there is continued arterial inflow. If the pulses are absent, an arteriogram should be performed to determine whether there are any injuries to major vascular structures. A determination is then made as to the timing of arterial repair and bony fixation. Re-establishing perfusion to the limb takes priority over fracture treatment.

3.3.6 Peripheral Vascular Injuries

Peripheral vascular injuries are not in themselves life-threatening providing that the bleeding is controlled. However, it is critical to assess whether ischaemia and vascular continuity are present, since this will influence the overall planning.

Every ED should have access to a simple flow Doppler monitor to assess pressures and flow. If there is any doubt over whether the vessel is patent, the ankle–brachial index should be measured, and if it is less than 0.9, an arteriogram is mandatory. Time and availability decide whether the patient can be transported to an angiography suite or should have an angiogram performed in the operating room or ED. Although it is desirable to do this in the angiography suite, it is not always possible, and the necessary equipment may not be available. If there is any doubt, consideration should be given to the use of the ED angiogram.[15]

3.4 SUMMARY

The decision of whether to operate in the ED or in the operating room should be made based on an overview of the urgency and the predicted outcome.

It is useful to have a well-thought-out plan for dealing with the critically injured trauma patient so that both clinical diagnosis and relevant investigations can be performed immediately, and an operative or non-operative therapeutic approach implemented.

There is no future in altering only the geographical site of death.

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Part 2

Physiology and the body's response to trauma

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Resuscitation Physiology 4

4.1 METABOLIC RESPONSE TO TRAUMA

4.1.1 Definition of Trauma

Physical injury is accompanied by systemic as well as local effects. Following trauma, the body responds locally by inflammation and by a general response which is often protective and which conserves fluid and provides energy for repair. Proper resuscitation may attenuate the response but will not abolish it.

The response is characterized by an acute catabolic reaction, which precedes the metabolic process of recovery and repair. This metabolic response to trauma traditionally was divided into an ebb and flow phase by Cuthbertson in 1932.[1]

The ebb phase is relatively short lived and corresponds to the period of severe shock characterized by depression of enzymatic activity and oxygen consumption.

After effective resuscitation has been accomplished with restoration of adequate oxygen transport, the flow phase comes into play. The flow phase can be divided into:

- A catabolic phase with fat and protein mobilization associated with increased urinary nitrogen excretion and weight loss.
- An anabolic phase with restoration of fat and protein stores, and weight gain.

The appropriate protective flow phase is characterized by:

- A normal or slightly elevated blood glucose level.
- Increased glucose production.
- Normal or slightly elevated free fatty acid levels, with increased flux.
- A normal or elevated insulin concentration.
- High normal or elevated levels of catecholamine and an elevated glucagon level.
- A normal blood lactate level.
- Elevated oxygen consumption.
- Increased cardiac output.
- Elevated core temperature.

These responses are marked by hyperdynamic circulatory changes, signs of inflammation, glucose intolerance, and muscle wasting.

4.1.2 Initiating Factors

The magnitude of the metabolic response depends on the degree of trauma and concomitant contributory factors such as infection, tissue necrosis, and pre-existing systemic disease. The response will also depend on the age and sex of the patient, the genetic composition, the underlying nutritional state, and the timing of treatment and its effectiveness. In general, the more severe the injury (i.e. the greater the degree of tissue damage), the greater the metabolic response.

The metabolic response alterations seem to be less aggressive in children and the elderly and in the premenopausal female. Starvation and nutritional depletion also modify the response. Patients with poor nutritional or immunological status (e.g. those with human immunodeficiency virus – HIV) have a reduced metabolic response to trauma compared to healthy well-nourished patients, while burns and severe traumatic brain injury cause a relatively greater response than other mechanical injuries.

Wherever possible, efforts should be made to reduce the magnitude and duration of the initial insult, since by doing so it may be possible to reduce the extent of the metabolic changes. Thus, aggressive resuscitation, control of pain and temperature, limiting acidosis, adequate devitalized tissue debridement, avoidance of unnecessary blood component administration with coagulopathy, and nutritional (preferably enteral) support are critical.

The precipitating factors can be broadly divided into:

4.1.2.1 HYPOVOLAEMIA

- Decrease in circulating blood volume.
- Increase in alimentary loss of fluid.
- Loss of interstitial volume.
- Extracellular fluid shift.

4.1.2.2 AFFERENT IMPULSES

- Somatic.
- Autonomic.
- Sympathetic ↑.
- Cholinergic ↓.

4.1.2.3 WOUND FACTORS: INFLAMMATORY AND CELLULAR

- Platelets – PF4.
- Neutrophils – superoxide, elastase.
- Macrophages/dendritic cells.
- Endothelial cells.
- Cytokines – interleukins IL1, IL2, IL6, IL10, IL-17, TNF.
- Chemokines – IL8.
- Eicosanoids – LTB4, LTC4, TXA_2, PGE_2.
- Damage associated molecular patterns (DAMPs) $HMGB_1$, HSP_{70}.

4.1.2.4 TOXINS/SEPSIS

- Endotoxins.
- Exotoxins.

4.1.2.5 FREE RADICALS

- Superoxide and derivatives.
- Nitrogen radicals.

4.1.2.6 HYPOVOLAEMIA

Hypovolaemia, specifically tissue hypoperfusion, is the most potent precipitator of the metabolic response. Hypovolaemia can be due to external losses, internal shifts of extracellular fluids, and changes in plasma osmolality. However, the most common cause is blood loss (see also Section 4.2).

The hypovolaemia will stimulate release of catecholamines, which in turn trigger the neuroendocrine response. This plays an important role in volume and electrolyte conservation and protein, fat, and carbohydrate catabolism.

4.1.2.7 AFFERENT IMPULSES

Hormonal responses are initiated by pain and anxiety. The metabolic response may be modified by administration of adequate analgesia, which may be parenteral, enteral, regional, or local. Somatic blockade may need to be accompanied by autonomic blockade, in order to minimize or abolish the metabolic response.

4.1.2.8 WOUND FACTORS

Endogenous factors may prolong or even exacerbate the systemic trauma insult, even though the primary cause is treated well. Tissue injury activates a diverse response via release of DAMPs activating toll-like receptors (TLR), and by release of multiple inflammatory mediators locally at the site of injury and/or infection through two predominant pathways:

- Humoral pathway.
- Cellular pathway.

Uncontrolled activation of endogenous inflammatory mediators and cells may contribute to a syndrome called the systemic inflammatory response syndrome (SIRS). The result of an excessive SIRS response is diffuse bystander organ injury.

Both humoral and cell derived activation products play a role in the pathophysiology of organ dysfunction.[2] It is important, therefore, to monitor post-traumatic biochemical and immunological abnormalities whenever possible, as a guide to direct and confirm the appropriateness of resuscitative interventions.

4.1.3 Immune Response

The immune response is complex and consists of an early enhanced upregulation of the primarily pro-inflammatory innate system and a concomitant prolonged suppression of the adaptive immune system. The magnitude of these responses is modified by the depth and duration of insult caused by the injury, as well as the patient's genetic composition and pre-existing comorbidities.

4.1.3.1 THE INFLAMMATORY PATHWAY

The inflammatory mediators of injury have been implicated in the induction of numerous cellular dysfunctions.

While neutrophils have been invoked as primary mediators of inflammatory processes for more than 100 years, we now recognize that these initial responses involve numerous cellular mediators including platelets, macrophages, endothelium, and epithelium.

4.1.3.1.1 Cytokines

The term cytokine refers to a diverse group of polypeptides and glycoproteins that are important mediators of inflammation. They are produced by a variety of cell types, but predominantly by leucocytes. Cytokines are generally divided into pro-inflammatory, and anti-inflammatory, but some have both properties, for example IL-6.

4.1.3.1.2 Pro-Inflammatory Cytokines

Certain cytokines, particularly TNF, IL-1, and IL-8 promote the inflammatory response by up-regulating expression of genes that generate the pro-inflammatory mediators. Pro-inflammatory cytokines also mediate inflammation by activating neutrophils, endothelium, and epithelium – all of which lead to tissue damage.

The TNF and IL-1 act synergistically to produce the acute innate immune response to ischaemia/reperfusion in many organs. TNF causes neutrophils to be attracted to injured epithelium, thereby helping to regulate the inflammatory response. It also stimulates endothelial cells to produce a cytokine subset known as chemokines (e.g. IL-8), which produce leukocyte migration into the tissues and IL-1 production. Like TNF, IL-1 is a primary responder in the inflammatory cascade, and its actions are similar to TNF, but it does not induce apoptosis or programmed cell death.

Interferon gamma (IFN-γ) is produced in response to antigens processed by macrophages, an event enhanced by IL-12. The IL-12 is produced by mononuclear phagocytes, and dendritic cells in response to intracellular microbes. IL-6 is produced by mononuclear phagocytes, endothelial cells, and fibroblasts, and acts in a pro-inflammatory manner by providing a potent stimulus for hepatocyte synthesis of acute phase proteins.

The IL-2, unlike the above cytokines which exert most of their influence via the innate immune system, stimulates acquired immunity, and has other immunomodulatory functions as well.

4.1.3.1.3 Anti-inflammatory Cytokines

The anti-inflammatory cytokines exert their effects by inhibiting the production of pro-inflammatory cytokines or countering their action. They reduce gene expression and mitigate or prevent numerous inflammatory effects.

The IL-10 is important in the control of innate immunity. It can prevent fever, pro-inflammatory cytokine release, and clotting cascade activation during endotoxin challenge. Other potent anti-inflammatory modulators include IL-4, IL-13, and transforming growth factor beta (TGF-β).

4.1.3.1.4 Modulation of Cytokine Activity in Sepsis, Systemic Inflammatory Response Syndrome (SIRS), and Compensatory Inflammatory Response Syndrome (CIRS)

Systemic inflammatory activity, which occurs in response to infectious or non-infectious stimuli, is the fundamental overall clinical phenomenon that can provoke whole body SIRS and can lead ultimately to multiple organ dysfunction syndrome (MODS) and multiple organ failure (MOF), which is associated with a mortality of up to 50%. It was initially suggested that SIRS and sepsis were attributable to an overwhelming pro-inflammatory innate immune response, mediated by TNF and numerous other cytokines. However, simultaneously, the body also mounts an endogenous counter- or anti-inflammatory reaction to restore homeostasis, which can then lead to CARS. Early, after severe injury or sepsis, the pro-inflammatory response predominates and SIRS and shock result. If the counter-inflammatory response leads to homeostasis the patient does well and recovers. However, if the suppressive response is excessive, it leads to immunosuppression, and greatly increased susceptibility to nosocomial infections that are frequently seen in the severely injured and critically ill patients.. The modern view is that, following injury, SIRS and CARS activate simultaneously.

The role of cytokines in sepsis is critical to outcomes and is very complex, with both pro-inflammatory and anti-inflammatory factors playing a role and determining clinical outcome.

4.1.3.1.5 Activated Protein C

Pro-inflammatory mediators have a role in triggering the clotting cascade by stimulating the release of tissue factor from monocytes and the vascular endothelium leading to thrombin formation and a fibrin clot. At the same time, thrombin stimulates many inflammatory pathways and suppresses natural anticoagulant responses by activating

thrombin-activatable fibrinolysis inhibitor (TAFI). This overall procoagulant response leads to microvascular thrombosis and is implicated in the multiple organ failure associated with sepsis. On the other hand, thrombin binding to endothelial thrombomodulin, generates activated protein C (APC), which is an endogenous anticoagulant. However, previous reports of benefit to providing exogenous APC in the setting of sepsis have subsequently been shown to provide no improvement in survival. While the concepts involved are no doubt important, our ability to appropriately modify their activity to benefit the critically ill patient remains elusive.

4.1.3.1.6 Eicosanoids

These compounds, derived from eicosapolyenoic fatty acids, are subdivided into prostanoids (the precursors of the prostaglandins), and leucotrienes (LT). Eicosanoids are synthesized from arachidonic acid (AA), which has been synthesized from phospholipids of cell walls, by the action of phospholipase A2 (in part, released by activated neutrophils). Cyclo-oxygenase converts arachidonic acid to prostanoids, the precursors of prostaglandins (PG), prostacyclins (PGI), and thromboxanes (TX). The term prostaglandins is used loosely to include all prostanoids. The leucotrienes are produced by the action of 5-lipoxygenase on AA with subsequent by-products produced by LTA4 hydrolase (i.e. LTB4 and LTC4 synthase). Eicosanoids modulate blood flow to organs and tissues by altering local balances between vasodilators and vasoconstricting mediators, and, in addition, directly stimulate certain immune cells.

The prostanoids (prostaglandins of the E and F series), PGI_2, and TX not only cause vasoconstriction (TXA_2 and PGF_1), but also vasodilatation (PGI_2, PGE_1 and PGE_2). TXA_2 activates and aggregates platelets and white cells, and PGI_2 and PGE_1 inhibit white cells and platelets. The leucotriene LTB4 is a very potent polymorphonucleocyte (PMN) chemo-attractant and activator, while LTC4 causes vasoconstriction, increased capillary permeability and bronchoconstriction.

4.1.3.2 THE CELLULAR PATHWAY

The classical pathway of complement activation involves an interaction between the specific antibody and the initial trimer of complement components C^1, C^4, and C^2. In the classical pathway, this interaction then cleaves the complement products C^3 and C^5, via proteolysis to produce the very powerful chemotactic factors C^{3a} and C^{5a}.

The so-called alternative pathway appears to be primarily involved following trauma. It is activated by properdin, and proteins D or B, to activate C^3 convertase, which generates the anaphylotoxins C^{3a} and C^{5a}. Its activation appears to be the earliest trigger for activating the innate immune cellular system and is responsible for aggregation of neutrophils and activation of basophils, mast cells, and platelets to secrete histamine and serotonin, which alter vascular permeability and are vasoactive. In trauma patients, the serum C^3 level is inversely correlated with the Injury Severity Score (ISS).[3] Measurement of C^{3a} is the most useful because the other products are more rapidly cleared from the circulation.

The short-lived fragments of the complement cascade, C^{3a} and C^{5a}, stimulate macrophages to secrete interleukin-l (IL-1) and proteolysis inducing factor (PIF), an active circulating cleavage product. These mediators cause proteolysis and lipolysis along with fever. The IL-1 activates T_4 helper cells to produce IL-2, which enhances the adaptive cell-mediated immunity. The IL-1 and PIF are also potent stimulators of the liver, bone marrow, spleen, and lymph nodes to produce acute-phase proteins, including complement, fibrinogen, α2-macroglobulin, and other proteins required for immune defence mechanisms.

There is also considerable cross-talk between the clotting cascade and inflammation. For example, activation of factor XII (Hageman factor A) stimulates kallikrein to produce bradykinin from bradykininogen, which affects capillary permeability and vaso-reactivity. Overall, the overlapping combination of these reactions causes the systemic inflammatory response. Kalligren can activate plasmin to promote fibrinolysis

4.1.3.3 TOXINS

Endotoxin is a lipopolysaccaride component of bacterial cell walls. Endotoxin and other bacterial and viral cell wall components are known to activate many immune cells, along with hepatocytes and myocardiocytes primarily via the TLR receptors. Activated cells release tumour necrosis factor (TNF) and a broad spectrum of potent mediators from macrophages. neutrophils, endothelial cells, and many others. Endotoxins cause vascular margination and sequestration of leukocytes, particularly in the capillary bed. At high doses, direct granulocyte destruction is seen.

4.1.3.4 PAMPS AND DAMPS

Injury causes a SIRS clinically much like sepsis. Multicellular animals detect pathogens via a set of

pattern recognition receptors (TLRs), which recognize pathogen-associated molecular patterns (PAMPs), which in turn activate innate immunocytes. Evidence is accumulating that trauma and its associated tissue damage are recognized at the cell level by a similar cell receptor-mediated detection of intracellular products released by injured and dying cells. The term 'alarmin' has been used to categorize these endogenous DAMPs that signal tissue and cell damage.[4] A major source of DAMPs is injury-induced release of mitochondrial products, including mitochondrial DNA, coined the 'enemies within'. Endogenous DAMPs and exogenous PAMPS therefore convey a similar message and elicit similar responses.[5] Surgical source control, whether for infection or necrotic tissue, is an attempt to minimize the host exposure to these toxic moieties.

4.1.3.5 FREE RADICALS

Oxygen radical (O_2^-) formation by white cells is a normal host defence mechanism. Diffuse activation after severe injury, however, may lead to excessive production by neutrophils and macrophages, with deleterious cellular effects on diffuse organ function. Nitric oxide (NO) is released by macrophages and endothelial cells, causing vasodilatation and decreased systemic vascular resistance. NO also combines with O_2^- to form a potent oxidizing agent. Toxic hydroxyl ion (OH^-) and hydrogen peroxide are also increased following sepsis or stress. Protective endogenous anti-oxidants are rapidly depleted following injury or sepsis leading to an even more enhanced cellular bystander injury.

4.1.4 Hormonal Mediators

In response to trauma, many circulatory hormones are altered. Adrenaline (epinephrine), noradrenaline (norepinephrine), cortisol, and glucagon are increased, while certain others are decreased. The sympathetic-adrenal axis is a major system by which the body's response to injury is activated.

4.1.4.1 HYPOTHALAMUS/PITUITARY

The hypothalamus is the highest level of integration of the stress response. The major efferent pathways of the hypothalamus are endocrine via the pituitary, and the efferent sympathetic and parasympathetic systems. In contrast, the cholinergic system is now recognized to have a variety of anti-inflammatory effects. The pituitary gland responds to trauma with increased levels of adrenocorticotrophic hormone (ACTH), prolactin, and growth hormone, while the remaining hormones are relatively unchanged.

Pain receptors, osmoreceptors, baroreceptors, and chemoreceptors stimulate or inhibit ganglia in the hypothalamus to induce sympathetic nerve activity. The neural endplates and adrenal medulla secrete catecholamines. Pain stimuli via the pain receptors also stimulate secretion of endogenous opiates, β-endorphin, and pro-opiomelanocortin (precursor of the ACTH molecule), which modifies the response to pain and reinforces the catecholamine effects. The β-endorphin has little effect but serves as a marker for anterior pituitary secretion.

Hypotension, hypovolaemia in the form of a decrease in left ventricular pressure, and hyponatraemia stimulate secretion of vasopressin, antidiuretic hormone (ADH) from the supra-optic nuclei in the anterior hypothalamus, aldosterone from the adrenal cortex, and renin from the juxtaglomerular apparatus of the kidney. The increase in aldosterone secretion results in conservation of sodium, and, thereby, water. As osmolality increases, the secretion of ADH increases, and more water is reabsorbed, thereby decreasing the serum osmolality (negative feedback control system).

Hypovolaemia stimulates receptors in the right atrium and hypotension stimulates receptors in the carotid artery. This results in activation of paraventricular hypothalamic nuclei, which secrete pituitary releasing hormone from the median eminence into capillary blood, which stimulates the anterior pituitary to ACTH. ACTH stimulates the adrenal cortex to secrete cortisol and aldosterone. Changes in glucose concentration influence the release of insulin from the β cells of the pancreas, and high amino-acid levels, the release of glucagon from the α cells.

4.1.4.2 ADRENAL HORMONES

Plasma cortisol and glucagon levels rise following trauma. The degree is related to the severity of injury. The function of glucocorticoid secretion in the initial metabolic response is uncertain, since the hormones have little direct action, and primarily they seem to augment the effects of other hormones such as the catecholamines.

4.1.4.3 PANCREATIC HORMONES

There is a rise in the blood sugar following trauma. The insulin response to glucose is reduced substantially with alpha-adrenergic stimulation and enhanced with beta-adrenergic stimulation.[6]

4.1.4.4 RENAL HORMONES

Aldosterone secretion is increased by several mechanisms. The renin-angiotensin mechanism is the most important. When the glomerular arteriolar inflow pressure falls, the juxtaglomerular apparatus of the kidney secretes renin, which acts with angiotensinogen to form angiotensin I. This is converted to angiotensin II, a substance that stimulates production of aldosterone by the adrenal cortex. Reduction in sodium concentration stimulates the macula densa, a specialized area in the tubular epithelium adjacent to the juxtaglomerular apparatus, to activate renin release. An increase in plasma potassium concentration also stimulates aldosterone release. Volume losses and a fall in arterial pressure stimulate release of ACTH via receptors in the right atrium and the carotid artery.

4.1.4.5 OTHER HORMONES

Atrial natriuretic factor (ANF) or peptide (ANP) is a hormone produced by the atria, along with brain or B-type natriuretic peptide (BNP) produced by the ventricular muscle cells, in response to an increase in vascular volume and thus distension and pressure.[7] ANF and BNP produce similar increases in glomerular filtration and pronounced natriuresis and diuresis to decrease intravascular volume by inhibition of aldosterone which also minimizes kaliuresis.

ANF and BNP also emphasize the heart's function as an endocrine organ.

4.1.5 Effects of the Various Mediators

4.1.5.1 HYPERDYNAMIC STATE

Following illness or injury, the systemic inflammatory response occurs, in which there is an increase in activity of the cardiovascular system, reflected as tachycardia, widened pulse pressure, and a greater cardiac output. There is an increase in the metabolic rate, with an increase in oxygen consumption, increased protein catabolism, and hyperglycaemia.

The cardiac index may exceed 4.5 L/min/m^2 after severe trauma in those patients able to respond. Ideally, decreases in vascular resistance accompany this increased cardiac output and there is an increase in oxygen delivery to the microcirculation. This hyperdynamic state elevates the resting energy expenditure to more than 20% above normal, total body oxygen consumption (VO_2) is increased and, due to the increase in metabolism, core temperature is increased. With an inadequate response, and a cardiac index of less than 2.5 L/min/m^2, oxygen consumption may fall to values of less than 100 mL/min/m^2 (normal = 120–160 mL/min/m^2). Endotoxins and anoxia may injure cells and limit their ability to utilize oxygen for oxidative phosphorylation.

The amount of adenosine triphosphate (ATP) synthesized by an adult is considerable. However, there is no reservoir of ATP or creatinine phosphate, and therefore, cellular injury and lack of oxygen results in rapid deterioration of processes requiring energy, and lactate is produced. Because of anaerobic glycolysis, only two ATP equivalents instead of 34 are produced from one mol of glucose in the Krebs cycle. Lactate is formed from pyruvate, which is the end-product of glycolysis. It is normally reconverted to glucose in the Cori cycle in the liver. However, in shock, the oxidation reduction (redox) potential declines and conversion of pyruvate to acetyl co-enzyme A for entry into the Krebs cycle is inhibited. Lactate therefore accumulates because of impaired hepatic gluconeogenesis, causing a metabolic acidosis.

Lactic acidosis after injury correlates with the ISS, and is an early and important clinical sign of acute blood loss, reflecting tissue hypoperfusion. Persistent lactic acidosis is indicative of inadequate resuscitation and predictive of the development of MOF and adult respiratory distress syndrome (ARDS).[8]

4.1.5.2 WATER AND SALT RETENTION

Secretion of ADH from the supra-optic nuclei in the anterior hypothalamus is stimulated by volume reduction and increased osmolality of the circulation. The latter is due mainly to increased sodium content of the extracellular fluid. Volume receptors are in the atria and pulmonary arteries, and osmoreceptors are located near ADH neurones in the hypothalamus. ADH acts mainly on the connecting tubules of the kidney but also on the distal tubules to promote reabsorption of water.

Aldosterone acts mainly on the distal renal tubules to promote reabsorption of sodium and bicarbonate and increased excretion of potassium and hydrogen ions.

Aldosterone also modifies the effects of catecholamines on cells, thus affecting the exchange of sodium and potassium across all cell membranes. The release of large quantities of intracellular potassium into the extracellular fluid may cause a significant rise in serum potassium especially if renal function is impaired. Retention of sodium and bicarbonate may produce metabolic alkalosis with impairment of the delivery of oxygen to the tissues. After injury, urinary sodium excretion may fall to 10–25 mmol/24 hours and potassium excretion may rise to 100–200 mmol/24 hours.

4.1.5.3 EFFECTS ON SUBSTRATE METABOLISM

4.1.5.3.1 Carbohydrates

Critically ill patients develop a glucose intolerance, which resembles that found in diabetic patients. This is a result of both increased mobilization and decreased uptake of glucose by the tissues. The turnover of glucose is increased, and the serum glucose is higher than normal.

As blood glucose rises during the phase of hepatic gluconeogenesis, blood insulin concentration rises, sometimes to very high levels. Provided that the liver circulation is maintained, gluconeogenesis will not be suppressed by hyperinsulinaemia or hyperglycaemia, because the accelerated rate of glucose production in the liver is required for clearance of lactate and amino acids, which are not able to be used for protein synthesis. This period of breakdown of muscle protein for gluconeogenesis and the resultant hyperglycaemia characterizes the catabolic phase of the metabolic response to trauma.

The glucose level following trauma should be monitored carefully in the intensive care unit (ICU). The optimum blood glucose level remains controversial, but the maximum level should be 10 mmol/L (see also Chapter 15). Excessive levels of glucose correlate directly with infectious complications, particularly in surgical or injury wounds. Control of the blood glucose is best achieved by titration with intravenous insulin, based on a sliding scale. However, because of the degree of insulin resistance associated with trauma, the quantities required may be considerably higher than normal. Nevertheless, over-aggressive control of blood glucose increases the risk of hypoglycaemia and must be avoided.

Enteral nutrition is preferred but parenteral nutrition may be required, and this will exacerbate the problem. However, glucose remains the safest energy substrate following major trauma: 60%–75% of the caloric requirements should be supplied by glucose, with the remainder being supplied as a fat emulsion.

4.1.5.3.2 Fat

A major source of energy following trauma is adipose tissue. Lipids stored as triglycerides in adipose tissue are mobilized when insulin falls below 25 units/mL. Initially, because of the suppression of insulin release by the catecholamine spike after trauma, as much as 200–500 g of fat may be broken down early after severe trauma.[9]

Catecholamines and glucagon activate adenyl cyclase in the fat cells to produce cyclic adenosine monophosphate (cyclic AMP). This activates lipase, which promptly hydrolyses triglycerides to release glycerol and fatty acids. Growth hormone and cortisol play a minor role in this process as well. Glycerol provides substrate for gluconeogenesis in the liver, which derives energy by β-oxidation of fatty acids, a process inhibited by hyperinsulinaemia.

The free fatty acids provide energy for all tissues and for hepatic gluconeogenesis.

4.1.5.3.3 Amino Acids

The intake of protein by a healthy adult is between 80 and 120 g of protein: 1 to 2 g protein/kg/day. This is equivalent to 13–20 g of nitrogen per day. In the absence of an exogenous source of protein, amino acids are principally derived from the breakdown of skeletal muscle protein. Following trauma or sepsis, the release rate of amino acids increases by three to four times. The process manifests as marked muscle wasting.

Cortisol, glucagon, and catecholamines play a role in this reaction. The mobilized amino acids are utilized for gluconeogenesis or oxidation in the liver and other tissues, but also for synthesis of acute-phase proteins required for immuno-competence, clotting, wound healing, and maintenance of cellular function.

After severe trauma or sepsis, as much as 20 g/day of urea nitrogen is excreted in the urine. Since 1 g urea nitrogen is derived from 6.25 g degraded amino acids, this protein wastage is up to 125 g/day.

One gram of muscle protein represents 5 g wet muscle mass. The patient in this example, would be losing 625 g of muscle mass per day. A loss of 40% of body protein is usually fatal, because failing immunocompetence leads to overwhelming infection. Nitrogen excretion usually peaks several days after injury, returning to normal after several weeks. This is a characteristic feature of the

metabolic response to illness. The most profound alterations in metabolic rate and nitrogen loss occur after burns and may persist for months.

4.1.5.3.4 The Gut

The intestinal mucosa requires rapid synthesis of amino acids. Depletion of amino acids results in atrophy of the mucosa causing failure of the mucosal barrier. This may lead to bacterial translocation from the gut to the portal system. The extent of bacterial translocation in trauma has not been defined.[10] The presence of food in the gut lumen is a major stimulus for mucosal cell growth. Food intake is invariably interrupted after major trauma, and the supply of glutamine may be insufficient for mucosal cell growth. Early nutrition (within 24–48 hours), and early enteral rather than parenteral feeding may prevent or reduce these events.

4.1.6 The Anabolic Phase

During this phase the patient is in positive nitrogen balance, regains weight, and restores fat deposits. The hormones, which contribute to anabolism, are growth hormones, androgens, and 17 beta-ketosteroids. The utility of growth hormone, and more recently, of insulin-like growth factor (IGF-1), in reversing catabolism following injury is critically dependent on adequate caloric intake.

4.1.7 Clinical and Therapeutic Relevance

Survival after injury depends on a balance between the extent of cellular damage, the efficacy of the metabolic response, and the effectiveness of treatment.

Tissue injury, hypoxia, pain, and toxins from invasive infection add to the initiating factor of hypovolaemia. The degree to which the body can compensate for injury is astonishing, although sometimes the compensatory mechanisms may work to the patient's disadvantage. Adequate resuscitation to shut off the hypovolaemic stimulus is important. However, once hormonal changes have been initiated, the effects of the hormones will not cease merely because hormonal secretion has been turned off by replacement of blood volume.

Mobilization and storage of the energy fuel substrates, carbohydrate, fats, and protein is regulated by insulin, balanced against catecholamines, cortisol, and glucagon. However, infusion of hormones has failed to cause more than a modest response. Rapid resuscitation, maintenance of oxygen delivery to the tissues, removal of devitalized tissue or pus, and control of infection are the cornerstones. The best metabolic therapy is excellent surgical care.

4.2 SHOCK

4.2.1 Definition of Shock

Shock is defined as inadequate delivery of oxygenated blood to the tissues, resulting in cellular hypoxia. This at first leads to reversible ischaemic-induced cellular injury. If the process is sufficiently severe or protracted, it ultimately results in irreversible cellular and organ injury and dysfunction. The precise mechanisms responsible for the transition from reversible to irreversible injury and death of cells are not clearly understood, although the biochemical/morphological sequence in the progression of ischaemic cellular injury has been well elucidated.[11] By understanding the events leading to cell injury and death, we may be able to intervene therapeutically in shock by protecting sub-lethally injured cells from irreversible injury and death.

4.2.2 Classification of Shock

The classification of shock is of practical importance if the pathophysiology is understood in terms that make a fundamental difference in treatment. Although the basic definition of shock, 'insufficient nutrient flow', remains inviolate, six types of shock, based on a distinction not only in the pathophysiology, but also in the management of the patients, are recognized:

1. Hypovolaemic.
2. Cardiogenic.
3. Cardiac compressive (e.g. cardiac tamponade).
4. Distributive (previously inflammatory) (e.g. septic shock).
5. Neurogenic.
6. Obstructive (e.g. mediastinal compression).

In principle, the physiological basis of shock is based on the following:

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate}$$

$$\text{Blood Pressure} \propto \text{Cardiac Output} \times \text{TotalPeripheral Resistance}$$

Table 4.1 Classes of Hypovalaemic Shock

Class	% Blood Loss	Volume	Pulse Rate	Blood Press.	Pulse Press.	Resp. Rate
Class I	15	<750 mL	<100	Normal	Normal	14–20
Class II	30	750–1500 mL	>100	Normal	Increased	20–30
Class III	40	2000 mL	>120	Decreased	Narrowed	30–40
Class IV	>40	>2000 mL	>140	Decreased	Narrowed	>35

Stroke volume is determined by the pre-load, the contractility of the myocardium, and by the afterload.

4.2.2.1 HYPOVOLAEMIC SHOCK

Hypovolaemic shock is caused by a decrease in the intravascular volume. This results in significant degeneration of both pressure and flow. It is characterized by significant decreases in filling pressures with a consequent decrease in stroke volume. Cardiac output is temporarily maintained by a compensatory tachycardia. With continuing hypovolaemia, the blood pressure is maintained by reflex increases in peripheral and, importantly, splanchnic vascular resistance and myocardial contractility mediated by neurohumoral mechanisms.

Hypovolaemic shock is divided into four classes (see Table 4.1).

Initially, the body compensates for shock, and Class I and Class II shock is compensated shock. When the blood volume loss exceeds 30% (Class III and Class IV shock), the compensatory mechanisms are no longer effective and the decrease in cardiac output causes a decreased oxygen transport to peripheral tissues. These tissues attempt to maintain their oxygen consumption by increasing oxygen extraction. Eventually, this compensatory mechanism also fails, and tissue hypoxia leads to lactic acidosis, hyperglycaemia, and failure of the sodium pump with swelling of the cells from water influx.

4.2.2.1.1 Clinical Presentation

The classic features of hypovolaemic shock are hypotension, tachycardia, pallor secondary to vasoconstriction, sweating, cyanosis, hyperventilation, confusion, and an oliguria. Cardiac function can be depressed without gross clinical haemodynamic manifestations. The heart shares in the total body ischaemic insult. Systemic arterial hypotension increases coronary ischaemia, causing rhythm disturbances and decreased myocardial performance. As the heart fails, left ventricular end-diastolic pressure rises, ultimately causing pulmonary oedema. Hyperventilation may maintain arterial PaO_2 at near normal levels but the $PaCO_2$ falls to 20–30 mm Hg (2.7–4 kPa). Later, pulmonary insufficiency may supervene from alveolar collapse and pulmonary oedema, resulting from damaged pulmonary capillaries, cardiac failure, or inappropriate fluid therapy.

Renal function is also critically dependent on renal perfusion. Oliguria is an inevitable feature of hypovolaemia. During volume loss, renal blood flow falls correspondingly with the blood pressure. Anuria sets in when the systolic blood pressure falls to around 50 mm Hg. Thus, urine output is a good indicator of peripheral perfusion. Oliguria in the hypovolaemic patient is a sign of renal success not failure.

4.2.2.2 CARDIOGENIC SHOCK

When the heart fails to produce an adequate cardiac output, even though the end diastolic volume is normal, cardiogenic shock is said to be present. Intravascular obstructive shock results when intravascular obstruction, excessive stiffness of the arterial walls, or obstruction of the microvasculature imposes an undue burden on the heart. The obstruction to flow can be on either the right or the left side of the heart. Causes include pulmonary embolism, air embolism, ARDS, aortic stenosis, calcification of the systemic arteries, thickening or stiffening of the arterial walls as a result of the loss of elastin and its replacement with collagen (as occurs in old age), and obstruction of the systemic microcirculation as a result of chronic hypertension or the arteriolar disease of diabetes.

Cardiac function is often impaired in shocked patients even if myocardial damage is not the primary cause. Reduced myocardial function in shock includes dysrhythmias, myocardial ischaemia from systemic hypotension and variations in blood flow, and myocardial lesions from high circulatory levels of catecholamines, angiotensin, and other myocardial depressant factors, such as DAMPs and other inflammatory mediators.

The reduced cardiac output can be a result of:

- Reduced stroke volume.
- Impaired myocardial contractility due to ischaemia, reperfusion induced oedema, infarction, cardiomyopathy or direct trauma.
- Altered ejection fraction.
- Coronary air embolism.
- Mechanical complications of acute myocardial infarction – acute mitral valvular regurgitation, ventricular septal rupture, or trauma.
- Arrhythmias.
- Conduction system disturbances (bradydysrhythmias and tachydysrhythmias).

Other forms of cardiogenic shock include those clinical examples in which the patient may have a nearly normal resting cardiac output but cannot raise the cardiac output under circumstances of stress because of poor myocardial reserves or an inability to mobilize those myocardial reserves due to pharmacologic beta-adrenergic blockade, for example propanolol for hypertension. Heart failure and dysrhythmias are discussed in depth elsewhere in this book.

4.2.2.2.1 Clinical Presentation

The clinical picture will depend on the underlying cause. Clinical signs of peripheral vasoconstriction are prominent, pulmonary congestion is frequent, and oliguria is almost always present. Pulmonary oedema may cause severe dyspnoea, central cyanosis, and crepitations, audible over the lung fields and lung oedema visible on x-rays. A systolic murmur appearing after myocardial infarction suggests mitral regurgitation or septal perforation.

Haemodynamic findings consist of a systolic arterial pressure less than 90 mm Hg, decreased cardiac output, usually less than 1.8 L/min/m^2, and a pulmonary arterial wedge pressure (PAWP) of greater than 20 mm Hg. However, cardiogenic shock can occur without the PAWP being elevated. This may be a result of excess diuretic therapy, plasma volume depletion by fluid lost into tissues (i.e. third spacing), or blood loss. Patients with relative hypovolaemia below the levels where there is a risk of pulmonary oedema, and, patients with significant right ventricular failure will also not have elevated PAWP. These patients, although their shock is cardiogenic, will respond dramatically to plasma volume expansion and will deteriorate if diuresis is attempted.

4.2.2.3 CARDIAC COMPRESSIVE SHOCK

The pathophysiology of cardiac compressive shock is very different from cardiogenic shock. External forces compress the thin-walled chambers of the heart (the atria and the right ventricle), the great veins (systemic or pulmonary), or any combination of these. Impaired diastolic filling will result. Clinical conditions capable of causing compressive shock include pericardial tamponade, tension pneumothorax, positive pressure ventilation with large tidal volumes or high airway pressures (especially in a hypovolaemic patient), an elevated diaphragm (as in pregnancy), displacement of abdominal viscera through a ruptured diaphragm, and the abdominal compartment syndrome (e.g. from ascites, abdominal distension, abdominal or retroperitoneal bleeding, or a stiff abdominal wall, as in a patient with deep burns to the torso).

The consequence of this compression is an increase in right atrial pressure, without an increase in volume, impeding venous return and reducing end diastolic volumes and provoking hypotension.

4.2.2.3.1 Clinical Presentation

Cardiac tamponade follows blunt or penetrating trauma and is a classic example of compressive cardiac shock. As a result of the presence of blood in the pericardial sac, the atria are compressed and cannot fill adequately. The systolic blood pressure is less than 90 mm Hg, there is a narrowed pulse pressure and a pulsus paradoxus exceeding 10 mm Hg. Distended neck veins may be present, unless the patient is hypovolaemic as well. Heart sounds are muffled. The limited compliance of the pericardial sac means that a very small amount (<25 mL blood) may be enough to cause decompensation. Similarly, tension pneumothorax can also produce compressive heart failure. In the patient with chest trauma and hypotension, the problem usually can be identified immediately, from decreased breath sounds, hyper-resonance of the affected side, and displacement of the trachea to the opposite side. Neck veins also may be distended. Immediate release of the tension, prior to waiting for an x-ray is required to prevent cardiac arrest.

4.2.2.4 DISTRIBUTIVE (INFLAMMATORY) SHOCK

Dilatation of the capacitance reservoirs in the body occurs with endotoxic shock, or prolonged hypovolaemic shock. Endotoxin can have a major effect on this form

of peripheral pooling and even though the blood volume is normal, the distribution of that volume is changed so that there is insufficient nutrient flow to meet aerobic metabolism needs.

Ultimately, all shock leads to cellular defect shock. Aerobic metabolism takes place in the cytochrome system in the cristae of the mitochondria. Oxidative phosphorylation in the cytochrome system produces high-energy phosphate bonds by coupling oxygen and glucose, forming the freely diffusable by-products carbon dioxide and water. Several poisons uncouple oxidative phosphorylation but the most common in clinical practice is endotoxin. Sepsis is frequent in hospitalized patients and endotoxic shock is distressingly common. There is fever; tachycardia may or may not be present, the mean blood pressure is usually below 60 mm Hg, yet the cardiac output varies between 3 and 6 L/m^2/min. This haemodynamic state is indicative of low peripheral vascular resistance.

In addition to maldistribution of blood volume due to increase in capacitance and low peripheral resistance as causes of hypotension in septic shock, other causes inhibiting the cardiovascular system maintenance of cardiac output at a level enough to maintain normal blood pressure in sepsis include:

- Hypovolaemia due to fluid translocation from the blood into interstitial spaces.
- Elevated pulmonary vascular resistance owing to ARDS.
- Bioventricular myocardial depression manifested by reduced contractility and an inability to increase stroke-work.

The ultimate cause of death in septic shock is failure of energy production at the cellular level as reflected by a decline in oxygen consumption. It is not only the circulatory insufficiency that is responsible for this, but also the impairment of cellular oxidative phosphorylation by endotoxin or endogenously produced superoxides. There is a narrowing of arterial-mixed venous oxygen difference as an indication of reduced oxygen extraction, which often precedes the fall of cardiac output. Inadequate oxidative phosphorylation leads to anaerobic glycogenolysis and a severe metabolic acidosis owing to lactate.

4.2.2.5 NEUROGENIC SHOCK

Neurogenic shock is a hypotensive syndrome in which there is loss of α-adrenergic tone and dilatation of the arterial and venous vessels. The cardiac output is normal, or may even be elevated, but because the total peripheral resistance is reduced, the patient is hypotensive. The consequence is a reduced perfusion pressure. In trauma it often occurs owing to spinal cord injury.

A simple example of this type of shock is syncope ('vaso-vagal syncope'). It is caused by a strong vagal discharge resulting in dilatation of the small vessels of the splanchnic bed. The next cycle of the heart has less venous return so that the ventricle will not fill, and the next stroke volume does not adequately perfuse the cerebrum, causing a faint. No blood is lost, but there is a sudden increase in the amount of blood trapped in one part of the circulation where it is no longer available for perfusion to an obligate aerobic glycolytic metabolic bed – the central nervous system.

4.2.2.5.1 Clinical Presentation

Commonly seen with high spinal cord injury, the patient usually has weakly palpable peripheral pulses, warm extremities, brisk capillary filling, and may be anxious. The pulse pressure is wide, with both systolic and diastolic blood pressure being low. Heart rate is below 100 beats per minute, and there may even be a bradycardia. However, the diagnosis of neurogenic shock should only be made once other causes of shock have been ruled out, since the common cause is injury, and there may be other injuries present causing a hypovolaemic shock in parallel.

4.2.2.6 OBSTRUCTIVE SHOCK

Intravascular obstructive shock results when intravascular obstruction, excessive stiffness of the arterial walls, or obstruction of the microvasculature imposes an undue burden on the heart. Because of the decreased venous return, the atrial filling is reduced with consequent hypotension. The obstruction to flow can be on either the right or the left side of the heart. Causes include pulmonary embolism, air embolism, ARDS, aortic stenosis, calcification of the systemic arteries, thickening or stiffening of the arterial walls as a result of the loss of elastin and its replacement with collagen (as occurs in old age), and obstruction of the systemic microcirculation as a result of chronic hypertension or the arteriolar disease of diabetes. The blood pressure in the pulmonary artery or the aorta will be high; the cardiac output will be low.

4.2.3 Measurements in Shock

In physics, flow is directly related to pressure and inversely related to resistance. This universal flow formula is not dependent on the type of fluid and is applied to the flow of electrons. In electricity, it is expressed as Ohm's law. This law applies just as appropriately to blood flow.

$$Flow = \frac{\text{Pressure}}{\text{Peripheral resistance}}$$

From this law it can be deduced that shock is just as much a state of elevated resistance as it is a state of low blood pressure. However, the focus should remain on flow rather than simply on pressure, since most drugs that result in a rise in pressure do so by raising the resistance, which in turn decreases flow while simultaneously increasing work and oxygen consumption by the myocardium.

4.2.3.1 CARDIAC OUTPUT

Blood flow is dependent on cardiac output. Three factors determine cardiac output:

- Pre-load or the volume entering the heart.
- Contractility of the heart.
- After-load or the resistance against which the heart must function to deliver the nutrient flow.

These three factors are interrelated to produce the systolic ejection from the heart. Up to a point, the greater the pre-load, the greater the cardiac output. As myocardial fibres are stretched by the pre-load the contractility increases according to the Frank-Starling principle. However, an excessive increase in pre-load leads to symptoms of pulmonary/systemic venous congestion without further improvement in cardiac performance. The pre-load is a positive factor in cardiac performance up the slope of the Frank-Starling curve, but not beyond the point of cardiac decompensation.

Contractility of the heart is improved by inotropic agents. The product of the stroke volume and the heart rate equals the cardiac output. Cardiac output acting against the peripheral resistance generates the blood pressure. Diminished cardiac output in patients with pump failure is associated with a fall in blood pressure. To maintain coronary and cranial blood flow there is a reflex increase in systemic vascular resistance to raise blood pressure. An exaggerated rise in systemic vascular resistance can lead to further depression of cardiac function by increasing ventricular after-load. After-load is defined as the wall tension during left ventricular ejection and is determined by systolic pressure and the radius of the left ventricle. Left ventricular radius is related to end-diastolic volume, and systolic pressure to the impedance to blood flow in the aorta, or total peripheral vascular resistance.

However, the critical emphasis in the definition of shock is on flow, we need to find improved methods to measure flow.

4.2.3.2 INDIRECT MEASUREMENT OF FLOW

In many patients in shock, simply laying a hand upon their extremities will help to determine flow by the cold clammy appearance of hypoperfusion owing to increased resistance. Probably the most important clinical observation to indirectly determine adequate nutrient flow to a visceral organ will be the urine output.

The kidney responds to decreased nutrient flow with several compensatory changes to protect its own perfusion. Over a range of blood pressure, the kidneys maintain a nearly constant blood flow. If the blood pressure decreases, the kidney's autoregulation of resistance results in dilatation of the vascular bed. It keeps nutrient flow constant by lowering the resistance even though the pressure has decreased. This allows selective shunting of blood to the renal bed.

For practical purposes, if a patient is producing a normal quantity of normal quality urine, then they are not in shock.

Another vital perfusion bed that reflects the adequacy of nutrient flow is the brain itself. Since adequate nutrient flow is a necessary, but not the only requirement for cerebration, normal consciousness also can be used to evaluate the adequacy of nutrient flow in the patient with shock.

4.2.3.3 DIRECT MEASUREMENTS

4.2.3.3.1 Central Venous Pressure

Placement of a central venous line that will allow accurate measurement of the hydrostatic pressure of the right atrium following fluid boluses can help differentiate between the different shock states. The actual measurement, except at the extremes, is frequently inaccurate in determining intravascular volume and less important than the change in value, especially in the acute

resuscitation of a patient. Normal is 4–12 cm water. A value below 4 cm indicates that the venous system is empty, and thus the pre-load is reduced, usually as a result of dehydration or hypovolaemia (blood loss), while a high value indicates that the pre-load is increased, either as a result of a full circulation or due to pump failure (e.g. cardiogenic shock due to aetiologies such as tension pneumothorax, cardiac tamponade, or myocardial contusion).

Generally, if a patient in shock has both systemic arterial hypotension and central venous hypotension, the shock is due to volume depletion. On the other hand, if central venous pressure is high though arterial pressure is low, shock is not due to volume depletion and is more likely due to pump failure.

4.2.3.3.2 Routes

Cannulation of the central venous system is generally achieved using the subclavian, jugular, or femoral route.

4.2.3.3.2.1 Subclavian Access

The subclavian route is preferred in the trauma patient, particularly when the status of the cervical spine is unclear. Ultrasonic guidance is not required, as visualization of the anatomy may be difficult. It is ideal for the intensive care setting, where occlusion of the access site against infection is required. Lines are easy to secure, and the incidence of line sepsis is lower than any other route. Pitfalls include arterial puncture and pneumothorax.

4.2.3.3.2.2 Internal Jugular Access

The internal jugular route or, occasionally, the external jugular route is the one most commonly utilized by anaesthesiologists, preferably under ultrasonic guidance. It provides ease of access, especially under operative conditions. However, there are significant dangers in the trauma patient, especially where the cervical spine has not yet been cleared, and other routes may be preferable. The ability to occlude the jugular site, especially in the awake patient in the ICU, is however, more limited, and there is greater discomfort for the patient.

4.2.3.3.2.3 Femoral Access

The femoral route is easy to access, especially when the line also will be used for venous transfusion. However, the incidence of femoral vein thrombosis is high, and the line should not be left beyond 48 hours because of the risk of infection. Pitfalls include placing the cannula inside the abdominal cavity. This can be particularly misleading if blood is present inside the abdominal cavity, since aspiration of the cannula will yield blood, and a false sense of security! The use of ultrasound can reduce catheter insertion complications.

4.2.3.3.3 Systemic Arterial Pressure

Systemic arterial pressure reflects the product of the peripheral resistance and the cardiac output. Measurement can be indirect or direct.

Indirect measurement involves the use of a blood pressure cuff with auscultation of the artery to determine systolic and diastolic blood pressure.

Direct measurement involves placement of a catheter into the lumen of the artery, with direct measurement of the pressure.

In patients in shock, with an elevated systemic vascular resistance, there is often a significant difference obtained between the two measurements. In patients with increased vascular resistance, low cuff pressure does not necessarily indicate hypotension. Failure to recognize this may lead to dangerous errors in therapy.

Cannulation of the brachial artery is not recommended because of the potential for thrombosis and for ischaemia of the lower arm and hand.

4.2.3.3.4 Pulmonary Arterial Pressure

The right-sided circulation is a valveless system through which flows the entire cardiac output from the right side of the heart. It is rarely used in the initial resuscitation phase.

4.2.3.3.5 Cardiac Output

Cardiac output can be measured with the thermodilution technique.[12] A thermodilution pulmonary artery catheter has a thermistor at the distal tip. When a given volume of a solution that is cooler than the body temperature is injected into the right atrium, it is carried by the blood past the thermistor, resulting in a transient fall in temperature. The temperature curve so created is analyzed, and the rate of blood flow past the thermistor (i.e. cardiac output) can be calculated. By measuring the mixed oxygen saturation in the pulmonary artery, blood oxygen extraction across the circulation can be determined.

4.2.4 Endpoints in Shock Resuscitation[13]

The ultimate measurement of the impact of shock must be at the cellular level. The most convenient measurement is a determination of the blood gases.

Measurement of PaO_2, $PaCO_2$, pH, base deficit (BD), and arterial lactate will supply information on oxygen delivery and utilization of energy substrates. Both PaO_2 and $PaCO_2$ are concentrations – partial pressure of oxygen and carbon dioxide in arterial blood. If the $PaCO_2$ is normal, there is adequate alveolar ventilation. CO_2 is one of the most freely diffusable gases in the body and is not overproduced or under-diffused. Consequently, its partial pressure in the blood is a measure of its excretion through the lung, which is a direct result of alveolar ventilation. The PaO_2 is a similar concentration but it is the partial pressure of oxygen in the blood and not the oxygen content. A concentration measure in the blood does not tell us the delivery rate of oxygen to the tissues per unit of time without knowing something of the blood flow that carries this concentration.

For evaluation of oxygen utilization, however, data are obtainable from arterial blood gases that can indicate what the cells are doing metabolically, which is the most important reflection of the adequacy of their nutrient flow. The pH is the hydrogen ion concentration, which can be determined easily and quickly. Loss of buffering by base excess also mirrors excess acid load. Nevertheless, the pH, BD, and the two carbon fragment metabolites are very important indicators of cellular function in shock.

In shock, there is a fundamental shift in metabolism. When there is adequate nutrient flow, glucose and oxygen are coupled to produce in glycolysis the high-energy phosphate bonds necessary for energy exchange. This process of aerobic metabolism also produces two freely diffusable by-products – carbon dioxide and water – both of which leave the body by excretion through the lungs and the kidneys. Aerobic metabolism is efficient, therefore, there is no accumulation of any products of this catabolism, and a high yield of ATP is obtained from this complete combustion of metabolites.

When there is inadequate delivery of nutrients and oxygen, as occurs in shock, the cells shift to anaerobic metabolism within 3–5 minutes. There are immediate consequences of anaerobic metabolism in addition to its inefficient yield of energy. In the absence of aerobic metabolism, energy extraction takes place at the expense of accumulating hydrogen ion, lactate, and pyruvate, which have toxic effects on normal physiology. These products of anaerobic metabolism can be seen as the 'oxygen debt'. There is some buffer capacity in the body that allows this debt to accumulate within limits, but it must ultimately be shut off by adequate oxygen and nutrient delivery.

Acidosis has significant consequences in compensatory physiology. Oxyhaemoglobin dissociates more readily as hydrogen ions increase; however, there is a significant toxicity of hydrogen ions as well. Despite the salutary effect on oxyhaemoglobin dissociation, the hydrogen ion has a negative effect on oxygen delivery. Catecholamines speed up the heart's rate and increase its contractile force, and the product of this inotropic and chronotropic effect is an increase in cardiac output. Catecholamines, however, are physiologically effective at alkaline or neutral pH. Therefore, an acid pH inactivates this catecholamine method of compensation for decreased nutrient flow. Lactate acidosis in the best single predictor of blood loss initially, and inadequate restoration of homeostasis after several hours in the ICU.

4.2.5 Post-Shock and Multiple Organ Failure Syndromes

Although the primary consequences of sepsis following trauma and shock is the development of multiple organ failure as discussed elsewhere in this book, it is important to briefly reiterate the usual sequence of events following shock to enable logical discussion of the management of shock.

The ultimate cause of death in shock is failure of energy production as reflected by a decline in oxygen consumption (VO_2) to less than 100 mL/min/m^2. Circulatory insufficiency is responsible for this fall in energy, compounded by impairment of cellular oxidative phosphorylation by endotoxin and endogenously produced substances, such as toxic oxides.

In shock, whether hypovolaemic or septic, energy production is insufficient to satisfy cellular requirements. In the presence of oxygen deprivation and cellular injury, the conversion of pyruvate to Acetyl Co-enzyme A (Acetyl CoA) for entry into the Krebs cycle is inhibited. Lactic acid accumulates and the oxidation reduction potential falls. Although lactate is normally used by the liver via the Cori cycle to synthesize glucose, hepatic gluconeogenesis may fail in hypovolaemic or septic shock because of hepatocyte injury and inadequate circulation. Terminally, the lactic acidosis cannot necessarily be corrected by improvement of circulation and oxygen delivery once the cells are irreparably damaged.

In the low-output shock-state, plasma concentrations of free fatty acids and triglycerides rise to high levels because ketone production by beta-oxidation

of fatty acids in the liver is reduced, suppressing the acetoacetate-to-betahydroxybutarate ratio in the plasma.

The post-shock sequel of inadequate nutrient flow, therefore, is progressive loss of function. The rate at which this loss occurs depends upon the cell's ability to switch metabolism, to convert alternate fuels to energy, the increased extraction of oxygen from haemoglobin, and the compensatory collaboration of failing cells and organs whereby nutrients may be shunted selectively to more critical systems. Not all cells are equally sensitive to shock nor similarly refractory to restoration of function when adequate nutrient flow is restored. As cells lose function, the reserves of the organ composed of those cells are depleted until impaired function of the organ results. These organs function in systems and a 'system failure' results. Multiple systems failure occurring in sequence leads to the collapse of the organism.

4.2.6 Management of the Shocked Patient

The primary goal of shock resuscitation is the early establishment of adequate oxygen delivery (DO_2). The calculated variable of DO_2 is the product of cardiac output, and arterial oxygen content (CaO_2).

By convention, CO is indexed to body surface area and expressed as a cardiac index (CI), and when multiplied by CaO_2, yields an oxygen delivery index (DO_2I). Normal DO_2I is roughly 450 mL/min/m^2.

CaO_2 and DO_2I are calculated as follows:

$$CaO_2(\text{mL}\cdot O_2/\text{dL} = [\text{Hb}](\text{g/dL.}) \times 1.38\,\text{mL}\cdot O_2/\text{g Hb} \times SaO_2(\%) + \left[PaO_2(\text{mm Hg}) \times 0.003\ \text{mL}\cdot O_2/\text{mm Hg}\right]$$

$$DO_2I(\text{mL/min/m}^2) = CI(\text{L/min/m}^2) \times CaO_2(\text{mL/dL}) \times 10\,\text{dL/L}$$

where

Hb	haemoglobin concentration
SaO_2	haemoglobin oxygen saturation
PaO_2	arterial oxygen tension
0.003	solubility of O_2 in blood.

Early work demonstrated that the 'survivor' response to traumatic stress is to become hyperdynamic. Supranormal resuscitation based on the DO_2I was therefore proposed. Subsequent randomized controlled trials have failed to demonstrate improved outcomes with goal directed supranormal therapy, and, in fact, this strategy increased ACS, MOF, and death.

Best practice guidelines for shock resuscitation are summarized by the large-scale collaborative project called 'Glue Grant' that provides standard operating procedures for clinical care. The 'Glue Grant' study for shock resuscitation suggests using a CI >3.8 L/min/m^2 as the resuscitation goal.[14]

The purpose for distinguishing the different pathophysiologic mechanisms of shock becomes important when treatment must be initiated. The final aim of treatment is to restore aerobic cellular metabolism. This requires restoration of adequate flow of oxygenated blood (which is dependent on optimal oxygenation of sufficient red blood cells, i.e. haematocrit and adequate cardiac output). The initial focus is securing a patent airway and controlling ventilation to prevent inadequate alveolar ventilation and oxygenation.

Restoration of optimal circulating blood volume, enhancing cardiac output using inotropic agents and/or mean arterial pressure (MAP) through vasopressors, the correction of acid-base disturbances and metabolic deficits, and the combating of sepsis, are all vital in the management of the shocked patient.

4.2.6.1 OXYGENATION

The severely traumatized, hypovolaemic, or septic patient has an oxygen demand that may exceed twice the normal. However, the traumatized shocked patient usually cannot generate the additional respiratory effort required, and therefore often develops respiratory failure followed by a lactic acidosis owing to tissue hypoxaemia.

In some patients, an oxygen mask may be enough to maintain oxygen delivery to the lungs. In more severe cases, endotracheal intubation and ventilatory assistance may be necessary. It is important to distinguish between the need for *intubation*, and the need for *ventilation*. Early intubation is preferable to cardiac collapse.

4.2.6.1.1 Airway Indications for Intubation

- Obstructed airway.
- Inadequate gag reflex.

4.2.6.1.2 Breathing Indications for *Intubation*

- Inability to breathe. (e.g. paralysis, either spinal or drug induced).
- Tidal volume less than 5 mL/kg.

4.2.6.1.3 Breathing Indications for *Ventilation*

- Inability to oxygenate adequately.
- PaO_2 less than 60 mm Hg (7.9 kPa) on 40% O_2 or SpO_2 of less than 90% on oxygen.
- A respiration rate of 30 breaths or more per minute and excessive ventilatory effort.
- A $PaCO_2$ of greater than 45 (6 kPa) mm Hg with metabolic acidosis, or greater than 50 mm Hg (6.6 kPa) with normal bicarbonate levels.

4.2.6.1.4 Circulation Indication for Intubation

- Systolic blood pressure less than 75 mm Hg despite resuscitation.

4.2.6.1.5 Disability Indications for Intubation

- High spinal injury with inability to breathe.
- Coma (GCS <9/15).

4.2.6.1.6 Environmental Indication for Intubation

- Core temperature of <32°C.

If ventilatory support is instituted the goals are relatively specific.

The respiratory rate should be adjusted to ensure a $PaCO_2$ of between 35 and 40 mm Hg (4.6–5.3 kPa). This will avoid respiratory alkalosis and a consequential shift of the oxyhaemoglobin dissociation curve to the left, which results in an increased haemoglobin affinity for oxygen, and significantly decreases oxygen availability to tissues, which will require increased cardiac output to maintain tissue oxygenation. Respiratory alkalosis also causes vasoconstriction of cerebral vessels and a further decrease in oxygen delivery to the CNS. For this reason, hyperventilation and hypocapnoea are no longer seen as appropriate for the management of brain injury.

The arterial PaO_2 should be maintained between 80–100 mm Hg (10.6–13.2 kPa) with the lowest possible inspired oxygen concentration.

It has also been shown that increased respiratory effort requires a disproportionate share of the total cardiac output for the respiratory muscles and, therefore, other organs are deprived of necessary blood flow and lactic acidosis is potentiated. Mechanical ventilation tends to reverse this lactic acidosis.

4.2.6.2 FLUID THERAPY FOR VOLUME EXPANSION

The preferred in hospital strategy is balanced blood component therapy. In the pre-hospital setting this strategy is also implemented in some countries (primarily Helicopter Emergency Services (HEMS) operations). Crystalloid, that is, Ringer lactate should be discouraged in the initial treatment of trauma patients, if balanced blood component therapy is available.

4.2.6.2.1 Hypotensive Resuscitation

In 1994, Bickell et al.[15] concluded that patients with penetrating torso trauma in hypovolaemic shock who were not given intravenous (i.v.) fluids during transport and emergency department evaluation had a better chance of survival than those who received conventional volume resuscitation. However, the only difference in survival was in the subgroup with pericardial tamponade. In animal studies, intravenous fluids have been shown to inhibit platelet aggregation, dilute clotting factors, modulate the physical properties of thrombus, and cause increases in blood pressure that can mechanically disrupt clots. Thus, the benefit of restricted resuscitation volumes was possibly because the reduced blood pressure limited the amount of blood loss. However, in the typical civilian trauma centre, blunt trauma is the most common form of injury and inadequate resuscitation leads to further organ injury due to inadequate oxygen delivery, particularly with traumatic brain injury (TBI). Thus, while the ideal approach is not known, the optimum systolic blood pressure for a patient with uncontrolled haemorrhage appears to be approximately 90 mm Hg, MAP should be approximately 70 mm Hg, for both the military environment, and, likely, the civilian setting also. However, a pre-hospital study by Schreiber et al. suggests the benefit of an even lower systolic blood pressure of 70 mm Hg.[16] Care must be exercised in cardiac patients and the elderly.

4.2.6.3 ROUTE OF ADMINISTRATION

4.2.6.3.1 Intravenous devices

In principle, with all intravenous lines, the shorter the line and the wider the diameter of the cannula, the faster will be the flow. For the same bore of line, flow rates are reduced:

14 G via peripheral cannula	Full flow
14 G via 20 cm central line	33% reduction in flow
14 G via 70 cm central line	50% reduction in flow

A minimum of two lines are required in the severely injured or hypotensive patient. In all cases of hypovolaemic shock, two large bore peripheral lines are essential. A central line is most useful for monitoring but can be used for transfusion as well. The monitoring line should be a central venous line, inserted ideally via the subclavian route. The subclavian route is preferable, since this approach avoids any movement of the head in a patient whose neck has not yet been cleared. The jugular and femoral routes are less preferable because of issues in securing the lines, and earlier sepsis at the insertion site owing to movement.

4.2.6.3.1.1 Intraosseous Devices

Intraosseous infusion is the process of injecting directly into the marrow of a bone to provide a non-collapsible entry point into the venous system. This technique is used in emergency and military situations to provide fluids and medication when intravenous access is not feasible. A comparison of intravenous, intramuscular, and intraosseous routes of administration concluded that in children, the intraosseous route is demonstrably superior to the intramuscular route, and comparable to the intravenous route.[17]

Insertion in adults requires less than a minute, and flow rates of up to 125 mL/min have been achieved. The devices are for emergency resuscitation and should be removed within 24 hours.

Suitable devices include the B.I.G.® gun (WaisMed, Houston, TX, USA) and the EZ-IO® (Vidacare Corp., San Antonio, TX, USA).

4.2.6.4 PHARMACOLOGIC SUPPORT OF BLOOD PRESSURE

Stroke volume is controlled by ventricular preload, after-load, and contractility. Ventricular preload is influenced primarily by the volume of circulating blood, but after-load and contractility can be enhanced by pharmacological agents. Reducing the systemic vascular resistance with vasodilators can be a very effective means of improving cardiac output when systemic pressures or cardiac filling pressures are normal or elevated, but is not currently recommended for acute trauma.

4.2.6.4.1 Noradrenaline (Norepinephrine)

The preferred inotropic agent for acute trauma is noradrenaline (norepinephrine). It is a sympathetic neurotransmitter with potent inotropic effects. It activates myocardial β-adrenergic receptors and vascular α-adrenergic receptors. It is used in the treatment of shock and hypotension characterized by low systemic vascular resistance and is unresponsive to fluid resuscitation.

4.2.6.4.2 Adrenaline (Epinephrine)

Adrenaline is a natural catecholamine with both α- and β-adrenergic agonist activity. The pharmacological actions are complex, and it can produce the following cardiovascular responses:

- Increased systemic vascular resistance.
- Increased systolic and diastolic blood pressure.
- Increased electrical activity in the myocardium.
- Increased coronary and cerebral blood flow.
- Increased strength of myocardial contraction.
- Increased myocardial oxygen requirement.

The primary beneficial effect of adrenaline is peripheral vasoconstriction, with improved coronary and cerebral blood flow. It works as a chronotropic and inotropic agent. The initial dose is 0.03 μg/kg/min, titrated upwards until the desired effect is achieved.

4.2.6.4.3 Dopamine

Dopamine hydrochloride is a chemical precursor, of noradrenaline, that stimulates dopaminergic, β_1-adrenergic and α-adrenergic receptors in a dose dependent fashion. Low doses of dopamine (<3 μg/kg/min) produce cerebral, renal, and mesenteric vasodilatation, and venous tone is increased. Urine output is increased, but there is no evidence to show that this is in any way protective to the kidneys.

At doses above 10 μg/kg/min, however, the α-adrenergic effects predominate. This results in marked increases in systemic vascular resistance, pulmonary resistance, and increases in preload due to marked arterial, splanchnic, and venous constriction. It increases systolic blood pressure without increasing diastolic blood pressure or heart rate.

Dopamine is used for haemodynamically significant hypotension in the absence of hypovolaemia.

4.2.6.4.4 Dobutamine

Dobutamine is a synthetic sympathomimetic amine that has potent inotropic effects by stimulating β_1- and α_1-adrenergic receptors in the myocardium. Dobutamine mediated increases in cardiac output also lead to a decrease in peripheral vascular resistance. At a dose of 10 μg/kg/min,

dobutamine is less likely to induce tachycardia than either adrenaline or isoproterenol. Higher doses may produce a tachycardia. Dobutamine increases cardiac output and its lack of induction of noradrenaline release means that there is a minimal effect on myocardial oxygen demand. There is also increased coronary blood flow. Dobutamine in low doses has also been used as a renal protective agent. There is little evidence to support its use on its own, but it may be helpful in improving renal perfusion as an adjunct to the administration of high dose adrenaline

Dobutamine and dopamine have been used together. The combination of moderate doses of both (7.5 μg/kg/min) maintains arterial pressure with less increase in pulmonary wedge pressure than dopamine alone.

4.2.7 Prognosis in Shock

The prognosis of the shocked patient depends on the duration of the shock, the underlying cause, and the pre-existing vital organ function. The prognosis is best when the duration is kept short by early recognition and aggressive correction of the circulatory disturbance and when the underlying cause is known and corrected.

Occasionally, shock does not respond to standard therapeutic measures. Unresponsive shock requires an understanding of the potential occult causes of persistent physiologic disturbances.

These correctable causes include:

- Under-appreciated volume losses with inadequate fluid resuscitation and a failure to assess the response to a fluid challenge.
- Erroneous presumption of overload when cardiac disease is also present.
- Hypoxia caused by inadequate ventilation, barotrauma to the lung, pneumothorax, or cardiac tamponade.
- Undiagnosed or inadequately treated sepsis.
- Uncorrected acid-base or electrolyte abnormalities.
- Endocrine failure like adrenal insufficiency or hypothyroidism.
- Drug toxicity.

4.2.8 Recommended Protocol for Shock

4.2.8.1 MILITARY EXPERIENCE

Recent military experience from the Iraq war has shown the value of 'damage control resuscitation.[18] (See also Chapter 6: Damage Control.) This implies that damage control techniques are used from the time of injury, minimizing the time between injury and care, controlling the bleeding and contamination through use of minimal clear fluids, early fresh whole blood, early resuscitation, and early damage control surgery. The military use of whole blood has minimized some of the risks of component therapy and has also shown that survival is improved. From this philosophy has come the change in protocol in civilian practice towards minimizing crystalloid resuscitation (restrictive or limited resuscitation; not hypotensive) and early use of blood and blood products to maintain the normal coagulation profile as much as possible.

4.2.8.2 INITIAL RESUSCITATION

A. Major trauma patients arriving in shock (SBP <90 mm Hg and/or HR >130 bpm) are initially considered to be in haemorrhagic shock. Correction should be aimed at damage control through surgical control of bleeding, and damage control resuscitation (see also Chapter 6).

B. Patients with major torso trauma requiring ongoing resuscitation should have a central venous line placed in the emergency department when time allows for it.

C. Early CVP >15 mm Hg (before extensive volume loading) suggests cardiogenic or cardiac compressive shock.

D. CVP <10 mm Hg despite volume loading suggests ongoing bleeding. Endpoints are currently vague. At present the rational compromise is volume limited resuscitation (SBP = 90 mm Hg and HR <130 bpm) with moderate volume loading until haemorrhage is controlled.

E. Patients at risk for trauma induced coagulopathy (TIC) should have a massive transfusion protocol initiated (see also Section 5.7).

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Transfusion in Trauma 5

Transfusion of blood and blood components is a fundamental part of trauma management and approximately 40% of the 13 million units of blood transfused in the United States each year are used in emergency resuscitation. Despite this, there is limited evidence to provide a rationale for administration of packed red blood cells (pRBCs) to trauma patients.

5.1 INDICATIONS FOR TRANSFUSION

5.1.1 Oxygen-Carrying Capacity

Anaemia is a decrease in the O_2-carrying capacity of blood, defined by a decrease in circulating red cell mass (to below 24 mL/kg in females and 26 mL/kg in males). Anaemia will result in an increase in cardiac output at a haemoglobin (Hb) level of <7 g/dL (4.0 mmol/L). Oxygen extraction increases as O_2 delivery falls, ensuring a constant O_2 uptake by the tissues. Normal humans can survive an 80% loss of red cell mass if they are normovolaemic and normothermic. The threshold for O_2 delivery to maintain adequate tissue oxygenation, is at a haematocrit of 10% and a Hb level of 3 g/dL (1.8 mmol/L) when breathing 100% O_2 and with a normal metabolic rate.

Volume-dependent markers (such as haematocrit and Hb) are poor indicators of anaemia because of the effect of dilution after fluid transfusion on their values, that is, they are relative values.

5.2 TRANSFUSION FLUIDS

5.2.1 Colloids

5.2.1.1 STARCHES

The use of starches is contraindicated in the actively bleeding patient, since starches deplete the von Willebrand/factor VIII complex, and may make the actively bleeding patient more coagulopathic from both factor depletion and dilution coagulopathy. Hydroxyethyl starch is an independent risk factor for acute kidney injury and death after blunt trauma.

5.2.1.2 ALBUMIN

Human albumin has not been evaluated for acute resuscitation, although animal experimentation suggests it may be appropriate. The Saline versus Albumin Fluid Evaluation (SAFE) study in 2007, tested saline versus albumin in the intensive care unit (ICU) and suggested an increased mortality in trauma patients and particularly in patients with traumatic brain injury (TBI). Albumin is not routinely used in trauma resuscitation.[1]

5.2.2 Blood

5.2.2.1 FRESH WHOLE BLOOD (FWB)

Humans are O_2-dependent organisms, and O_2 depletion causes major damage within minutes. Thus, in the exsanguinating patient, red blood cells (RBCs) are transfused in order to improve O_2 transport, although older blood does not carry oxygen well. Evidence from military and civilian trauma studies has suggested the advantage of fresh whole blood (FWB) in the resuscitation and survival of the exsanguinating patient.[2] The rationale is that fresh whole blood has more function than purely that of an O_2 transport medium and provides:

- Oncotic pressure (from plasma).
- Clotting factors and platelets (PLT).
- Temperature homeostasis (from warm circulating fluid).
- Fresh whole blood offers blood at close to 37°C, RBCs, plasma, and platelets in natural proportions,

to cover the need of the exsanguinating patient for O_2 and oncotic pressure. A 500 mL unit of FWB has:

- A haematocrit of 38%–50%.
- 50,000–400,000/mm^3 fully functional platelets.
- 100% activity of clotting factors diluted only by the anticoagulant.
- Excellent oxygen carrying ability.

In addition, the viability and flow characteristics of fresh RBCs are better than their stored counterparts that have metabolic depletion and membrane dysfunction. However, FWB, unless in a military environment with availability of large numbers of healthy, pre-screened, young blood carriers, is generally not available. The levels of clotting factors V and VIII in FWB decline quickly for 24 hours after collection. The rate of decline then slows until clinically subnormal levels are reached within 7–14 days. It is because FWB contains these factors and is so effective in the correction of coagulopathy, that it is recommended for massive transfusion. The other clotting factors remain stable in stored blood. Fresh whole blood has lost most of its platelets after 3 days of storage. Fresh whole blood can, if warmed, be transfused within 24 hours, and considered still fresh if stored at 4°C for 48 hours.

5.2.2.2 PACKED RED BLOOD CELLS

Previous haemorrhage management transfused excessive amounts of crystalloids, which diluted native clotting factors, causing hypocoagulation.[3] This additional fluid aggravated the coagulopathy initiated from the moment of injury due to:

- Loss of warm blood and replacement with cooler fluid, resulting in decreased body temperature.
- Hypoperfusion, resulting in anaerobic metabolism, increased lactic acid production, and a decrease in pH.

Biochemical reactions within the body require a specific and narrow temperature and pH range to proceed. The coagulation cascade does not proceed, even in the presence of all the clotting factors, when the tissue pH is below 7.2 and temperature below 34°C. These factors are called trauma induced coagulopathy (TIC),[4] and differs from disseminated intravascular coagulopathy, which may develop after hours or days, when the septic component adds its consequences to trauma.

5.2.3 Component Therapy (Platelets, Fresh Frozen Plasma, Cryoprecipitate)

5.2.3.1 PLATELETS

A fall in platelet count occurs somewhat later than the loss of clotting factors. Hypothermia affects platelet adhesion more than enzymes, above 34°C, while hypothermia affects all aspects of coagulation below 34°C. There is general agreement that the indications for platelet transfusion are:

- *Prophylaxis*: If the platelet count <15,000/mm.
- *Pre-surgery*: Platelet count <50,000/mm^3.
- *Active bleeding*: Platelet count <100,000/mm^3.
- 1 unit increases the platelet count by 10,000/mm^3 platelets.

1 (5 units) mega-unit of apheresis platelets increases the platelet count by 50,000/mm^3.

5.2.3.2 FRESH FROZEN PLASMA

Massively bleeding trauma patients will need fresh frozen plasma (FFP) early. This is different from most recommendations, which are based on more controlled circumstances, and is founded on computer simulation of the amount of FFP required to avoid excessive plasma dilution compromising haemostasis.

Current evidence suggests that most patients will require 1 unit of FFP for every unit of blood transfused. A unit of FFP also contains most of the citrate anticoagulant from the unit of blood from which it was originally derived. It contains about 0.5 g fibrinogen, and normal levels of pro- and anticoagulants. Solvent-detergent-related/freeze-dried plasma carries about 20% less of the above per unit given. Potential advantages are:

- It contains all coagulation factors, but not all in equal concentration.
- It is preferred to cryoprecipitate, which contains 50% content of most normal coagulation factors, with the exception of fibrinogen, factor VIII and von Willebrand factor.

5.2.3.3 CRYOPRECIPITATE

Cryoprecipitate contains fibrinogen, von Willebrand factor/factor VIII complex, and fibrin stabilizing factor XIII. Cryoprecipitate may not be required in all cases of

trauma. One unit (250 mL) of FFP contains 0.5 g fibrinogen; 1 unit of cryoprecipitate contains 0.25 g fibrinogen, but in 10 mL (rather than 250 mL). Therefore, in most cases, FFP will meet the needs required. However, if a rapid increase in the amount of fibrinogen is required, cryoprecipitate is a useful adjunct.

The CRYOSTAT trials are currently in progress: CRYOSTAT-1 was a feasibility study, which suggested that early cryoprecipitate therapy maintained acceptable blood fibrinogen levels during active bleeding, with a signal for reduced mortality in the treatment arm of the study. CRYOSTAT-2 will test the effect of early cryoprecipitate (within 90 minutes of admission) compared to standard blood transfusion therapy and is due for completion in 2020.

5.2.3.4 FIBRINOGEN CONCENTRATE

Fibrinogen concentrate can be used to correct acquired hypofibrinogenaemia in trauma. There are several guidelines, but evidence is lacking regarding treatment efficacy and safety in trauma.

5.3 EFFECTS OF TRANSFUSING BLOOD AND BLOOD PRODUCTS

Stored pRBCs (stored for a maximum of 42 days with current US Food and Drug Administration approved storage solutions) develop defects proportionate to the duration of storage that assume greater clinical significance when transfused rapidly, or in large quantities, such as in critically ill patients.

5.3.1 Metabolic Effects

- There is a storage related decreased ATP which precedes red blood cell membrane deformability and its survival during storage.
- Degradation of 2,3-diphosphoglycerate (2,3-DPG) has occurs after 7–10 days in storage. 2,3-DPG is an enzyme affecting the affinity of Hb for O_2. After 7 days of storage, the O_2-transporting ability of Hb drops by two-thirds. Adenine added to pRBCs may restore levels of 2,3-DPG *in vivo* after transfusion.
- Increased ammonia release occurs owing to the release of intracellular protein after disruption of the red cell membrane during storage.

5.3.2 Effects of Microaggregates

This remains controversial; however, microfilters are no longer used during transfusion to remove microaggregates or red cell debris after membrane rupture. Microaggregates are created from:

- Red cell membrane instability which leads to cell rupture.
- Increased amounts of microaggregates (platelets/leukocytes/fibrin debris) in the buffy coat.
- Impaired pulmonary gas exchange and adult respiratory distress syndrome (ARDS) and transfusion-related lung injury (TRALI) can occur.
- Reticulo-endothelial system (RES) depression.
- Activation of complement and coagulation cascades.
- Production of vasoactive substances.
- Antigenic stimulation.
- Acute-phase response.

5.3.3 Hyperkalaemia

Serum potassium levels rise in stored blood as the efficiency of the Na^+/K^+ pump decreases. Transfused blood may have a potassium concentration of >40 mmol/L. Transient hyperkalaemia may occur as a result, but often does not need correction.

5.3.4 Coagulation Abnormalities

Fresh frozen plasma contains all the clotting factors of the coagulation cascade. Thawed plasma is FFP brought to 4°C and stored for 5 days, this timeline being based on the lifetime of factors V and VIII. Recent studies have shown that thawed plasma stored at this temperature retains significant clotting function for up to 14 days.

- Thrombocytopenia and a loss of factors V and VIII in stored blood may contribute to the coagulopathy. Platelets have a short half-life, and their functioning is usually minimal after 3–5 days of storage.
- Levels of clotting factors V and VIII decline quickly for 24 hours after collection. The rate of decline slows until clinically subnormal levels are reached at 7–14 days.
- Packed red cells do not contain platelets as these are generally spun off, and whole blood has lost most of its platelets after 3 days of storage. Spontaneous bleeding rarely occurs if the platelet count is greater

than 30,000/mm³. Levels as low as this are seen after the replacement of one to two times the total blood volume and may result from dilution. Despite this, the body has large reserves of platelets, sequestrated in the spleen, liver, and endothelium that are mobilized when there is a need.
- In whole blood, platelets may contribute to microaggregates that find their way to the lungs. Their presence is less evident in packed red cells. Transfusion of pooled platelets carries a greater risk of infection, as several donors have contributed to a single pack of platelets.

It is important to detect trauma induced coagulopathy as early as possible, and coagulation tests, such as prothrombin time/international ratio (PT/INR), activated partial thromboplastin time (APTT), fibrinogen concentration, and platelet counts have traditionally been used. However, there is a striking lack of evidence to support the use of conventional coagulation tests (CCTs) to monitor blood component use in trauma. Increasingly, the transfusion of blood components is guided by viscoelastic haemostatic assays (VHA) such as thrombo-elastography (TEG or RoTEM). This is most relevant where surgical bleeding is controlled; however, in the face of continued bleeding, despite surgical control, blood products may need to be given empirically. VHA may be performed at point of care and provide results within minutes.

5.3.5 Other Risks of Transfusion

5.3.5.1 TRANSFUSION-TRANSMITTED INFECTIONS

- Hepatitis A, B, C, and D.
- Human immunodeficiency virus 'window period'.
- Cytomegalovirus.
- Atypical mononucleosis and a swinging temperature that can be present for 7–10 days post-transfusion.
- Malaria.
- Brucellosis.
- Yersinia.
- Syphilis.

5.3.5.2 HAEMOLYTIC TRANSFUSION REACTIONS

- Incompatibility: ABO, rhesus (blood type), and 26 other surface antigens (screen for these).
- To very cold blood, overheated blood, or pressurized blood.
- Immediate generalized reaction (plasma).

5.3.5.3 IMMUNOLOGICAL COMPLICATIONS

- Major incompatibility reaction (usually caused by 'wrong blood' owing to administrative errors).
- Graft-versus-host disease: TRALI.
- Immunomodulation: reports on transplant and oncology patients have provided evidence that transfusion induces a regulatory immune response in the recipient that increases the ratio of suppressor to helper T cells. These changes may render the trauma patient more susceptible to infection.

5.3.5.4 FACTORS IMPLICATED IN HAEMOSTATIC FAILURE

- *Hypothermia*: Blood is stored at 4°C, but body temperature is 37°C, so the body needs to provide 1255 kJ of energy to heat each unit of blood to body temperature.
- Acidosis (from citrate and lactate).
- Dilution, depletion, and decreased production of red cells and platelets.
- *Diffuse intravascular coagulation*: There is a consumption of clotting factors and platelets within the circulation, which are trapped in the microvascular thrombi created due to fibrin disposition.
- *Extrinsic*: Tissue thromboplastins, for example, blunt trauma and surgery, and burns.
- *Intrinsic*: Endothelial injury, endotoxin, hypothermia, hypoxia, acidosis, and platelet activation.
- Fibrinolysis.
- Consumption of red cells and platelets.
- Protein C activation.

Despite the extensive list quoted above, there is limited evidence regarding the risks of pRBC transfusion, although pRBC transfusion is an independent risk factor for:

- Increased nosocomial infections (wound infection, pneumonia, sepsis).
- Multiple organ failure and systemic inflammatory response syndrome.
- Longer ICU and hospital length of stay, increased complications, and increased mortality.
- Pre-storage leukocyte depletion of RBC transfusion reduces complication rates, some studies showing a reduction in infectious complications.
- There is a relationship between transfusion and TRALI and ARDS.
- Transfusion and pRBC and FFP increase the risk for DVT in trauma patients.

Blood has effects and side effects, some of which are 'bad', and we should use it in a rational and restrictive way in most patients. In trauma haemorrhage, there is no good alternative so far and prioritizing balanced transfusion as part of haemostatic resuscitation secures clotting and oxygenation. The less 'bad' blood may be FWB, and its surrogate components transfusion (which amounts to a reconstitution of whole blood). However, FWB is not generally available outside the military context, and may be logistically difficult to obtain in civilian practice.

5.4 CURRENT BEST TRANSFUSION PRACTICE

5.4.1 Initial Response

1. Aggressively pursue the diagnosis and treatment of haemorrhage.
2. Titrate administered fluids to maintain a lower than normal blood pressure (hypotensive resuscitation), until control of haemorrhage is achieved (see also Chapter 6).
3. Measure and closely follow serum lactate and arterial pH as indicators of the state of systemic perfusion.
   - If normal, attempt to maintain perfusion.
   - If abnormal, attempt a gradual improvement without elevating the blood pressure and aggravating the haemorrhage.
4. Maintain normothermia.
5. Control ventilation to achieve O_2 saturation of 99%–100% and a normal end-tidal carbon dioxide level.
6. Aim for a target Hb of 7–9 g/dL (4–5.5 mmol/L) and a normal prothrombin time at the time that haemorrhage is controlled. Consider maintaining a higher Hb concentration in older patients and in those with known ischaemic disease.
7. If massive transfusion is likely, attempt from the outset to maintain the composition of whole blood. Use early RBC, plasma, and platelet transfusion.

Blood pressure and heart rate are the current standard-of-care monitors of shock resuscitation in the field, and in the emergency department when associated with serum lactate or base excess (base deficit). Both are, however, insensitive markers of early compensated shock; alternative monitors are needed for assessing the adequacy of tissue perfusion, with a view to avoiding both under-resuscitation and over-resuscitation. The challenge is to identify as early as possible those patients who are not responding to early interventions. Blind and aggressive volume loading in the hope of normalizing blood pressure and heart rate, without appropriate emphasis on the control of haemorrhage, sets the stage for the so-called bloody vicious cycle, the abdominal compartment syndrome, or multiple organ failure (MOF).

5.4.2 Reduction in the Need for Transfusion

Blood is a scarce (and expensive) resource and is also not universally safe. Reducing the need for transfusion is the best way to limit complications:

- Treat the cause, that is, stop bleeding, and avoid hypothermia and acidosis.
- Treat deficiencies and complications as they arise. There is no evidence to support prophylactic therapy with FFP, platelets, etc.;[5] however, replacement of components becomes of great importance in massive transfusion.
- Follow a restrictive transfusion policy in ICU. One large multicentre trial documented a significantly lower mortality rate for critically ill patients managed with a restrictive transfusion strategy and a transfusion threshold of 7 g/dL (5 mmol/L) Hb.[6] However, this assumes normovolaemia, absence of ongoing bleeding, and an absence of pre-existing cardiovascular disease or severe brain injury.
- Develop a capacity for cell salvage.

5.4.3 Transfusion Thresholds

There is no level I evidence indicating the ideal trigger for transfusion in trauma patients. In general, the following guidelines apply:[7]

1. Identify the critically ill patient with a Hb less than 7 g/dL (5 mmol/L) or a haematocrit below 21%.
2. If the Hb is less than 7 g/dL, transfusion with pRBCs is appropriate.[8] For patients with severe cardiovascular disease, and trauma patients with ongoing bleeding or haemodynamic instability, a higher threshold of 8–10 g/dL (6–7 mmol/L) is appropriate, although this remains a non-evidence-based extrapolation of the TRICC study where they specifically excluded actively bleeding patients.
3. If the Hb is less than 7 g/dL, assess the patient for hypovolaemia. If this is found, administer

intravenous fluids to achieve normovolaemia, and reassess the Hb level.
4. If the patient is not hypovolaemic, determine whether there is evidence of impaired O_2 delivery.
5. If impaired O_2 delivery is present, consider cardiac output monitoring, using a Vigileo® (Edwards Life Sciences, Irvine, CA), or a similar monitor.
6. If impaired O_2 delivery is not present, monitor Hb as appropriate.

5.4.4 Transfusion Ratios

While principle largely favours FWB, blood component transfusion is the best feasible alternative in most civilian situations.

Military trauma[9] studies suggest transfusing pRBC:FFP:platelets in a proportion of 1:1:1, with life threatening bleeding, but the only randomized clinical trial failed to establish improved survival at 2 hours or 30 days.

The optimal ratio of RBCs to FFP remains, however, controversial. Currently, a ratio of 1:1:1/RBC:FFP:platelets appears to be reasonable.[10,11] If apheresis platelets (usually containing 5 or 6 units of platelets) are supplied, this ratio will become 5:5:1 or 6:6:1.

5.4.5 Adjuncts to Enhance Clotting

There has been extensive interest in the provision of adjuncts to enhance clotting as part of the resuscitation of the trauma patient. These include:

5.4.5.1 RECOMBINANT ACTIVATED FACTOR VII (rFVIIa)

Interest has focused on recombinant activated factor VIIa (rVIIa®) (NovoSeven). This was initially developed as an adjunct for the treatment of haemophilia. However, following its successful use in controlling the bleeding in a trauma patient, there was considerable interest in its use. A large multi-centre trial in 2005[12] showed a reduction in red cell transfusion requirements in blunt trauma patients, and the drug has been used extensively 'off-label'. A further large trial in 2008 showed a reduction in blood product usage of 3.6 units in blunt injury, but the study sample was too small to show significance on mortality, or for penetrating injury.[13] Consequently, rFVIIa is not widely used, but it is still utilized in certain countries, and in some military situations. A suitable protocol appears in Table 5.1.

5.4.5.2 TRANEXAMIC ACID (TXA)

Tranexamic acid is indicated for prolonged bleeding (empirically) or when there is evidence of hyperfibrinolysis (measured using TEG or RoTEM – see below).

The CRASH-2 trial showed a significant reduction in mortality with the use of tranexamic acid;[14] however, although the trial involved very large numbers of subjects, fewer than half the patients required red cell transfusion, in those who were transfused, the two arms utilized the same amount of blood, and the mortality rate in both arms did not correlate with that in other studies. In addition, no injury severity comparisons were included. Study of the use of TXA in the military context showed improved coagulation and survival, especially in those patients requiring massive transfusion (The MATTERs Study).[15]

However, a recent study indicated that most severely injured patients have a fibrinolysis shutdown (i.e. become hypercoagulable), so that TXA may have no effect.[16] No clear benefit has been shown when used in an urban environment, with access to major trauma centres.[17] The association between TXA treatment and an increased risk of vascular occlusive events is still unknown.

Current recommendations suggest administration of TXA:

- Within 3 hours from time of injury.
- At a dose of 1 gram intravenously administered over 10 minutes, then 1 gram intravenously administered over 8 hours.
- In adult trauma patients with severe haemorrhagic shock (SBP <75 mm Hg), with known predictors of fibrinolysis, or verified fibrinolysis by TEG (LY30).[18]

5.4.5.3 DESMOPRESSIN (DDAVP)

Desmopressin potentiates the function of platelets and is indicated only for functional platelet disorders, secondary to platelet inhibitors such as aspirin, clopidogrel, ticegrelor, prasugerl, etc., renal or hepatic failure, haemophilia-A and von Willebrand's disease.

5.4.6 Monitoring the Coagulation Status: Traditional and VHA

Ideally, the use of blood components should be guided by laboratory tests of clotting function. This is part of the concept of personalized medicine, or in trauma as

Table 5.1 Guidelines for the Use of Recombinant Activated Factor VII (rFVIIa) in Trauma

Definition

This guideline describes the use of rFVIIa as an *adjunct* in the management of coagulopathy following trauma with massive bleeding or the need to enter the massive transfusion protocol.

Issue

The blood bank will issue the required rFVIIa for administration immediately **after completion** of the 6th and 12th units of transfused blood.

Limitation

rFVIIa should **only** be used:

- **If all *surgical* bleeding has been controlled.**
- **In the presence of active bleeding.**
- **Where possible, its use should be backed up with a thromboelastogram (TEG).**
- Increased R (reaction) time despite fresh frozen plasma.
- After transfusion of >6 units of blood.
- If the platelet count is >50,000/mm^3.
- If the pH is >7.2.
- If the temperature is >34°C.

Blood specimens

Disseminated intravascular coagulopathy screen:

- Full blood count and platelets.
- Fibrinogen.
- TEG or RoTEM or if not available.
- Prothrombin time, activated partial thromboplastin time, thrombin time, international normalized ratio, D-dimer.

Dose

The dose of rFVIIa should be 90 μg/kg, but may be as high as 120 μg/kg:

- Round UP to the nearest 1.2 mg.

  (Example: a 75 kg male receives 75 × 90 μg/kg = 6.75 mg rFVIIa. Round up to 7.2 mg.)

If the patient continues to bleed:

- Repeat the dose after 1 hour and after 3 hours from first dose.
- Repeat the dose after completion of the **12th** unit of transfused blood.

End points of administration

The first of:

- Cessation of bleeding;

  or
- Three doses.

part of the concept of goal-directed therapy, giving the patient only what is needed and avoiding transfusions that are not needed. Haemostasis, according to the cell-based model, is described in the phases of initiation, amplification, and propagation, from clot formation to clot lysis, with participation of all circulating plasmatic and cellular components. Thrombin generation is central for clot development and strength. It primarily occurs on the surface of activated platelets and, hence, platelets and thrombin generation are closely related to the development of coagulopathy.

5.4.6.1 TRADITIONAL ASSAYS

- International normalized ratio (INR) – extrinsic.
- Partial thromboplastin time (PTT) – intrinsic.
- D-dimer values (fibrinolysis).

All the above are cost effective if frequently done, but specimens are heated to the normal *in vivo* temperature of 37°C. All are time consuming. Therefore, the conventional approach is inappropriate for the hypothermic trauma patient, whose coagulation status

changes rapidly with alterations in pH and temperature. Therefore, TEG/ROTEM is more effective at covering the assessment need in the trauma patient and providing an adjunct to goal-directed haemostasis.

5.4.6.2 VISCOELASTIC HAEMOSTATIC ASSAYS (VHA): THROMBOELASTOGRAPHY (TEG)/ROTARY THROMBOELASTOMEROGRAPHY (ROTEM)[19,20]

The VHA technology results in a visual profile, or trace, and variables with a reference value, see Figure 5.1a,b. Briefly, the collected whole blood sample is placed in a special designed small cup (approximately 1 cc). Inside the cup is suspended a pin connected to a detector system (a torsion wire in TEG, an optical detector in ROTEM), and the cup and pin are oscillated relative to each other with movement initiated from either the cup (TEG) or the pin (ROTEM). As fibrin strings form between the cup and pin, the transmitted rotation from the cup to pin (TEG) or the impedance of the rotation of the pin (ROTEM) are detected at the pin and a trace is generated as seen in Figure 5.2. The standard assays are with kaolin activation in TEG, kaolin + tissue factor in RapidTEG® and tissue factor or kaolin activation, respectively, in the ExTEM and InTEM assays in ROTEM, and several other dedicated assays are available from both technologies.

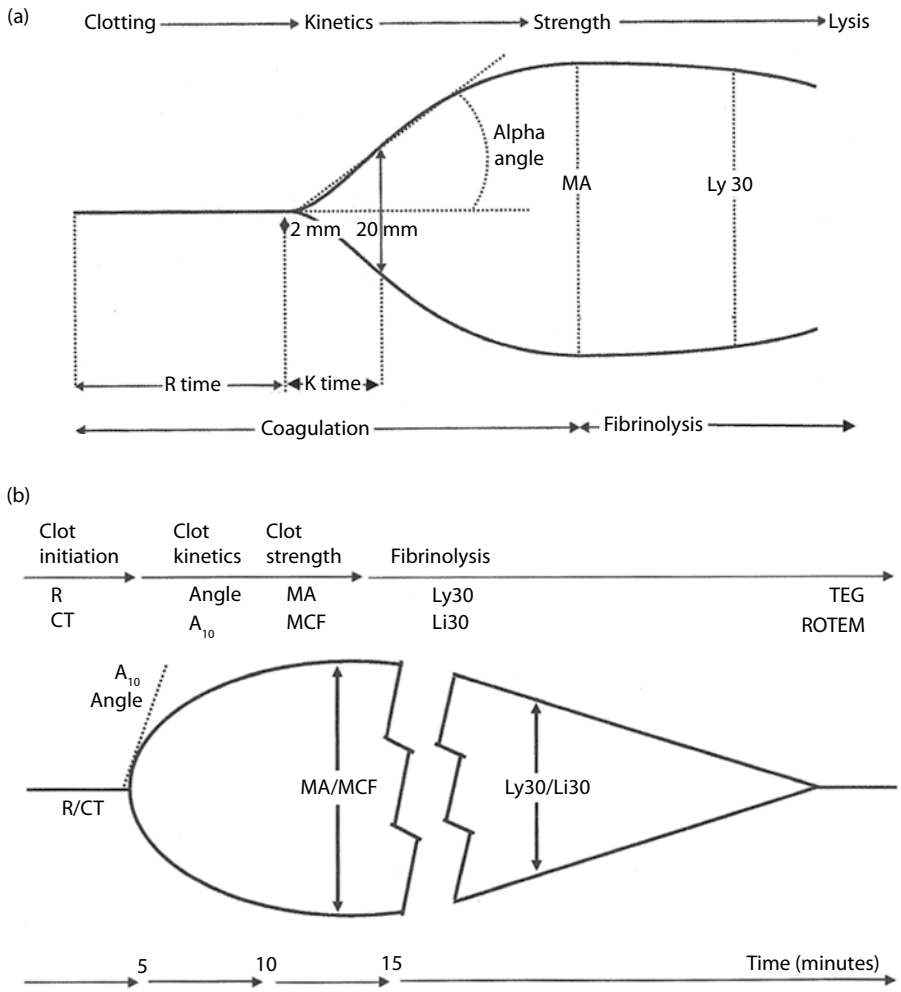

Figure 5.1 (a) Thromboelastogram. (b) RoTEM appearance. Abbreviations: α (alpha) angle, clot strength; A10, amplitude after 10 minutes; CT, clotting time; K time, kinetic time; Ly30/Li30, fibrinolysis after 30 minutes; MA, maximum amplitude – maximum clot strength; MCF, maximum clot firmness; R time, reaction time.

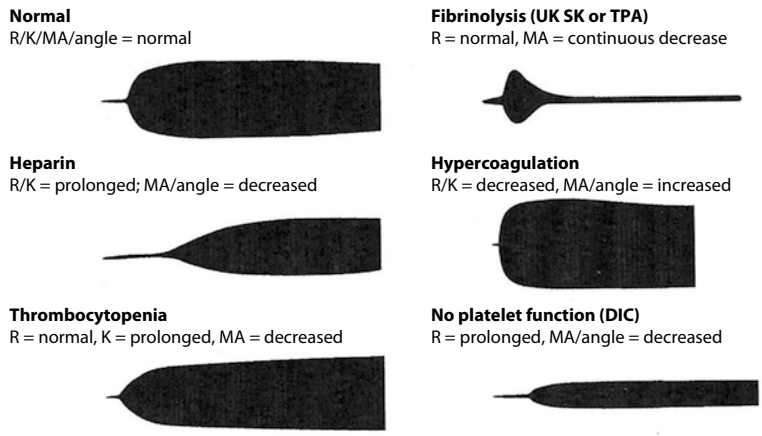

Figure 5.2 Abnormal appearances of the thromboelastogram. Abbreviations: DIC, disseminated intravascular coagulopathy; K, kinetic time; MA, maximum amplitude; R, reaction time; SK, streptokinase; TPA, tissue plasminogen activator; UK, urokinase.

The contribution of fibrinogen to clot strength can be evaluated in the functional fibrinogen assay in TEG and the FibTEM assay in ROTEM. VHAs are portable bedside devices but these are several advantages in running these in a standardized lab by skilled technicians.

The result is available within 3–10 minutes, in the form of a curve (Figure 5.1a or b). Several parameters can be measured (Table 5.2):

- R time (reaction time)/clotting time – the latency from the time at which the blood is placed in the cup until the clot begins to form.
- The α (alpha) angle – the progressive increase in clot strength.
- K time (kinetic time) – the K time starts where the R time ends and ends when the curve is at 20 mm amplitude.
- MA (maximum amplitude) – the maximal clot strength.
- Ly30 – Amount of thrombolysis after 30 minutes, expressed as percent of the MA.

Table 5.2 Interpretation of the Parameters of the TEG and RoTEM

Measurement	TEG Parameter (Normal Range)	ROTEM Parameter (Normal Range)
Clotting factor activity	R time (3–8 minutes)	CT_{EXTEM} (38–79 seconds) CT_{INTEM} (100–240 seconds)
Kinetics to maximum clot strength	K time (1–3 mm)	
Rate of increase in clot strength	α (alpha) angle (55–69 degrees)	$A10_{EXTEM}$ (43–65 mm)
Maximum strength of the clot	MA (51–69 mm)	MCF_{EXTEM} (50–72 mm)
Fibrinolysis at 30 minutes	Ly30 (<4%)	LI30 (94%–100%)
Fibrinogen activity (level)	FF_{MA} (14–24 mm)	FibTEM MCF (9–16 mm)

Abbreviations: Thrombelastography (TEG) – α, alpha angle; FF_{MA}, functional fibrinogen; K, K time; Ly30, fibrinolysis after 30 minutes; MA, maximum amplitude; R, reaction time. Rotational thromboelastometry (ROTEM) – A10, amplitude after 10 minutes; Fib, fibrinogen; LI30, fibrinolysis after 30 minutes; MCF, maximum clot firmness.

The VHAs allow for goal-directed haemostatic therapy, thereby only treating with what is needed. See Table 5.3 for one of several international validated algorithms. Furthermore, it is possible to decide if the bleeding is surgical or coagulopathic/pathological, which

Table 5.3 Goal-Directed Administration of Haemostatic Products and Medication, Based on TEG and ROTEM

TEG	RoTEM	Coagulopathy	Treatment Options
R 10–14 minutes	ExTEM CT 80–100 seconds InTEM CT 200–240 seconds	Coagulation factors ↓	FFP 20 mL/kg
R >14 minutes	ExTEM CT >100 seconds InTEM CT >240 seconds	Coagulation factors ↓↓	FFP 30 mL/kg rFVIIA (see Table 5.1)
FF_{MA} 7–14 mm	FibTEM MCF 6–9 mm	Fibrinogen ↓	FFP 20 mL/kg or cryoprecipitate 3 mL/kg or fibrinogen concentrate 20 mg/kg
FF_{MA} 0–7 mm	FibTEM MCF 0–6 mm	Fibrinogen ↓↓	FFP 30 mL/kg or cryoprecipitate 5 mL/kg or fibrinogen concentrate 30 g/kg
K (kinetic) time	>4 minutes		Cryoprecipitate 5 mL/kg or fibrinogen concentrate 30 mg/kg or rFVIIA (see Table 5.1)
α angle	<65°		Cryoprecipitate 5 mL/kg or fibrinogen concentrate 30 mg/kg or DDAVP
MA 45–49 mm and FF_{MA} >14 mm	ExTEM A_{10} 35–42 mm and FibTEM ≥10 mm ExTEM MCF <50 mm and FibTEM ≥10 mm	Platelets ↓	Platelets 5 mL/kg
MA <45 mm and FF_{MA} >14 mm	ExTEM A_{10} <35 mm and FibTEM ≥10 mm	Platelets ↓↓	Platelets 10 mL/kg
Ly30 >3 (−8) %	ExTEM Li 30 <94%	Hyperfibrinolysis	TXA 10 Gm or 10–20 mg/kg
Difference in R Hep TEG versus standard TEG R >2 minutes	InTEM CT/HepTEM CT > 1,25	Heparinization	Protamine 50–100 mg or FFP 10–20 mL/kg

Abbreviations: TXA, tranexamic acid; FFP, fresh frozen plasma; rFVIIA, recombinant factor VIIa.

clotting factors are missing, the function of platelets, and whether fibrinolysis is evolving normally. Transfusion of blood components, coagulation factors and additional medication can be administered rationally, based on the results.

The TEG offers substantial support to decision-making during the resuscitation, as it gives real-time accurate information on the coagulation status of the trauma patient, and facilitates the differentiation between pathological abnormality, and surgically correctable bleeding.

5.5 AUTOTRANSFUSION

Intra-operative and post-operative blood salvage and alternative methods for decreasing transfusion may lead to a significant reduction in allogenic blood usage.

Autotransfusion eliminates the risk of incompatibility and the need for crossmatching; the risk of transmission of disease from the donor is also eliminated. Autotransfusion is a safe and cost-effective method of sustaining RBC mass while decreasing demands on the blood bank. However, cell salvage in trauma patients is logistically challenging as, in the trauma patient, autotransfusion typically involves the collection of blood shed into wounds, body cavities – especially the chest, and drains.

Modern autotransfusion devices are basically of two types:

- Collection of blood that is collected, anticoagulated with heparin or citrate, and then run through a system in which it is washed and centrifuged, before being re-transfused.

  Reinfusion after filtration is less labour intensive and provides blood for transfusion quickly. Whole blood is returned to the patient with platelets and proteins intact, but free Hb and procoagulants are also reinfused. A high proportion of the salvaged blood is returned to the patient, and the most recent devices do not require mixing of the blood with an anticoagulant solution. In-line filters are essential when autotransfusion devices are used. These filters remove gross particles and macroaggregates during collection and reinfusion, thus minimizing microembolization.[21]
- Cell-washing and centrifugation techniques require a machine and (usually) a technician to be the sole operator. This latter requirement can limit the utility of the devices in everyday practice. The cell washing cycle produces red cells suspended in saline with a haematocrit of 55%–60%. This solution is relatively free of free Hb, procoagulants and bacteria. However, bacteria have been shown to adhere to the iron in the Hb molecule and washing therefore does not eliminate the risk of infection.

To a degree, the simpler the system, the less likely it is that problems will occur. In elective situations, nurses, technicians or anaesthesia personnel can participate in the autotransfusion process. In emergency situations without additional personnel, such participation may not be possible. Systems that process reclaimed RBCs may require trained technicians, particularly if the procedure is used infrequently.

In practical terms, bleeding from the chest seems ideal for immediate autotransfusion as the contents of thoracic cavity are sterile, in contrast with abdominal bleeding, where visceral injury and contamination may coexist. The simplest effective method is to use the sterile chest drain container. Use saline to create the fluid valve at the end of a chest drainage tube, to which is added 1000 IU fractionated heparin. The contents of the bottle may be hung and (using a microfilter to collect microaggregates) immediately reintroduced intravenously.

Autotransfusion is generally contraindicated in the presence of bacterial or malignant cell contamination (e.g. open bowel, infected vascular prostheses, etc.), unless no other RBC source is available, and the patient is in a life-threatening situation. However, there are several studies showing the practice may not be as unreasonable as previously thought.[22]

Cell salvage techniques have been shown to be cost-effective and useful in some trauma patients (e.g. in splenic trauma with significant blood loss), but further studies are indicated to clarify the indications.

5.6 RED BLOOD CELL SUBSTITUTES[23]

The ideal pRBC substitute is cheap, has a long shelf-life, is universally compatible and well tolerated, and has an O_2 delivery profile identical to that of blood. Significant effort has been made to find a suitable substitute that could, essentially, be treated as an artificial O_2 carrier.

Artificial O_2 carriers can be grouped into perfluorocarbon (PFC) emulsions and modified Hb solutions. The native Hb molecule needs to be modified in order to decrease its O_2 affinity and to prevent rapid dissociation of the native $alpha_2$–$beta_2$ tetramer into $alpha_2$–$beta_2$ dimers.

5.6.1 Perfluorocarbons

Perfluorocarbons are carbon–fluorine compounds that are completely inert and have low viscosity but dissolve large amounts of gas. They do not mix with water and therefore need to be produced as emulsions. Unlike the sigmoid relationship of Hb, they exhibit a linear relationship with O_2; therefore, their efficacy relies on maintaining a high PaO_2; however, PFCs unload O_2 well. They do not expand the intravascular volume and can only be given in small volumes as they overload the reticulo-endothelial system. Once thought to hold potential, they have so far not been found to confer additional benefit compared with crystalloid solutions, especially as there is a significant incidence of side effects.

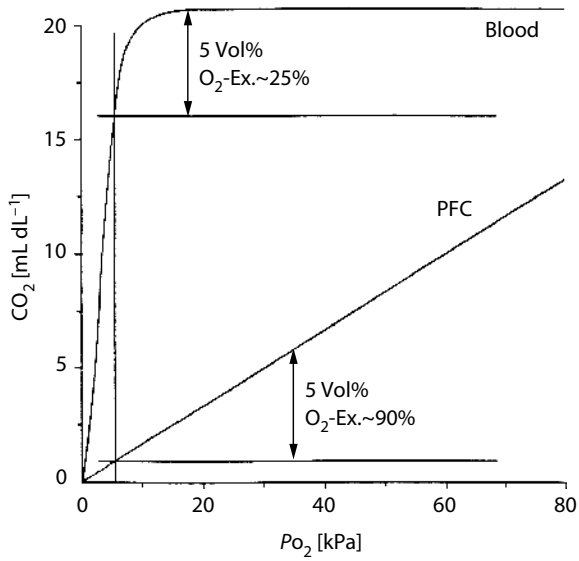

Figure 5.3 Oxygen transport characteristics of haemoglobin and perfluorocarbons.

5.6.2 Haemoglobin Solutions

Although free haemoglobin can transport O_2 outside of the red cell membrane, it is too toxic to be clinically useful. Techniques have been developed to remove the need for the red cell membrane and create haemoglobin-based oxygen carriers (HBOCs).

5.6.2.1 LIPOSOMAL HAEMOGLOBIN SOLUTIONS

These are based on the encapsulation of Hb in liposomes. The mixing of phospholipid and cholesterol in the presence of Hb yields a sphere with Hb at its centre. These liposomes have O_2 dissociation curves similar to red cells, with low viscosity and their administration can transiently produce high circulating levels of Hb.

Problems associated with HBOCs relate to effects on vasomotor tone, which appears to be modulated by the carriers' interaction with nitric oxide, causing significant vasoconstriction.

5.6.2.2 POLYMERIZED HAEMOGLOBIN SOLUTIONS (HUMAN-OUTDATED/BOVINE RBCS)

Techniques have been developed for crosslinking the haemoglobin molecules, initially with a di-aspirin crosslink and recently as a haemoglobin polymer. Both human and bovine Hb have been used.

Considerable research has taken place in the past decade with regard to the development of synthesized Hb solutions. Bovine-derived Hb (Hemopure) is approved for clinical use in South Africa. Currently, the products have not been licensed for use in the trauma patient.

The O_2 transport characteristics of modified Hb solutions and PFC solutions are fundamentally different (Table 5.3). The Hb solutions exhibit a sigmoid O_2 dissociation curve similar to that of blood, while PFC emulsions are characterized by a linear relationship between the partial pressure and the content of O_2. Haemoglobin solutions therefore provide O_2 transport and unloading characteristics similar to blood. This means that at a relatively low arterial O_2 pressure, substantial amounts of O_2 are being transported. In contrast, relatively high arterial O_2 partial pressures are necessary to maximize the O_2 transport of PFC emulsions (Figure 5.2).

Note that 5% O_2 can be offloaded by both blood and PFCs, PFC O_2 being more completely offloaded than blood-transported O_2 (Figure 5.3 and Table 5.4).

5.6.3 Future Evolution

The last few years' research on the use of the oxygen nanobubbles gives promising results on the reversal of tumour hypoxia that potentiates the effectiveness of therapy. Nano-engineering may open up new currently inconceivable ways to transfer oxygen to the hypoxic tissue.

Table 5.4 Advantages and Disadvantages of Haemoglobin-Based Solutions Compared with Hydrocarbons

Haemoglobin-Based Solutions	PFC-Based solutions
Advantages • Carries and unloads O_2 • Few and mild side effects • No known organ toxicity	**Advantages** • Carries and unloads O_2 • Sigmoidal O_2 dissociation curve • 100% FiO_2 not mandatory for maximum potency • Easy to measure

Disadvantages
• 100% FiO_2 *is* mandatory for maximum potency
• Additional colloid is often necessary with potential side effects
	Disadvantages
	• Side effects
	  • Vasoconstriction
	  • Interference with laboratory methods (colourimetric)

5.7 MASSIVE HAEMORRHAGE/ MASSIVE TRANSFUSION[24]

5.7.1 Definition

Blood volume is approximately 70 mL/kg. Massive transfusion is defined as:

- The replacement of 100% of the patient's blood volume in less than 6 hours.
- The administration of 50% of the patient's blood volume in 1 hour.

There is a danger of death when blood loss is more than 150 mL per minute or 50% of blood volume in 20 minutes. Each trauma unit should have a policy for massive transfusion, which should be activated as soon as a potential candidate is admitted.

Also germane to the initial period of massive blood transfusion are the potential complications of acidosis, hypothermia, and hypocalcaemia. Hypothermia (<34°C) causes platelet sequestration and inhibits the release of platelet factors that are important in the intrinsic clotting pathway. In addition, it has consistently been associated with a poor outcome in trauma patients. Core temperature often falls insidiously because of exposure at the scene and in the emergency department, and because of the administration of resuscitation fluids stored at ambient temperature.

The use of bicarbonate in the treatment of systemic acidosis remains controversial. Administration of sodium bicarbonate may cause a leftward shift of the oxyhaemoglobin dissociation curve, reducing tissue O_2 extraction, and may worsen intracellular acidosis caused by carbon dioxide production. Use of sodium bicarbonate in the acidotic trauma patient is associated with an increase in mortality.[25]

Hypocalcaemia caused by citrate binding of ionized calcium does not occur until the blood transfusion rate exceeds 100 mL per minute (equivalent to 1 unit every 5 minutes). Decreased serum levels of ionized calcium depress myocardial function before impairing coagulation. Calcium gluconate or calcium chloride should be reserved for cases in which there is electrocardiographic (ECG) evidence of QT interval prolongation or, in rare instances, for cases of unexplained hypotension during massive transfusion. or where the active (ionized) fraction is >1 mmol/L on a blood-gas result.

5.7.2 Massive Transfusion Protocol (MTP)

An algorithm of coordinated action incorporating many hospital departments (surgery, blood bank, ICU, anaesthesiology) is activated upon the arrival of a trauma patient with massive haemorrhage. The protocol provides roles for the personnel, actions to be taken, and medications and blood products to be transfused. The target is the increase of survival of these patients. The basis of these protocols has been the knowledge recently acquired from modern battlefields, which has dramatically changed the way we manage these patients (Table 5.5).

5.8 HAEMOSTATIC ADJUNCTS IN TRAUMA

5.8.1 Overview

Haemostatic substances can be used after surgical haemostasis in trauma surgery to secure the surface of the

Table 5.5 Guidelines for Massive Transfusion

Definition

- The replacement of 100% of the patient's blood volume in less than 6 hours.
- The administration of 50% of the patient's blood volume in 1 hour.

Activation

The protocol will be activated **automatically by the blood bank** after 2 units of packed red blood cells (pRBCs) have been issued to a patient, *and* a request for a further four (4) units of blood or more is subsequently requested within any 24-hour period. A prospective tool utilizing PR >120 bpm and BP <90 *and* free blood in the abdomen can be used.[19]

Activation can also be done at the discretion of the treating physician.

It is essential the protocol activation to be based on criteria available on admission and not on parameters calculated after hours (e.g. blood loss) since by that time the salvation potential is zeroed.

Blood specimens

Group and crossmatch:

- Leukodepleted blood should be used wherever it is available.
- Crossmatched blood if available.
- Uncrossmatched group O blood.

The following **baseline blood specimens** are required

- Full blood count including platelets.
- Prothrombin time (PT), activated partial thromboplastin time (aPTT), thrombin time, International Normalized Ratio (INR), fibrinogen, D-dimer, thromboelastogram (TEG) or RoTEM.

The following are required **after every 6 units of transfused blood:**

- Repeat baseline blood samples.
- Full TEG or RoTEM.

Avoid hypothermia (patient and transfused fluid)

- Use an appropriate blood warmer.
- Keep the patient warm using an appropriate patient-warming device.
- Maintain a warm environment.

Blood and blood products

The blood bank will issue the following products (as part of a 2 or 6 unit 'massive transfusion pack'):

NB: Multiple '**2-unit packs**' are preferable as they can be returned if the 'cold chain' is intact

- Two units or 6 units of pRBCs using the *freshest blood available*.
- Two units or 6 units of **thawed** fresh frozen plasma (FFP).
- Two units or 5/6 units of platelets (apheresis unit – individual laboratory dependent).

or

- For every 5 or 6 units of blood issued.
- One apheresis unit of platelets ('platelet mega-unit'). NB: may be 5 or 6 units of pooled platelets.

Administration

Microaggregate filters are **not** advised

Once administration of the 'massive transfusion pack' blood is begun, administer all the above in a **1:1:1 ratio,** (blood:FFP:platelets) or **6:6:1 / 5:5:1 / 4.4.1,** (blood:FFP:apheresis mega-platelet unit – depends on local interpretation of a megaplatelet unit). After every 6 units of red cells, if ongoing bleeding or need for transfusion is present:

- Give a further 4 units of FFP if PT or APTT is >1.5 times mid-normal or according to TEG/RoTEM.
- Give 10 units of cryoprecipitate if fibrinogen <1 g/L or according to TEG/RoTEM.
- Give 10 mL 10% calcium chloride ***only*** if the above additional doses are given.
- Give at least 1 Megaunit of pooled platelets if the platelet count is <75,000/mm^3.

Return all unused 'massive transfusion packs' to the blood bank as soon as possible.

(*Continued*)

Table 5.5 (*Continued*) Guidelines for Massive Transfusion

End points of transfusion

- Any active surgical bleeding has been controlled.
- No further need for red cells.
- Temperature >35°C.
- pH >7.3.
- Fibrinogen >1.5 g/L.
- INR better than 1.5, PT less than 16 seconds, aPTT less than 42 seconds.
- Haemoglobin 8–10 g/dL (4–6 mmol/L).

wound. Tissue adhesives are used alone or in combination with other haemostatic measures.

The main indications for using adhesives are:

- To arrest minor oozing of blood.
- To secure the wound area to prevent subsequent bleeding.

Various forms of fibrin sealing are available and are suitable for treating injuries, especially of the parenchymatous organs. The different presentations make some suitable for superficial bleeding surfaces, and others easier to apply in deep lacerations. Some are readily available, while preparation is time-consuming in others. It is important that the surgeon knows what haemostatic agents are available and how and where they can be used.

5.8.2 Tissue Adhesives

5.8.2.1 FIBRIN

Of the adhesives currently available, fibrin glue is the most suitable for treating injuries to the parenchymatous organs and retroperitoneum. It is also possible to make autologous fibrin from the patient's own blood (Vivostat system; Vivolution A/S Birkeroed, Denmark); the fibrin is applied with a sprayer. The necessary volume of blood (125 mL) can already be drawn in the emergency room, and the autologous adhesive is ready within 30 minutes.

Fibrin sealing is based on the transformation of fibrinogen to fibrin. Fibrin promotes clotting, tissue adhesion, and wound healing through interaction with the fibroblasts. The reaction is the same as in the last phase of blood clotting. One such heterologous fibrin is Tisseel/Tissucol (Baxter Hyland Immuno, Vienna, Austria). Heterologous fibrin is a biological two-component adhesive and has high concentrations of fibrinogen and factor XIII, which, together with thrombin and calcium, result in clotting. Resorption time and resistance to tearing depend on the size and thickness of the glue layer, and on the proportion by volume of the two components. The fibrin sealant is best applied with a sprayer or syringe injection system such as the Tissomat sprayer (Baxter Hyland Immun, Rochester, MN, USA).

5.8.2.2 TACHOSIL®

TachoSil® (Baxter Corporation, Deerfield, IL, USA), is a fixed, ready-to-use combination of a collagen sponge coated with a dry layer of the human coagulation factors fibrinogen and thrombin, making it easy to employ. It is most suitable for oozing from the raw surfaces of solid organs or to seal air leaks from lung injuries. It is available in most countries in Europe and Australasia.

5.8.2.3 HEMOPATCH®

Similarily, HemoPatch® (Baxter Hyland Immuno, Vienna) has recently become available.

HemoPatch® is a soft, thin, and flexible patch consisting of a porous collagen matrix, which facilitates clotting, coated on one side with a thin protein-binding layer (pentaerythritol polyethylene glycol ether tetrasuccinimidyl glutarate (NHS-PEG)) which allows rapid adhesion (within 2 minutes) via electrophilic crosslinking. The patch consequently has a dual-method mechanism of action, in which the two components interact to achieve hemostasis by sealing off the bleeding surface and initiating the body's own clotting mechanisms. The patch is resorbed within 6–8 weeks.

5.8.2.4 COLLAGEN FLEECE

Even after surgical haemostasis, deep parenchymal injuries can require a resorbable tamponade; here, collagen fleece (e.g. TissoFleece, Baxter, Vienna) is suitable. Collagen fleece is composed of heterologous collagen fibrils obtained from devitalized connective tissue and

is fully resorbable. Collagen fleece promotes the aggregation of thrombocytes when in contact with blood. The platelets degenerate and liberate clotting factors, which in turn activate haemostasis. The spongy structure of the collagen stabilizes and strengthens the coagulate. Another alternative for deep parenchymal injuries is FloSeal (Baxter Corporation, Deerfield, IL, USA).

Fibrin glue and collagen fleece are used preferentially to treat slight oozing of blood. Before application, the bleeding surface should be tamponaded and compressed with a warm pad for a few minutes. Immediately after removal of the pad, air alone is first sprayed, followed by short bursts of fibrin. This creates a surface that is free from blood and nearly dry when the fibrin glue is sprayed onto it. A dry field is essential for most fibrin sprays in order to secure adequate haemostasis.

If collagen fleece is to be applied, a thin layer of fibrin is sprayed onto the fleece, which, in turn, is pressed onto the wound. After a few moments compression, the fleece is sprayed with fibrin glue. The thickness of the fibrin layer will depend on the size and depth of the injury.

5.8.3 Other Haemostatic Adjuncts

5.8.3.1 CHITOSAN (CELOX (MEDITRADE LTD., CREWE, UK)/ HEMCON (HEMCON MEDICAL TECHNOLOGIES, PORTLAND, OR, USA))

Chitosan is a granular product made from a natural polysaccharide derived from chitin from shellfish. Chitosan is the deacetylated form of chitin. In the form of an acid salt, chitosan demonstrates mucoadhesive activity. Chitosan stops bleeding by bonding with red blood cells and gelling with fluids to produce a sticky pseudoclot. This reaction is not exothermic and has been used successfully within body cavities. Chitosan is broken down by enzymatic action within the body to produce glucosamine. The dressing is sold as pads or bandages.

5.8.3.2 MINERAL ZEOLYTE (QUIKCLOT® (Z-MEDICAL CORPORATION, WALLINGFORD CT, USA))

Mineral zeolyte, when made moist, produces an exothermic reaction that seals blood vessels and results in haemostasis. The initial preparation was in the form of granules and was very exothermic, resulting in significant tissue damage. The current preparation is presented in bags ('tea-bags') that are packed into the wound and may cause less damage.

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Damage Control 6

6.1 INTRODUCTION

Damage control has as its objective, delay in imposition of additional surgical stress at a moment of physiological frailty.[1] The goal is to focus on the body physiology, and align the objectives of the surgical intervention (specifically to stop bleeding and control contamination), with the resuscitation objectives as an overarching damage control principle.

Damage control resuscitation (DCR), damage control surgery (DCS), and damage control orthopaedics (DCO) are a deliberate and pre-emptive set of non-traditional manoeuvres used to reverse the pre-terminal effect of exsanguination, massive injury, and shock. The primary goal is to temporize management of major injuries with directed resuscitation and staged surgery, to allow for resuscitation and restoration of normal physiology.

The concept of damage control is not new, and livers were packed more than 100 years ago by Pringle. However, with the failure to understand the underlying physiological rationale, the results were disastrous. Over time, the concept has evolved into the modern technique of better resuscitation (DCR), a staged laparotomy (DCS), with establishment of intra-abdominal pack tamponade, control of contamination, delayed definitive management, and the restoration of normal physiological parameters and coagulation. The use of adjuncts, such as angiography and embolization, has been beneficial where packing of the viscera has failed (e.g. as in uncontrolled pelvic bleeding or soft tissue destruction).

Damage control concepts are not restricted to the abdomen and extend to every cavity in the body as well as vascular damage control. The need for damage control in children is much less common; however, they are much more prone to hypothermia given larger surface area relative to their smaller blood volume. Although the physiological parameters are very different, the principles are the same. At the other end of the spectrum, the application of damage control to the elderly who have decreased physiological reserve and a high morbidity and mortality has also been successful, with survival of greater than 50% when damage control is applied to this group.[2] Damage control surgery may be performed in smaller hospitals before transfer to a larger centre. Damage control surgery procedures, on properly selected patients, can be life-saving, and may have to be performed in any hospital admitting trauma cases.

Damage control can also be applied to the general surgery population in patients with intra-abdominal catastrophes such as perforated viscous pancreatitis, ischemia, or gastrointestinal haemorrhage. DCS allows for rapid source control with the ability to re-evaluate the source easily. It also allows for frequent re-evaluation of bowel viability in the setting of ischaemia. Finally, and possibly most importantly, DCS decreases (but does not eliminate) the incidence of abdominal compartment syndrome (see also Section 17.10). This patient population often receives a massive amount of intravenous volume during the initial resuscitation, which results in bowel oedema and increased intra-abdominal pressure. A temporary abdominal closure lowers the risk of this potentially devastating process.

The application of damage control principles has influenced military care and disaster planning. The need to triage mass casualty scenarios makes an abbreviated surgical and damage control approach advantageous in maximizing the use of limited resources to many patients in a restricted time span or space. A recent prospective observational military study reported that damage control was utilized in 77% abdominal operations performed in Afghanistan and was safe.[3] The study concluded that DCR and DCS was the optimal approach to abdominal war injury and should be considered in logistical planning for future military operations. Events such as the Boston Marathon bombing showed that the critical patient surge placed on hospitals within minutes of an incident was well handled with good outcomes when the abbreviated DCS strategy was used to manage all patients.[4]

6.2 DAMAGE CONTROL RESUSCITATION

The optimal strategy for the management of the haemorrhaging patient is now termed DCR and is a critical adjunct to the application of DCS, with which it occurs in parallel.[5] During World War II, it was noted that there were improved outcomes with slightly lower blood pressures (owing to the reduction in blood loss). Permissive hypotension reduces blood loss, the need for additional fluids which might cause a dilutional coagulopathy, and avoids the associated acidosis, hypothermia, and clotting derangements.

DCR is the proactive, anticipatory treatment of the coagulopathy, hypothermia, and acidosis that presents with critical injury and shock. DCR priorities include the following:[6–8]

- Permissive hypertension with resuscitation only to a pressure that allows organ *perfusion* ('hypotensive resuscitation'), rather than achieving *normotension*.

  Permissive hypotension is a strategy to reduce blood loss by limiting systolic pressure to the minimum necessary to maintain perfusion of vital organs. In massively bleeding patients, raising blood pressure to normal levels before achieving surgical haemostasis has been shown to enhance bleeding by displacing clots formed during the body's attempt at primary haemostasis ('popping the clot'). The strategy is not new, and was reported during the World War I, when it was stated that 'if the pressure is raised before the surgeon is ready to check any bleeding that may take place, blood that is sorely needed may be lost'. Although safe limits of blood pressure in terms of preservation of organ perfusion are unknown, a systolic blood pressure of 80 mm Hg, and in case of concomitant severe brain trauma, a mean arterial pressure (MAP) of over 80 mm Hg are recommended. While there is no hard evidence for these limits, it is very clear that such low pressures should be maintained for the shortest possible time in order to limit vital organ ischaemia. The importance of early surgical haemostasis and the need for the DCR concept being applied must be emphasized.
- Minimizing the use of crystalloid and early use of blood products during resuscitation – facilitated by use of a massive transfusion protocol (see also Section 5.7).
- Blood product administration with liberal use of fresh frozen plasma (FFP) and platelets.

  The optimal ratio of blood component therapy has yet to be determined, but a ratio approaching 1:1:1 has been suggested. In certain environments properly cross-matched whole blood may also be used.

  Early and aggressive administration of blood and blood products has shown to improve survival after trauma-related haemorrhagic shock. Lost blood volume should be replaced by blood products aiming at near equal ratios of packed red blood cell (pRBC): FFP:platelets.[9,10] Such regimens have demonstrated improved outcomes. Data suggest that plasma-based resuscitation when compared to crystalloids is better at preserving endothelial integrity. FFP administration after haemorrhagic shock has anti-inflammatory properties and a potential glycocalyx-restoring capacity.[11]
- Targeting coagulopathy with goal-directed haemostasis, using viscoelastic assays such as TEG or RoTEM, if available, may be used to guide product therapy.
- Restoration of normothermia.

  Hypothermia below 35°C has a profound impact on surgical site infection rates after trauma laparotomy. Hypothermia also adversely affects coagulation as well as cardiac output and function in most bodily organs.

The recent EAST guidelines on DCR[12] addressed the requirements (Table 6.1).

The implementation of a massive transfusion protocol (MTP) is recommended, which will allow the ready availability of blood products (see also Chapter 5). Availability of an MTP has shown to be associated with a reduction of organ failure and improved 30-day survival after severe trauma.[13]

6.3 DAMAGE CONTROL SURGERY

DCS is a technique whereby the surgeon minimizes operative time and surgical intervention in the grossly unstable patient. The primary reason for this is to minimize hypothermia, metabolic acidosis, and coagulopathy ('the lethal triad'), which are often apparent after the patient has sustained massive tissue destruction, or significant blood volume loss. The focus is exclusively on

Table 6.1 Evidence-Based Guidelines for Aspects of DCR

	Question	Guidelines
1	In adult patients with severe trauma, should a massive transfusion protocol (MT)/damage control resuscitation (DCR) versus no MT/DCR be used to decrease mortality or total blood products used?	In adult patients with severe trauma we *recommend* the use of a massive transfusion/damage control resuscitation protocol in comparison to no protocol to reduce mortality.
2	In adult patients with severe trauma, should a high ratio of plasma to red blood cells (PLAS:RBC) and platelet to red blood cells (PLT:RBC) versus a low ratio be administered to decrease mortality or total blood products used?	In adult patients with severe trauma we *recommend* targeting a high ratio of plasma and platelets to red blood cells as compared to a low ratio to reduce mortality. This is best achieved by transfusion equal amounts of red blood cells, plasma, and platelets, during the early empirical phase of resuscitation.
3	In adult patients with severe trauma, should the haemostatic adjunct recombinant factor VIIa (rVIIa) versus no rVIIa be administered to decrease mortality, total blood products used, or MT? Does the use of rVIIa increase venous thromboembolism (VTE) rates?	In adult patients with severe trauma we *cannot recommend* for or against the use of rVIIa as a haemostatic adjunct in comparison to no rVIIa. We feel that the use rVIIa needs further study with particular attention to optimal doses and the timing of administration, relative to blood product administration. As with other haemostatic agents, the VTE rates need to be more carefully evaluated with the use of a defined surveillance protocol.
4	In adult patients with severe trauma, should the hemostatic adjunct tranexamic acid (TXA) versus no TXA be administered to decrease mortality, total blood products used, or MT? Does the use of TXA increase VTE rates?	There is no clear universal mortality benefit to TXA. In adult patients with severe trauma we *conditionally recommend* the use of TXA as an in-hospital haemostatic adjunct in comparison to no TXA. These recommendations only apply to the use of TXA in a hospital setting pending the results of pre-hospital trials. As with other haemostatic agents, the VTE rates need to be more carefully evaluated with the use of a defined surveillance protocol.

life-saving surgical procedures (bleeding and contamination control), thus permitting more successful resuscitation and normalization of the patient's physiology. After a post-operative intensive care resuscitation and stabilization period, the patient is returned to the operating room as soon as physiologically possible, for definitive surgical care (e.g. restoration of bowel continuity). Although the principles are sound, extreme care needs to be exercised to avoid over-utilization of the concept, causing secondary insults to viscera. Furthermore, surgery must be appropriate, to minimize activation of the inflammatory cascade and the consequences of systemic inflammatory response syndrome (SIRS) and organ dysfunction.

Damage control can be divided into five distinct stages: this begins with patient selection (Stage 1) and continues through potential late abdominal wall reconstruction (Stage 5).

6.3.1 Stage 1: Patient Selection

Proper patient selection is crucial to optimize outcomes following damage control surgery. All patients should undergo a very rapid trauma evaluation, and appropriate DCR. The duration of this stage is dictated by the patient's physiological stability as well as the underlying pathology. Delays to the operating

room must be avoided. Rapid manoeuvres to control external bleeding (tourniquets, digital control of bleeding, etc.), are indicated as transition to the operating room occurs.

'The treatment of bleeding is to stop the bleeding...'

In any hospital managing trauma or a high number of emergency surgical cases, protocols for massive transfusion supporting haemostatic resuscitation should be in place and supported by anaesthesia, blood bank, and intensive care staff (see also Chapter 5). The surgeon is well advised to call for an additional surgeon to assist, if available.

The indications to consider in selecting a patient for damage control are:

- Haemodynamic instability:
  - Systolic blood pressure <90 mm Hg and not responding to resuscitation.
- Temperature <35°C.
- Metabolic instability:
  - Temperature <35°C.
  - pH <7.2.
  - Base excess ≥−5 and worsening.
  - Serum lactate ≥5 mmol/L.
- Coagulopathy:
  - Abnormal viscoelastic haemostatic assays (VHA): TEG or RoTEM.
  - Prothrombin time (PT) >16 seconds.
  - Partial thromboplastin time (PTT) >60 seconds.
- Surgical anatomy:
  - Complex life-threatening injuries (e.g. major vascular injury or moderate vascular injury with complex hollow viscus injuries, complex liver injury, exsanguinating retroperitoneal pelvis, multi-cavitary exsanguination etc.).
  - Anticipated need for a time-consuming surgical procedure in a patient with a suboptimal response to resuscitation.
  - Inability to perform the definitive repair in a timely fashion.
  - Demand for non-operative control of other injuries, for example a fractured pelvis.
  - Inability to approximate the abdominal incision
- Environment and/or resource demands
  - Blood requirement requiring an MTP.
  - Operating time greater than 60 minutes.
- Logistics:
  - Multiple patients/mass casualty situation.
- Minimal resources:
  - (e.g. personnel, medical equipment, safety concerns).

It is critical to identify these potential scenarios before the patient becomes metabolically unsalvageable. The decision to deploy a damage control physiological-based approach should be done to PREVENT this metabolic deterioration from occurring in the first place. It can often be a decision that is made at the very beginning of the surgical intervention.

If resources allow, the use of a hybrid operating room expedites definitive haemorrhage control and can complement damage control therapy in the exsanguinating patient.

Irrespective of the setting, a coagulopathy is the single most common reason for abortion of a planned procedure or curtailment of definitive surgery. It is important to abort the surgery *before* the coagulopathy becomes obvious.

The technical aspects of the surgery are dictated by the injury pattern.

6.3.2 Stage 2: Operative Haemorrhage and Contamination Control

See also: The Trauma Laparotomy (Chapter 9.1).

6.3.2.1 INITIAL INCISION

A very large midline laparotomy incision is necessary to allow for wide retraction of the abdominal wall and rapid visualization of the liver, abdominal great vessels, and all other retroperitoneal structures. Two experienced surgeons are recommended. Rapid control of the largest source of blood loss is critical and in those cases with several sources of bleeding and manual pressure, packing, and temporary ligation may be necessary. Each case is unique in presentation; however, the principle is to control active large blood loss first.

6.3.2.2 HAEMORRHAGE CONTROL

The operative process works through the following (see also Section 9.1):

- Arrest arterial and major venous bleeding. Failure to control ongoing bleeding will lead to the patient's demise:

- Major arterial bleeding must be controlled at the index procedure. A temporary intravascular shunt is preferred for named vessels; however, ligation may be required to save an exsanguinating patient.
- Resuscitative endovascular balloon occlusion of the aorta (REBOA) may be effective in controlling arterial haemorrhage in difficult access areas such as the pelvis, the retroperitoneum, or the kidney (see also Section 15.3).

- Temporary intravascular shunting (see Figure 6.1)
  - Use tubing approximately 50% of the diameter of the vessel being shunted.
  - Allow 3–4 cm of the tube in each end of the vessel.
  - The shunt can either be a straight (linear) shunt (Figure 6.1a), for example, for iliac vessels, portal vein, etc., or a 'pig-tail' shunt (Figure 6.1b) where access is difficult (e.g. superior mesenteric artery, or some temporary mobility is expected (e.g. shunting a superficial popliteal or distal femoral artery prior to orthopaedic repair.
- Tamponade using wraps or packs.
- Occlusion of inflow into the bleeding organ (e.g. Pringle's manoeuvre for bleeding liver).
- Repair or ligation of accessible blood vessels.
- Intra-operative or post-operative catheter directed embolization.

6.3.2.3 CONTROL OF CONTAMINATION (HOLLOW VISCUS ORGANS)

- Defects in the bowel or hollow viscous organs can be controlled with simple suturing or stapler applications.
- Damaged segments can be resected with clamps or staples.
- Biliary and genitourinary injuries can be temporized with external drainage (T-tube, ureterostomy, etc.).
- Pancreatic injuries should be widely drained and packed.

Avoid definitive repair, restoration of intestinal continuity, stoma formation, and creation of feeding access at this stage.

6.3.2.4 COPIOUS WASHOUT

At the end of the procedure, before temporary closure, the abdominal cavity must be washed out with copious (several litres of normal saline, especially if there has been contaminations by faeces, for example). After washing out, suck out all fluid. Leave the cavity as dry as possible.

6.3.2.5 TEMPORARY ABDOMINAL CLOSURE (TAC)

The abdominal cavity should be closed to prevent heat and moisture loss, and to protect the viscera.

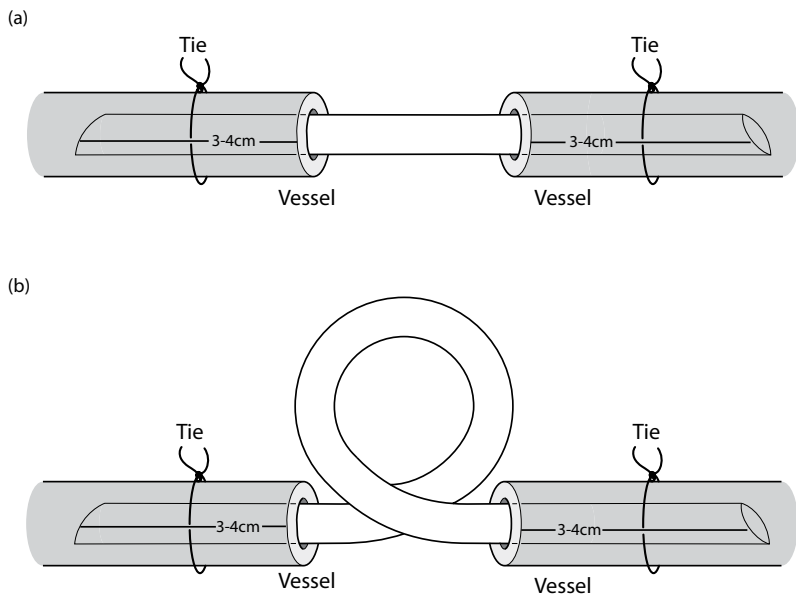

Figure 6.1 Diagram of an intravascular shunt. (a) Straight shunt; (b) 'pig-tail' shunt.

Delayed closure is required when any combination of the factors listed above under damage control, or re-look surgery, exists. Patients with multiple injuries who have undergone protracted surgery with massive volume resuscitation to maintain haemodynamic stability will often develop tissue interstitial oedema. This may predispose to the development of abdominal compartment syndrome (ACS) or may simply make primary closure of the sheath impracticable. In addition, significant enteric or other contamination will raise the risk of intra-abdominal sepsis, or the extent of tissue damage may raise doubt over the viability of any repair; these conditions usually mandate planned re-laparotomy and thus a temporary closure.

In these circumstances, a temporary abdominal closure is required. The needs of such a closure can be summarized as:

- Quick to do.
- Cheap.
- Keeps the abdominal contents inside the abdominal cavity.
- Allows drainage of fluid.
- Minimizes sepsis.
- Facilitates delayed primary fascial closure.

Vacuum-based closure is recommended for the management of the open abdomen. A review of the literature available suggests that negative-pressure wound vacuum therapy may have improved facial closure rates and may be associated with improved outcomes over alternative abdominal closure techniques (mesh, Velcro, bag-type dressings).

Many temporary closure systems are available, of which the most cost effective is the 'sandwich technique' first described by Schein in 1986[14] and was popularized by Rotundo and Schwab.[1]

A sheet of self-adhesive incise drape (Opsite® – Smith and Nephew, London, UK, Steri-Drape® or Ioban® – 3M Corporation, St Paul, MN, USA) is placed flat, sticky side up, and an abdominal swab is placed upon it. The objective is to create a non-stick membrane on one side of the abdominal swab to place against the visceral organs. The size should be large enough that, when inserted, the sheet will extend laterally to the paracolic gutters, and 10 cm cranial and caudal to the incision. The edges are folded over to produce a composite sheet with a membrane (i.e. the drape) on one side, and an abdominal swab on the other. The membrane is utilized as an on-lay with the margins 'tucked in' under the edges of the open sheath as far as the paracolic gutters, with only the membrane in contact with the bowel.

Pitfalls

Table 6.2 Dos and Don'ts

DO
• Cover only one side of the swab. Covering both sides impedes drainage by preventing capillary wicking through the weave of the swab. • Insert the sandwich, plastic side against the bowel. • Insert the sandwich as a 'diamond', with the points tucked in at top and bottom of the incision, and laterally.
DO NOT
• Make any holes (slits) in the membrane. Holes would allow the suction to be transmitted directly to the serosa of the bowel, with possible risk of fistula formation. • Have a vacuum above a maximum of 25 mm Hg. Higher suction pressures, especially in a cold hypotensive patient, transmitted directly on to the bowel may exceed capillary pressure. • Preferably avoid the commercial equivalents (e.g. VAC® – Kinetic Concepts Incorporated (KCI), San Antonio, TX, USA or Renasys® – Smith and Nephew, London UK), currently. These should be reserved for the definitive closure of a wound, both for cost reasons, and the higher vacuum suction pressure often present.

The appreciable drainage of serosanguinous fluid that occurs is best dealt with by placing a pair of drainage tubes (e.g. sump-type nasogastric tubes or closed-system suction drains) through separate stab incisions, with the tips placed in the caudal end of the incision. The tubes lie on either side of the incision, under the edge of the anterior abdominal peritoneum, and the gauze of the swab., and utilizing continuous low-vacuum suction.

This arrangement is covered by an occlusive incise drape applied to the skin, thus providing a closed system (the 'sandwich'; Figures 6.2 and 6.3).

The 'Bogota bag' and towel clips, etc. are no longer used, and for temporary abdominal closure, no sutures should be placed in the sheath, nor skin.

The timing of transfer of the patient from the operating theatre to the intensive care unit (ICU) is critical. Prompt transfer is cost-effective; premature transfer is counter-productive. Control of bleeding and

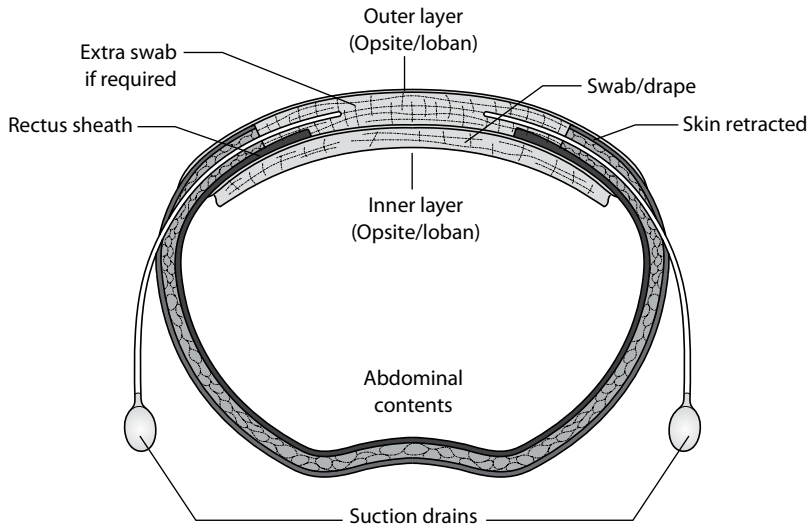

Figure 6.2 Diagrammatic representation of the sandwich technique for temporary abdominal closure.

contamination must be achieved. On the other hand, once haemostasis has been properly achieved, it may not be necessary to abort the procedure in the same fashion.

In the operating room, efforts must be started to reverse all the associated adjuncts, such as acidosis, hypothermia, and hypoxia, and it may be possible to improve the coagulation status through these methods alone. Adequate time should still be allowed for this, following which reassessment of the abdominal injuries should take place, as it is not infrequent to discover further injuries or ongoing bleeding.

6.3.3 Stage 3: Physiological Restoration in the ICU

Priorities in the ICU are as follows.

6.3.3.1 RESTORATION OF BODY TEMPERATURE

- Passive rewarming using warming blankets, convection blankets, warmed fluids, heat lamps, elevation of the ambient room temperature, etc.
- Active rewarming with lavage of the chest or abdomen.

6.3.3.2 OPTIMIZATION OF OXYGEN DELIVERY

- Volume loading to restore circulating blood volume.
- Haemoglobin optimization to a haemoglobin level of 8–10 g/dL (80–100 g/L), (4–6 mmol/L).
- Monitoring of cardiac output.
  - Ultrasonic cardiac output devices.
  - Cardiac arterial output monitors.
  - Correction of acidosis to a pH >7.3.
- Measurement and correction of lactic acidosis to less than 2.5 mmol/L.
- Inotropic support as required, but not prioritized over *adequate* blood product resuscitation.

6.3.3.3 CORRECTION OF CLOTTING PROFILES

- Blood component repletion.
- Assessment and normalization of any coagulopathy.
  - Coagulation studies.
- Thromboelastography (TEG) or rotational thromboelastometry (RoTEM).

6.3.3.4 IMPROVEMENT OF PHYSIOLOGICAL ENDPOINTS: AS EVIDENCED BY

- Lactate clearance.
- SvO_2.
- Urine output.
- Haemodynamics.
- Resolution of acidaemia.
- Inotropic support decreased once intravascular volume has been repleted.

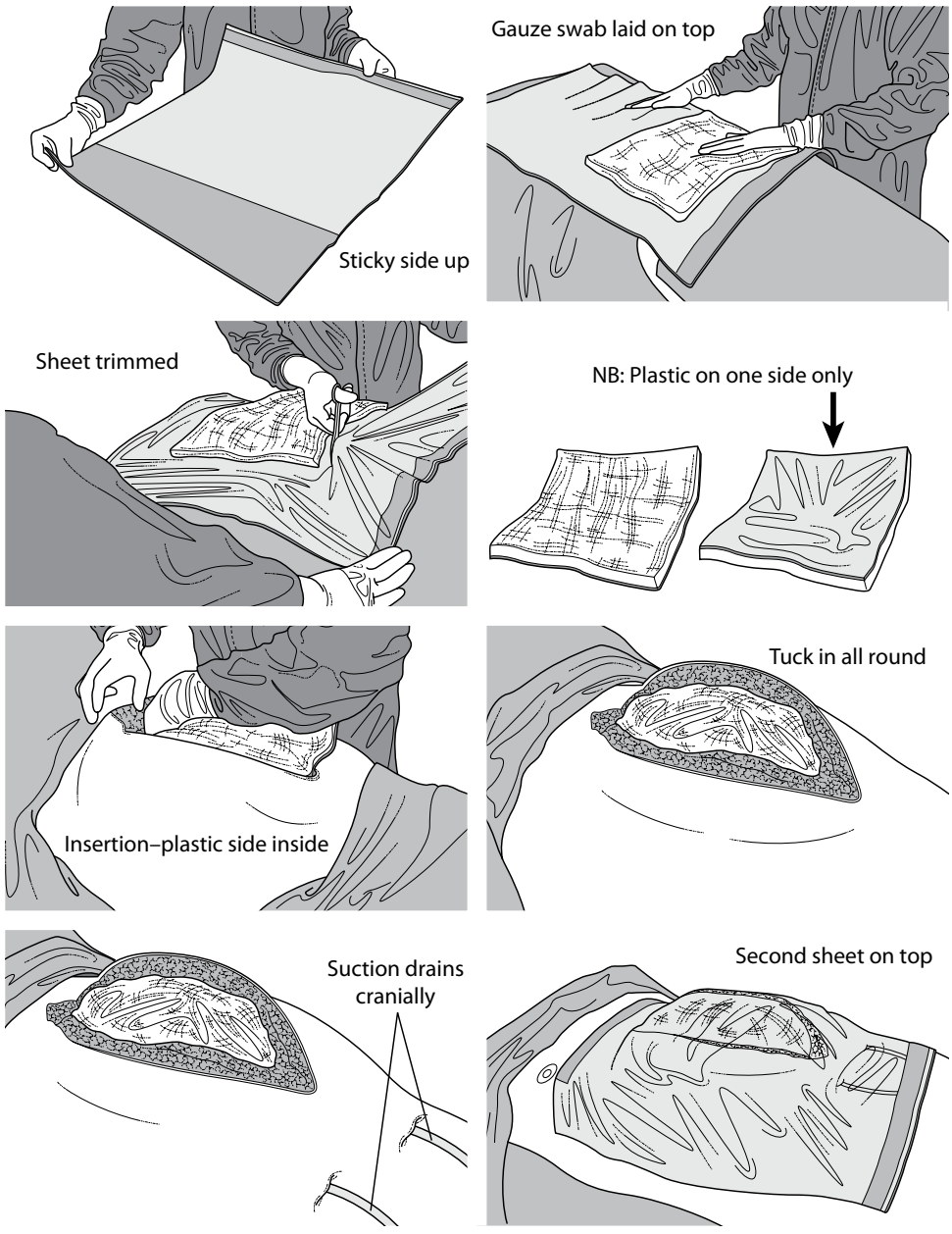

Figure 6.3 Sandwich technique for temporary abdominal closure.

6.3.3.5 MONITORING FOR AND MINIMIZING THE INCIDENCE OF INTRA-ABDOMINAL HYPERTENSION (AH) AND ABDOMINAL COMPARTMENT SYNDROME (ACS) (SEE ALSO SECTION 17.10)

- Measurement of intra-abdominal pressure (IAP).
  - Foley (bladder) catheter.
  - Intragastric catheter.

6.3.3.6 RECOGNITION OF ADDITIONAL INJURIES

- Review of injury mechanism, presentation, and those steps in the standard trauma resuscitation, along with diagnostics and therapies that were not completed to facilitate damage control.

- Tertiary survey.
- Additional imaging as needed.
- Review of history, co-morbid conditions, and medications.

6.3.4 Stage 4: Definitive Surgery

The patient is returned to the operating theatre as soon as physiological stability has been achieved. Ideally this would be within 24–48 hours, as delaying beyond 48 hours can be detrimental; however, the timing for this is determined by:

- The indication for damage control in the first place.
- The pattern of injury.
- The physiological response to resuscitation, warming, and evaluation as above.

Patients with ongoing bleeding despite correction of the other parameters require immediate return to the operating theatre or angiography suite.

Patients who develop major abdominal compartment syndrome must undergo re-look surgery early, and any further underlying causes must be corrected. This can and does occur even in the patient with a temporary abdominal closure.

Every effort must be made to return *all* patients to the operating theatre within 24–36 hours of their initial surgery. By leaving matters longer, other problems such as acute respiratory distress syndrome, SIRS, and sepsis may intervene (cause or effect) and may preclude further surgery.

6.3.4.1 THE 'RE-LOOK LAPAROTOMY'

The re-look laparotomy may be:

- *Planned*, that is, decided upon at the time of the initial procedure and usually for reasons of contamination, doubtful tissue viability, for retrieval of intra-abdominal packs or for further definitive surgery after a damage control exercise.
- *On demand*, that is, when evidence of intra-abdominal complication develops. In these cases, the principle applies of re-operation 'when the patient fails to progress according to expectation'. Failure to act in these circumstances may have dire consequences in terms of morbidity and mortality.

Re-exploration should be thorough, with careful evaluation for previously undiagnosed injuries. If the patient's physiological parameters deteriorate again, then the damage control philosophy should be reapplied with aggressive resuscitation, abbreviated laparotomy, and possible adjunctive procedures such as angiography, pelvic stabilization, or evaluation of extra abdominal injury. In many cases, repacking combined with aggressive correction of physiological endpoints including coagulopathy, along with further warming and coagulopathy, are necessary.

If a stoma (colostomy or ileostomy) is needed, the stoma is positioned more laterally, between the musculature of the anterior and mid axillary lines. The rectus fascia and musculature are thereby spared for future reconstruction, and the stoma is positioned well away from the midline wound, thus minimizing contamination. As the abdominal wall is reapproximated the stoma will move toward the anterior midline.

6.3.5 Stage 5: Abdominal Wall Closure

Once the patient has completed definitive surgery and no further operations are contemplated, the abdominal wall can be closed. In most series most patients had the fascia closed after the first re-look, or within 7 to 8 days from injury. This varies greatly depending on patient size and body habitus, time to achieve physiological stabilization, volume of fluid used to resuscitate, and injuries sustained.

6.3.5.1 DELAYED PRIMARY ABDOMINAL CLOSURE

Delayed closure will be required once the reasons for the temporary surgery have been removed or treated. This is usually possible after an interval of 24–48 hours (or longer). Usually, the abdominal wall can be closed in layers using normal closure techniques. However, this should only be performed when no further surgery is contemplated and should be accomplished without any fascial tension.

If abdominal viscera are protruding above the fascia in the supine anaesthetized patient, further management with temporary closure (vacuum assisted) is recommended. Return to the operating room after another 24–48 hours of intensive care and fluid mobilization in most cases will then allow primary fascial closure. Both surgeon and anaesthetist must be aware

of the danger of causing ACS as a result of the closure, and intra-abdominal or respiratory pressures must be closely monitored.

6.3.5.2 SECONDARY ABDOMINAL CLOSURE

If, for various reasons, a delayed primary closure is not possible, then it will be necessary to accept (but minimize) the defect that results. The skin of the abdominal wall and the underlying fascia retract, and thus prevent a primary closure from being achieved. During the period of the open abdomen, in this situation, several techniques are usually combined to get to a point where the fascia can be closed without tension and safely. The goal is to achieve fascial closure, and subsequent skin closure.

Methods involved which will minimize or reduce the eventual defect include:

- Vacuum-assisted closure:
  - Commercial closures have been shown to reduce the defect and assist in closure of the sheath. These include V.A.C. (Kinetic Concepts Inc., San Antonio, TX, USA), and Renasys (Smith and Nephew, London, UK).[15]
- Skin-only closure:
  - Grafts using Vicryl® mesh (Johnson and Johnson, New Brunswick, NJ, USA) or Gore-Tex® sheets (W L Gore and Associates Inc., Flagstaff, AZ, USA) or other synthetic sheets.
  - Biologic meshes – human dermal matrix (AlloDerm® – LifeCell, Bridgewater, NJ, USA) or porcine dermal matrix (Permacol – Covidien, Mansfield, MA, USA).
  - Split-thickness skin grafts directly on granulated bowel or mesh.

6.3.5.3 SECONDARY CLOSURE

If delayed primary closure is not possible after several days or a week, there are several options:

- Closure of skin only, allowing the formation of a hernia.
- Biological material such as human (AlloDerm) or porcine (Permacol) dermal matrix used as mesh early to prevent hernia formation, providing skin coverage.
- Continued vacuum-assisted temporary abdominal closure such as V.A.C.® (Kinetic Concepts Inc., KCI. San Antonio, TX, USA) and Renasys® (Smith and Nephew, London, UK) until granulation over bowel occurs, for subsequent split-thickness skin grafting.
- If a synthetic mesh is left *in situ*, skin coverage of the resulting defect by split-grafting or flap transfer – *never use a polypropylene only mesh.*
- For large hernias, often the need for later reconstruction using different techniques such as component separation and flap construction.
- A Wittmann patch.
- A multitude of tension assist devices that are now available.

An absorbable mesh of polyglycolic acid such as Vicryl® (Johnson and Johnson, New Brunswick, NJ, USA) elicits minimal tissue reaction and in-growth. Thus, it has a low risk of infection or fistula formation.

Membranes such as polytetrafluoroethylene (PTFE) (Gore-Tex® – W L Gore and Associates, Flagstaff, AZ, USA), which elicit minimal tissue reaction and in-growth and thus minimize risk of infection or fistula formation, can be used, but this is considerably costlier. Recently, composite meshes have shown promise. The mesh can then have a skin graft placed upon it (or even directly on bowel), and definitive abdominal wall reconstruction can take place at a later stage.

Should any mesh be used and left *in situ*, however, the resulting defect will require skin coverage by split-grafting or flap transfer.

6.3.5.4 PLANNED HERNIA

A planned hernia approach aims at skin coverage with subsequent delayed abdominal wall reconstruction. Conditions favouring a planned hernia strategy include the inability to reapproximate the retracted abdominal wall edges, sizeable tissue deficit, risk of tertiary abdominal compartment syndrome, inadequate infection source control, anterior enteric fistula, and poor nutritional status of the patient. Various techniques have been applied. Autologous split-thickness skin grafting over the visceral content of the abdomen allows coverage of the exposed bowel. Maturation of the skin graft requires about 6–12 months, after which the grafted skin can be easily removed from the bowel surface for reconstruction.

Bridging mesh is often employed, and absorbable mesh is preferred. Non-absorbable (polypropylene mesh,

etc.) has been abandoned because of infection and high rates of fistula formation.

More commonly a component separation is performed. A large relaxing incision is placed in the exterior oblique muscle component of the anterior rectus muscle bilaterally. This can be combined with mobilization of the rectus muscle from the posterior fascial sheath to provide local advancement across the hernia defect. Modification of this technique has been described with division of the internal oblique to the arcuate line. Large abdominal wall defects can be reconstructed with pedicular or microvascular flaps. The most commonly used is the tensor fascia lata (TFL) flap.

6.3.6 **Outcomes**

In a recent study of 88 damage control patients with a mean injury severity score of 34, Brenner et al. reported of the 63 survivors, 81% had gone back to work and resumed normal daily activities.[16]

6.4 DAMAGE CONTROL ORTHOPAEDICS[17]

It used to be orthopaedic standard of care that patients who were too sick, should have their surgery delayed for up to two weeks. However, with the arrival of DCS techniques, even in the critically ill, it became apparent that morbidity, post-operative complications, lengths of stay, and outcomes were improved by a damage control approach, with early (even temporary) fixation. Fracture care (possibly because of the large energy required to break the bone in the first place), plays a large part in minimizing the body's systemic inflammatory response to trauma.

The goals of damage control orthopaedics are to limit ongoing haemorrhage and soft tissue through efficient fracture stabilization, while minimizing additional physiological insult. Care is to follow normal damage control principles, such as avoiding the triad of hypothermia, coagulopathy, and acidosis, and minimizing secondary injury to organ systems (kidney, brain, etc.). External fixation is employed for long-bone fractures and pelvic injuries. In addition to wound toilet and removal of devitalized tissues, surgical management of haemorrhage and fasciotomies for potential or actual compartment syndrome are performed.

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Part 3

Anatomical and organ system injury

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The Neck 7

7.1 OVERVIEW

The high density of critical vascular, aerodigestive, and neurological structures within the neck makes the management of penetrating injuries difficult and contributes to the morbidity and mortality seen in these patients.

Before World War II, non-operative management of penetrating neck trauma resulted in mortality rates of up to 15%. Therefore, the exploration of all neck wounds penetrating the platysma muscle became mandatory. However, in recent years, numerous centres have challenged this principle of mandatory exploration, since up to 50% of neck explorations may be negative for significant injury.

7.2 MANAGEMENT PRINCIPLES: PENETRATING CERVICAL INJURY

7.2.1 Initial Assessment and Definitive Airway

Patients with signs of significant neck injury with active bleeding or an expanding haematoma, will require prompt exploration. However, initial assessment and management of the patient should be carried out according to Advanced Trauma Life Support® principles.

The major initial concern in any patient with a penetrating neck wound is early control of the airway. Supplemental oxygen is critical. Oxygenate the patient with simple measures first (i.e. basic airway opening techniques).

Intubation in these patients is complicated by the possibility of associated cervical spine injury, laryngeal trauma, and large haematomas in the neck. Appropriate protective measures for possible cervical spine injury must be implemented. While it is important to protect the unstable/potentially unstable cervical spine with immobilization, a rapid risk/benefit assessment must be done in the presence of a deteriorating airway. Cervical spine immobilization techniques make endotracheal intubation more difficult.

The route of intubation must be carefully considered in these patients since it may be complicated by distortion of anatomy, haematoma, dislodging of clots, laryngeal trauma, and cervical spinal injury. It may be possible to place an endotracheal tube over a bronchoscope, enter the trachea under direct vision, and then slide the endotracheal tube into place. Use of the fibre-optic portable video laryngoscope (Glidescope Go®, Verathon, Bothell, WA, USA) may prove useful.

Recent UK military experience in Afghanistan is that most patients can be managed with a rapid sequence induction of anaesthesia followed by endotracheal intubation with a relatively small tube (e.g. size 6.5 mm or 7.0 mm in an adult male) aided by a gum-elastic bougie or stylet. The caveat is that a surgeon is scrubbed and ready to perform a cricothyroidotomy if it becomes immediately obvious that the trachea cannot be intubated following rapid sequence induction (RSI).

Patients have also been managed by a gas induction of anaesthesia (supplemented with small boluses of ketamine – 10 mg IV), laryngoscopy while still breathing, then endotracheal intubation.

Patients with signs of significant neck injury, and those whose condition is unstable, should be explored urgently in the operating room with good light, good instruments, and good assistance, once rapid initial assessment has been completed and the airway has been secured. Tracheostomy has no place in the emergency department. Cricothyroidotomies should be converted to formal tracheostomies within about 48 hours.

Pitfalls

- Be aware that bleeding into the airway will rapidly obscure the view of the cords.
- Laryngeal mask airways (LMAs) do NOT provide a definitive airway in the presence of complex neck injuries, but can be used to attempt oxygenation prior to endotracheal tube intubation or surgical airway.
- Endotracheal intubation should be considered at a very early stage in the presence of a large or expanding haematoma, before the anatomy is lost. Subsequent cricothyroidotomy or emergency percutaneous tracheostomy (only to be considered if the enabling environment, with extensive experience in the technique, is available), can be technically difficult as by entering the haematoma planes, torrential bleeding via the skin incision may result.
- *NB: The use of paralysing agents in these patients should be used as a last resort, and with extreme caution, since the airway may be held open only by the patient's own use of muscles.* Abolishing the use of muscles in such patients may result in the immediate and total obstruction of the airway and, with no visibility owing to the presence of blood, may result in catastrophe. Ideally, local anaesthetic spray should be used with sedation, and a cricothyroidotomy (below the injury) should be considered when necessary.

Managing complicated airway injuries should be rehearsed along with back-up plans for alternate techniques to secure the airway if the initial plan fails. The decision as to what technique is the most appropriate should be made rapidly and jointly by the surgeon, anaesthetist, and trauma team leader.

There should be no hesitation in performing an emergency cricothyroidotomy should circumstances warrant it.

7.2.2 Control of Haemorrhage

Control of haemorrhage should be done by direct pressure where possible. If the neck wound is *not* bleeding, do not probe or finger the wound, as the clot may be dislodged. If the wound is actively bleeding, the bleeding should be controlled by digital pressure or if ineffective, a Foley catheter may be inserted into the wound and the balloon inflated. This is often useful for stopping haemorrhage deep in the neck. Occasionally, the wound may need to be sutured around the catheter to produce the required tamponade.

Pitfall

This may result in converting external haemorrhage into concealed bleeding deep within the neck if tamponade is not achieved and will further compromise an already compressed airway. Hence the importance of securing an early definitive airway. More than one Foley catheter is sometimes needed.

7.2.3 Injury Location

Division of the neck into anatomical zones (Figure 7.1) helps the categorization and management of neck wounds:

- *Zone I* extends from the level of the sternal notch to the lower border of the cricoid cartilage. Within zone I lie the great vessels, the trachea, the oesophagus, the thoracic duct, and the upper mediastinum and lung apices.
- *Zone II* includes the area between the cricoid cartilage and the angle of the mandible. Enclosed within its region are the carotid and vertebral arteries, jugular veins, pharynx, larynx, oesophagus, and trachea.
- *Zone III* includes the area above the angle of the mandible to the base of the skull, the pharynx, and the distal extracranial carotid and vertebral arteries, as well as segments of the jugular veins.

Injuries in zone II are readily evaluated and exposed operatively. In trauma centres with a high volume of penetrating trauma, a decision on immediate surgery can be reached on clinical grounds without any pre-operative investigations. Zones I and III are difficult to assess clinically and to access operatively. Therefore, in those two zones it is important in the physiologically stable patient

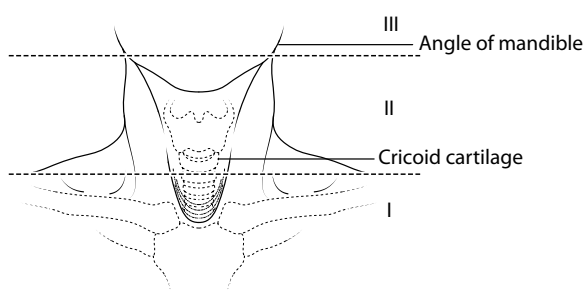

Figure 7.1 Anterior view showing zones of the neck.

to proceed with diagnostic work-up so that the presence or absence of injury is established, the possibility of dealing with it without an operative procedure is decided (e.g. embolization of bleeding artery), as should operation be necessary, this will be facilitated by knowing the type of injury and its topography.

Pitfall

Assuming that a wound in one zone means an injury in that zone. Not infrequently, a wound in one zone is associated with the injury in quite another area. For example, a long knife may penetrate the skin in zones I or II, but the injury may lie in the superior mediastinum. Similarly, bullets and missile fragments often transgress zone boundaries. Either may cross the midline, giving rise to contralateral injured structures.

7.2.4 Mechanism

Gunshot wounds carry a higher risk of major injury than stab wounds, because of their tendency to penetrate more deeply and their ability to damage tissue outside the tract of the missile owing to cavitation, or a percussion wave injury.

7.2.5 Frequency of Injury

The carotid artery and internal jugular vein are the most frequently injured vessels. Owing to its relatively protected position, the vertebral artery is involved less frequently. The larynx and trachea, and pharynx and oesophagus are frequently injured. The spinal cord is damaged less often, but injury should not be ruled out until examination has confirmed its integrity.

7.2.6 Use of Diagnostic Studies

In the stable patient without indications for immediate neck exploration, additional studies are often obtained, including computed tomography (CT) angiography, endoscopy, contrast radiography, and bronchoscopy.

7.2.6.1 COMPUTED TOMOGRAPHY SCANNING WITH CONTRAST/COMPUTED TOMOGRAPHY ANGIOGRAPHY

Modern CT scanners have provided a means of creating a three-dimensional (3D) images of high quality, and CT scanning is now the investigation of choice for penetrating injury in the stable patient. Angiography has been largely replaced by CT-angiography and should include both internal and external carotid arteries on both sides (four vessel CT angiography), as well as the vertebral arteries, preferably with 3D reconstruction. All patients with zone I and III neck injuries should undergo CT angiography provided their vital signs are stable.

7.2.6.2 ANGIOGRAPHY

Especially in zone I or zone III injuries, where surgical exposure can be difficult, open angiography allows arterial embolization, as well as stenting of a particular vessel.

Using a selective approach to the management of zone II wounds, angiography is useful in excluding carotid injuries, especially with soft signs of injury, including stable haematoma and history of significant bleeding, or when the wound is near the major vessels.

7.2.6.3 OTHER DIAGNOSTIC STUDIES

The selective management of penetrating neck wounds involves evaluation of the oesophagus, larynx, and trachea. Modern fluoroscopy machines with water-soluble contrast of an iso-oncotic nature (e.g. Omnipaque) will detect 99% of clinically relevant oesophageal injuries.[1] Flexible endoscopy may increase the yield even further. Laryngoscopy and bronchoscopy are useful adjuncts in localizing or excluding injury to the hypopharynx or trachea. A recent development is 3D CT reconstruction of the trachea and bronchi can replace endoscopy in suspected injuries of these anatomical structures.

7.3 MANAGEMENT

7.3.1 Mandatory versus Selective Neck Exploration

Recommendations for the management of patients with penetrating cervical trauma depend on the zone of injury and the patient's clinical status. If the platysma is not penetrated, the patient may be observed. Mandatory exploration for penetrating neck injury in patients with hard signs of vascular or aerodigestive tract injury is still appropriate.

Missed injuries are associated with a high morbidity and mortality due to missed visceral injuries, and the negligible morbidity caused by negative exploration are important; however, exploration of all stab wounds of the

neck may yield a high rate of non-therapeutic procedures. Thus, the selective management of penetrating neck wounds based on CT evaluation has been recommended.

7.3.2 Management Based on Anatomical Zones

- Zone I injuries require angiography because of the increased association of vascular injuries with penetrating trauma to the thoracic outlet. Angiography helps the surgeon plan the surgical approach.
- Zone II injuries may require angiography if the vertebral vessels are thought to be injured. Venous phasing should be requested.
- Zone III injuries require angiography because of the relationship of the blood vessels to the base of the skull. Often these injuries can be best managed by either non-operative techniques or manoeuvres remote from the injury site such as balloon tamponade or embolization.

Pitfall

Angiography will *not* rule out significant injury to the trachea or oesophagus.

7.4 ACCESS TO THE NECK

The operative approach selected to explore neck injuries is determined by the structures known or suspected to be injured. Surgical exploration should be done formally and systematically in a fully equipped operating room under general anaesthesia with endotracheal intubation. Blind probing of wounds or mini-explorations in the emergency department should **never** be attempted.

7.4.1 Position

Active bleeding from a penetrating wound should be controlled digitally. Penetrating wounds to the neck should not be probed, cannulated or locally explored because these procedures may dislodge a clot and cause uncontrollable bleeding or air embolism. Skin preparation should include the entire chest and shoulder, extending above the angle of the mandible. The face, neck, and anterior chest should be prepped and widely draped. The patient is placed in the supine position on the operating table with the arms tucked at the sides. If possible, the head should be extended and rotated to the contralateral side. A sandbag may be placed between the shoulder blades, and the neck extended and rotated away from the side of injury – provided that the cervical spine has been cleared pre-operatively (Figure 7.2).

7.4.2 Incision

Always expect the worst! Plan the incision to provide optimal access for early vascular source control or immediate access to the airway. The most universally applicable approach is via an incision along the anterior border of sternomastoid, which can be lengthened proximally and distally, extended to a median sternotomy or augmented with lateral extensions (Figure 7.3).

Figure 7.2 Approach to the left side of the neck – patient positioning.

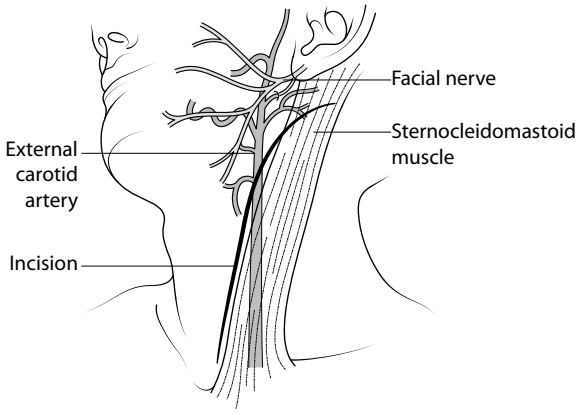

Figure 7.3 Approach to the left side of the neck – initial incision.

Having made the sternocleidomastoid muscle incision, platysma is divided, and the sternomastoid is retracted laterally to expose the fascial sheath covering the internal jugular vein. The vein cannot be mobilized until the common facial vein is divided. Omohyoid muscle is the only strap muscle that crosses the carotid sheath obliquely, and acts as a landmark for the common carotid artery. Lateral retraction of the jugular vein and underlying carotid artery allows access to the trachea, oesophagus, and thyroid, and medial retraction of the carotid sheath and its contents will allow the dissection to proceed posteriorly to the prevertebral fascia and vertebral arteries. Posterior to the carotid sheath, the sympathetic chain lies on longus colli, which separates it from the transverse processes of the cervical vertebrae (Figure 7.4).

7.4.3 Surgical Access

7.4.3.1 ZONE I

Zone I vascular injuries at the base of the neck require aggressive management. Frequently, uncontrollable haemorrhage will require immediate thoracotomy for initial proximal control. In an unstable patient, quick exposure often may be achieved via a median sternotomy and a supraclavicular extension.

The location of the vascular injury will dictate the definitive exposure. For right-sided great vessel injuries, a median sternotomy with a supraclavicular extension allows optimal access. On the left side, a left anterolateral thoracotomy may provide initial proximal control. Further operation for definitive repair may require a sternotomy, or extension into the right side of the chest or up into the neck. 'Trapdoor' or 'clamshell' incisions are ***not*** recommended. They are often difficult to perform, do not significantly improve the exposure, but significantly increase the morbidity postoperative disability. Avoid injury to the phrenic and vagus nerves as they enter the thorax. In the stable patient in whom the vascular injury has been confirmed by CT, the right subclavian artery or the distal two-thirds of the left subclavian artery can be exposed through an incision immediately superior to the medial third of the clavicle. Injuries to the vessels behind

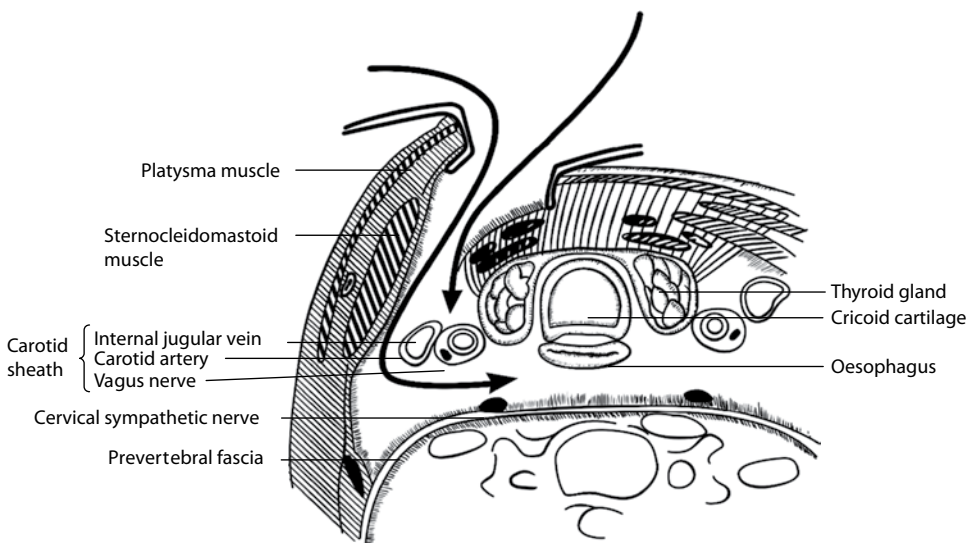

Figure 7.4 Approach to the left side of the neck showing retraction of the platysma and sternomastoid muscles.