Manual of Definitive Surgical Trauma Care

SECOND EDITION

Edited by Kenneth D. Boffard

International Association for the Surgery of Trauma and Surgical Intensive Care
Manual of Definitive Surgical Trauma Care
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Unless they deal with major trauma on a particularly frequent basis, few surgeons can attain and sustain the level of skill necessary for decision-making in major trauma. This includes both the intellectual decisions and the manual dexterity required to perform all the manoeuvres for surgical access and control. These can be particularly challenging, and may be infrequently required, yet rapid access to and control of sites of haemorrhage after trauma can be a life-saving surgical intervention. Many situations require specialist trauma expertise, yet often this is simply not available within the time frame in which it is required.

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 an understanding of the following:

- The causation of injuries 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 (Howard Champion, USA; Stephen Deane, Australia; Abe Fingerhut, France; David Mulder, Canada; and Don Trunkey, USA), members of the International Society of Surgery – Société Internationale de Chirurgie (ISS-SIC) – and the International Association for Trauma Surgery and Intensive Care (IATSIC), met in San Francisco during a meeting of the American College of Surgeons. It was apparent that there was a specific need for further surgical 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. Particular attention needed to be directed to those who were senior trainees or had completed their training.

It was believed that a short course focusing on the life-saving surgical techniques and surgical decision-making was required for surgeons, in order further to train the surgeon who dealt with major surgical trauma on an infrequent basis to deal with major trauma. This course would meet a worldwide need, and would supplement the well-recognized and accepted American College of Surgeon Advanced Trauma Life Support (ATLS®) course. The experience that Sten Lennquist had gained offering 5-day courses for surgeons in Sweden was integrated into the programme development, and prototype courses were offered in Paris, Washington and Sydney.

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. A unique feature of the Course is that, although the principles are standardized, once the Course has been established nationally in a country, it can be modified to suit the needs and circumstances of the environment in which the care takes place. The Education Committee of IATSIC has an International DSTC™ Sub-committee which oversees the quality and content of the courses. However, the concept of the Definitive Surgical Trauma
Care Course has remained the same. In addition to the initial ‘founding’ countries (Australia, Austria and South Africa), courses have been delivered in more than 15 countries across the world, with the new participants joining the IATSIC programme each year. The course is designed to support those who, whether through choice or necessity, must deal with major trauma, and may not necessarily have the experience of expertise required. The requirements for a DSTC™ Course or the establishment of a DSTC™ programme can be found in Appendix C of this book.

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.

An Editorial Board, made up of those who have contributed to the DSTC™ programme, 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.

This second edition had been revised and updated, taking into account new evidence-based information. It is dedicated to those who care for the injured patient and whose passion is to do it well.

Ken Boffard
Editor
Part 1

Introduction
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Injury (trauma) remains a major health problem worldwide and, in many countries, it continues to grow. The care of the injured patient should ideally be a sequence of events involving education, prevention, acute care and rehabilitation. In addition to improving all the aspects of emergency care, improved surgical skills will save further lives and contribute to minimizing disability.

The standard general surgical training received in the management of trauma is often deficient, partly because traditional surgical training is increasingly organ specific, concentrating on ‘superspecialties’ such as vascular, hepatobiliary or endocrine surgery, and partly because, in most developed training programmes, there is a limited exposure to the range of injured patients.

1.1 THE NEED FOR A DEFINITIVE SURGICAL TRAUMA CARE COURSE

Advanced Trauma Life Support Course of the American College of Surgeons

The Advanced Trauma Life Support (ATLS®) programme is probably the most widely accepted trauma programme in the world, with almost 50 national programmes currently taking place.

Yuen and Chung report that over 180 physicians in Hong Kong have been trained in ATLS. Kobayoshi describes how many surgeons in Japan have a high standard of surgical skills before entering traumatology, but each emergency care centre sees only a few hundred major trauma cases per year. Many trauma cases, especially those associated with non-penetrating trauma, are treated non-surgically, resulting in insufficient operative exposure for the training of young trauma surgeons.

Barach et al. reported that the ATLS® Course was introduced to Israel in 1990, and that over 4000 physicians have been trained. In 1994, the scientific board of the Israeli Medical Association accredited the ATLS® programme and mandated that all surgical residents become ATLS certified. There is also a combat trauma life support course for active military doctors.

Rennie described the need for training health officers in emergency surgery in Ethiopia. Much of the pathology in rural Ethiopia is secondary to trauma, and there is a real need for trauma surgical education.

Arreola-Risa et al. reported that there are no formal post-residency training programmes in Mexico. The ATLS® Course has been successfully implemented and has a 2-year waiting list.

Jacobs described the development of a trauma and emergency medical services (EMS) system in Jamaica. There is a significant need for a formalized trauma surgical technical educational course that could be embedded in the University of the West Indies.

Trauma continues to be a major public health problem, both in the pre-hospital setting and within the hospital system.

In addition to increasing unrest politically and socially, resulting in the increasing use of firearms for personal violence, the car has become a substantial cause of trauma worldwide. These socio-economic determinants have resulted in a large number of injured patients. Although, undoubtedly, prevention of injury will play one of the major roles in the reduction of mortality and morbidity caused by trauma, there will also be the need to minimize secondary injury to patients as a result of inadequate or inappropriate management.

There is thus the increasing need to provide the surgical skills and techniques necessary to resuscitate and manage these patients surgically – the period after ATLS is complete.

In the USA, trauma affects both young and elderly people. It is the third leading cause of death for all ages and the leading cause of death from age 1 to 4.4 People
under the age of 45 account for 61 per cent of all injury fatalities and 65 per cent of hospital admissions. However, people aged 65 and older are at a higher risk of both fatal injury and more protracted hospital stay. About 50 per cent of all deaths occur minutes after injury, and most immediate deaths are because of massive haemorrhage or neurological injury. Post-mortem data has demonstrated that central nervous system injuries account for 40–50 per cent of all injury deaths and haemorrhage accounts for 30–35 per cent. Motor vehicles and firearms accounted for 29 and 24 per cent of all injury deaths in 1995.9,10

In South Africa there is a high murder rate (56 per 100 000 population) and a high motor vehicle accident rate.11

There are other areas of the world, such as Australia and the UK, where penetrating trauma is unusual, and sophisticated injury prevention campaigns have significantly reduced the volume of trauma. However, there is a significant amount of trauma from motor vehicles, falls, recreational pursuits and among elderly people. The relatively limited exposure of surgeons to major trauma provides, as a mandate, a requirement for designated trauma hospitals and specific skill development in the management of major trauma.

Furthermore, there are multiple areas of the developing world – the West Indies, South America and Africa – where general surgical training may not necessarily include extensive surgical education and psychomotor technical expertise on trauma procedures. There are other countries where thoracic surgery is not an essential part of general surgical training. Therefore, a general surgeon called upon to control thoracic haemorrhage definitively may not have had the required techniques incorporated into formal surgical training.

Various techniques for stabilization of fractures, and pelvic fixation, may have an important place in the initial management of trauma patients.

For these reasons the course needs to be flexible in order to accommodate the local needs of the country in which it is being taught.

Military conflicts occur in numerous parts of the world. These conflicts involve not only superpowers, but also the military of a large number of other countries. It is essential that the military surgeon be well prepared to manage any and all penetrating injuries that can occur on the battlefield. The increasing dilemma that is faced by the military is that conflicts are, in general, small and well contained, and do not produce casualties in large numbers or on a frequent basis. For this reason, it is difficult to have a large number of military surgeons who can immediately be deployed to perform highly technical surgical procedures in the battlefield arena, or under austere conditions.

It is increasingly difficult for career military surgeons to gain adequate exposure to battlefield casualties, or indeed penetrating trauma in general, and increasingly many military training programmes are looking to their civilian counterparts for assistance.

These statistics mandate that surgeons responsible for the management of these injured patients, whether military or civilian, be skilled in the assessment, diagnosis and operative management of life-threatening injuries. There remains a poorly developed appreciation among many surgeons 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, and partly because of other interests, many surgeons quite simply no longer have the expertise to deal with such life-threatening situations.

## 1.2 COURSE OBJECTIVES

By the end of the course, the student will have received training to allow:

- Enhanced surgical decision-making in trauma
- Enhanced surgical techniques in the management of major trauma.

## 1.3 DESCRIPTION OF THE COURSE

A prerequisite of the Definitive Surgical Trauma Care (DSTC™) Course is a complete understanding of all the principles outlined in a general surgical training, and also the ATLS® Course. For this reason, there are no lectures on the basic principles of trauma surgery or on the initial resuscitation of the patient with major injuries.

The course consists of a core curriculum, designed to be a two-and-a-half-day activity. In addition to the core curriculum, there are a number of modules that can be added to the course to allow it to be more suited to local conditions in the area in which it is being taught.

The course consists of a number of components:

- Didactic lectures: designed to introduce and cover key concepts of surgical resuscitation, end points and overview of best access to organ systems.
• Cadaver sessions: 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.

• Animal laboratories: where possible, use is made of live, anaesthetized animals, prepared for surgery. The instructor introduces various injuries. The objects of the exercise are both to improve psychomotor skills and to teach new techniques for preservation of organs and control of haemorrhage. The haemorrhagic insult is such that it is a challenge to both the veterinary anaesthetist and the surgeon to maintain a viable animal. 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.

1.4 SUMMARY

The course is therefore designed to prepare the relatively fully trained surgeon to manage difficult, surgically created injuries, which mimic the injuries that might present to a major trauma centre. The course fulfils the educational, cognitive and psychomotor needs for mature surgeons, surgical trainees and military surgeons, 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 civilian arena.

REFERENCES

1 Yuen WK, Chung CH. Trauma care in Hong Kong. Trauma Q 1999;14:241–7.
7 Jacobs LM. The development and implementation of emergency medical and trauma services in Jamaica. Trauma Q 1999;14:221–5.
Part 2

Physiology and Metabolism
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2.1 METABOLIC RESPONSE TO TRAUMA

2.1.1 Definition of trauma

Bodily injury is accompanied by systemic as well as local effects. Any stress, including injury, surgery, anaesthesia, burns, vascular occlusion, dehydration, starvation, sepsis, acute medical illness or even severe psychological stress, will initiate the metabolic response to trauma.\(^1,2\)

Following trauma, the body responds locally by inflammation and by a general response which is 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 was divided into an ebb and flow phase by Cuthbertson.\(^3\)

The ebb phase corresponds to the period of severe shock characterized by depression of enzymatic activity and oxygen consumption. Cardiac output is below normal, core temperature may be subnormal and a lactic acidosis is present.

It is characterized by the following metabolic changes:

- An elevated blood glucose level
- Normal glucose production
- Elevated free fatty acid levels
- A low insulin concentration
- Elevated levels of catecholamines and glucagon
- An elevated blood lactate level
- Depressed oxygen consumption
- Below-normal cardiac output
- Below-normal core temperature.

The ebb phase is dominated by cardiovascular instability, alterations in circulating blood volume, impairment of oxygen transport and heightened autonomic activity. Emergency support of cardiopulmonary performance is the paramount therapeutic concern.

After effective resuscitation has been accomplished and restoration of satisfactory oxygen transport has been achieved, 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 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 flux increased
- 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.

2.1.2 Initiating factors

The magnitude of the metabolic response depends on the degree of trauma and concomitant contributory factors such as drugs, sepsis and underlying systemic disease. The response will also depend on the age and sex of the patient, the underlying nutritional state, the timing of treatment, and its type and 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 seems to be less aggressive in children and elderly people and in premenopausal women. Starvation and nutritional depletion also modify the response. Patients with poor nutritional or immunological status (e.g. those with human immunodeficiency virus) have a reduced metabolic response to trauma compared with well-nourished patients.

Burns cause a relatively greater response than other injuries of comparable extent, probably because of the propensity for greater continued volume depletion and heat loss.

Wherever possible, efforts must be made to prevent or reduce the magnitude of the initial insult, because by doing so it may be possible to reduce the nature of the response, which, although generally protective, may also be harmful. Thus, aggressive resuscitation, control of pain and temperature, and adequate fluid and nutritional provision are critical.

The precipitating factors can broadly be divided into the following.

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

2.1.2.2 AFFERENT IMPULSES
- Somatic
- Autonomic.

2.1.2.3 WOUND FACTORS: INFLAMMATORY AND CELLULAR
- Neutrophils
- Cytokines
- Eicosanoids
- Prostanoids
- Leukotrienes
- Macrophages
- Interleukin-1 (IL-1)
- Proteolysis-inducing factor (PIF).

2.1.2.4 TOXINS/SEPSIS
- Endotoxins
- Exotoxins.

2.1.2.5 FREE RADICALS

2.1.2.6 HYPOVOLAEMIA

It is said that hypovolaemia, specifically involving tissue hypoperfusion, is the most potent precipitator of the metabolic response. Hypovolaemia can also be the result of external losses, internal shifts of extracellular fluids, and changes in plasma osmolality. However, the most common cause is blood loss secondary to surgery or traumatic injury.

<table>
<thead>
<tr>
<th>Class of shock</th>
<th>Percentage blood loss</th>
<th>Volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>&lt; 750</td>
</tr>
<tr>
<td>II</td>
<td>30</td>
<td>750–1500</td>
</tr>
<tr>
<td>III</td>
<td>40</td>
<td>2000</td>
</tr>
<tr>
<td>IV</td>
<td>&gt; 40</td>
<td>&gt; 2000</td>
</tr>
</tbody>
</table>

Class III or IV shock is severe and, unless treated as a matter of urgency, will make the situation much worse (see Section 2.2, Shock).

The hypovolaemia will stimulate catecholamines, which in turn trigger the neuroendocrine response. This plays an important role in volume and electrolyte conservation, and protein, fat and carbohydrate catabolism. Early fluid and electrolyte replacement, and parenteral or enteral surgical nutrition, administering amino acids to injured patients losing nitrogen at an accelerated rate, and giving fat and carbohydrates to counter caloric deficits, may modify the response significantly. However, the variety of the methods available should not distract the surgeon from his or her primary responsibility of adequate resuscitation.

2.1.2.7 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.

2.1.2.8 WOUND FACTORS

Endogenous factors may prolong or even exacerbate the surgical insult, despite the fact that the primary cause
can be treated well. Tissue injury activates a specific response, along two pathways:

1. Inflammatory (humoral) pathway
2. Cellular pathway.

Uncontrolled activation of endogenous inflammatory mediators and cells may contribute to this syndrome.

Both humorally and cell-derived activation products play a role in the pathophysiology of organ dysfunction. It is important, therefore, to monitor post-traumatic biochemical and immunological abnormalities whenever possible.

2.1.3 Immune response

2.1.3.1 THE INFLAMMATORY PATHWAY

The inflammatory mediators of injury have been implicated in the induction of membrane dysfunction. That leukocytes have been involved in inflammatory processes has been known for more than 100 years. Initially these are neutrophils, which also play a major role in reperfusion injury.

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 leukocytes. Cytokines are generally divided into proinflammatory cytokines and anti-inflammatory cytokines. Discussion of cytokines is often complicated with confusing nomenclature. Many cytokines were found in different settings, e.g. tumour necrosis factor (TNF) is also known as cachectin. Current nomenclature follows a more consistent system. The term ‘interleukin’ (IL) refers to a substance that acts between leukocytes and is used together with a number, e.g. IL-6.

Proinflammatory cytokines

Certain cytokines, particularly TNF, IL-1 and IL-8, promote the inflammatory response by upregulating expression of genes encoding phospholipase A₂, cyclooxygenase-2 (COX-2) or inducible nitric oxide (NO) synthase, thereby increasing the proinflammatory mediators. Proinflammatory cytokines mediate neutrophils action, causing cell migration into the tissues and activating neutrophils degranulation – all of which lead to tissue damage.

Tumour necrosis factor and IL-1 act synergistically to produce the acute innate immune response to invading micro-organisms. Tumour necrosis factor 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 and IL-12), 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 cannot induce apoptosis.

Interferon γ (IFN-γ) is produced in response to antigen, an event enhanced by IL-12. It activates macrophages. Interleukin 12 is produced by mononuclear phagocytes and dendritic cells in response to intracellular microbes. Interleukin 6 is produced by mononuclear phagocytes, endothelial cells and fibroblasts, and acts in a proinflammatory manner by providing a potent stimulus for hepatocyte synthesis of acute phase proteins.

Interleukin 2, unlike the above cytokines that exert most of their influence via the innate immune system, mediates acquired immunity and has an immunomodulatory function as well.

Anti-inflammatory cytokines

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

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

Modulation of cytokine activity in sepsis, systemic inflammatory response syndrome and compensatory inflammatory response syndrome

Systemic inflammatory activity, which may occur in response to infectious or non-infectious stimuli, is the fundamental phenomenon in which systemic inflammatory response syndrome (SIRS) occurs in response to a stimulus, and the whole-body inflammatory response can lead ultimately to multisystem organ dysfunction syndrome (MODS), which is associated with a mortality rate higher than 50 per cent. It was initially suggested that SIRS and sepsis were attributable to an overwhelming
proinflammatory immune response, moderated by TNF and other cytokines. Another view suggests that the body also mounts a countering anti-inflammatory reaction, which can then lead to compensatory inflammatory response syndrome (CARS). It may be that, when the proinflammatory response predominates, SIRS and shock result and, when the anti-inflammatory response predominates, CARS, immunosuppression and increased susceptibility to infection result.

It is clear that the role of cytokines in sepsis is very complex, with both proinflammatory and anti-inflammatory factors playing a role, and determining clinical outcome.

**Activated protein C (Xigris®)**

Proinflammatory factors 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 response by activating thrombin-activatable fibrinolysis inhibitor (TAFI). This procoagulant’s response leads to microvascular thrombosis and is implicated in the multiple organ failure associated with sepsis.

Protein C is an endogenous anticoagulant that reduces the generation of thrombin, and also exerts an anti-inflammatory effect. Decreased levels are usually present in sepsis and are associated with an increased mortality. A multicentre phase III trial comprising 1690 patients documented a 6.1 per cent reduction in total mortality in patients treated with recombinant human activated protein C (Xigris®). Taken as a whole, the current data suggests that, in view of the side-effect profile and potential complications of activated protein C, its use should be considered only in patients who have severe sepsis and are at high risk for death.

**Eicosanoids**

These compounds, derived from eicosapolyenoic fatty acids, may be subdivided into prostanoids (the precursors of the prostaglandins), leukotrienes (LTs) and thromboxanes. Eicosanoids are synthesized from arachidonic acid, which has been synthesized from phospholipids of damaged cell walls, white blood cells and platelets, by the action of phospholipase A2. Cyclooxygenase converts arachidonic acid to prostanoids, the precursors of prostaglandin (PG), prostacyclin (PGI) and thromboxanes (Tx). The term ‘prostaglandins’ is used loosely to include all prostanoids. The leukotrienes and prostanoids derived from the arachidonic acid cascade play an important role in inflammation. Eicosanoids modulate blood flow to organs and tissues by altering local balances between production of vasodilators and production of vasoconstricting components.

**Prostanoids**

The prostanoids (prostaglandins of the E and F series), PGI₂ and Tx synthesized from arachidonic acid by cyclooxygenase (in TxA₂), endothelial cells, white cells and platelets cause not only vasoconstriction (TxA₂ and PGF₁), but also vasodilatation (PGI₂, PGE₁ and PGE₂). Thromboxane A₂ activates and aggregates platelets and white cells, and PGI₂ and PGE₁ inhibit white cells and platelets.

**Leukotrienes**

Lipoxygenase, derived from white cells and macrophages, converts arachidonic acid to leukotrienes (LTB₄, LTC₄ and LTD₄). The leukotrienes cause vasoconstriction, increased capillary permeability and bronchoconstriction.

**Thromboxanes**

These are agents that cause contraction of vascular smooth muscle and platelet aggregation.

**2.1.3.2 THE CELLULAR PATHWAY**

There are a number of phagocytic cells (neutrophils, eosinophils and macrophages), but the most important of these are polymorphonuclear leukocytes and macrophages. Normal phagocytosis commences with chemotaxis, which is the primary activation of the metabolic response, via the activation of complement.

The classic pathway of complement activation involves an interaction between the initial antibody and the initial trimer of complement components C₁, C₄ and C₂. In the classic pathway, this interaction then cleaves the complement products C₃ and C₅, via proteolysis, to produce the very powerful chemotactic factors C₃a and C₅a.

The so-called alternative pathway seems to be the main route after trauma. It is activated by properdin, and proteins D or B, to activate C₃ convertase, which generates the anaphylotoxins C₃a and C₅a. Its activation
appears to be the earliest trigger for activating the 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 C3 level is inversely correlated with the Injury Severity Score (ISS). Measurement of C3a is the most useful because the other products are more rapidly cleared from the circulation. The C3a/C3 ratio has been shown to correlate positively with outcome in patients after septic shock. The short-lived fragments of the complement cascade, C3a and C5a, stimulate macrophages to secrete IL-1 and its active circulating cleavage product PIF. These cause proteolysis and lipolysis with fever. Interleukin 1 activates T4-helper cells to produce IL-2, which enhances cell-mediated immunity. Interleukin 1 and PIF are potent mediators stimulating cells of the liver, bone marrow, spleen and lymph nodes to produce acute phase proteins, which include complement, fibrinogen, \( \alpha_2 \)-macroglobulin and other proteins required for defence mechanisms.

Monocytes can produce plasminogen activator, which can adsorb to fibrin to produce plasmin. Thrombin generation is important because of its stimulatory properties on endothelial cells.

Activation of factor XII (Hageman factor A) stimulates kallikrein to produce bradykinin from bradykinogen, which also affects capillary permeability and vasoactivity. A combination of these reactions causes the inflammatory response.

### 2.1.3.3 TOXINS

Endotoxin is a lipopolysaccharide component of bacterial cell walls. Endotoxin causes vascular margination and sequestration of leukocytes, particularly in the capillary bed. At high doses, granulocyte destruction is seen. A major effect of endotoxin, particularly at the level of the hepatocyte, may be to liberate TNF in the macrophages.

Toxins derived from necrotic tissue or bacteria, either directly or via activation of complement system, stimulate platelets, mast cells and basophils to secrete histamine and serotonin.

### 2.1.3.4 FREE RADICALS

Oxygen radical (\( O_2^- \)) formation by white cells is a normal host defence mechanism. Changes after injury may lead to excessive production of oxygen free radicals, released by neutrophils and macrophages, with deleterious effects on organ function. Nitrogen oxide (NO) is also released by macrophages, causing vasodilatation and decreased systemic vascular resistance. NO combines with \( O_2^- \) to form a potent oxidizing agent, which can oxidize the catecholamine ring. Hydroxyl ions (OH-) and hydrogen peroxide are also increased following sepsis or stress.

### 2.1.4 Hormonal mediators

During trauma, several hormones are altered. Epinephrine (adrenaline), norepinephrine (noradrenaline), cortisol and glucagon are increased, while certain others are decreased. The sympathetic–adrenal axis is probably the major system by which the body’s response to injury is activated. Many of the changes are caused by adrenergic and catecholamine effects, and catecholamines are increased after injury.

#### 2.1.4.1 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.

The pituitary gland responds to trauma with two secretory patterns. Adrenocorticotropic hormone (ACTH), prolactin and growth hormone levels increase. The remainder 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, \( \beta \)-endorphin and pro-opiomenocortin (precursor of the ACTH molecule), which modify the response to pain and reinforce the catecholamine effects. The \( \beta \)-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 supraoptic nuclei in the anterior hypothalamus, aldosterone from the adrenal cortex and renin from the juxtaglomerular apparatus of the kidney.
As osmolality increases, the secretion of ADH increases and more water is reabsorbed, thereby decreasing the 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 releasing hormone from the median eminence into capillary blood, which stimulates the anterior pituitary to secrete ACTH. This, in turn, 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.

Plasma levels of growth hormone are increased. However, the effects are transitory and have little long-term effect.

2.1.4.2 ADRENAL HORMONES

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

With passage into the later phases after injury, a number of metabolic effects take place. Glucocorticoids exert catabolic effects such as gluconeogenesis, lipolysis and amino acid breakdown from muscle. Catecholamines also participate in these effects by mediating insulin and glucose release, and the mobilization of fat.

There is an increase in aldosterone secretion, and this results in a conservation of sodium and, thereby, water.

Catecholamines are released in copious quantities after injury, primarily stimulated by pain, fear and baroreceptor stimulation.

2.1.4.3 PANCREATIC HORMONES

There is a rise in the blood sugar after trauma. The insulin response to glucose in normal individuals is reduced substantially with α-adrenergic stimulation, and enhanced with β-adrenergic stimulation.10

2.1.4.4 RENAL HORMONES

Aldosterone secretion is increased by several mechanisms. The rennin–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 decrease and a fall in arterial pressure stimulate release of ACTH via receptors in the right atrium and the carotid artery.

2.1.4.5 OTHER HORMONES

Atrial natriuretic factor (ANF) or atriopeptin is a hormone produced by the atria, predominantly the right atrium of the heart, in response to an increase in vascular volume.11 It produces an increase in glomerular filtration and pronounced natriuresis and diuresis. It also produces inhibition of aldosterone secretion, which minimizes kaliuresis and causes suppression of ADH release.

Before the discovery of ANF, it was suggested that a hormone, a third factor, was secreted after distension of the atria, which complemented the activity of two known regulators of blood pressure and blood volume: the hormone aldosterone and filtration of blood by the kidney. Atrial natriuretic factor has also emphasized the heart’s function as an endocrine organ. It has great therapeutic potential in the treatment of intensive care patients who are undergoing parenteral therapy.

2.1.5 Effects of the various mediators

2.1.5.1 HYPERDYNAMIC STATE

After 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 per m² after severe trauma or infection in those patients who are able to respond adequately. Decreases in vascular resistance
accompany this increased cardiac output. This hyperdynamic state elevates the resting energy expenditure to more than 20 per cent above normal. In an inadequate response, with a cardiac index of less than 2.5 L/min per m², oxygen consumption may fall to values of less than 100 mL/min per m² (normal = 120–160 mL/min per m²). Endotoxins and anoxia may injure cells and limit their ability to utilize oxygen for oxidative phosphorylation.

The amount of 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. As a result of anaerobic glycolysis, only 2 ATP equivalents instead of 34 are produced from 1 mole of glucose in the citrate cycle.

Lactate is formed from pyruvate, which is the endproduct 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-coenzyme A for entry into the citrate cycle is inhibited. Lactate therefore accumulates because of impaired hepatic gluconeogenesis, causing a severe metabolic acidosis.

A persistent lactic acidosis in the first 3 days after injury not only correlates well with the Injury Severity Score (ISS), but also confirms the predictive value of lactic acidosis in subsequent adult respiratory distress syndrome (ARDS).12

Accompanying the above changes is an increase in oxygen delivery to the microcirculation. Total body oxygen consumption \( (\text{VO}_2) \) is increased. These reactions produce heat, which is also a reflection of the hyperdynamic state.

2.1.5.2 WATER AND SALT RETENTION

The oliguria that follows injury is a consequence of the release of ADH and aldosterone.

Secretion of ADH from the supraoptic nuclei in the anterior hypothalamus is stimulated by volume reduction and increased osmolality. The latter is the result mainly of increased sodium content of the extracellular fluid. Volume receptors are located in the atria and pulmonary arteries, and osmoreceptors are located near ADH neurons in the hypothalamus. Antidiuretic hormone 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. In addition, aldosterone 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 h and potassium excretion may rise to 100–200 mmol/24 h.

2.1.5.3 EFFECTS ON SUBSTRATE METABOLISM

Carbohydrates

Critically ill patients develop a glucose intolerance that resembles that found in pregnancy and in patients with diabetes. This is a result of both increased mobilization and decreased uptake of glucose by the tissues.13 The turnover of glucose is increased and the serum glucose is higher than normal.

Glucose is mobilized from stored glycogen in the liver by catecholamines, glucocorticoids and glucagon. Glycogen reserves are limited, and glucose can be derived from glycogen for 12–18 h only. Early on, the insulin blood levels are suppressed (usually lower by 8 units/mL) by the effect of adrenergic activity of shock on degranulation of the \( \beta \) cells of the pancreas. Thereafter, gluconeogenesis is stimulated by corticosteroids and glucagon. The suppressed insulin favours the release of amino acids from muscle, which are then available for gluconeogenesis. Growth hormone inhibits the effect of insulin on glucose metabolism.

Thyroxine (\( \text{T}_4 \)) also accelerates gluconeogenesis, but triiodothyronine (\( \text{T}_3 \)) and \( \text{T}_4 \) levels are usually low or normal in severely injured patients.

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 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 after trauma should be carefully monitored. A hyperglycaemia may exacerbate ventilatory insufficiency, and may provoke an osmotic diuresis and hyperosmolality. The optimum blood glucose level is between 4 and 10 mmol/L. 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.

Parenteral nutrition may be required, and this may exacerbate the problem. However, glucose remains the best energy substrate after major trauma: 60–75 per cent of the caloric requirements should be supplied by glucose, with the remainder being supplied using a fat emulsion.

**Fat**

The principal source of energy after trauma is adipose tissue. Lipids stored as triglycerides in adipose tissue are mobilized when insulin falls below 25 units/mL. As a result of the suppression of insulin release by the catecholamine response after trauma, as much as 200–500 g fat may be broken down daily after severe trauma. Tumour necrosis factor and possibly IL-1 play a role in the mobilization of fat stores.

Catecholamines and glucagon activate adenylyl cyclase in the fat cells to produce cyclic adenosine 3′:5′-monophosphate (cAMP). 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.

Ketones are released into the circulation and are oxidized by all tissue except the blood cells and the central nervous system (CNS). Ketones are water soluble and will pass the blood–brain barrier freely, permitting rapid CNS adaptation to ketone oxidation.

Free fatty acids provide energy for all tissues and for hepatic gluconeogenesis. Carnitine, synthesized in the liver, is required for the transport of fatty acids into the cells.

There is a limit to the ability of traumatized patients to metabolize glucose, and a high glucose load makes management of the patient much more difficult. For this reason, nutritional support of traumatized patients requires a mixture of fat and carbohydrates.

**Amino acids**

The intake of protein by a healthy adult is between 80 and 120 g of protein – 1–2 g protein/kg per day. This is equivalent to 13–20 g nitrogen/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. This process appears to be induced by PIF, which has been shown to increase by as much as eight times in these patients. The process manifests as marked muscle wasting.

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

Certain amino acids such as glutamic acid, asparagine and aspartate can be oxidized to pyruvate, producing alanine, or to 2-oxoglutarate, producing glutamine. The others must first be deaminated before they can be used. In the muscle, deamination is accomplished by transamination from branched-chain amino acids. In the liver, amino acids are deaminated by urea that is excreted in the urine. After severe trauma or sepsis, as much as 20 g/day of urea nitrogen is excreted in the urine. As 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 muscle mass/day. A loss of 40 per cent of body protein is usually fatal, because failing immunocompetence leads to overwhelming infection. Cuthbertson showed that nitrogen excretion and hypermetabolism peaked 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.

To measure the rates of transfer and utilization of amino acids mobilized from muscle or infused into the circulation, the measurement of central plasma clearance rate of amino acids (CPCR-AA) has been developed. Using this method, a large increase in peripheral production and central uptake of amino acids into the liver has been demonstrated in injured patients,
especially if sepsis is also present. The protein-depleted patient can be improved dramatically by parenteral or enteral alimentation, provided that adequate liver function is present. Amino acid infusions in patients who ultimately die cause plasma amino acid concentration to rise to high levels with only a modest increase in CPCR-AA. This may be a result of hepatic dysfunction caused by anoxia or toxins liberated by bacteria responsible for sepsis. Possibly, inhibitors that limit responses to IL-1 and PIF may be another explanation.

The gut

The intestinal mucosa has a rapid synthesis of amino acids. Depletion of amino acids results in atrophy of the mucosa, causing failure of the mucosal antibacterial barrier. This may lead to bacterial translocation from the gut to the portal system and is probably one cause of liver injury, overwhelming infection and multistystem failure after severe trauma. The extent of bacterial translocation in trauma has not been defined. The presence of food in the gut lumen is a major stimulus for mucosal cell growth. Food intake is invariably interrupted after major trauma. The supply of glutamine may be insufficient for mucosal cell growth, and there may be an increase in endotoxin release, bacterial translocation and hypermetabolism. Early nutrition (within 24–48 h), and early enteral rather than parenteral feeding, may prevent or reduce these events.

2.1.6 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 17β-ketosteroids. The utility of growth hormone and also, more recently, of insulin-like growth factor (IGF-I) in reversing catabolism after injury is critically dependent on adequate caloric intake.

2.1.7 Clinical and therapeutic relevance

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

Hypovolaemia caused by both external losses and internal shifts of extracellular fluid seems to be the major initiating trigger for the metabolic sequence. Fear and pain, tissue injury, hypoxia and toxins from invasive infection add to the initiating factor of hypovolaemia. The degree to which the body is able to 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. 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.

Thus, once the metabolic effects of injury have begun, therapeutic or endogenous restitution of blood volume may lessen the severity of the metabolic consequences but cannot prevent them.

Mobilization and storage of the energy fuel substrates, carbohydrate, fats and protein are 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.

Therapy should be aimed at removal of the factors triggering the response. Thorough resuscitation, elimination of pain, surgical debridement and, where necessary, drainage of abscesses and appropriate antibiotic administration, coupled with respiratory and nutritional support to aid defence mechanisms, are of fundamental importance.

2.1.8 References

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2.2 SHOCK

2.2.1 Definition of shock

Shock is defined as inadequate circulation of blood to the tissues, resulting in cellular hypoxia. This at first leads to reversible ischaemically 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 fairly well elucidated.1 By understanding the events leading to cell injury and death, we may be able to intervene therapeutically in shock by protecting sublethally injured cells from irreversible injury and death.2

2.2.2 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 (cardiac tamponade)
4 Inflammatory (previously septic shock)
5 Neurogenic
6 Obstructive (mediastinal compression).

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

Cardiac output = Stroke volume × Heart rate
Blood pressure ∝ Cardiac output × Total peripheral resistance.

Stroke volume is determined by the preload and the contractility of the myocardium, as well as the afterload.

2.2.2.1 HYPOVOLEMIC 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 vascular
resistance and myocardial contractility mediated by neurohumoral mechanisms.

Hypovolaemic shock is divided into four classes, as shown in Table 2.2.

Initially, the body compensates for shock, and class I and class II shock are compensated shock. When the blood volume loss exceeds 30 per cent (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.

Clinical presentation

The classic features of hypovolaemic shock are hypotension, tachycardia, pallor secondary to vasoconstriction, sweating, cyanosis, hyperventilation, confusion and 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 PaO₂ at near normal levels but the PaCO₂ falls to 2.7–4.0 kPa (20–30 mmHg). 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 falls to 50 mmHg. Urine output is a good indicator of peripheral perfusion.

<table>
<thead>
<tr>
<th>Class</th>
<th>Percentage blood loss</th>
<th>Volume (mL)</th>
<th>Pulse rate (/min)</th>
<th>BP</th>
<th>Pulse pressure</th>
<th>RR (/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>&lt; 750</td>
<td>&lt; 100</td>
<td>Normal</td>
<td>Normal</td>
<td>14–20</td>
</tr>
<tr>
<td>II</td>
<td>30</td>
<td>750–1500</td>
<td>&gt; 100</td>
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<td>Increased</td>
<td>20–30</td>
</tr>
<tr>
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<td>40</td>
<td>2000</td>
<td>&gt; 120</td>
<td>Decreased</td>
<td>Narrowed</td>
<td>30–40</td>
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<tr>
<td>IV</td>
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<td>&gt; 2000</td>
<td>&gt; 140</td>
<td>Decreased</td>
<td>Narrowed</td>
<td>&gt; 35</td>
</tr>
</tbody>
</table>

BP, blood pressure; RR, respiratory rate.

2.2.2.2 CARDIOGENIC SHOCK

When the heart fails to produce an adequate cardiac output, even though the end-diastolic volume is normal, then cardiogenic shock is said to be present.

Cardiac function is impaired in such shocked patients even if myocardial damage is not the primary cause. Reduced myocardial function in shock includes dysrhythmias, myocardial ischaemia from systemic hypertension and variations in blood flow, and myocardial lesions from high circulatory levels of catecholamines, angiotensin and possibly a myocardial depressant factor.

The reduced cardiac output can be a result of:

- Reduced stroke volume
- Impaired myocardial contractility as in ischaemia, infarction and cardiomyopathy
- Altered ejection volume
- Mechanical complications of acute myocardial infarction – acute mitral valvular regurgitation and ventricular septal rupture
- Altered rhythms
- Conduction system disturbances (bradydysrhythmias and tachydysrhythmias).

Other forms of cardiogenic shock include those clinical examples in which the patient may have an almost 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 as a result of pharmacological β-adrenergic blockade, e.g. propanolol for hypertension. Heart failure and dysrhythmias are discussed in depth elsewhere in this book.

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 radiographs.

Signs on cardiac examination depend on the underlying cause. A systolic murmur appearing after myocardial infarction suggests mitral regurgitation or septal perforation.

Haemodynamic findings consist of a systolic arterial pressure < 90 mmHg, decreased cardiac output, usually < 1.8 L/m² per min, and a pulmonary arterial wedge pressure (PAWP) > 20 mmHg (2.7 kPa). Sometimes cardiogenic shock occurs without the PAWP being elevated. This may be a result of diuretic therapy or plasma volume depletion by fluid lost into the lungs. Patients with relative hypovolaemia below the levels where there is a risk of pulmonary oedema and, finally, patients with significant right ventricular infarction and right heart 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 diuretics are given.

2.2.2.3 CARDIAC COMPRESSION SHOCK

External forces compress the thin-walled chambers of the heart (the atria and the right ventricle), great veins (systemic or pulmonary), great arteries (systemic or pulmonary) or any combination of these. Impaired diastolic filling occurs. Clinical conditions capable of causing compressive shock include pericardial tamponade, tension pneumothoraces, 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 with no increase in volume, impeding venous return and provoking hypotension.

Clinical presentation

The presence of cardiac tamponade usually follows blunt or penetrating trauma. 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 <90 mmHg, there is a narrowed pulse pressure and a pulsus paradoxus exceeding 10 mmHg. 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 sufficient to cause decompensation.

2.2.2.4 INFLAMMATORY SHOCK

This same dilatation of the capacitance reservoirs in the body occurs with endotoxic 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 where aerobic metabolism is needed.

In the ultimate analysis, 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 very 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 mmHg, yet the cardiac output varies between 3 and 6 L/m² per min. This haemodynamic state is indicative of low peripheral vascular resistance.

In addition to low peripheral resistance as a cause of hypotension in septic shock, there are three other causes of the inability of the cardiovascular system to maintain the cardiac output at a level sufficient to maintain normal blood pressure:

1. Hypovolaemia caused by fluid translocation from the blood into interstitial spaces
2. Elevated pulmonary vascular resistance resulting from ARDS
3. 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. Anaerobic glycolysis and a severe metabolic acidosis caused by lactic acidaemia result. The mechanisms responsible for the phenomena observed in sepsis and endotoxic shock are discussed in detail above.

2.2.2.5 NEUROGENIC SHOCK

Neurogenic shock is a hypotensive syndrome in which there is loss of $\alpha$-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 may be reduced perfusion pressure.

A very simple example of this type of shock is syncope (‘vasovagal 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 will 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 the obligate aerobic glycolytic metabolic bed – the CNS.

Clinical presentation

The patient may have weakly palpable peripheral pulses, warm extremities and brisk capillary filling, and may be alert or anxious. The pulse pressure is wide, with both systolic and diastolic blood pressures being low. Heart rate is below 100 beats/min, and there may even be a bradycardia. The diagnosis of neurogenic shock should be made only once other causes of shock have been ruled out, because the common cause is injury, and there may be other injuries present causing a hypovolaemic shock in parallel.

2.2.2.6 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. As a result 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. The commonest causes is tension pneumothorax. Other causes include pulmonary valvular stenosis, 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.

Clinical presentation

In the patient with hypotension, the problem can usually be identified immediately, from decreased breath sounds, hyperresonance of the affected side and displacement of the trachea to the opposite side. The neck veins may be distended.

2.2.3 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:

\[
\text{Flow} = \frac{\text{Pressure}}{\text{Peripheral resistance}}. 
\]

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 because most drugs that result in a rise in pressure do so by raising the resistance, which in turn decreases flow.

2.2.3.1 CARDIAC OUTPUT

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

1. preload or the volume entering the heart,
2. contractility of the heart,
3. afterload 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 preload, the greater the cardiac output. As
myocardial fibres are stretched by the preload, the contractility increases according to the Frank–Starling principle. However, an excessive increase in preload leads to symptoms of pulmonary/systemic venous congestion without further improvement in cardiac performance. The preload 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 afterload. Afterload 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.

As the emphasis in the definition of shock is on flow, we should be looking for ways to measure flow.

2.2.3.2 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. However, probably the most important clinical observation to determine adequate nutrient flow to a visceral organ indirectly 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 an almost 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.

If the blood pressure falls further and a true decrease in flow across the glomeruli occurs, then the renin–angiotensin mechanism is triggered. Renin from the juxtaglomerular apparatus acts upon angiotensin from the liver. The peptide is cleaved by renin and a decapptide results, which in the presence of converting enzyme clips off two additional amino acids to produce the octapeptide angiotensin II, one of the most potent vasopressors known. The third step is that the same octopeptide stimulates the zona glomerulosa of the adrenal cortex to secrete aldosterone, which causes sodium retention and results in volume expansion. The kidney thus has three methods of protecting its perfusion: autoregulation, pressor secretion and volume expansion. When all three compensatory mechanisms have failed, there is a decrease in the quality and quantity of urine as a function of nutrient flow to this organ. Urine flow is such an important measurement of flow in the patient in shock that we can use this to define the presence or absence of shock. For practical purposes, if the patient is producing a normal quantity of normal quality urine, he or she is not in shock.

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

2.2.3.3 DIRECT MEASUREMENTS

Central venous pressure

Between the groin or axillae and the heart the veins do not have any valves, so measurement of the pressure in this system, at the level of the heart, will reflect the pressure in the right atrium, and therefore the filling pressure of the heart.

Placement of a central venous line that will allow accurate measurement of the hydrostatic pressure of the right atrium after fluid boluses can help differentiate between the different shock states. The actual measurement is less important than the change in value, especially in the acute resuscitation of a patient: normal is 4–12 cmH2O. A value < 4 cmH2O indicates that the venous system is empty, and thus the preload is reduced, usually as a result of dehydration or hypovolaemia, whereas a high value indicates that the preload is increased, as a result of either a full circulation or pump failure (e.g. cardiogenic shock caused by aetiologies such as tension pneumothorax, cardiac tamponade or myocardial contusion).

As a general rule, if a patient in shock has both systemic arterial hypotension and central venous
hypotension, the shock is the result of volume depletion. On the other hand, if central venous pressure is high, though arterial pressure is low, shock is not caused by volume depletion and is more likely to be the result of pump failure.

Cannulation of the central venous system is generally achieved using the subclavian, jugular or femoral route. The subclavian route is the preferred one in the trauma patient, particularly when the status of the cervical spine is unclear. It is ideal for the intensive care setting, where occlusion of the access site against infection is required. The safest technique is that utilized by the Advanced Trauma Life Support (ATLS®) programme.6

The internal jugular route or, occasionally, the external jugular route is the one most commonly used by anaesthetists. 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 intensive care unit (ICU) is, however, more limited, and there is greater discomfort for the patient.

The subclavian route is reliable, easy to maintain and relatively safe. Pitfalls include arterial puncture and pneumothorax.

**Technique of subclavian line insertion**7

1 Place the patient in a supine position, at least 15° head-down to distend the neck veins and prevent an air embolism. Do not move the patient's head.
2 Cleanse the skin and drape the area.
3 Use lidocaine 1 per cent at the injection site to effect local anaesthesia.
4 Introduce a large calibre needle, attached to a 10 mL syringe with 1 mL saline in it, 1 cm below the junction of the middle and medial thirds of the clavicle.
5 After the needle has been introduced, with the bevel of the needle upwards, expel the skin plug that may occlude the needle.
6 The needle and syringe are held parallel to the frontal plane.
7 Direct the needle medially, slightly cephalad and posteriorly, behind the clavicle, towards the posterosuperior angle to the sternal end of the clavicle. (Aim at a finger placed in the suprasternal notch.)
8 Advance the needle while gently withdrawing the plunger of the syringe.
9 When a free flow of blood appears on the syringe, rotate the bevel so that it faces caudally and remove the syringe. Occlude the needle to avoid any chance of air embolism.
10 Introduce the guidewire while monitoring the ECG for abnormalities.
11 Insert the catheter over the guidewire to a predetermined length. The tip of the catheter should be at the entrance to the right atrium. In an adult, this is approximately 18 cm.
12 Connect the catheter to intravenous tubing.
13 Affix securely to the skin and cover with an occlusive dressing.
14 Obtain a chest radiograph to confirm position.

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

**Technique of femoral line insertion**8

1 Place the patient in a supine position.
2 Cleanse the skin.
3 Locate the femoral vein by locating the femoral artery. The vein lies immediately medial to the artery.
4 If the patient is awake, infiltrate the puncture site with lidocaine 1 per cent.
5 Introduce a large-calibre needle, attached to a 10 mL syringe containing 1 mL saline. The needle, directed towards the patient's head, should enter the skin directly over the femoral vein.
6 The needle and syringe are held parallel to the frontal plane.
7 Direct the needle cephalad and posteriorly at 45° to the skin, and slowly advance the needle, while withdrawing the plunger of the syringe.
8 When a free flow of blood appears on the syringe, remove it. Occlude the needle to avoid any chance of air embolism.
9 Insert the catheter over the guidewire to a predetermined length. The tip of the catheter should be at the entrance to the right atrium. In an adult, this is approximately 30 cm.
10 Connect the catheter to intravenous tubing.
11 Affix securely to the skin and cover with an occlusive dressing.

12 Obtain a chest radiograph to confirm position.

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.

Arterial Doppler ultrasonography can be used for measuring arterial blood pressure. Only measurement of the systolic blood pressure is possible. However, the Doppler correlates well with the direct measurement pressure.

The sites for arterial cannulation vary. The radial artery is the most common site. It is usually safe to use, provided that adequate ulnar collateral flow is present. It is important both medically and legally to do an Allen test, compressing both radial and ulnar arteries, and releasing the ulnar artery to check for collateral flow. Thrombosis of the radial artery is quite common, although ischaemia of the hand is rare.

The dorsalis pedis artery is generally quite safe. Cannulation of the brachial artery is not recommended because of the potential for thrombosis and for ischaemia of the lower arm and hand.

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.

Catheterization can be performed easily and rapidly at the bedside, using a balloon-tipped, flow-directed, thermodilution catheter. In its passage from the superior vena cava through the right atrium, from which it migrates into the right ventricle on a myocardial contraction, the balloon tip enters the pulmonic valve exactly like a pulmonary embolus, until the balloon-tipped catheter wedges in the pulmonary artery. Additional side holes are provided in the catheter, allowing measurement of pressure in each right-sided chamber, including right arterial pressure, right ventricular pressure, pulmonary and pulmonary wedge pressure.

The tip of the catheter is placed in the pulmonary artery, and then the occlusive balloon is inflated. This has the effect of occluding the lumen. As a result, the pressure transmitted via the catheter represents pulmonary venous pressure and, thus, left atrial pressure. The wedged pulmonary arterial pressure is a useful approximation of left ventricular end-diastolic pressure (LVEDP), which usually correlates with left ventricular end-diastolic volume (LVEDV).

In addition to direct measurement of pressures, a pulmonary artery catheter allows the following:

- Measurement of cardiac output by thermodilution
- Sampling of pulmonary arterial (mixed venous) blood.

**Technique of insertion of a pulmonary artery catheter using the internal jugular route**

**Equipment**

1. Lidocaine
2. Swan–Ganz catheter set: commercial pack
3. Calibrated pressure transducer with continuous heparin flush and connecting tubing
4. Visible oscilloscope screen showing both ECG and pressure tracings
5. A dedicated assistant (e.g. a nurse).

**Technique**

1. Prepare all supplies at the bedside.
2. Calibrate the transducer for a pressure range 0–50 mmHg.
3. Remove all pillows from behind the patient, and turn the patient’s head to the left.
4. Make sure airway and breathing are acceptable. Patient should be on oxygen, preferably also monitored on pulse oximetry.
5. Tilt the bed head-down to distend the jugular vein.
6. Prepare and drape the skin, allowing access from below the clavicle to the mastoid process.
7. Locate the right carotid pulse, and infiltrate over the area with local anaesthetic at the apex of the triangle between the sternal and clavicular heads of sternomastoid muscle.
8 Insert a 16 gauge needle beneath the anterior border of sternomastoid, aiming towards the right nipple, to place the needle behind the medial end of the clavicle and to enter the right internal jugular vein.

9 Pass the J-wire through the needle and advance the wire until well into the vein.

10 Remove the needle, and enlarge the skin site with a no. 11 scalpel blade, followed with the dilator provided in the set.

11 Attach an intravenous solution to the introducer and suture the introducer to the skin.

12 Connect and flush the catheter to clear all air and to test all balloons, ports, etc. Move the catheter to confirm that the trace is recorded.

13 Insert the catheter into the introducer. If it has a curve, ensure that this is directed anteriorly and to the left. Insert to the 20 cm mark. This should place the tip in the right atrium.

14 Inflate the balloon.

15 Advance the catheter through the right ventricle to the occlusion pressure position. In most adults, this is at the 45–55 cm mark.

16 Deflate the balloon. The pulmonary artery waveform should appear, and with slow inflation the occlusion waveform should return. If this does not occur, advance and then withdraw the catheter slightly.

17 Attach the sheath to the introducer.

18 Apply a sterile dressing.

19 Confirm correct placement with a chest radiograph.

Cardiac output

Cardiac output can be measured with the thermodilution technique. 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 analysed, and the rate of blood flow past the thermistor (i.e. cardiac output) can be calculated. By estimating oxygen saturation in the pulmonary artery, blood oxygen extraction can be determined.

2.2.4 Metabolism in shock

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 $\text{PaO}_2$, $\text{PaCO}_2$, pH and arterial lactate will supply information on oxygen delivery and utilization of energy substrates. Both $\text{PaO}_2$ and $\text{PaCO}_2$ are concentrations – the partial pressure of oxygen and carbon dioxide in arterial blood. If the $\text{PaCO}_2$ is normal, there is adequate alveolar ventilation. Carbon dioxide is one of the most freely diffusible gases in the body and is not over-produced 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 $\text{PaO}_2$ 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 carried this concentration.

For evaluation of oxygen utilization, however, data are obtainable from arterial blood gases which can indicate what the cells are doing metabolically – 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. The lactate and pyruvate concentrations can be measured, but this is more time-consuming. The pH 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 – CO₂ and water – both of which leave the body by excretion through the lung and the kidney. Aerobic metabolism is efficient, so 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 min. 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 ions, 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 paid off.

Acidosis has significant consequences in compensatory physiology. In the first instance, oxyhaemoglobin dissociates more readily as the concentration of hydrogen ions increases. 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 are, however, physiologically effective at alkaline or neutral pH, so an acid pH inactivates this catecholamine method of compensation for decreased nutrient flow, e.g. if a catecholamine such as isoproterenol is administered to a patient in shock, it would increase myocardial contractility and heart rate and also dilate the periphery to increase nutrient flow to these ischaemic circulation areas. However, the ischaemic areas have shifted to anaerobic metabolism, accumulating hydrogen ion, lactate and pyruvate. When the circulation dilates, this sequestered oxygen debt is dumped into the central circulation and the drop in pH inactivates the catecholamines circulatory improvement as effectively as if the infusion of the agent had been interrupted.

2.2.5 Post-shock sequence and multiple organ failure syndromes

Although the consequences of sepsis after trauma and shock, the metabolic response to trauma and multiple organ failure are discussed in detail elsewhere in this book, it is important briefly to reiterate the usual sequence of events after shock to enable logical discussion of its management.

The ultimate cause of death in shock is failure of energy production as reflected by a decline in oxygen consumption (VO₂) to < 100 mL/m² per min. Circulatory insufficiency is responsible for this energy, compounded by impairment of cellular oxidative phosphorylation by endotoxin and endogenously produced substances – superoxides.

In shock, whether hypovolaemic or septic, energy production is insufficient to satisfy requirements. In the presence of oxygen deprivation and cellular injury, the conversion of pyruvate to acetyl-CoA for entry into the citrate 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 and septic shock because of hepatocyte injury and inadequate circulation. The lactic acidemia cannot 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 β oxidation of fatty acids in the liver is reduced, suppressing the acetoacetate:β-hydroxybuturate ratio in the plasma.

The post-shock sequel of inadequate nutrient flow is, therefore, progressive loss of function. The rate at which this loss occurs depends upon the cell's ability to switch metabolism, to convert alternative 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 or 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.

2.2.6 Management of the shocked patient

The primary goal of shock resuscitation is the early establishment of adequate oxygen delivery (DO₂). The
calculated variable of \( \text{DO}_2 \) is the product of cardiac output, and arterial oxygen content (\( \text{CaO}_2 \)).

By convention, cardiac output (\( \text{CO} \)) is indexed to body surface area and expressed as a cardiac index (\( \text{CI} \)); when multiplied by \( \text{CaO}_2 \), it yields an oxygen delivery index (\( \text{DO}_2I \)). Normal \( \text{DO}_2I \) is roughly 450 mL/min per m\(^2\).

\( \text{CaO}_2 \) and \( \text{DO}_2I \) are calculated as follows:

\[
\text{CaO}_2 \text{ (mL O}_2/\text{dL)} = [\text{Hb}] \text{(g/dL)} \times 1.38 \text{ mL O}_2/\text{g Hb}
\times \text{SaO}_2(\%) + [\text{PaO}_2 \text{ (mmHg)}] \times 0.003 \text{ mL O}_2/\text{mmHg} \\
\text{DO}_2I \text{ (mL/min per m}^2\text{)} = \text{CI (L/min per m}^2\text{)} \times \text{CaO}_2 \text{ (mL/dL)} \times 10 \text{ dL/L}
\]

where \( \text{Hb} \) = haemoglobin concentration, \( \text{SaO}_2 \) = haemoglobin oxygen saturation, \( \text{PaO}_2 \) = arterial oxygen tension and 0.003 = solubility of \( \text{O}_2 \) in blood.

Early work demonstrated that the ‘survivor’ response to traumatic stress is to become hyperdynamic. Supranormal resuscitation based on the \( \text{DO}_2I \) was therefore proposed. Subsequent randomized controlled trials have failed to demonstrate improved outcomes with goal-directed supranormal therapy, and this strategy may even be harmful. The ‘Glue Grant’ study for shock resuscitation\(^{16} \) suggests using a \( \text{CI} > 3.8 \text{ L/min per m}^2 \) as the resuscitation goal.

The purpose of distinguishing the different pathophysiological mechanisms of shock becomes important when treatment has to 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 and adequate cardiac output) and restoration of aerobic cellular metabolism. These aims can be achieved by securing a patent airway and controlling ventilation if alveolar ventilation is inadequate.

Restoration of optimal circulating blood volume, enhancing cardiac output through the use of inotropic agents or increasing systemic vascular resistance through the use of 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.

Best practice guidelines for shock resuscitation are summarized in a large-scale collaborative project to provide standard operating procedure for clinical care—the so-called ‘Glue Grant’.\(^{16} \)

### 2.2.6.1 OXYGENATION\(^{17} \)

The traumatized, hypovolaemic or septic patient has an oxygen demand that often exceeds twice normal. The traumatized shocked patient usually cannot exert the additional respiratory effort required, and therefore often develops respiratory failure followed by a lactic acidosis as a result of tissue hypoxaemia.

In some patients, an oxygen mask may be enough to maintain efficient 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.

**Airway indications for intubation**
- Obstructed airway
- Inadequate gag reflex.

**Breathing indications for intubation**
- Inability to breathe (e.g. paralysis, either spinal or drug induced)
- Tidal volume < 5 mL/kg.

**Breathing indications for ventilation**
- Inability to oxygenate adequately
- \( \text{PaO}_2 < 8 \text{ kPa (60 mmHg)} \) on 40 per cent \( \text{O}_2 \)
- \( \text{PaO}_2 < 90 \) per cent on oxygen
- A respiration rate of \( \geq 30 \) breaths/min
- Excessive ventilatory effort
- A \( \text{PCO}_2 > 6 \text{ kPa (45 mmHg)} \) with metabolic acidosis or \( > 6.7 \text{ kPa (50 mmHg)} \) with normal bicarbonate levels.

**Circulation indication for intubation**
- Systolic blood pressure < 75 mmHg despite resuscitation.

**Disability indications for intubation**
- Spinal injury with inability to breathe
- Coma (Glasgow Coma Scale [GCS] < 8/15).

**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 PaCO₂ of between 4.7 and 5.3 kPa (35 and 40 mmHg). 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 CO to maintain tissue oxygenation.

The PaO₂ should be maintained between 10.6 and 13.3 kPa (80 and 100 mmHg) with the lowest possible oxygen concentration.

It has been shown that respiratory muscles require a disproportionate share of the total CO and, therefore, other organs are deprived of necessary blood flow and lactic acidosis is potentiated. Mechanical ventilation tends to reverse this lactic acidosis.

**2.2.6.2 FLUID THERAPY FOR VOLUME EXPANSION**

There is considerable controversy with regard to the type of fluid to be administered for volume expansion in hypovolaemic shock. Despite many studies, minimal convincing evidence exists that favours any specific fluid regimen. Balanced salt solutions (BSSs) are effective volume expanders for the initial resuscitation of patients with shock. For most patients, Ringers’ lactate solution is the preferred crystalloid solution. The lactate acts as a buffer and is eventually metabolized to CO₂ and water. However, septic patients with significant hepatic dysfunction do not metabolize lactate well and, for these patients, other BSSs are preferred.

In hypovolaemic shock, a volume of solution in excess of measured losses is generally required. In principle, three times the volume of BSS is given per unit of blood lost. A bolus dose of 2000 mL BSS (e.g. Ringers’ lactate) in adults is given and the response of pulse rate, blood pressure and urinary output monitored. If this fails to correct haemodynamic abnormalities, additional crystalloid solution and blood are indicated, because crystalloids in large quantities will ultimately cause a dilutional effect that can decrease the blood’s oxygen-carrying capacity. It is true that the restored vascular volume will increase the CO and thus maintain tissue oxygenation. This increased CO can be sustained by the normal heart but, in the diseased heart or the elderly patient, it is safer to give blood earlier to obviate the possibility of cardiac failure. In many countries, packed red blood cells with crystalloid solutions instead of whole blood are given because the blood banking industry in those countries has changed to component therapy to the extent that whole blood replacement is not readily available for large volume transfusion.

**Crystalloids or colloids?**

Crystalloids are cheaper, with fewer side effects. Colloids are more expensive, and have more side effects. However, their rate of excretion is much slower than that of crystalloids, so that the volume remains in the circulation for longer. Balanced salt solutions are said to have a half-life in the circulation of 20 min, whereas Gelofusine® has a half-life of 4–6 h. However, additional considerations relate to rate of infusion, and the problem with most cases of hypovolaemic shock is that inadequate volumes of resuscitation fluid are infused in the time available. Thus, there are advantages in using a fluid that does not leave the circulation as fast. However, a recent Cochrane review of the available trial data comparing crystalloids and colloids for resuscitation after trauma showed no improvement in survival with colloids, and therefore their use cannot be supported at present. Lactated Ringers’ solution is the currently preferred crystalloid. As yet, no advantages have been shown to the use of newer formulations utilizing pyruvate or acetate. Physiological or 0.9 per cent saline results in an increase of hyperchloraemic metabolic alkalosis.

**Resuscitation versus no resuscitation**

In 1994, Bickell et al. concluded that injured patients in hypovolaemic shock who were not given intravenous fluids during transport and emergency department evaluation had a better chance of survival than those who received conventional treatment. 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 clot. This was possibly because the reduced blood pressure reduced the amount of bleeding that took place. The optimum systolic blood pressure for a patient with uncontrolled haemorrhage would appear to be between 90 and 100 mmHg.

**Hypertonic saline**

Hypertonic saline solutions containing up to 7.5 per cent sodium chloride (compared with 0.9 per cent for physiological saline) show promise for resuscitating patients in
situations where large-volume resuscitation with isotonic solutions is impossible (e.g. combat, events involving mass casualties and pre-hospital trauma care). Hypertonic solutions provide far more blood volume expansion than isotonic solutions and result in less cellular oedema. Several randomized controlled trials have evaluated the use of hypertonic saline in the resuscitation of hypovolaemia. In all the trials, patients resuscitated with saline survived longer than those resuscitated in the conventional fashion. Also, the patients did best when the hypertonic saline was given as the initial therapy and the patients most likely to benefit were those with head injuries. Hypertonic saline may be more effective when mixed with a small amount of an oncotically active molecule such as dextran. The combination of hypertonic saline and a colloid is most likely to be beneficial when definitive surgery may be delayed.

Blood substitutes
Blood substitutes, including haemoglobin-based preparations and perfluorocarbons, have several potential advantages. No crossmatching is necessary, disease transmission is not an issue and shelf-life is extended. Several haemoglobin substitutes are being evaluated, but at present remain experimental and are not generally approved for human use in trauma.

2.2.6.3 ROUTE OF ADMINISTRATION

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 (Table 2.3) are reduced:

- 14 gauge via peripheral cannula: full flow
- 14 gauge via 30 cm central line: 33 per cent reduction in flow
- 14 gauge via 70 cm central line: 50 per cent reduction in flow.

A minimum of two lines is required. 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 via the subclavian, jugular or femoral route. In blunt polytrauma, the subclavian route is preferable, because this avoids any movement of the head in a patient whose neck has not yet been cleared.

<table>
<thead>
<tr>
<th>Cannula size (gauge)</th>
<th>Crystalloid</th>
<th>Colloid</th>
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</thead>
<tbody>
<tr>
<td>8.5 French</td>
<td>1000</td>
<td>600</td>
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<tr>
<td>14</td>
<td>125</td>
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<td>20</td>
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<td>17</td>
</tr>
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</table>

2.2.6.4 Pharmacological support of blood pressure

Stroke volume is controlled by ventricular preload, afterload and contractility. Preload is mainly influenced by the volume of circulating blood, but afterload and contractility can be manipulated by pharmacological agents. Reducing the systemic vascular resistance with vasodilators can be a very effective means of improving CO when systemic pressures or cardiac filling pressures are normal or elevated.

Use of vasoactive agents should be restricted to ‘non-responders’.

Epinephrine (adrenaline)
Epinephrine is a natural catecholamine with both α- and β-adrenergic agonist activity. The pharmacological actions are complex and 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 epinephrine 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 per min, titrated upwards until the desired effect is achieved. In trauma patients, it is often used in conjunction with dobutamine.

Dopamine
Dopamine hydrochloride is a chemical precursor of norepinephrine (noradrenaline), which stimulates dopaminergic, β₁-adrenergic and α₁-adrenergic receptors.
in a dose-dependent fashion. Low doses of dopamine (< 3 μg/kg per 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 per min, the α-adrenergic effects predominate. This results in marked increases in systemic vascular resistance, pulmonary resistance and increases in preload as a result of 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.

**Dobutamine**

Dobutamine is a synthetic sympathomimetic amine that has potent inotropic effects by stimulating β₁- and α₁-adrenergic receptors in the myocardium. There is only a mild vasodilatory response. Dobutamine-mediated increases in cardiac output also lead to a decrease in peripheral vascular resistance. At a dose of 10 μg/kg per min, dobutamine is less likely to induce tachycardia than either epinephrine or isoproterenol. Higher doses may produce a tachycardia. 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 epinephrine.

Dobutamine increases cardiac output and its lack of induction of norepinephrine release means that there is a minimal effect on myocardial oxygen demand. There is also increased coronary blood flow.

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

**Isoproterenol**

Isoproterenol hydrochloride is a synthetic sympathomimetic amine with a particularly strong chronotropic effect. Newer inotropic drugs such as dobutamine have largely superseded isoproterenol in most settings.

**Nitroprusside**

Sodium nitroprusside is a potent peripheral vasodilator with effects on both venous and arterial smooth muscle, and has balanced vasodilating effects on both circulations, thus minimizing adverse effects on arterial blood pressure. Its effects are seen almost immediately, and cease when it is stopped.

**Digoxin**

Digoxin enhances cardiac contractility but its use in shock is limited because it takes considerable time to act. In the ICU situation, digoxin is usually reserved for the treatment of atrial flutter and supraventricular tachycardias.

### 2.2.5 Metabolic Manipulations

The endogenous opiate β-endorphin appears to be involved in the hypotension and impaired tissue perfusion that occur in both hypovolaemic and septic shock states, because elevations in this substance can be demonstrated at the time that these physiological changes take place.

Naloxone, an opiate antagonist, has been shown to elevate blood pressure and CO, and to improve survival significantly in septic and haemorrhagic shock models. Early results in shock patients have supported these findings. Prostaglandins have also been implicated in shock. They may play a role in the pathophysiology of shock by vasodilatation or vasoconstriction of the microcirculation with shunting of blood. Experimental evidence exists that cyclo-oxygenase inhibitors such as indometacin and ibuprofen can improve the haemodynamic state in experimental shock.

### 2.2.7 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 physiological disturbances.

These correctable causes include:

- Under-appreciated volume need 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 such as adrenal insufficiency or hypothyroidism
• Drug toxicity.

2.2.8 Recommended protocol for shock

2.2.8.1 Initial resuscitation

A Major trauma patients arriving in shock (systolic blood pressure [SBP] < 90 mmHg and/or heart rate [HR] > 130 beats/min) are managed using ATLS® protocols.

B Major torso trauma patients requiring ongoing resuscitation should have a central venous line placed in the emergency department.

C Early central venous pressure (CVP) > 15 mmHg (before extensive volume loading) suggests cardiogenic shock.

D CVP < 10 mmHg despite volume loading suggests ongoing bleeding. Endpoints are currently vague. At present the rational compromise is hypotensive resuscitation (SBP > 90 mmHg and HR < 130 beats/min) with moderate volume loading until haemorrhage is controlled.

E Boluses of Ringers' lactate should be continued and, when the amount exceeds 30 mL/kg, blood should be administered.

F Protocols for massive transfusion should be established (see Section 9.5.2).

2.2.8.2 Resuscitation in the ICU

A On arrival, a decision is made on continuing resuscitation using serial vital signs.

B For patients not responding to ongoing volume loading or transfusion, pulmonary artery catheterization is warranted.

C Intubation should be considered if not already present.

D If CI > 3.8 L/min per m² the patient should be monitored appropriately.

E Haemoglobin should be maintained at between 8 and 10 g/dL.

F Pulmonary capillary wedge pressure (PCWP) > 15 mmHg may enhance cardiac performance.

G After obtaining an optimal PCWP, if CI < 3.8 L/min per m² then infusion of a vasodilating inotropic agent should be considered. Dobutamine is recommended as the preferred agent, commencing at a dose of 5 μg/kg per min. If the patient does not tolerate the vasodilatation, an agent such as dopamine should be considered.

H Occasionally an inotropic agent with vasoconstrictive effects such as norepinephrine or epinephrine) may be required.

2.2.9 References


2.3 BLOOD TRANSFUSION IN TRAUMA

Transfusion of blood and blood components is a fundamental part of our treatment of injured patients. Approximately 40 per cent of 11 million units of blood transfused in the USA each year are used in emergency resuscitation. There is little level I evidence to provide a rationale for administration of packed red blood cells (PRBCs) to trauma patients.

2.3.1 Indications for transfusion

2.3.1.1 OXYGEN-CARRYING CAPACITY

Anaemia is a decrease in the O₂-carrying capacity of blood, and is defined by a decrease in circulating red cell mass (to < 24 mL/kg in females and 26 mL/kg in males). Anaemia will result in an increase in cardiac output at Hb 4.5–7 g/dL. Oxygen extraction (ER\(\text{O}_2\)) increases as \(\text{O}_2\) delivery (\(\text{DO}_2\)) falls, ensuring a constant \(\text{O}_2\) uptake (\(\text{VO}_2\)) by tissues. Threshold for \(\text{O}_2\) delivery is at a haematocrit of 10 per cent and Hb of 3 g/dL, breathing 100 per cent O₂ and normal metabolic rate.

2.3.1.2 VOLUME EXPANSION

Normal humans can survive 80 per cent loss of red cell mass if normovolaemic. Volume-dependent markers such as packed cell volume (PCV) and Hb are poor indicators of anaemia because of the effect of dilution on their values, i.e. they are relative values.

2.3.1.3 COMPONENT THERAPY (PLATELETS, FRESH FROZEN PLASMA OR FFP)

Platelet transfusion
- Prophylaxis: platelet count < 15 000 mm\(^{-3}\)
- Pre-surgery: platelet count < 50 000 mm\(^{-3}\)
- Active bleeding: platelet count < 100 000 mm\(^{-3}\)
• 1 unit increases count by 5000 platelets
• Resistant if original cause not controlled.

Fresh frozen plasma
• Contains all coagulation factors
• Preferred to cryoprecipitate which contains 50 per cent of coagulation factors (haemophilia – fibrinogen, factor VIII – von Willebrand’s factor).

2.3.2 Effects of transfusing blood and blood products

2.3.2.1 STORED BLOOD DEVELOPS STORAGE DEFECTS THAT ASSUME GREATER CLINICAL SIGNIFICANCE WHEN TRANSFUSED RAPIDLY, OR IN LARGE QUANTITIES

- ↓ ATP
- ↑ 2,3-Diphosphoglycerate (DPG) degradation
- ↓ affinity for oxygen
- Membrane instability
- ↑ potassium (K+) release
- ↑ ammonia release
- Microaggregates (platelet/leukocyte/fibrin thrombi) in buffy coat.

2.3.2.2 EFFECTS OF MICROAGGREGATES

- Impaired pulmonary gas exchange, ARDS
- Reticuloendothelial system (RES) depression
- Activation of complement, coagulation cascades
- Vasoactive substances
- Antigenic stimulation
- Acute phase response.

2.3.2.3 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 40–70 mmol/L. Transient hyperkalaemia may occur as a result. Serum potassium levels rise in stored blood as the efficiency of the Na⁺/K⁺ pump decreases. Transfused blood may have a potassium concentration of 40–70 mmol/L. Transient hyperkalaemia may occur as a result.

2.3.2.4 COAGULATION ABNORMALITIES

- Thrombocytopenia and loss of factors V and VIII in stored blood may contribute to problems with coagulation.

- Levels of clotting factors V and VIII decline quickly for 24 h after collection. The rate of decline slows until clinically subnormal levels are reached at 7–14 days. It is because fresh whole blood will contain these factors that it is recommended for massive transfusion. The other clotting factors remain stable in stored blood.
- Packed red cells do not contain platelets, because 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 > 30 000 mm⁻³. 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 seems to have large reserves of platelets.
- 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.

2.3.2.5 COAGULATION STUDIES

- Ideally, the use of blood components should be guided by laboratory tests of clotting function. This may be appropriate where surgical bleeding is controlled and the operating field appears dry. However, in the face of continued oozing, when obvious surgical bleeding has been controlled, blood products may need to be given empirically.

2.3.3 Other risks of transfusion

2.3.3.1 TRANSFUSION-TRANSMITTED INFECTIONS

- Hepatitis A, B, C and D
- HIV ‘window period’
- Cytomegalovirus (CMV)
- Atypical mononucleosis and swinging temperature 7–10 days post-transfusion
- Malaria
- Brucellosis
- Yersinia infection
- Syphilis.

2.3.3.2 HAEMOLYTIC TRANSFUSION REACTIONS

- Incompatibility: ABO, rhesus (type) and 26 others (screen)
• Frozen blood, overheated blood, pressurized blood
• Immediate generalized reaction (plasma).

2.3.3.3 IMMUNOLOGICAL COMPLICATIONS
• Major incompatibility reaction (usually caused by ‘wrong blood’ as a result of administrative errors).

2.3.3.4 POST-TRANSFUSION PURPURA

2.3.3.5 GRAFT-VERSUS-HOST DISEASE
• Transfusion-related acute lung injury.

2.3.3.6 IMMUNOMODULATION
• Reports on transplant recipients 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.

2.3.3.7 HAEMOSTATIC FAILURE
• Relates to degree of tissue injury (tissue thromboplastins), hypothermia and acidosis, and less to amount of blood (dilution)
• Dilution, depletion and decreased production
• Hypothermia (1 unit 4°C →37°C) =1255 kJ
• Acidosis (citrate, lactate)
• Diffuse intravascular coagulation: consumption of clotting factors and platelets within the circulation, causing microvascular obstruction as a result of fibrin deposition via two pathways of coagulation
• Extrinsic: tissue thromboplastins, e.g. blunt trauma, surgery
• Intrinsic: endothelial injury, endotoxin, burns, hypothermia, hypoxia, acidosis and platelet activation.

2.3.4 What to do?

2.3.4.1 REDUCE 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 the complications:
• Treat the cause, i.e. urgent surgery to stop bleeding, avoid hypothermia and acidosis.
• Treat deficiencies and complications as they arise; there is no evidence to support prophylactic therapy with FFP, platelets, etc., but patients with very major injuries may require empirical transfusion
• Follow a restrictive transfusion policy in the ICU. A recent multicentre trial documented a significantly lower mortality rate for critically ill patients managed with a restrictive transfusion strategy. However, this assumes normovolaemia, absence of ongoing bleeding and absence of pre-existing cardiovascular disease.
• Develop a capacity for cell salvage.

2.3.4.2 INVESTIGATE AND MONITOR THE COAGULATION STATUS
• Fibrinogen degradation products (FDPs)
• International normalized ratio (INR) – extrinsic
• Partial thromboplastin time (PTT) – intrinsic
• D-dimer (fibrin deposition).

2.3.4.3 TRANSFUSION THRESHOLDS
• Packed cells for O₂-carrying capacity.

There is no level 1 evidence indicating the ideal trigger for transfusion in trauma patients. In general, the following guidelines apply:
1. Identify the critically ill patient with a haemoglobin < 7 g/dL (or haematocrit < 21 per cent)
2. If Hb < 7 g/dL, transfusion with PRBCs is appropriate for patients with severe cardiovascular disease, and trauma patients with ongoing bleeding or haemodynamic instability, a higher threshold (< 8–10 g/dL is appropriate
3. If Hb > 7 g/dL, assess the patient for hypovolaemia; if so, administer intravenous fluids to achieve normovolaemia and reassess haemoglobin
4. If the patient is not hypovolaemic, determine whether there is evidence of impaired oxygen delivery
5. If impaired oxygen delivery is present, consider pulmonary artery catheter placement
6. If impaired oxygen delivery is not present, monitor haemoglobin as appropriate.

2.3.4.4 COAGULOPATHY

Thrombocytopenia and loss of factors V and VIII in stored blood may contribute to problems with
coagulation. The other clotting factors remain stable in stored blood.

Packed red cells do not contain platelets, because 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 > 30,000 mm\(^{-3}\). Levels as low as this are seen after the replacement of one or two times the body blood volume and may result from dilution. Despite this, the body seems to have large reserves of platelets. Platelet counts need to be done after the replacement of every 20 units of blood, and replacement should be considered if the platelet level is < 50,000 mm\(^{-3}\).

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 platelets carries a greater risk of infection, because several donors have contributed to a single pack of platelets.

Levels of clotting factors V and VIII decline quickly for 24 h after collection. The rate of decline slows until clinically subnormal levels are reached at 7–14 days. It is because fresh whole blood will contain these factors that it is recommended for massive transfusion.

Increased levels of factor VIII can be produced by the liver under conditions of stress if it remains well perfused. Hypotension and hypothermia will reduce the ability to do this.

Coagulation studies

Ideally, the use of blood components should be guided by laboratory tests of clotting function. This may be appropriate where surgical bleeding is controlled and the operating field appears dry. However, in the face of continued oozing, when obvious surgical bleeding has been controlled, blood products may need to be given empirically.

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 40–70 mmol/L. Transient hyperkalaemia may occur as a result. The presence of acidosis and hypercapnia will tend to replace the K\(^+\) in the cell.

2.3.4.5 ARTIFICIAL CLOTTING FACTORS

There has been extensive interest in the provision of artificial clotting factors as part of the resuscitation of the trauma patient. Interest has focused on recombinant activated factor VIIa (NovoSeven). This was initially developed as an adjunct for the treatment of haemophilia. However, after its successful use in controlling the bleeding in a trauma patient, there has been considerable interest in its use. A large multicentre trial\(^1\) showed reduction in red cell transfusion requirement in blunt trauma patients, and the drug has been used extensively ‘off-label’. It is currently the subject of a very large multicentre trial in severely injured patients with ongoing, non-surgically correctable bleeding from torso trauma.

2.3.5 Massive transfusion

2.3.5.1 DEFINITION

Massive transfusion is defined as:

- replacement of 100 per cent of the patient’s blood volume in less than 24 h
- the administration of 50 per cent of the patient’s blood volume in 1 h.

There is a danger of death when blood loss is > 150 mL/min or 50 per cent of blood volume in 20 min. Each trauma unit should have a massive transfusion policy, which should be activated as soon as a potential candidate is admitted. A full, suitable, proposed massive transfusion protocol appears in Section 9.5.2.

2.3.6 Autotransfusion

Autotransfusion eliminates the risk of incompatibility and the need for crossmatch; the risk of transmission of disease from the donor is also eliminated. Autotransfusion is a safe and cost-effective method of sustaining red blood cell mass while decreasing demands on the blood bank. However, cell salvage of trauma patients is fraught with difficulty. In trauma, autotransfusion typically involves collection of blood shed into wounds and body cavities, and drains.

Autotransfusion is generally contraindicated in the presence of bacterial or malignant cell contamination (e.g. open bowel, infected vascular prostheses) unless no other red blood cell source is available and the patient is in a life-threatening situation.

Modern autotransfusion devices are basically of two types:
Blood is collected, mixed with an anticoagulant, typically citrate, and returned. The blood is collected, anticoagulated with heparin, and then run through a system in which it is washed and centrifuged, before being re-transfused.

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 red blood cells may require trained technicians, particularly if the procedure is used infrequently.

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 salvaged blood is returned to the patient and the most recent devices do not require mixing of the blood with an anticoagulant solution. Inline filters are absolutely essential when autotransfusion devices are used. These filters remove gross particles and macroaggregates during collection and reinfusion, thus minimizing microembolization.

Cell washing and centrifugation techniques require a machine and (usually) a technician to be the sole operator. This 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 per cent. 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.

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

### 2.3.7 Transfusion: red blood cell substitutes

The ideal blood substitute is cheap, has a long shelf-life, is universally compatible, well tolerated and has an O\textsubscript{2} delivery profile identical to blood. Significant effort has been made to find a suitable substitute, which, essentially, should be treated as an artificial O\textsubscript{2} carrier.

Artificial O\textsubscript{2} carriers can be grouped into perfluoro-carbon emulsions (PFCs) and modified Hb solutions. The native Hb molecule needs to be modified in order to decrease O\textsubscript{2} affinity and to prevent rapid dissociation of the native $\alpha_2\beta_2$ tetramer into $\alpha_2\beta_2$ dimers.

#### 2.3.7.1 PERFLUOROCARBONS

Perfluorocarbons are carbon–fluorine compounds that are completely inert and with 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 haemoglobin, they exhibit a linear relationship with oxygen, so their efficacy relies on maintaining a high $P_aO_2$; however, PFCs unload oxygen well. They do not expand the intravascular volume and can only be given in small volumes because they overload the reticuloendothelial system. Once thought to hold potential, to date they have not been found to confer additional benefit compared with crystalloid solutions, especially as there is a significant incidence of side effects.

#### 2.3.7.2 HAEMOGLOBIN SOLUTIONS

**Liposomal haemoglobin solutions**

This is based on the encapsulation of haemoglobin 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\textsubscript{2} dissociation curves similar to red cells, with low viscosity, and their administration can transiently produce high circulating levels of Hb.

Problems associated with Hb-based O\textsubscript{2} carriers relate to effects on vasomotor tone, which appears to be modulated by the carriers' interaction with NO, causing significant vasoconstriction.

**Stroma-free haemoglobin (human-outdated/bovine red blood cells)**

These are known as Hb-based oxygen carriers (HBOCs). Although free Hb can transport O\textsubscript{2} outside its cell membrane, it is too toxic to be clinically useful. Techniques have been developed for removing the red cell membrane products, and cross-linking the haemoglobin, initially with a di-aspirin link and recently as an Hb polymer. Both human and bovine haemoglobins have been used.

Considerable research is currently taking place with regard to the development of artificial Hb. After some initially discouraging results, current efforts with a
bovine-derived Hb (Hemopure) and human Hb (Polyheme) have produced some dramatic improvement. Currently the products are not licensed for human use in the trauma patient.

No level 1 evidence has yet appeared to support the use of Hb substitutes rather than blood. The O₂ transport characteristics of modified Hb solutions and PFC solutions are fundamentally different. The Hb solutions exhibit a sigmoid O₂ dissociation curve similar to that of blood, whereas PFC emulsions are characterized by a linear relationship between O₂ partial pressure and O₂ content. Hb solutions therefore provide O₂ transport and unloading characteristics similar to blood. This means that, at a relatively low PaO₂, substantial amounts of O₂ are being transported. In contrast, relatively high PaO₂ values are necessary to maximize the O₂ transport of PFC emulsions.

Note that 5 per cent O₂ can be offloaded by both blood and PFCs. Perfluorocarbon O₂ is more completely offloaded than blood-transported O₂.

### 2.3.8 References


### 2.3.9 Recommended reading


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### Haemoglobin-based solutions

**Advantages**
- Carries and unloads O₂
- Sigmoidal O₂ dissociation curve
- 100 per cent FiO₂ not mandatory for maximum potency
- Easy to measure

**Disadvantages**
- Side effects:
  - vasoconstriction
  - interference with laboratory methods (colorimetric)

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### PFC-based emulsions

**Advantages**
- Carries and unloads O₂
- Few and mild side effects
- No known organ toxicity

**Disadvantages**
- 100 per cent FiO₂ is mandatory for maximal efficacy
- Additional colloid often necessary with potential side effects
2.4 HYPOTHERMIA

Hypothermia is almost always a complication of trauma and, thus, after major trauma there is rapid heat loss. Although hypothermia may itself cause cardiac arrest, it is also protective to the brain through a reduction in metabolic rate, with marked depression of cerebral blood flow and oxygen requirements. Oxygen consumption is reduced by 50 per cent at a core temperature of 30°C. Victims may appear to be clinically dead because of marked depression of brain and cardiovascular function, and the potential for resuscitation with full neurological recovery is theoretically possible. The American Heart Association Guidelines recommend that the hypothermic patient who appears dead should not be considered so until a near normal body temperature is reached. However, hypothermia on balance is extremely harmful to trauma patients, especially by virtue of the way it alters oxygen delivery. Therefore, the patient must be warmed as soon as possible, and heat loss minimized at all costs.

2.4.1 Rewarming

Hypothermia is common after immersion injury. Rewarming must take place with intensive monitoring. Patients who have spontaneous respiratory effort and whose hearts are beating, no matter how severe the bradycardia, should not receive unnecessary resuscitation procedures. The hypothermic heart is very irritable and fibrillates easily. Patients with a core temperature of < 29.5°C are at high risk for ventricular arrhythmias, and should be rewarmed as rapidly as possible. Recent studies have not shown any increase in ventricular arrhythmias with rapid rewarming.

A hypothermic heart is resistant to both electrical and pharmacological cardioversion, especially if the core temperature is < 29.5°C and cardiopulmonary resuscitation (CPR) should be continued if necessary.

If the core temperature is > 29.5°C and fibrillation is present, one attempt at electrical cardioversion should be made. If this is ineffective, intravenous bretylium may be helpful.

Patients with a core temperature of between 29.5 and 32°C generally can be passively rewarmed, and if haemodynamically stable may be rewarmed more slowly. However, active core rewarming is still generally required.

Patients with a core temperature of > 32°C can generally be rewarmed using external rewarming.

Methods of rewarming include the following.

External

- Remove wet or cold clothing and dry the patient
- Infrared (radiant) heat
- Electrical heating blankets
- Warm air heating blankets.

Note that, in the presence of hypothermia, ‘space blankets’ are ineffective, because there is minimal intrinsic body heat to reflect.

Internal

- Heated, humidified respiratory gases to 42°C
- Warmed intravenous fluids to 37°C
- Gastric lavage with warmed fluids (usually saline at 42°C)
- Continuous bladder lavage with water at 42°C.
- Peritoneal lavage with potassium-free dialysate at 42°C (20 mL /kg every 15 min)
- Intrapleural lavage
- Extracorporeal rewarming.
It is recommended that resuscitation should not be abandoned while the core temperature is subnormal, because it may be difficult to distinguish between cerebroprotective hypothermia and hypothermia resulting from brain-stem death.

2.5 END POINTS OF RESUSCITATION

The first end point is control of the cause

Traditional end points of resuscitation have been limited to pulse rate, blood pressure, Hb, and urea and electrolyte levels, but these were truly limited in their scope of interpretation. Limitations were induced by the pre-existing functional status of the patient, use of inotropes, effects of positive end-expiratory pressure, haemodilution, hyperventilation, etc. These occur in a patient as a result of reperfusion injury, the acute phase response, the risk of a second hit phenomenon, coagulation deficiency, abdominal compartment syndrome and immune compromise induced by trauma. Packing the abdomen and closing it inherently introduces an abdominal compartment syndrome with resultant increases in the peak airway pressure and a decrease in the glomerular filtration rate.

Central venous pressure monitoring measures provide an indication of trends in the right atrium, without much indication of events in the lungs and heart. i.e. major target organs in shock. Resuscitation to supramaximal endpoints based on monitoring cardiac function (PAWP catheter) introduced by Shoemaker was subsequently shown not to improve survival. The ability of the cardiovascular system to compensate far outstrips the ability of the gut and other organ systems to respond to physiological challenges. Urine output (> 1 mL/kg per h) is a sensitive indicator of visceral perfusion but its value may be compromised by renal damage associated with trauma or drugs. Global parameters of resuscitation, e.g. base deficit and lactate levels, may be modified by alcohol and drugs. Base deficit is currently the most readily accessible and commonly used global parameter of resuscitation and roughly correlates with survival, days spent in the ICU, and amount of blood required. Gastric tonometry is currently the most specific indicator of gut perfusion, but lacks sensitivity with respect to other tissue beds, requires careful attention and is expensive.

The critical phases to determine the quality of resuscitation occur at the end of surgery and the initial phase of stay in the ICU. These efforts cannot be left solely to other disciplines because the urgency of adequate resuscitation, the deleterious effects of inotropic drugs and assessment of ongoing haemorrhage may not be readily understood. Joint decision-making is essential. Time to determine return to the operating theatre is ultimately a surgical decision taken when the goals of resuscitation are met, irrespective of time of the day or time since the original procedure.

2.5.1 Admission to the ICU

There are no definite indicators for admission of injured patients to the ICU, only guidelines. In general, admission to intensive care with elective ventilation is mandatory following damage control surgery as a result of the physiological debility induced by the indications for, and the effects of, damage control. These include:

- Respiratory failure with a PaO₂/FiO₂ ratio (PF ratio) < 200, peak airway pressures > 38 cmH₂O
- Hypothermia < 34°C as a result of acidosis, myocardial depression, coagulopathy, etc., induced by global enzyme malfunction
- Metabolic acidosis with a base deficit of > 6.0 or a pH < 7.1
- Massive blood transfusion (see definition above)
- Coagulation deficiency with an INR or PTT value of ≥ 2 or platelet count < 50 000 because of the risk of ongoing haemorrhage
- Requirement for inotropes to maintain CO
- Evidence of abdominal compartment syndrome (ACS).

Normalization of the above parameters will also constitute adequate resuscitation, along with normalizing standard haemodynamic parameters and urine output > 1 mL/kg per h.

Other guidelines to admit to ICU and ventilate relate to individual injuries or pre-existing disease and include:

- Inability to maintain an airway, e.g. severe facial fractures or GCS < 8.
- The need to obtain initial control of PO₂ and PCO₂ in head-injured patients, because avoidance of hypoxia and maintenance of normocapnia (PaCO₂ of
4.7–5.3 kPa or 35–40 mmHg) are known to improve survival in head-injured patients
- Injury Severity Score (ISS) > 25 implies a mortality rate of > 20 per cent; charts are available as guidelines
- Major burns
- Any major co-morbid disease or extremes of age will upgrade any of the above indicators.

Elective admission to ICU for severely injured patients has been shown to result in an average of 4 ICU days whereas salvage admission resulted in an average of 7.5 ICU days. The cost benefit is obvious.

Physiological or intervention-based scores, e.g. APACHE (Acute Physiology and Chronic Health Evaluation II) or TRISS (Trauma Score in association with ISS), retrospectively measure outcome in populations and are particularly insensitive for determining outcome in individual trauma patients. The implications of admitting patients for potentially futile care must be carefully considered, e.g. in severe head injuries (GCS 3 or 4) or complete quadriplegia levels C3–4 or above. Guidelines should be developed beforehand by all healthcare disciplines involved.

Admission to ICU for severe trauma or after damage control surgery requires active surgical input including:
- Hyperventilation to improve acidosis
- Pressure-controlled ventilation to limit barotrauma and to limit the effects of ACS
- Intravesical pressure monitoring to identify ACS
- High-capacity (e.g. Level One™ apparatus) infusions of the freshest blood available to a Hb of 8–10 g per cent
- Platelet infusions to ensure a platelet count > 50 000 mm⁻³
- Infusion of cryoprecipitate as colloid to provide clotting factors, e.g. to INR/PTT < 1.5
- Active rewarming using warm a air blanket (Bair Hugger), warmed fluids, warmed ventilation and cavity lavage.

Limiting inotropes to the minimum required to maintain satisfactory output, e.g. mean arterial pressure > 70 mmHg, urine output 1–2 mL/kg per h and central venous pressures showing a rising trend.

The patient should be returned to theatre in case of deterioration, or failure to improve – usually as a result of the development of intra-abdominal compartment syndrome or ongoing haemorrhage. Return to theatre for definitive surgery should take place as soon as the goals of resuscitation are met.

2.5.1.1 KEY REFERENCE

2.5.2 Endpoints of ventilation

There are three main indications for ventilation:
1 hypoxia
2 ventilatory insufficiency
3 compromised or threatened airway.

Hypoxia implies decreased delivery of oxygen to the tissues.

$$\text{DO}_2 = \text{CI} \times [\text{Hb}] \times 1.39 \times \text{SaO}_2.$$ 

Therefore, delivery is compromised when the CI, Hb or Hb-carrying capacity is low, not only when the PaO₂ or saturation is low.

Hypoxic hypoxia implies that the PaO₂ is low and occurs with ventilation–perfusion mismatch, alveolar hypoventilation, low inspired O₂ fraction, diffusion abnormality and, in the presence of ventilation–perfusion mismatch, a low cardiac output.

Ventilation–perfusion mismatch and diffusion defects occur with parenchymal disease such as pulmonary contusion or oedema, aspiration and ARDS. Alveolar hypoventilation occurs with neuromuscular disease, overdose of drugs or alcohol, airway obstruction or a severe metabolic alkalosis.

Delivery is reduced not only with severe anaemia, but also with a reduced carrying capacity as occurs with carbon monoxide or cyanide poisoning. These conditions have a normal PaO₂ but, if delivery is below a critical level, they will present with a metabolic acidosis.

The presence of hypoxia therefore includes assessment of oxygenation and acid–base status.

Oxygenation is assessed by means of the PaO₂ (mmHg)/FiO₂ ratio (normal 400–500) or by the saturation. Circulatory or anaemic hypoxia by the pH and the HCO₃⁻. Adequacy of ventilation is assessed by the PaCO₂. However, an elevated value is not necessarily an acute problem provided that the pH is abnormal, e.g.:

- ↑ CO₂; ↑ HCO₃⁻; normal pH – chronic obstructive pulmonary disease (COPD)
- ↑ CO₂; ↑ HCO₃⁻; ↓ pH – acute on chronic
- ↑ CO₂; HCO₃⁻ normal; ↓ pH – acute ventilatory insufficiency
When to ventilate?

First, recognize that a problem might exist:

- Agitated, belligerent or obtunded patients
- Patients with respiratory distress or noisy breathing
- Those with CNS depression or head injury
- Severe maxillofacial injury
- Those with severe chest injury, fractures or flail chest
- The hypotensive patient or the patient who has been in a smoky environment.

Those patients who should be ventilated include those with:

- Apnoea
- Head injury (GCS > 8)
- ↑CO$_2$ and respiratory acidosis
- PaO$_2$ < 4 kPa (60 mmHg) on re-breathing mask
- Haemodynamic instability or cardiac arrest
- Severe metabolic acidosis pH < 7.2.

2.5.3 End points of circulation

2.5.3.1 PATHOPHYSIOLOGY

The traditional aetiological approach to shock is simple, clear, logical, straightforward, readily understandable, and generally accepted by most textbooks and educators. The problem with this concept of shock is that real life is not this simple … if it is to be maximally effective, therapy must address all components of the disturbed circulation.

An insult of sufficient magnitude results in inadequate organ perfusion and tissue oxygenation, i.e. shock. In addition to the vasomotor response, the metabolic response to any insult, e.g. haemorrhage, is remarkably similar, and consists of the acute phase response:

- Hormonal – diabetogenic state
- Immune – immunosuppression
- Coagulation – hypercoagulability
- Anticoagulation – clot lysis
- Temperature – pyrexia
- Leukocyte activation/ aggregation – inflammatory response
- Arachidonic acid derivatives (complement, prostaglandins).

Hypovolaemic shock results in vasoconstriction, anaerobic metabolism and clogging of the microcirculation.

Anaerobic metabolism results in production of 2 mol ATP (47 kcal) per mole of oxidized glucose as opposed to 36 mol (673 kcal)/mol glucose oxidized. Resuscitation by increasing the intravascular volume results in release of the products of anaerobic metabolism, including metabolic acids (predominantly lactate), and radicals. Radicals cause further damage by reacting with the nearest electron donors, especially in endothelial cells – thereby increasing vascular permeability. Subsequent adverse events result in logarithmic activation of primed leukocytes – causing an enhanced acute phase response. Depending on the magnitude, duration and repetition of the insult, the response is one or all of the following:

- Systemic inflammatory response syndrome (SIRS)
- Septic syndrome
- Multiple organ dysfunction syndrome (MODS).

In addition to these global phenomena, the ability of the cardiovascular system far outstrips the ability of other organ systems to respond to resuscitation. The key principle is to resuscitate patients, including all their organ systems and tissue beds, not only their cardiovascular systems and certainly not just their systolic blood pressure.

2.5.3.2 VOLUME RESUSCITATION

There is no level 1 evidence of improved survival after resuscitation using crystalloids versus colloids. Colloids remain in the system longer and exert a colloid osmotic pressure. Theoretically, colloids buy more time (hours) to address the cause of shock. Low-volume hypertonic saline/saline dextran (<200 mL) may be of benefit to improve survival only in isolated head injuries, at the risk of sodium overload. Hypotensive resuscitation (blood pressure [BP] 90) until the bleeding is controlled may be of benefit provided that there is only a short interval between injury and control of bleeding. Subset analysis of the original work showed benefit to be confined to penetrating cardiac injury.

2.5.3.3 BLOOD PRESSURE

Traditional manual measurements of BP are notoriously inaccurate. We are interested in perfusion (flow) but measure a linear dimension thereof (pressure). Mean arterial pressure (MAP) is the true driving pressure for flow, does not change as the pressure waveform moves distally, and is not altered so much by distortions
generated in recording systems. At low pressures, electrically monitored non-invasive BP yields spuriously low values – use a BP cuff and the Doppler probe, or preferably intra-arterial catheters. Aim for a MAP <70 mmHg.

2.5.3.4 TEMPERATURE

There is no enzyme system that functions under hypothermic conditions. Temperature <34°C is associated with a mortality rate of 32 per cent. Temperature <32°C in trauma patients is associated with mortality rate of >60 per cent. One degree centigrade of temperature loss results in an increase in metabolic requirements of 12 per cent in an ATP-depleted patient. This must be prevented at all costs.

2.5.3.5 SERUM LACTATE/BASE DEFICIT

Lactate level is the best clinical indicator of the degree of tissue ischaemia – even in the absence of clinical shock. Persistence of an elevated serum lactate after 24 h, despite resuscitation is associated with ARDS (50 per cent), multisystem organ failure (36 per cent) and mortality (43 per cent). Failure to establish lactate levels of <2.5 mg/dL within 24 h may be an indicator to use pulmonary capillary wedge pressure (PCWP) to guide inotrope and volume resuscitation. Hyperglycaemia, epinephrine infusion and infection may also elevate lactate levels. Base deficit is probably the poor person’s alternative to lactate determinations.

2.5.3.6 URINE VOLUME

Urine volume is a sound indicator of organ perfusion, because the kidney takes up 25 per cent of cardiac output. Pain, stress and morphine will increase ADH secretion and limit urine volume. A urine volume of 0.5–1.5 mL/kg per h without diuretics is normal.

2.5.3.7 HAEMOGLOBIN/HAEMATOCRIT (SEE MASSIVE BLOOD TRANSFUSION, SECTION 9.5.2)

Both of these measures are relative to intravascular volume. Oxygen-carrying capacity is reconstituted in a matter of hours – depending on the age of the blood. There is no level 1 evidence identifying the ideal Hb level in trauma patients. There is no difference in survival between patients (admitted to surgical ICU) transfused to Hb 8–10 g/dL and a higher level.

2.5.3.8 CENTRAL VENOUS PRESSURE

Normal CVP is 0–10 cmH₂O. Central venous pressure is an indicator, not a parameter, of cardiac filling. Central venous pressure (and PCWP) correlate poorly with volume of blood lost (vasoconstriction, decreased ventricular compliance). In general, use a bolus method, e.g. Ringers’ lactate 50 mL/h or colloid 250 mL/h, in previously healthy patients, until CVP fails to increase for 30 min. For previously compromised patients, half the volume of bolus is suggested. Avoid the use of starch-based solutions because of their depletion of certain clotting factors, especially factor VIII.

2.5.3.9 OXYGEN EXTRACTION RATIO

The oxygen extraction ratio defines the efficiency with which oxygen is extracted compared to the amount delivered, and therefore gives an indication of tissue perfusion:

\[ ERO_2 = \frac{VO_2}{DO_2} \times 100 \]

(normal = 0.2–0.3).

A value of 0.5–0.6 implies critical O₂ delivery, resulting in inadequate tissue perfusion. This results in anaerobic metabolism and increased lactate production:

\[ ERO_2 = SaO_2 - SvO_2 = SaO_2 - CvO_2 \]

Hypovolaemia = 0.3–0.5

Hypovolaemic shock = < 0.5.

\( SvO_2 \) is the mixed venous oxygen saturation.

2.5.3.10 GAS EXCHANGE

\( SaO_2 \) measured by pulse oximetry differs by <3 per cent from \( SaO_2 \) measured by co-oximetry, and is accurate down to Hb 3 g/dL and blood pressure 30 mmHg. End-tidal \( PaCO_2 \) exhibits a linear relationship to cardiac output with a correlation coefficient of 0.87 during resuscitation, and can be monitored non-invasively.

2.5.3.11 INTRA-ABDOMINAL HYPERTENSION (SEE SECTION 3.5)

Intra-abdominal compartment syndrome is a syndrome of decreased cardiac output, ventilation and renal function following an increase in the intra-abdominal pressure. Pressure is measured by instilling 50 mL saline into the bladder and measuring at the level of symph-
ysis pubis. Pressure is normally < 12 mmHg. A pressure > 20 mmHg indicates incipient ACS and need to relieve intra-abdominal pressure.

2.5.3.12 COAGULATION

STOP THE BLEEDING including the OOZING!

(See below on Massive blood transfusion – Section 9.5.2.)

2.5.3.13 PULMONARY CAPILLARY WEDGE PRESSURE

Equivalent to left atrial pressure (LAP) or LVEDP. Resuscitating to supranormal values has been shown to differentiate between responders (corresponding to survivors) and non-responders (equivalent to mortalities), and not to improve outcome. The current approach is to resuscitate to normal values.

The PCWP catheter is used to monitor the following situations:

- Non-responders to volume correction
- Guiding inotrope therapy
- Lactate > 2.5 mg/dL after maximal resuscitation
- Pre-existing cardiac or pulmonary disease.

2.5.3.14 GASTRIC MUCOSAL PH

This measures local tissue (hypo)perfusion. A saline balloon is placed in the stomach. Equalization time is allowed, the gastric content is aspirated and the pH is measured (using a blood gas analysis). A pH < 7.2 implies hypoperfusion of the splanchnic area.

Gastric mucosal PCO₂ is largely derived from gastric mucosal lactate, i.e. gastric mucosal pH measures lactate levels in gastric mucosa.

2.5.4 References


2.5.5 Recommended reading


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Part 3

Decision-making
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3.1 Resuscitation in the Resuscitation Room

3.1.1 Ideal Practice

Patients with life-threatening injuries represent about 10–15 per cent of all patients hospitalized for injuries.1 Some authors have defined extensive trauma as a patient who has an injury severity score (ISS) > 15.2–4 This is not useful for triage purposes, but for this discussion extensive trauma can be defined as an ISS > 25. Most patients in this category have multisystem trauma. For triage purposes, information available in the prehospital phase and primary survey should be used.

A standardized approach, utilizing the ‘MIST’ handover, should be used (Table 3.1).

Table 3.1 The ‘MIST’ handover

<table>
<thead>
<tr>
<th>M</th>
<th>Mechanism of injury</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>Injuries observed</td>
</tr>
<tr>
<td>S</td>
<td>Vital signs</td>
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<tr>
<td>T</td>
<td>Therapy instituted</td>
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</tbody>
</table>

Deforming and destructive injuries can be obvious, but the surgeon or physician initially treating the patient must promptly conduct a systematic work-up, so that all wounds, including occult mortal injuries, can be treated optimally.

The principles of management for patients suffering major trauma are:

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

Time is working against the resuscitating physician. Sixty-two per cent of all trauma patients die within the first 4 hours of hospitalization.3 Most of these patients either bleed to death or die from primary or secondary injuries to the central nervous system. To reduce this mortality, the surgeon must promptly guarantee ventilation and restore tissue perfusion, identify the sites of haemorrhage and stop it, diagnose and evacuate mass intracranial lesions, and treat cerebral oedema. 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.

Unfortunately, collecting information requires time. Time usually is not available and the work-up of the critically injured patient must often 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. We use ATLS as a paradigm for assessment, resuscitation and prioritization of the patient’s injuries.

General guiding times in the emergency department (ED) are:

- For the unstable patient: 15 min
- For the stable patient: 30 min
- The unstable patient should either be in the operating room or the intensive care unit (ICU) within 15 min
- The stable patient should be in the ICU’s computed tomography (CT) scanner within 30 min.

3.1.2 Resuscitation

Resuscitation is divided into two components: the primary survey and initial resuscitation, and 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 operative intervention immediately.

3.1.2.1 Primary survey

The priorities of the primary survey are:

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

Airway

Patients with extensive trauma who are unconscious or in shock benefit from immediate endotracheal intubation. The cervical spine must not be excessively flexed or extended during intubation to prevent spinal cord injury. Oral endotracheal intubation is successful in most injured patients. A few patients may require nasotracheal intubation performed by an experienced physician. During intubation, firm compression of the cricoid cartilage against the cervical spine occludes the oesophagus and reduces the risk of aspirating vomitus.

On rare occasions, bleeding, deformity or oedema from the maxillofacial region will require cricothyroidotomy or tracheostomy. Patients likely to require a surgical airway include those with laryngeal fractures. The airway priorities are to clear the upper airway, establish high-flow oxygen initially with a bag–mask and proceed immediately to endotracheal intubation in most and surgical airway in a few.

Breathing

Patients with respiratory compromise are not always easy to detect. Simple parameters such as the respiratory rate (RR) and adequacy of breathing on simple clinical parameters should be examined within the first minute of arrival. One of the most important things is to detect a tension pneumothorax, necessitating direct drainage by needle thoracotomy and a thoracic tube.

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 are cool and pale, lack venous filling and have poor capillary refill. The pulse will be thready and consciousness diminished. 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, there are five possibilities:

1. Tension pneumothorax
2. Pericardial tamponade
3. Myocardial contusion
4. Myocardial infarct
5. Air embolism.

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

Pericardial tamponade is most commonly encountered in patients with penetrating injuries to the torso. Approximately 25 per cent 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 a very few patients with equivocal findings. Pericardiocentesis is an occasionally useful diagnostic or therapeutic aid. Proper treatment is immediate thoracotomy, preferably in the operating room, although ED thoracotomy can be life saving.

Myocardial contusion is a rare cause of cardiac failure in the trauma patient. Myocardial infarction from coronary occlusion is not uncommon in elderly people.

Air embolism is a syndrome that has relatively recently been appreciated as important in injured patients, and represents air in the systemic circulation caused by a bronchopulmonary venous fistula. Air embolism occurs in 4 per cent of all major thoracic injuries. Thirty-five per cent of the time it is caused by blunt trauma, usually a laceration of the pulmonary parenchyma by a fractured rib. In 65 per cent of patients it is the result of gunshot 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, and then followed by expansion of the intravascular volume. Open cardiac massage, intravenous epinephrine (adrenaline) and venting of the left heart and aorta with a needle to remove residual air may be required. The pulmonary injury is definitively treated 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 access to the circulation
- To obtain a blood sample from the patient
- To determine where the volume loss is occurring
- To give resuscitation fluids
- To prevent and treat coagulopathy
- To prevent hypothermia.

Access is usually via the subclavian route, but alternative ways to gain access to the circulation are by a surgical cut-down on the saphenous vein at the ankle or via bilateral percutaneous femoral vein cannulation with a large-bore catheter or an 8-French introducer, more commonly used for passing a pulmonary artery catheter.

As soon as the first intravenous line has been established, baseline blood work is obtained that includes haematocrit, toxicology, blood type and crossmatch, 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 by rapid chest radiograph, 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. Common sense dictates that, if the patient’s chest radiograph is normal and the femur not fractured, the patient who remains in shock must be suspected of having ongoing haemorrhage in the abdomen or pelvis. Most of these unstable patients would require immediate laparotomy to avoid death from haemorrhage.

The fourth volume priority for the resuscitating physician is to order resuscitation fluids, starting with balanced salt solution and adding type-specific whole blood as soon as possible.

The fluid used to resuscitate a hypotensive patient will depend on the patient’s response to fluid load. The ‘rapid responder’ may require no more than crystalloid to replace the volume deficit. The ‘transient responder’ may need the addition of colloid or blood. The only practical way to measure atrial filling pressure in the ED, and immediately in the operating room, is by a central venous pressure (CVP) monitor. In elderly patients with extensive traumatic injuries, placing a pulmonary artery catheter in the ICU may be prudent because it will be used to direct a sophisticated multifactorial resuscitation. 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 tests.

Crystalloid, synthetic colloids such as gelatins and dextrans, as well as blood, are available to replace volume in hypotensive patients. It is clear that patients requiring massive transfusions need the oxygen-carrying capacity of red cells. Data suggests that trauma leads to leaky cells in the pulmonary capillary bed, and the use of colloid puts these patients at further risk. In non-trauma patients where the pulmonary vasculature is not at risk, colloids would be beneficial. It is likely that most trauma patients will receive a mix of crystalloid, synthetic colloid and blood.

Bickell et al. found that the survival in patients with a penetrating torso trauma was improved if fluid replacement was delayed. They suggested that immediate volume replacement in these patients might disrupt blood clot that had obliterated a bleeding vessel.

Research continues on the use of perfluorocarbons and stroma-free haemoglobins as effective substitutes for blood.

Although whole blood is preferred, it is indeed difficult to obtain whole blood from modern blood banks, forcing the use of blood components. With modest blood loss, infusion of balanced salt solutions and packed red
cells is all that is needed. In the exsanguinating patient, type O blood should be given, whereas in a stable patient it is prudent to wait for typed and crossmatched blood. With extensive haemorrhage and massive transfusion (more than one blood volume), component therapy must be directed by monitoring specific defects such as thrombocytopenia, hypofibrinogenaemia, and factor V or VIII deficiency. The criteria for adequate resuscitation are simple and straightforward:

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

Neurological status

The next priority during the primary survey is quickly to assess neurological status and to initiate diagnostic and treatment priorities. The key components of a rapid neurological evaluation are:

- To determine level of consciousness
- To observe size and reactivity of the pupils
- To check eye movements and oculovestibular responses
- To document skeletal muscle motor responses
- To determine the pattern of breathing
- To perform a peripheral sensory examination.

A decreasing level of consciousness is the single most reliable indication that the patient potentially has 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 him- or herself 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 brain-stem 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 six components and ensuring that the four primary reflexes (ankle, knee, biceps and triceps) are assessed, and repeating this exam at frequent intervals, it is possible both to diagnose and to 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 a significant neurological injury. Computed tomography of the head is the definitive test for head injury and should be done as soon as possible.

Environment

Hypothermia

The body temperature of trauma patients decreases rapidly and, if the ‘on-scene time’ has been prolonged, e.g. 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. 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 before 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 after the primary survey include:

- Chest radiograph
- Plain film of the pelvis.

Selected patients, e.g. those with haematuria or significant deceleration injury, may require a single film intravenous urogram (IVU) to confirm bilateral nephrograms. It must be emphasized that resuscitation should not cease during these films and the resuscitating team must wear protective lead aprons. Optimally, the radiological facilities are juxtaposed to the ED, although the basic radiographs can all be obtained with a portable machine.

Finally, if the patient stabilizes, secondary survey and diagnostic studies are done. However, if the patient remains unstable, he or she should be promptly taken to the operating room or surgical ICU.
3.2 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.

The first priority is to establish an airway, usually with an endotracheal tube, which will allow an assistant to ventilate the patient effectively with reduced risk of aspiration. The second priority is to ensure that oxygenation and ventilation are adequate. Finally, large-bore intravenous access is established, usually with 14-gauge intravenous cannulae in the upper extremities. If there is need for further intravenous access, an 8.5-French catheter can be introduced into the central venous system through either the groin or the subclavian approach. This access will allow for large volumes of crystalloid and blood to be given immediately. The goal is to transfuse warm fluid at a rate of up to a litre per minute. Basic life-saving procedures are instituted. It is essential to complete a secondary assessment of the patient rapidly to identify all life-threatening and other injuries.

An effective plan is to identify the patient’s probability of survival. Once it has been determined that there is a high likelihood of death, it is useful to make a mental judgement of when death will occur if the patient is not treated:

- Immediately
- Within the next 5 min
- Within the next hour
- Within the next day.

As a basic consideration, for all major trauma victims with a systolic blood pressure of less than 90 mmHg, there is a 50 per cent likelihood of death which, in a third of cases, will occur within the next 30 min. If the patient has arrested, then cardiopulmonary resuscitation (CPR) should be initiated while other life-saving manoeuvres are being contemplated.

If death is likely to occur in the next 5 min, it is essential to determine in which body cavity the lethal event will occur.

A simple dictum is that a surgeon should be able to direct the medical examiner to the right body cavity for the cause of death.
If death is likely to occur in the next hour, there is time to proceed with an orderly series of investigations and, time permitting, radiological or other diagnostic aids, to determine precisely what is injured, and to effect an operative plan for the management for this life-threatening event.

### 3.2.1 Craniofacial injuries

It is unusual but possible to exsanguinate from a massive scalp laceration. 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.

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 plegia is diagnostic of a mass lesion with sufficiently significant intracranial pressure to induce coning. This requires immediate decompression. Time can be saved by hyperventilation to induce hypocapnia and concomitant vasoconstriction.

Attention should be paid to monitoring the end-tidal CO\(_2\) (ET\(\text{CO}_2\)), as a proxy for \(\text{PaCO}_2\). \(\text{PaCO}_2\) should not be allowed to fall below 3.99 kPa (30 mmHg). 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 for the determination of a specific site for the mass lesion and the type of haematoma. This directs 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. This should not delay any other diagnostic or therapeutic procedures.

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

### 3.2.2 Chest trauma

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

Transected aorta is usually diagnosed with a widened mediastinum and confirmed with an arteriogram or CT (see section on chest injuries). Once the diagnosis has been made, it is essential to repair the aorta in the operating room, as soon as possible. In general, it is useful to maintain control of hypotension in the 100 mmHg range so as not to precipitate free rupture from the transection. **Note that abdominal injury generally takes priority over thoracic aortic injury.**

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

The diagnosis of cardiac tamponade is frequently difficult to make clinically. It is usually associated with hypotension and elevated JVP. There are usually muffled heart sounds, but this is difficult to hear in a noisy resuscitation suite. Placing a central line with resultant high venous pressures can confirm the diagnosis. If ultrasonography is available, this is an extremely helpful diagnostic adjunct. Once the diagnosis has been made, if the patient is hypotensive, the tamponade needs to be relieved. In the event of a penetrating injury to the heart or blunt rupture of the heart, there is usually a substantial clot in the pericardium. A needle pericardiocentesis may be able to aspirate a few millilitres of blood and this, along with rapid volume resuscitation to increase preload, can buy enough time to move to the operating room.

It is far better to perform a thoracotomy in the operating room, either through an anterolateral approach or as 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 range 40 mmHg or lower despite volume resuscitation, there is no choice but to proceed immediately with a left anterior thoracotomy in an attempt to relieve the tamponade and control the penetrating injury to the heart. If there is an obvious penetrating injury to either the left or right ventricle, a Foley catheter can be introduced into the hole and the balloon distented to create tamponade. The end of the Foley catheter should be clamped. **Great care should be taken to apply minimal traction on the Foley catheter – just enough to allow sealing. Excessive traction will pull the catheter out and extend the wound by tearing**
the muscle. Once the bleeding has been controlled, the wound can be easily sutured with pledgetted sutures.

Massive haemorrhage from intercostal vessels secondary to multiple fractured ribs frequently will stop without surgical intervention. This is also true for most bleeding from the pulmonary system. It is essential to attempt to collect shed blood from the hemithorax into an autotransfusion collecting device, so that the massive haemothorax can be immediately autotransfused to the patient.

3.2.3 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. A definitive diagnosis can be made with a grossly positive diagnostic peritoneal lavage (DPL), ultrasonography or CT. The decision to operate for bleeding should be based on the haemodynamic status.

Diagnostic peritoneal lavage is easy to perform and gives a highly specific but insensitive answer immediately. Ten millilitres of grossly positive blood requires immediate laparotomy.

Ultrasonography is a useful tool because it is specific for blood in the peritoneum, but it is operator dependent and does not usually give a definitive answer as to which organ is injured or the severity of injury.

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

There is increasingly a philosophy of observing the lesser severity grades of injury to the liver and spleen if the patient does not have persistent hypotension.

3.2.4 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 the pneumatic anti-shock garment (PASG), can be used. There are now external fixating devices, such as the C-clamp, which can be placed in the resuscitation suite; these immediately return the pelvis to its normal anatomy. As the pelvis is realigned it compresses the haematoma in the pelvis. As blood is incompressible there is a rapid rise of intrahaematoma pressure, which quickly exceeds venous and arterial pressure. As about 85 per cent of pelvic bleeding is venous, or directly from the fracture, compressing the haematoma usually stops most of the bleeding from the pelvis.

If the patient continues to be hypotensive, resuscitation should continue and an angiogram performed if the haemodynamic situation allows. This identifies significant arterial bleeding in the pelvis, which can then be embolized immediately. In the absence of bleeding from the pelvis, an arteriogram of the solid organs in the abdomen can be performed for diagnostic purposes as well as to assess the potential for embolization. If the patient is exsanguinating from the pelvic injury, consider damage control surgery with packing of the pelvis before angiography. Recent reports indicate that extraperitoneal pelvic packing is more effective than intraperitoneal packing.

3.2.5 Long bone fractures

Long bone fractures, particularly of the femur, can bleed significantly. The immediate treatment for a patient who is hypotensive as a result of 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 pulses are absent, an arteriogram should be performed to determine whether there are any injuries to major vascular structures. A determination is then made about the timing of arterial repair and bony fixation.

3.2.6 Peripheral vascular injuries

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

Every ED should have access to a simple flow Doppler to assess pressures and flow. If there is any doubt as to
whether the vessel is patent, an arteriogram is mandatory. Although it is desirable to do this in the angiography suite, this 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.

3.2.7 Summary

The decision as to whether to operate in the ED or 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 potentially dying trauma patient, so that both clinical diagnosis and relevant investigations can be performed immediately, and a surgical or non-surgical therapeutic approach implemented.

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

3.3 CURRENT CONTROVERSIEST

3.3.1 Pre-hospital resuscitation

There are two recent studies; one from Ben Taub Hospital in Houston and the other from Los Angeles County Hospital, that suggest that ‘scoop and run’ is superior to any advanced life support (ALS) techniques exercised in the pre-hospital setting. Both of these studies are controversial and must be carefully analysed before appropriate changes are made in our current pre-hospital care. The study in Houston is problematic in that it addressed only penetrating injuries and the response time was approximately 10 min. If minimal fluid resuscitation was carried out, the outcome was better, measured by either mortality or the development of multiple organ failure. Clearly, it has been known for some time that control of major vascular injuries is imperative before massive resuscitation. Nevertheless, this study does re-emphasize some important principles although it does not support the application of these principles to other emergency medical services (EMS) settings, particularly rural areas, or to blunt trauma.

The other study was from Los Angeles County, which showed that patients transported by private vehicle did better than those patients transported by ambulance. Again, one must be circumspect in interpreting this data. There is no question that by transporting in a private vehicle there is an element of time that is gained – specifically, the ambulance response time and any delays incurred at the scene from treatment. One has to weigh the advantages of rapid transport within different geographical settings and whether or not certain ALS techniques (airway) outweigh the disadvantages.

3.3.2 Systemic inflammatory response syndrome (SIRS)

Two large studies have shown that 50 per cent of patients with ‘sepsis’ are abacteraemic. It is also recognized that the aetiology in these abacteraemic patients may be burns, pancreatitis, significant soft tissue and destructive injuries to tissue, particularly when associated with shock. The common theme through all of these various injuries and sepsis is that the inflammatory cascade has been initiated and runs amok. Once the inflammatory response is initiated it leads to systemic symptoms that may or may not be beneficial or harmful. The primary symptoms associated with systemic inflammatory response syndrome (SIRS) include:

- Temperature: < 36°C or > 38°C
- Heart rate: > 90/min
- RR: > 20/min
- Deranged arterial gases: $\text{PCO}_2 < 32$ mmHg
- White blood count: > $12.0 \times 10^9/L$ or < $4.0 \times 10^9/L$ or 0.10 immature neutrophils.

Patients who have one or more of these primary components are thought to have SIRS. A further classification of SIRS is that sepsis is SIRS plus documented infection. Severe sepsis is sepsis plus organ dysfunction, hypoperfusion abnormalities or hypotension. Finally, septic shock is defined as septic-induced hypotension despite fluid resuscitation. It is now recognized that there are a number of messengers associated with SIRS, including cytokines, growth factors and cell surface adhesion molecules. Equally important components of the expression of SIRS are the genetic cellular events, including the transcriptases and other proteins associated with the upregulation and downregulation of gene expression. It is now appreciated that, if these cytokines and cell adhesion molecules are in proper balance, beneficial effects take place during the inflammatory response.

Conversely, if there is a dysregulation or dyshomeostasis of these various cytokines and growth factors,
harmful effects may take place, damaging organs, and may lead to patient death. This dysregulation may affect vascular permeability, chemotaxis, vascular adherence, coagulation, bacterial killing and all the components of tissue remodelling. Some of the corollary concepts that have grown out of our understanding of SIRS is that the inflammatory cascade is not to be interpreted as harmful. It is only when the dysregulation occurs that it is a problem in patient management. The second concept is that cytokines are messengers and we must not kill the messenger. Whether or not we can control them by either upregulation or downregulation remains to be proven by careful human studies.

3.3.3 Head injury

A recent monograph on practice guidelines for the management of head injury has challenged our current non-surgical management.\(^1\) Using a careful review of the surgical literature, a team of neurosurgeons using standards, guidelines and options have determined that some of the ‘standard’ treatment cannot be justified by the literature. An example is hyperventilation, which is almost routinely used in order to control vascular volume within the cranial vault. Other examples include the head-up position, mannitol, barbiturates and the efficacy of maintaining mean arterial pressure (MAP) in order to increase cerebral perfusion pressure. Such practice guidelines are extremely useful to emergency physicians and surgeons.

3.3.4 Specific organ injury\(^2\)

Myocardial contusion has been better defined and a practice guideline has been developed. Aortic disruption has become controversial from a diagnostic standpoint with the introduction of CT and transoesophageal echocardiography. At this time it is probably best to consider an arteriogram still as the gold standard. Aortic disruption is further complicated by the best methodology to avoid paraplegia. It is most probable that it cannot be avoided entirely because it probably represents an anatomical anomaly in about 5–8 per cent of the population and there is currently no way to predict this 5–8 per cent and optimally prevent it. Splenic injuries remain controversial but have been further defined. It is now clear that anatomical injury severity scales do not correlate with physiological states and it is not possible to predict, from the injury severity scales, which organs can be salvaged and which cannot. It is also appreciated that splenectomy in the adult does not have the risk originally assumed. Multiple papers have described the management of civilian colon injuries and, although there seems to be some unanimity of opinion that not all patients require a colostomy, it has not been defined by practice guidelines precisely which patients benefit from a colostomy.

3.3.5 References


3.4 DAMAGE CONTROL

The concept of ‘damage control’ (also known as ‘staged laparotomy’) has as its objective the delay in imposition of additional surgical stress at a moment of physiological frailty.

Briefly stated, this is a technique where the surgeon minimizes operative time and intervention in the grossly unstable patient. The primary reason is to minimize hypothermia and coagulopathy, and to return the patient to the operating room in a few hours after stability has been achieved in an ICU setting. Although the principles are sound, extreme care has to be exercised over use of the concept so that we do not cause secondary insults to viscera. Furthermore, enough appropriate surgery has to be carried out in order to minimize activation of the inflammatory cascade and the consequences of SIRS and organ dysfunction.

The concept is not new, and livers were packed as much as 90 years ago but, with a failure to understand the underlying rationale, the results were disastrous. The concept was reviewed and the technique of initial abortion of laparotomy, establishment of intra-abdominal pack tamponade and then completion of the procedure once coagulation had returned to an acceptable level proved to be life saving. The concept of staging applies both to routine and to emergency procedures, and can apply equally well in the chest, pelvis and neck as in the abdomen.
3.4.1 **Stage 1: patient selection**

The indications for damage control generally can be divided into the following:

- Inability to achieve haemostasis
- Combined vascular, solid and hollow organ injury
- Inaccessible major venous injury, e.g. retrohepatic vena cava
- Anticipated need for a time-consuming procedure
- Demand for non-surgical control of other injuries, e.g. fractured pelvis
- Inability to approximate the abdominal incision
- Desire to reassess the intra-abdominal contents (directed relook)
- Evidence of decline of physiological reserve:
  - Temperature < 34°C
  - pH < 7.2
  - Serum lactate > 5 mmol/L
  - Prothrombin time (PT) > 16 s
  - Partial thromboplastin time (PTT) > 60 s
  - > 10 units blood
  - Systolic BP < 90 mmHg, for > 60 min
  - Operating time > 60 min.

Irrespective of setting, a coagulopathy is the single most common reason for abortion of a planned procedure, or the 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.

3.4.2 **Stage 2: operative haemorrhage and contamination control**

**Surgical objectives**

The primary objectives are as follows.

**Haemorrhage control**

Arrest bleeding, and the resulting (causative) coagulopathy. Procedures for haemorrhage control include:

- Repair or ligation of accessible blood vessels
- Occlusion of inflow into the bleeding organ (e.g. Pringle’s manoeuvre for bleeding liver)
- Tamponade using wraps or packs
- Intraoperative or postoperative embolization
- Intravascular shunting.

**Technique (see also Trauma laparotomy)**

During the period of time that the packs have been placed, it is important to place further intravenous lines and other monitoring devices as required. Hypothermia should be anticipated and the necessary corrective measures taken. After haemodynamic stability has been achieved, the packs in the two lower abdominal quadrants can be removed.

The packs in the left upper quadrant are then removed and, if there is splenic haemorrhage, a decision must be made whether to preserve or sacrifice the spleen, or temporarily to control haemorrhage with a vascular clamp placed across the hilum.

Finally, the packs are removed from the right upper quadrant and injury to the liver is assessed.

**Contamination control**

If there is associated faecal soiling this should be controlled. Limit contamination and the sequelae thereof.

- Ligation or stapling of bowel
- Resection of damaged segment with clips, clamps or staples.

**Temporary abdominal closure**

Close the abdomen to limit heat and fluid loss, and to protect viscera; this depends on whether the abdomen can be approximated to achieve closure. If not, the following options are available:

- ‘Sandwich’ (Vacpac) technique
- Bogota bag (temporary Silo bag)
- Mesh closure (e.g. with Vicryl mesh)
- Towel clips (now obsolete).

The timing of the transfer of the patient from the operating theatre to the ICU is critical. Prompt transfer is cost-effective and premature transfer is counterproductive. In addition, once haemostasis has been properly achieved, it may not be necessary to abort the procedure in the same fashion. Conversely, there are some patients, with severe head injuries, in whom the coagulopathy is induced secondary to severe irreversible cerebral damage, and further surgical energy is futile.

In the operating theatre, 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, because it is not infrequent to discover further injuries or ongoing bleeding.
3.4.3 Stage 3: physiological restoration in the ICU

The following are priorities in the ICU.

3.4.3.1 Restoration of Body Temperature

- Passive rewarming using warming blankets, warmed fluids, etc.
- Active rewarming with lavage of chest or abdomen.

3.4.3.2 Correction of Clotting Profiles

Blood component repletion.

3.4.3.3 Optimization of Oxygen Delivery

- Haemoglobin optimization to Hb 8–10 g/dL
- Swann–Ganz pulmonary artery wedge pressure (PAWP) monitoring.
- Swann–Ganz pulmonary capillary wedge pressure (PCWP) monitoring
- Correction of acidosis to pH > 7.3
- Measurement and correction of lactic acidosis to < 2.5 mmol/L
- Inotropic support as required.

3.4.3.4 Avoidance of the Abdominal Compartment Syndrome

Measurement of intra-abdominal pressure:

- Foley (bladder) catheter
- Intragastric catheter.

3.4.4 Stage 4: operative definitive surgery

The patient is returned to the operating theatre as soon as stage 3 is achieved. The time is determined by:

- The indication for damage control in the first place
- The injury pattern
- The physiological response.

Patients who develop major abdominal compartment syndrome (ACS) must be relooked at early, and any further underlying causes corrected.

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

The relook operation should be carried out thoroughly, suspecting further, previously undiagnosed, abdominal injury. If the patient’s physiological parameters deteriorate again, then further damage control should take place.

3.4.5 Stage 5: abdominal wall reconstruction if required

Once the patient has received definitive surgery, and no further operations are contemplated, the abdominal wall can be closed.

Methods involved include:

- Primary closure
- Closure of the sheath, leaving the skin open.
- Silo bag (Bogota bag), with subsequent gradual closure
- Grafts with Vicryl mesh, Gore-Tex sheets or other synthetic sheets.

3.4.6 Recommended reading


3.5 Abdominal Compartment Syndrome

3.5.1 Introduction

Raised intra-abdominal pressure (IAP) has far-reaching consequences for the physiology of the patient. There have been major developments in our understanding of
IAP and intra-abdominal hypertension (IAH). Raised IAP has far-reaching consequences for the physiology of the patient. The syndrome that results is known as ACS. Increasingly it is being recognized that ACS is not uncommon in trauma patients and failure to consider its prevention, detect it in a timely fashion and treat it aggressively result in a high mortality rate.

3.5.2 Definition

The first World Congress on ACS was held in 2005 and an internal consensus agreement relating to definitions is shown in Table 3.2.

Specifically ACS is defined as a sustained IAP \leq 20 \text{ mmHg} \text{ (with or without an abdominal perfusion pressure} < 60 \text{ mmHg)} \text{ that is associated with new organ dysfunction/failure.}

3.5.3 Pathophysiology

The incidence of IAH in postoperative trauma patients ranges from 20 per cent to 50 per cent. It is common after many forms of emergency surgery. The causes of acutely increased IAP are usually multifactorial and are shown in Table 3.3. In addition to the direct causes shown in Table 3.3, hypothermia, acidosis and overall injury severity will further exacerbate the problem.

3.5.4 Causes of raised IAP

See Table 3.3.

<table>
<thead>
<tr>
<th>Table 3.3 Causes of raised intra-abdominal pressure</th>
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<tbody>
<tr>
<td>Massive resuscitation</td>
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<td>Major intra-abdominal and retroperitoneal haemorrhage</td>
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<td>Tissue oedema secondary to insults such as ischaemia and sepsis</td>
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<td>Paralytic ileus</td>
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<td>Ascites</td>
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3.5.5 Effect of raised IAP on individual organ function

3.5.5.1 RENAL

In 1945, Bradley, in a study of 17 volunteers, demonstrated that there was a reduction in renal plasma flow
and glomerular filtration rate (GFR) in association with increased IAP. In 1982, Harman showed that, as IAP increased from 0 to 20 mmHg in dogs, the GFR decreased by 25 per cent. At 40 mmHg, the dogs were resuscitated and their cardiac output returned to normal. However, their GFR and renal blood flow did not improve, indicating a local effect on renal blood flow.

The situation in seriously ill patients may, however, be different and the exact cause of renal dysfunction in the ICU is not clear, as a result of the complexity of critically ill patients. In a recent study, it was reported that, of 20 patients with increased IAP and renal impairment, 13 already had impairment before the IAP increased.

The most likely direct effect of increased IAP is an increase in the renal vascular resistance, coupled with a moderate reduction in cardiac output. Pressure on the ureter has been ruled out as a cause, because investigators have placed ureteric stents with no improvement in function. Other factors that may contribute to renal dysfunction include humeral factors and intraparenchymal renal pressures.

The absolute value of IAP that is required to cause renal impairment is probably in the region of 15 mmHg. Maintaining adequate cardiovascular filling pressures in the presence of increased IAP also seems to be important.

### 3.5.5.2 Cardiovascular

Increased IAP reduces cardiac output, as well as increasing CVP, systemic vascular resistance, pulmonary artery pressure and PAWP. Cardiac output is affected mainly by a reduction in stroke volume, secondary to a reduction in preload and an increase in afterload. This is further aggravated by hypovolaemia. Paradoxically, in the presence of hypovolaemia, an increase in IAP can be temporarily associated with an increase in cardiac output. It has been identified that venous stasis occurs in the legs of patients with abdominal pressures < 12 mmHg. In addition, recent studies of patients undergoing laparoscopic cholecystectomy show up to a fourfold increase in renin and aldosterone levels.

### 3.5.5.3 Respiratory

In association with increased IAP, there is diaphragmatic stenting, which exerts a restrictive effect on the lungs with reduction in ventilation, decreased lung compliance, increase in airway pressures and reduction in tidal volumes.

In critically ill ventilated patients, the effect on the respiratory system can be significant, resulting in reduced lung volumes, impaired gas exchange and high ventilatory pressures. Hypercapnia can occur and the resulting acidosis can be exacerbated by simultaneous cardiovascular depression as a result of raised IAP. The effects of raised IAP on the respiratory system in the ICU can sometimes be life-threatening, requiring urgent abdominal decompression. Patients with true ACS undergoing abdominal decompression demonstrate a remarkable change in their intraoperative vital signs.

### 3.5.5.4 Visceral Perfusion

Interest in visceral perfusion has increased with the popularization of gastric tonometry, and there is an association between IAP and visceral perfusion as measured by gastric pH. This has been confirmed recently in 18 patients undergoing laparoscopy, in whom reduction of between 11 and 54 per cent in blood flow was seen in the duodenum and stomach, respectively, at an IAP of 15 mmHg. Animal studies suggest that reduction in visceral perfusion is selective, affecting intestinal blood flow before, for example, adrenal blood flow. We have demonstrated, in a study of 73 post-laparotomy patients, that IAP and pH are strongly associated, suggesting that early decreases in visceral perfusion are related to levels of IAP as low as 15 mmHg.

### 3.5.5.5 Intracranial Contents

Raised IAP can have a marked effect on intracranial pathophysiology and causes severe increases in intracranial pressure (ICP).

### 3.5.6 Measurement of IAP

The gold standard for IAP measurement involves the use of a urinary catheter. The patient is positioned flat in the bed and a standard Foley catheter is used with a T-piece bladder pressure device attached between the urinary catheter and the drainage tubing. This piece is then connected to a pressure transducer, on-line to the monitoring system. The pressure transducer is placed in the midaxillary line and the urinary tubing is clamped. Approximately 50 mL isotonic saline is inserted into the bladder via a three-way stopcock. After zeroing, the pressure on the monitor is recorded.
Increasingly it is recognized that IAP is not a static measurement and should be measured continuously. In addition, whether IAP is measured intermittently or continuously, consideration for abdominal perfusion measurement should be given.

**ABDOMINAL PERFUSION PRESSURE MEASUREMENT**

Similar to the concept of cerebral perfusion pressure, calculation of the ‘abdominal perfusion pressure’ (APP), which is defined as MAP minus the IAP, assesses not only the severity of IAP present, but also the adequacy of the patient’s abdominal blood flow. APP has been studied as a resuscitation end point in four clinical trials, which demonstrated statistically significant differences in APP between survivors and non-survivors with IAH/ACS. Cheatham et al.,2 in a retrospective trial of surgical and trauma patients with IAH (mean IAP 22 ± 8 mmHg), concluded that an APP > 50 mmHg optimized survival based on receiver-operating characteristic curve analysis. APP was also superior to global resuscitation end points such as arterial pH, base deficit, arterial lactate and hourly urinary output, in its ability to predict patient outcome. Malbrain et al.3–5 in three subsequent trials in mixed medical–surgical patients (mean IAP 10 ± 4 mmHg) suggested that 60 mmHg represented an appropriate resuscitation goal. Persistence of IAH and failure to maintain an APP ≥ 60 mmHg by day 3, after development of IAH-induced acute renal failure, was found to discriminate between survivors and non-survivors.

**TIPS FOR IAP MEASUREMENT**

A strict protocol and staff education on the technique and interpretation of IAP are essential. Very high pressures (especially unexpected ones) are usually caused by a blocked urinary catheter.

The volume of saline instilled into the bladder is not critical, but should be ± 50 mL. A CVP manometer system can be used but it is more cumbersome than on-line monitoring. The size of the urinary catheter does not matter. Elevation of the catheter and measuring the urine column provide a rough guide and are simple to perform.

If the patient is not lying flat, IAP can be measured from the pubic symphysis. Real-time continuous monitoring of IAP is effective and shows trends as well as actual pressures.

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**3.5.7 Treatment**

**3.5.7.1 PREVENTION**

To avoid ACS developing in the first place, in the ED, concepts of damage control coupled with adequate pre-hospital information will help identify patients at high risk even before they arrive in the ED.

**3.5.7.2 GENERAL SUPPORT**

There are a number of key principles in the management of patients with potential ACS:

- Regular (< 8-hourly) monitoring of IAP
- Optimization of systemic perfusion and organ function in the patient with IAH
- Institution of specific medical procedures to reduce IAP and the end-organ consequences of IAH/ACS
- Early surgical decompression for refractory IAH.

**3.5.7.3 REVERSIBLE FACTORS**

The second aspect of management is to correct any reversible cause of ACS, such as intra-abdominal bleeding. Massive retroperitoneal haemorrhage is often associated with a fractured pelvis and consideration should be given to measures that would control haemorrhage such as pelvic fixation or vessel embolization. In some cases, severe gaseous distension or acute colonic pseudo-obstruction can occur in ICU patients. This may respond to drugs such as neostigmine but, if it is severe, surgical decompression may be necessary. A common cause of raised IAP in ICU is related to the ileus. There is little that can be actively done in these circumstances, apart from optimizing the patient’s cardiorespiratory status and serum electrolytes.

Remember that the ACS is often only a symptom of an underlying problem. Abdominal evaluation for sepsis is a priority and this obviously should include a rectal examination, as well as investigations such as ultrasonography and CT. Surgery is the obvious mainstay of treatment in patients whose rise in IAP is the result of postoperative bleeding.

**3.5.8 Surgery for raised IAP**

As yet, there are few guidelines for exactly when surgical decompression is required in the presence of raised IAP. Some studies have stated that abdominal
decompression is the only treatment and that it should be performed early in order to prevent ACS. This is an overstatement and not supported by level I evidence.

The indications for abdominal decompression are related to correcting pathophysiological abnormalities, as much as achieving a precise and optimum IAP.

Indications for performing TAC include:

- Abdominal decompression
- Facilitate re-exploration in abdominal sepsis
- Inability to close the abdomen
- Prevention of ACS.

A large number of different techniques have been used to facilitate a temporary abdominal closure, including intravenous bags, Velcro, silicone and zips. Whatever technique is used, it is important that effective decompression be achieved with adequate incisions.

3.5.8.1 Tips for surgical decompression for raised IAP

- Early investigation and correction of the cause of raised IAP
- Ongoing abdominal bleeding with raised IAP requires urgent operative intervention
- Reduction in urinary output is a late sign of renal impairment. Gastric tonometry may provide earlier information on visceral perfusion
- Abdominal decompression requires a full-length abdominal incision
- The surgical dressing should be closed using a sandwich technique by use of two suction drains placed laterally to facilitate fluid removal from the wound
- If the abdomen is very tight, pre-closure with a Silo bag should be considered.

Unfortunately, clinical infection is common in the open abdomen and the infection is usually polymicrobial. Particular care needs to be taken in patients undergoing post-aortic surgery because the aortic graft may become colonized. The mesh in this situation should be removed and the abdomen left open. It is desirable to close the abdominal defect as soon as possible. This is often not possible as a result of persistent tissue oedema.

3.5.9 Future

The concept of IAP measurement and its significance are increasingly important in the ICU and are rapidly becoming part of routine care. Patients with raised IAP require close and careful monitoring, aggressive resuscitation and a low index of suspicion for requirement of surgical abdominal decompression.

The formation of the World Society on ACS (2007) has been a major advance with production of consensus definitions, formation of a research policy, with multicentre trials, and the publishing of the consensus guidelines on ACS.

3.5.10 References


3.5.11 Recommended reading

Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM Intra-abdominal hypertension after life-threatening penetrating...


3.6 CLOSURE OF THE ABDOMEN

3.6.1 Objectives

- Understanding the general principles of abdominal closure
- Understanding the various techniques and indications for their use
- Understanding the indications for reoperation or ‘re-laparotomy’ (‘second-look laparotomy’).

3.6.2 Introduction: general principles of abdominal closure

In most cases, the standard and most efficient means of access to the injured abdomen is through a vertical (midline) laparotomy. On completion of the intra-abdominal procedures, it is important to prepare for the closure adequately. This preparation includes:

- Careful evaluation of adequacy of haemostasis and/or packing
- Copious lavage and removal of debris within the peritoneum and wound
- Placement of adequate and appropriate drains if indicated
- Ensuring that the instrument and swab counts are completed and correct.

It is worth remembering the principles of wound closure, the most important of which is avoidance of undue tension in the tissues of the closure.

3.6.3 Choosing the optimal method of closure

Thal and O'Keefe¹ state that the optimal closure technique is chosen on the basis of five principal considerations and list these as follows:

1. The stability of the patient (and therefore the need for speed of closure)
2. The amount of blood loss both before and during operation
3. The volume of intravenous fluid administered
4. The degree of intraperitoneal and wound contamination
5. The nutritional status of the patient and possible intercurrent disease.

These factors will also dictate the decision to plan for a re-laparotomy, which will naturally influence the method chosen for closure.

3.6.4 Techniques for closure

The most commonly used technique at present is that of mass closure of the peritoneum and sheath using a monofilament suture with a continuous (preferable because relatively quick) or an interrupted (discontinuous) application. Either absorbable material (e.g. 1-polydioxanone loop) or non-absorbable material (e.g. nylon) is used. Chromic catgut is not a suitable material.

No difference is found to exist between the two methods with respect to rate of dehiscence, but the extent of disruption is generally more limited with the interrupted technique.

3.6.4.1 PRIMARY CLOSURE

Primary closure of the abdominal sheath (or fascia), subcutaneous tissue and skin is obviously the desirable goal, and may be achieved when the conditions outlined above are optimal, i.e. a stable patient with minimal
blood loss and volume replacement, no or minimal contamination, no significant intercurrent problems and a patient in whom surgical procedures are deemed to be completed with no anticipated subsequent operation.

Should any reasonable doubt exist regarding these conditions at the conclusion of operation, it would be prudent to consider a technique of delayed closure.

Whichever method is used, the most important technical point is that of avoiding excessive tension on the tissues of the closure.

Retention sutures should be avoided at all costs and a wound that seems to require these is not suitable for primary closure.

Remember the ‘1 cm–1 cm’ rule as described by Leaper et al.2 (the so-called Guildford technique). This spacing seems to minimize tension in the tissues, thus also minimizing the compromise of circulation in the area and using the minimum acceptable amount of suture.

Skin closure as a primary manoeuvre may be done in a case with no or minimal contamination, using monofilament sutures or staples. These have the advantage of speed and, although being less haemostatic, nevertheless allow for a greater degree of drainage past the skin edges and less tissue reaction.

3.6.4.2 TEMPORARY ABDOMINAL CLOSURE (TAC)

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

Under these circumstances, a TAC is required. The needs of such a closure can be summarized as:

- Quick to do
- Cheap
- Keeps abdominal contents inside the abdominal cavity
- Allows drainage of fluid
- Minimizes sepsis.

The ‘sandwich technique’ was first described by Schein et al.3 in 1986 and popularized by Rotondo et al.4

A sheet of self-adhesive incise drape (Opsite, Steridrape or Ioban) is placed flat, and an abdominal swab placed on it. The edges are folded over, to produce a sheet with membrane on one side and abdominal swab on the other. If extra firmness is required, an abdominal drape can be used.

The membrane is used as an on-lay with the margins ‘tucked in’ under the edges of the open sheath, with the membrane in contact with the bowel.

The appreciable drainage of serosanguineous 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 onto the membrane and using continuous low-vacuum suction.

This arrangement is covered by an occlusive incise drape applied to the skin, thus providing a closed system (Figure 3.2).

An acceptably cheap and readily available membrane-type material is provided by use of a sterile polyethylene intravenous infusion bag cut open – the ‘Bogota bag’.

3.6.5 Re-laparotomy

The circumstances under which re-laparotomy is undertaken have been touched on above. It is, however, worthwhile recollecting that the ‘second look’ may be:

- Planned, i.e. 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, i.e. when evidence of intra-abdominal complication develops. In these cases, the principle applies of reoperation ‘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.

At present, and despite decades of recognition of the importance to outcome of these considerations, the decision to plan re-laparotomy or to re-explore upon demand often remains a subjective clinical judgement. There is thus ongoing work to attempt to define more accurately and establish standardized objective criteria for this important surgical decision-making process.

Studies to evaluate the significance of recognized quantifiable variables such as age, co-morbidities, mechanism of injury, time from injury to operation, initial (presenting) blood pressure, number of intra-abdominal injuries, associated extra-abdominal injuries, requirements for crystalloid and blood transfusion, and the Penetrating Abdominal Trauma Index (PATI – see Appendix B3.5) score is currently under way.

### 3.6.6 Delayed closure

Delayed closure will be required once the reasons for the temporary surgery have been removed or treated. This is usually undertaken by secondary suturing after an interval of 72 h (or longer).

The use of laterally placed, relaxing, ‘counter-incisions’ should seldom be necessary to achieve an acceptable degree of approximation with this technique of temporary closure.

It is expected that virtually all patients in whom such temporary closures are used will undergo re-exploration with subsequent definitive sheath closure. Should a mesh be used and left in situ, however, the resulting defect will require skin coverage by split grafting or flap transfer.

Delayed primary closure is usually possible but, under other circumstances, an inlay will be required.

Non-absorbable meshes (Marlex and Mersilene): both allow tissue in-growth, but with the disadvantages of predisposing to local sepsis and enteric fistulization when employed as a direct on-lay onto the bowel.

Absorbable mesh of polyglycolic (e.g. Vicryl) acid and membranes (PTFE [polytetrafluoroethylene] or Gore-Tex): elicit minimal tissue reaction and in-growth and thus the risk of infection or fistula formation, but are considerably more costly. Recently, composite meshes have shown promise. The mesh can then have a skin graft placed on it (or even directly on bowel), and definitive abdominal wall reconstruction can take place at a later stage.

### 3.6.7 References


### 3.6.8 Recommended reading


Part 4

Specific Organ Injury
4.1 OVERVIEW

4.1.1 Introduction

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

4.1.2 Management principles

Current management of penetrating cervical injuries depends on several factors.

4.1.2.1 INITIAL ASSESSMENT

Patients with signs of significant neck injury will require prompt exploration. However, initial assessment and management of the patient should be carried out according to Advanced Trauma Life Support (ATLS®) principles.

The major initial concern in any patient with a penetrating neck wound is early control of the airway. 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.

A characteristic of neck injuries is rapid airway obstruction and often difficult intubation. The key to management is early intubation, which has the above-mentioned complications. The route of intubation must be carefully considered in these patients because it may be complicated by distortion of anatomy, haematoma, dislodging of clots, laryngeal trauma and a significant number of cervical spinal injuries.

Note that use of paralysing agents in these patients is contraindicated, because the airway may be held open only by the patient’s 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 caused by 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.

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 because a clot may be dislodged. If the wound is actively bleeding, the bleeding should be controlled by digital pressure or, as a last resort, by a Foley catheter.

Patients with signs of significant neck injury, or who are unstable, should be explored urgently once rapid initial assessment has been completed and the airway secured. There should be no hesitation in performing an emergency cricothyroidotomy should circumstances warrant it. Tracheostomy should be considered as a planned procedure in the operating theatre. Cricothyroidotomies should be converted to formal tracheostomies within about 48 h.

4.1.2.2 INJURY LOCATION

Division of the neck into anatomical zones (Figure 4.1) helps in the categorization and management of neck wounds.

Zone I extends from the bottom of the cricoid cartilage to the clavicles and thoracic outlet. Within zone I lie the great vessels, trachea, oesophagus, thoracic duct, and 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 and the distal extracranial carotid and vertebral arteries, as well as segments of the jugular veins.

Injuries in zone II are readily evaluated and easily exposed operatively. Adequate exposure of injuries in zone I or III can be difficult, so the diagnostic work-up may be more extensive than for zone II injuries.

4.1.2.3 MECHANISM

Gunshot wounds carry a higher risk of major injury than stab wounds because of their tendency to penetrate deeper and their ability to damage tissue outside the tract of the missile as a result of cavitation.

4.1.2.4 FREQUENCY OF INJURY

The carotid artery and internal jugular vein are the most frequently injured vessels. As a result of its relatively protected position, the vertebral artery is involved less frequently. The larynx and trachea, and the pharynx and oesophagus, are frequently injured, whereas the spinal cord is involved less often.

4.1.3 Mandatory versus selective neck exploration

Recommendations for 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 simply observed. Mandatory exploration for penetrating neck injury in patients with hard signs of vascular or aerodigestive tract injury is still appropriate, however.

Mandatory exploration of all stable patients is controversial. Significant morbidity and mortality caused by missed visceral injuries, as well as the negligible morbidity caused by negative exploration, are reasons to operate on all patients with penetrating wounds. However, exploration for all stab wounds of the neck may yield a high rate of negative findings. Thus, selective management of penetrating neck wounds with thorough non-surgical evaluation has been recommended. It is clear that missed injuries are associated with a high morbidity and mortality.

In the stable patient with a wound that penetrates the platysma, either mandatory exploration or non-surgical evaluation (angiography, oesophagography, bronchoscopy and thorough endoscopy) is appropriate.

4.1.4 Use of diagnostic studies

In the stable patient with no indications for immediate neck exploration, additional studies are often obtained, including angiography, endoscopy, contrast radiography and bronchoscopy. (A few recent studies have even suggested that asymptomatic patients can be observed safely by serial examination, but this is a highly selective approach.)

4.1.4.1 COMPUTED TOMOGRAPHY (CT) WITH CONTRAST/CT ANGIOGRAPHY

Modern 16–64 slice CT scanners have provided a means of creating a three-dimensional image of high quality. Use of contrast has allowed CT angiography. Computed tomography is now the investigation of choice for penetrating injury in the stable patient.

4.1.4.2 ANGIOGRAPHY

This should be used especially in zone I or III injuries, where surgical exposure can be difficult. Angiography is invaluable to plan the conduct of operation. It should visualize both internal and external carotid arteries on both sides (‘four-vessel angiography’) as well as the vertebral arteries on both sides.
Using a selective approach to 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 in close proximity to the major vessels.

4.1.4.3 OTHER DIAGNOSTIC STUDIES

The selective management of penetrating neck wounds involves evaluation of the oesophagus, larynx and trachea. Either contrast oesophagography or oesophagoscopy alone will detect 60 per cent of oesophageal injuries. The two tests used together increase diagnostic accuracy to almost 90 per cent. Laryngoscopy and bronchoscopy are useful adjuncts in localizing or excluding injury to the hypopharynx or trachea.

4.1.5 Treatment based on anatomical zones

4.1.5.1 VASCULAR INJURIES

If associated injuries allow, a bolus of 5000–10 000 units heparin should be given before any of the arteries in the neck are occluded. As they have no branches, the common and internal carotid arteries can be safely mobilized for some distance from the injury to ensure a tension-free repair.

All patients with zone I and III neck injuries should undergo angiography as soon as vital signs are stable. 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 III injuries require angiography because of the relationship of the blood vessels to the base of the skull. Often these injuries can best be managed by either non-surgical techniques or manoeuvres remote from the injury site such as balloon tamponade or embolization. Injuries in zone II may require angiography if vertebral vessels are thought to be injured. However, angiography will not rule out significant injury to the venous system, trachea or oesophagus.

The patient is placed in the supine position on the operating table with the arms tucked at the side. Active bleeding from a penetrating wound should be controlled digitally. However, 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. If possible, the head should be extended and rotated to the contralateral side. A sandbag may be placed between the shoulder blades.

Zone III injuries, at the very base of the skull, are complex and should be explored with great care. Access is often extremely difficult. On rare occasions it may not be possible to control the distal stump of a high internal carotid artery injury. Although techniques for mandibular dislocation may be helpful, bleeding from this injury can be controlled both temporarily and permanently by inserting a Fogarty catheter into the distal segment. The catheter is secured, transected and left in place. It may be necessary to control the internal carotid artery from within the cranial cavity.

Zone II injuries are explored by an incision made along the anterior border of the sternocleidomastoid muscle as for carotid endarterectomy. An extended collar incision or bilateral incisions along the anterior edge of the sternocleidomastoid muscles may be used for wounds that traverse both sides of the neck. Proximal and distal control of the blood vessel is obtained. If the vessel is actively bleeding, direct pressure is applied to the bleeding site while control is obtained. Use of anticoagulation is optional. If there are no injuries that preclude its use, heparin may be given in the management of carotid injuries. Vascular shunts are rarely needed in patients with carotid injuries, especially if the distal clamp is applied proximal to the bifurcation of the internal and external carotid arteries. Repair techniques for cervical trauma do not differ significantly from those used for other vascular injuries:

- The intraoperative decisions are influenced by the patient's preoperative neurological status. If the patient has no neurological deficit preoperatively, the injured vessel should be repaired. (The one exception may be if complete obstruction of blood flow is found at the time of surgery, because restoration of flow may cause distal remobilization or haemorrhagic infarction.)
- Surgical management of the patient with a carotid injury and a preoperative neurological deficit is controversial. Vascular reconstruction should be performed in patients with mild-to-moderate deficits in whom retrograde flow is present. Ligation is recommended for patients with severe preoperative neurological deficits more than 48 h old, and without evidence of retrograde flow at the time of operation.
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 may often 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 surgery for definitive repair may require a sternotomy, or extension into the right side of the chest or up into the neck. Trapdoor incisions are not recommended, because they are often difficult to perform and do not significantly improve the exposure, although they significantly increase the postoperative disability.

Care must be taken to 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 angiography, the right subclavian artery or the distal two-thirds of the left subclavian artery can be exposed through an incision immediately superior to the clavicle. The clavicle can be divided at its midpoint, or occasionally it may be necessary to resect the medial half of the clavicle. This is rarely necessary:

- Injuries to the internal jugular vein should be repaired if possible. In severe injuries that require extensive debridement, ligation is preferred. Venous interposition grafts should not be performed.
- Vertebral artery injuries are generally found to have been injured only on angiographic study. These rarely require surgical repair, because they are best dealt with by angioembolization. Operative exposure may be difficult and require removal of the vertebral lamina in order to access it for ligation. If a vertebral artery injury is found at operation, the area around the injury should be packed. If this tamponades the bleeding, the patient should be transferred from the operating room to the radiology suite for angiography and embolization of the vertebral artery.

As with all vascular injuries of the neck, a useful adjunct is to have a size 3 or 4 Fogarty catheter available. This can be passed up into the vessel to obtain temporary occlusive control.

There is increasing support for treatment of carotid artery injuries with intraluminal stenting. This approach is now commonly used for central as well as for more distal peripheral vascular lesions. It has also been applied to the management of traumatic injuries to the thoracic aorta and selected injuries to the peripheral and visceral vasculature. Not surprisingly, stenting has also been used for the management of carotid artery injuries, particularly injuries to the distal internal carotid artery that are not easily approached surgically. Overall, stents are most frequently used in situations where arterial lesions are not surgically accessible or when anticoagulation is contraindicated.

### 4.1.5.2 Tracheal Injuries

Injuries to the trachea should be closed in a single layer with absorbable sutures. Larger defects may require a fascia flap. These injuries should be drained.

### 4.1.5.3 Pharyngeal and Oesophageal Injuries

Oesophageal injuries are often missed at neck exploration. Injuries to the hypopharynx and cervical oesophagus may also be difficult to diagnose preoperatively. Perforations of the hypopharynx or oesophagus should be closed in two layers and widely drained. For devastating oesophageal injuries requiring extensive resection and debridement, a cutaneous oesophagostomy for feeding, and pharyngostomy for diversion, may be necessary.

### 4.1.6 Rules

The first concern in the patient with a penetrating injury of the neck is early control of the airway. The next concern is to stop bleeding, by either digital pressure or the use of a Foley catheter. The stability of the patient decides the appropriate diagnostic and treatment priorities. Never make the operation more difficult than necessary by inadequate exposure. Adequate exposure of the area involved is critical.

### 4.1.7 Recommended reading


4.2 ACCESS TO THE NECK

The surgical 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.

4.2.1 Incision

Always expect the worst, and plan the incision to provide optimal access for early proximal vascular control or immediate access to the airway. The most universally applicable approach is via an anterior sternomastoid incision, which can be lengthened proximally and distally, extended to a median sternotomy or augmented with lateral extensions. The patient is positioned supinely with a bolster between the shoulders, the neck extended and rotated away – provided that the cervical spine has been cleared preoperatively. The face, neck and anterior chest should be prepped and draped widely.

The incision is made along the anterior border of the sternocleidomastoid muscle and carried through the platysma into the investing fascia. The muscle is freed and retracted laterally to expose the fascial sheath covering the internal jugular vein. Lateral retraction of the jugular vein and underlying carotid 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.

4.2.2 Carotid

Exposure of the carotid is obtained by ligating the middle thyroid and common facial veins and retracting the internal jugular laterally, together with the sternocleidomastoid. The vagus nerve posterior in the carotid sheath, and the hypoglossal nerve anteriorly, must be preserved. The occipital artery and inferior branches of the ansa cervicalis may be divided. To expose the carotid bifurcation, the dissection is carried upwards to the posterior belly of the digastric muscle, which is divided behind the angle of the jaw. Access to the internal carotid can be improved by dividing the sternocleidomastoid muscle near its origin at the mastoid.

Care must be taken not to injure the accessory nerve where it enters the sternomastoid muscle 3 cm below the mastoid, and the glossopharyngeus nerve crossing anteriorly over the internal carotid artery. More distal exploration of the internal carotid artery may require unilateral mandibular subluxation or division of the ascending ramus. The styloid process may be excised after division of the stylohyoid ligament, and styloglossus and stylopharyngeus muscles. The facial nerve lies superficial to these muscles and must be preserved. To reach the internal carotid where it enters the carotid canal, part of the mastoid bone can be removed. Fortunately, this is rarely required. Figures 4.2 and 4.3 show the surgical approach to the neck.

The proximal carotid artery is exposed by division of the omohyoid muscle between the superior and inferior bellies. More proximal control may require a midline sternotomy.

4.2.3 Midline visceral structures

The trachea, oesophagus and thyroid are approached by retracting the carotid sheath laterally. The inferior thyroid artery should be divided laterally near the carotid

![Figure 4.2 Approach to the left side of the neck with divided sternomastoid and digastric muscles.](image-url)
and the thyroid lobe is lifted anteriorly to expose the trachea and oesophagus posteriorly.

Oesophageal identification is aided by passing a large dilator or nasogastric tube. The recurrent laryngeal nerves should be carefully preserved: the left nerve runs vertically in the tracheo-oesophageal groove, but the right nerve runs obliquely across the oesophagus and trachea from inferolateral to superomedial. Both nerves are at risk of injury with circumferential mobilization of the oesophagus. Bilateral exposure of the midline structures may require transverse extension of the standard incision.

### 4.2.4 Root of the neck

The structures at the root of the neck can be approached by extending the incision laterally above the clavicle. The clavicular head of the sternocleidomastoid is divided and the supraclavicular fat pad is cleared by blunt dissection. This reveals the scalenus anterior muscle, with the phrenic nerve crossing it from the lateral side. Division of scalenus anterior, with preservation of the phrenic nerve, allows access to the second part of the subclavian artery. The distal subclavian can be exposed by dividing the clavicle at its midpoint, and dissecting away the subclavius muscle and fascia. The clavicle should not be resected because this leads to considerable morbidity. To fix the divided bone, the periosteum should be approximated with strong polyfilament absorbable sutures.

### 4.2.5 Collar incisions

Horizontal or “collar” incisions placed either over the thyroid or higher up over the thyroid cartilage are useful to expose bilateral injuries, or injuries limited to the larynx or trachea. The transverse incision is carried through the platysma, and sub-platysmal flaps are then developed: superiorly up to the thyroid cartilage notch and inferiorly to the sternal notch. The strap muscles are divided vertically in the midline and retracted laterally to expose the fascia covering the thyroid. The thyroid isthmus can be divided to expose the trachea. A high collar incision, placed over the larynx, is useful for repairing isolated laryngeal injuries.

### 4.2.6 Vertebral arteries

The proximal part of the vertebral artery is approached via the anterior sternomastoid incision, with division of the clavicular head of the sternomastoid. The internal jugular vein and common carotid artery are mobilized and the vein is retracted medially, and the artery and nerve are retracted laterally. The proximal vertebral artery lies deeply between these structures. The vertebral artery is crossed by branches of the cervical sympathetic chain and, on the left side, by the thoracic duct. The inferior thyroid artery crosses in a more superficial plane just before the vertebral artery enters its bony canal.

Access to the distal vertebral artery is challenging and rarely needed. The contents of the carotid sheath are retracted anteromedially and the prevertebral muscles longitudinally split over a transverse process above the level of the injury. The anterior surface of the transverse process can be removed with a small rongeur, or a J-shaped needle may be used to snare the artery in the space between the transverse processes.

The most distal portion of the vertebral artery can be approached between the atlas and the axis after division...
of the sternocleidomastoid near its origin at the mastoid process. The prevertebral fascia is divided over the transverse process of the atlas. With preservation of the C2 nerve root, the levator scapulae and splenius cervicis muscles are divided close to the transverse process of the atlas.

The vertebral artery can now be visualized between the two vertebrae, and may be ligated with a J-shaped needle. Neck exploration wounds are closed in layers after acquiring homoeostasis. Drainage is usually indicated, mainly to prevent haematomas and sepsis.
5.1 OVERVIEW

5.1.1 Objectives

To familiarize the practitioner with:

- The spectrum and types of thoracic injury
- The pathophysiology associated with thoracic injury
- The applied surgical anatomy of the thorax
- The surgical approaches to the thorax and the applied techniques.

5.1.2 Introduction: the scope of the problem

Thoracic injury constitutes a significant problem in terms of mortality and morbidity. In the USA during the early 1990s, there were approximately 180,000 deaths per annum from injury. Several investigators have shown that 50 per cent of fatal injuries are the result of primary brain injury, 25 per cent of fatal accidents are caused by chest trauma and, in another 25 per cent (including brain injury), thoracic injury contributes to the primary cause of mortality.\(^1\)

Somewhat less clearly defined is the extent of appreciable morbidity after chest injury, most usually the long-term consequences of hypoxic brain damage. There are a number of important points to be made in this regard:

- A definite proportion of these deaths occur virtually immediately (i.e. at the time of injury), e.g. rapid exsanguination after traumatic rupture of the aorta in blunt injury or major vascular disruption after penetrating injury.
- Of survivors with thoracic injury who reach hospital, a significant proportion die in hospital as the result of misassessment or delay in the institution of treatment. These deaths occur early as a consequence of shock, or late as the result of adult respiratory distress syndrome (ARDS), multiple organ failure (MOF) and sepsis.
- Most life-threatening thoracic injuries can be simply and promptly treated after identification by needle or tube placement for drainage. These are simple and effective techniques that can be performed by any physician.
- Approximately 40 per cent of penetrating thoracic injuries and 20 per cent of blunt thoracic injuries will require definitive surgery.

Emergency department thoracotomy (ERT) has distinct and specific indications; these virtually always relate to patients in extremis with penetrating injury. Indiscriminate use of ERT will not alter the mortality and morbidity, but will increase the risk of communicable disease transmission to health workers.

Injuries to the chest wall and thoracic viscera can directly impair oxygen transport mechanisms. Hypoxia and hypovolaemia resulting from thoracic injuries may cause secondary injury to patients with brain injury, or may directly cause cerebral oedema.

Conversely, shock and/or brain injury can secondarily aggravate thoracic injuries and hypoxaemia by disrupting normal ventilatory patterns or causing loss of protective airway reflexes and aspiration.

The lung is a target organ for secondary injury following shock and remote tissue injury. Microemboli formed in the peripheral microcirculation embolize to the lung causing ventilation-perfusion mismatch and right heart failure. Tissue injury and shock can activate the inflammatory cascade, which can contribute to pulmonary injury (reperfusion).

5.1.3 The spectrum of thoracic injury

Thoracic injuries are grouped into two types.
5.1.3.1 IMMEDIATELY LIFE-THREATENING INJURIES

- Airway obstruction resulting from any cause but specifically from laryngeal or tracheal disruption with obstruction or extensive facial bony and soft tissue injuries
- Impaired ventilation caused by tension pneumothorax, open pneumothorax or flail chest
- Impaired circulation caused by massive haemothorax or pericardial tamponade
- Air embolism.

5.1.3.2 POTENTIALLY LIFE-THREATENING INJURIES

- Blunt cardiac injury (previously termed ‘myocardial contusion’)
- Pulmonary contusion
- Traumatic rupture of the aorta (TRA)
- Traumatic diaphragmatic herniation (TDH)
- Tracheobronchial tree disruption
- Oesophageal disruption
- Simple haemothorax
- Simple pneumothorax.

The entity of the ‘traversing mediastinal wound’ in penetrating injury warrants specific mention. Injuries of this type frequently involve damage to a number of the mediastinal structures and are thus more complex in their evaluation and management.

5.1.4 Pathophysiology of thoracic injuries

The well-recognized pathophysiological changes occurring in patients with thoracic injuries are essentially the result of:

- Impairment of ventilation
- Impairment of gas exchange at the alveolar level
- Impairment of circulation caused by haemodynamic changes.

The approach to the patient with thoracic injury must therefore take all these elements into account.

Specifically, hypoxia at cellular or tissue level results from inadequate delivery of oxygen to the tissues with the development of acidosis and associated hypercapnia. The late complications resulting from misassessment of thoracic injuries are directly attributable to these processes.

Many forces can act on the torso to cause injury to the outer protective layers or the contained viscera. Penetrating trauma is most often the result of 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 the result of 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 viscous organ. In contrast, missile injuries can cause more extensive tissue destruction, related to the kinetic energy (KE), which is expressed as:

$$\text{KE} = \frac{1}{2} m v^2$$

where \( m \) = mass, \( v \) = velocity. Perhaps more appropriate is the concept of ‘wounding energy’ (WE), expressed as:

$$\text{WE} = \frac{1}{2} m (v_{\text{EN}} - v_{\text{EX}})^2$$

where \( m \) = mass, \( v_{\text{EN}} \) = velocity on entry and \( v_{\text{EX}} \) = velocity on exit.

Velocity is important in determining final kinetic energy. If the exit velocity is high, very little injury is imparted to the tissue. Thus, bullets are designed so that, on impact, the missile expands or shatters, imparting all of its energy to the tissue. Other characteristics of the missile may contribute to tissue destruction, including yaw, tumble and pitch. It has been appreciated that tumbling may be particularly important in higher-velocity weapons (> 800 m/s). Shotgun blasts can be the most devastating because almost all of the energy is imparted to the tissue.

Penetrating chest injuries should be obvious. Exceptions include small puncture wounds such as those caused by ice picks. Bleeding is generally minimal secondary to the low pressure within the pulmonary system. Exceptions to these management principles include wounds to the great vessels as they exit over the apex of the chest wall to the upper extremities, or injury to any systemic vessel that may be injured in the chest wall such as the internal mammary or intercostal vessels.

Penetrating injuries to the mid-torso generate more controversy. They require a fairly aggressive approach, particularly with anterior wounds. If the wound is between one posterior axillary line and the other, and obviously penetrates the abdominal wall, laparotomy is indicated. If the wound does not obviously penetrate, an option is to explore the wound under local anaesthesia to determine whether or not it has penetrated the peritoneal lining or the diaphragm. If peritoneal penetration has occurred, laparotomy is indicated. Other options include laparoscopy or thoracoscopy to determine whether the diaphragm has been injured.
Patients arrive in two general physiological states:

1. Haemodynamically stable
2. Haemodynamically unstable.

In the patient with penetrating injury to the upper torso who is haemodynamically unstable, and the bleeding is occurring into the chest cavity, it is important to insert a chest tube as soon as possible during the initial assessment and resuscitation. In the patient in extremis who has chest injuries or where there may be suspicion for a transmediastinal injury, bilateral chest tubes may be indicated. A radiograph is not required to insert a chest tube, but is useful after the chest tubes have been inserted to confirm proper placement.

In patients who are haemodynamically stable, a radiograph remains the gold standard for diagnosis of pneumothorax or haemothorax. In these patients it is preferable to have the radiograph completed before placement of a chest tube. The decrease in air entry may not be the result of a pneumothorax, and especially following blunt injury may be caused by a ruptured diaphragm with bowel or stomach occupying the thoracic cavity.

5.1.5 Applied surgical anatomy of the chest

It is useful to broadly view the thorax as a container with an inlet, walls, a floor and contents.

5.1.5.1 THE CHEST WALL (FIGURE 5.1)

This is the bony ‘cage’ constituted by the ribs, thoracic vertebral column and sternum, with the clavicles anteriorly and the scapula posteriorly. The associated muscle groups and vascular structures (specifically the intercostal vessels and the internal thoracic vessels) are the other components.

Remember the ‘safe area’ of the chest. This triangular area is the thinnest region of the chest wall in terms of musculature, and is the area of choice for tube thoracotomy insertion. In this area, there are no significant structures within the walls that may be damaged; however, take note of the need to avoid the intercostal vascular and nerve bundle on the under-surface of the rib.

5.1.5.2 THE CHEST FLOOR

This is formed by the diaphragm with its various openings. This broad sheet of muscle with its large, trefoil-shaped central tendon has hiatuses, through which pass the aorta, the oesophagus, and the inferior vena cava, and is innervated by the phrenic nerves. The oesophageal hiatus also contains both vagus nerves. The aortic hiatus contains the azygos vein and the thoracic duct.

During normal breathing, the diaphragm moves about 2 cm but can move up to 10 cm in deep breathing. During maximum expiration, the diaphragm may rise as high as the fifth intercostal space, so any injury below this space may involve the abdominal cavity.

5.1.5.3 THE CHEST CONTENTS (FIGURE 5.2)

These are:
- The left and right pleural spaces containing the lungs, lined by the parietal and visceral pleurae, respectively
- The mediastinum and its viscera, located in the centre of the chest. The mediastinum itself has anterior, middle, posterior and superior divisions. The superior mediastinum is contiguous with the thoracic inlet and zone 1 of the neck.

Tracheobronchial tree

The trachea extends from the cricoid cartilage at the level of the sixth cervical vertebra, to the carina at the level of the upper border of the fifth thoracic vertebra, where it bifurcates. The right main bronchus is shorter, straighter and at less of an angle compared with the left
The chest

The mediastinum

Superior

Anterior

Middle

Posterior

The chest | 77

side. It lies just below the azygos–superior vena caval junction and behind the right pulmonary artery.

Lungs and pleurae

The right lung constitutes about 55 per cent of the total lung mass and has oblique and transverse fissures that divide it into three lobes. The left lung is divided into upper and lower lobes by the oblique fissure. Both lungs are divided into bronchopulmonary segments corresponding to the branches of the lesser bronchi, and are supplied by branches of the pulmonary arteries. The right and left pulmonary arteries pass superiority in the hilum, anterior to each respective bronchus. There are superior and inferior pulmonary veins on each side, the middle lobe usually being drained by the superior vein.

The pleural cavities are lined by parietal and visceral pleura. The parietal pleura lines the inner wall of the thoracic cage, and the visceral pleura is intimately applied to the surface of the lungs and reflected onto the mediastinum to join the parietal pleura at the hilum.

Heart and pericardium

The heart lies in the middle mediastinum, extending from the level of the third costal cartilage to the xiphisternal junction. Most of the anterior surface of the heart is represented by the right atrium and its auricular appendage superiorly, and the right ventricle inferiorly. The aorta emerges from the cranial aspect and crosses to the left as the arch. The pulmonary artery extends cranially and bifurcates in the concavity of the aortic arch. The left pulmonary artery is attached to the concavity of the arch of the aorta, just distal to the origin of the left subclavian artery, by the ligamentum arteriosum. The pericardium is a strong fibrous sac that completely invests the heart and is attached to the diaphragm inferiorly. Pericardial tamponade can be created by < 50 mL and up to > 200 mL of blood.

The aorta and great vessels

The thoracic aorta is divided into three parts: the ascending aorta, arch and descending aorta. The innominate artery is the first branch from the arch, passing upwards and to the right, posterior to the innominate vein. The left common carotid artery and left subclavian artery arise from the left side of the arch.

Oesophagus

The oesophagus, approximately 25 cm long, extends from the pharynx to the stomach. It starts at the level of the sixth cervical vertebra and passes through the diaphragm about 2.5 cm to the left of the midline at the level of the eleventh thoracic vertebra. The entire intrathoracic oesophagus is surrounded by loose areolar tissue, which allows for rapid spread of infection if the oesophagus is breached.

Thoracic duct

The duct arises from the cysterna chyli overlying the first and second lumbar vertebrae. It lies posteriorly and to the right of the aorta and ascends through the oesophageal hiatus of the diaphragm between the aorta and the azygos vein, anterior to the right intercostal branches from the aorta. It overlies the right side of the vertebral bodies, and injury can result in a right-sided chylothorax. It drains into the venous system at the junction of the left subclavian and internal jugular veins.

From a functional and practical point of view, it is useful to regard the chest in terms of a 'hemithorax and its contents', both in evaluation of the injury and in choosing the option for access. Figures 5.3 and 5.4 illustrate the hemithoraces and their respective contents.
5.1.6 Paediatric considerations

- In children, the thymus may be very large and care should be taken to avoid damage to it.
- The sternum is relatively soft and can be divided using a pair of heavy scissors.
- Intercostal drains should be tunnelled subcutaneously to facilitate later removal. The child may not cooperate with a Valsalva manoeuvre and pressure on the tract may prevent iatrogenic pneumothorax.

5.1.7 Diagnosis

Penetrating injuries to the chest may be clinically obvious. It is, however, important to log roll the patient to make sure that the entire back has been examined. Log rolling is just as important in patients with penetrating trauma as it is in blunt trauma, until injuries to the thoracic or lumbar spine have been ruled out. 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 or not 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 per cent of the time. Experience with forensic pathology is required to be more accurate.

The surgeon should auscultate each hemithorax, noting whether there are diminished or absent breath sounds. Whenever possible, a chest radiograph is obtained early in the patient with penetrating injuries. This is the key diagnostic study and, in most instances, it will reveal the presence of a pneumothorax and haemothorax. Furthermore, missiles may often leave metallic fragments outlining the path of the bullet. Areas of pulmonary contusion are also indicators of the missile tract. 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 the surgeon in determining the missile track. It can also be useful for stab wounds. Tracking the missile helps the surgeon determine which visceral organs may be injured and, in particular, whether or not there is potential transgression of the diaphragm and/or mediastinum.

Ultrasonography has a role primarily in determining whether or not a patient has intra-pericardial blood.
Using the FAST (focused abdominal sonography for trauma) technique, this blood can be detected. Similarly, transoesophageal echocardiography is a useful adjunct in determining whether tamponade is present in the haemodynamically stable patient. Computed tomography (CT) is not routinely used in patients with penetrating chest injury, except in stable patients with transmediastinal wounds. It might have some use in determining the extent of pulmonary contusion caused by higher-velocity injuries or shotgun blasts, but is not generally indicated in the initial resuscitation or treatment. Arteriography can be quite useful in the haemodynamically stable patient with penetrating injuries to the thoracic outlet or upper chest. This can detect arteriovenous fistulas and false aneurysms.

5.1.8 Management

5.1.8.1 CHEST DRAINAGE

Chest tubes are placed according to the technique described in the ATLS® programme. The placement is in the anterior axillary line, via the fifth intercostal space. Care must be taken to avoid placement of the drain through breast tissue, or the pectoralis major muscle.

In the conscious patient, a weal of 1 per cent lidocaine is placed on the skin, followed by a further 5–10 mL subcutaneously, and down to the pleura. Adequate local anaesthesia is very important. The chest is prepped and draped in the usual way and after topical analgesia an incision is made over about 2 cm in length, on to the underlying rib. Using blunt dissection, the tissue is lifted upwards, off the rib, and penetration is made over the top of the rib towards the pleura.

In this manner, damage to the intercostal neurovascular bundle is avoided. Once the incision has been made the wound is explored with the index finger in adults and the fifth finger in children. This ensures that the chest cavity has been entered and also allows limited exploration of the pleural cavity. Once the tract has been dilated with the finger a large (34 or 36 F) chest tube is inserted directed upwards and towards the posterior gutter. This provides optimal drainage of both blood and air. When the chest tube is in place it is secured to the chest wall with a size 0 monofilament suture and appropriate bandage and tape.

All connectors are taped to prevent inadvertent disconnection or removal of the chest tube. After the chest tube has been placed, it is prudent to obtain an immediate chest film to assess the adequate removal of air and blood and the position of the tube. If for any reason blood accumulates and cannot be removed, another chest tube is inserted. Persistent air leak or bleeding should alert the surgeon that there is significant visceral injury that may require surgical intervention. The surgeon should be aware that the blood may be entering the chest via a hole in the diaphragm.

Complications of tube thoracostomy include wound tract infection and empyema. With meticulous aseptic techniques the incidence of both these should be well under 1 per cent. Routine antibiotics are not a substitute for good surgical technique.

5.1.8.2 NON-OPERATIVE MANAGEMENT

As noted above, non-operative management can be used in most penetrating injuries. These patients should be observed in a monitored setting to ensure haemodynamic stability, monitoring of ventilatory status and output of blood from the pleural cavity.

Non-surgical management of mid-torso injuries is problematic until injury to the diaphragm or abdominal viscera has been ruled out. Thoracoscopy and laparoscopy have been successful in diagnosing diaphragm penetration. Laparoscopy may have a small advantage in that, if the diaphragm has been penetrated, it also allows some assessment of intraperitoneal viscera. It should be noted, however, that in some studies up to 25 per cent of penetrating injuries to hollow viscous organs have been missed at laparoscopy. In many ways thoracoscopy is better for assessment of the diaphragm, particularly in the right hemithorax. The disadvantage is that once an injury has been detected it does not rule out associated intraperitoneal injuries.

Failures of non-operative management include patients who continue to bleed from the pleural cavity and those patients who go on to develop a clotted thorax. If placement of additional chest tubes does not remove the thoracic clots, thoracoscopy is indicated to aid in the removal of these clots. Optimally, this should be done within 72 h of injury before the clot becomes too adherent to be safely removed by thoracoscopy.

5.1.8.3 OPERATIVE MANAGEMENT

In general, patients who have penetrating injuries to the torso should be left in the supine position in the operating room. The importance of this cannot be
overemphasized. The surgeon must be prepared to extend incisions up into the neck or along the supraclavicular area if there are thoracic outlet injuries. Similarly, once it has been determined that the diaphragm has been penetrated or there are associated injuries to the lower torso, it is important for the patient not be in a lateral decubitus position that would compromise exploration of the peritoneal cavity or pelvis. The surgeon must be comfortable in dealing with injuries on both sides of the diaphragm.

The trauma patient must be prepared and the drapes positioned over a large area so that the surgeon can expeditiously gain access to any body cavity and properly place drains and chest tubes. The entire anterior portion and both lateral aspects of the torso should be prepared with antiseptic solution and draped so that the surgeon can work in a sterile field from the neck and clavicle above to the groins below, and table top to table top laterally. Prepping should not involve more than a few minutes and is preferably carried out before induction of anaesthesia so that, if deterioration should occur, immediate laparotomy or thoracotomy can be carried out.

For emergency thoracotomy, an anterior lateral thoracotomy in the fifth intercostal space is preferred. Most often, this is done on the left chest, particularly if it is a resuscitative thoracotomy. The rationale for this left thoracotomy is that posterior myocardial wounds will necessitate traction of the heart. If this is done through a median sternotomy and the heart is lifted, decreased venous return and fatal dysrhythmia may occur. In patients who are in extremis, and a left thoracotomy has been performed that turns out to be inadequate for the extensive injuries, there should be no hesitation to extend this into the right chest in a ‘clamshell’ fashion, which gives excellent exposure to all intrathoracic viscera. Occasionally, a right anterolateral thoracotomy is indicated in emergencies if air embolism is suspected (see below).

In patients who are haemodynamically stable, a median sternotomy is often the best incision when the visceral injury is undetermined or if there may be multiple injuries. An alternative is the butterfly or clamshell incision, which gives superb exposure to the entire thoracic viscera. Sternotomy is generally preferred for upper mediastinal injuries or injuries to the great vessels as they exit the thoracic outlet. The sternotomy can be extended up the sternocleidomastoid muscle or laterally along the top of the clavicle. Resection of the medial half of the clavicle exposes most of the vessels, except possibly for the proximal left subclavian. When this diagnosis is known it is best approached by a left posterior lateral thoracotomy. In an emergency, it may be necessary to go through a fourth or fifth intercostal space for a left anterolateral thoracotomy. Care should be taken in women not to transect the breast.

An adjunctive measure to exploratory thoracotomy, after injuries have been dealt with, is the pleural toilet. It is extremely important to evacuate all clots and foreign objects. Foreign objects can include clothing, wadding of shotgun blasts and any spillage from hollow viscous injury. In general, it is best to place a right-angled chest tube to drain the diaphragmatic sulcus and a straight tube to drain the posterior gutter up towards the apex. These chest tubes should be placed so that they do not exit the chest wall at the bed line. All chest tubes are sutured to the skin with a 0 monofilament suture. Another useful adjunct is to inject 0.25 per cent bupivacaine (Marcaine) into the intercostal nerve posteriorly, in the inner space of the thoracotomy and intercostal nerves just above and below the thoracotomy. This provides excellent analgesia in the immediate postoperative period. This can then be supplemented with a thoracic epidural if necessary after the initial 12 h of the postoperative period.

Emergency department thoracotomy is indicated in the agonal or dying patient with thoracic injuries. The best results have been obtained with penetrating injuries to the torso, but some authors report up to 5 per cent salvage in patients with blunt injuries. Specific indications include resuscitative thoracotomy from hypovolaemic shock, suspected pericardial tamponade and air embolism. Patients who have signs of life in the prehospital setting and arrive with an electrical complex are also candidates. Exceptions include those patients who have associated head injuries with exposure or extrusion of brain tissue from the injury. The intent of the emergency thoracotomy is either to aid in resuscitation or to control bleeding and bronchopulmonary vein fistulas (air embolism).

### 5.1.8.4 MANAGEMENT OF SPECIFIC INJURIES

The incidence of open pneumothorax or significant chest wall injuries after civilian trauma is quite low, certainly less than 1 per cent of all major thoracic injuries. Although all penetrating wounds are technically open pneumothoraces, the tissue of the chest wall serves as an effective seal. True open pneumothorax is most often
associated with close-range shotgun blasts and high-velocity missiles. There is usually a large gaping wound commonly associated with frothy blood at its entrance. Respiratory sounds can be heard with to-and-fro movement of air. The patient often has air hunger and may be in shock from associated visceral injuries.

The wound should be immediately sealed with an occlusive clean or sterile dressing such as petroleum-soaked gauze, thin plastic sheets, and sealed on three sides to create a valve, or even aluminium foil as a temporary dressing. Once the chest wound has been sealed, it is important to realize that a tube thoracostomy may be immediately necessary because of the risk of converting the open pneumothorax into a tension pneumothorax, if there is associated parenchymal injury to the lung. Large gaping wounds will invariably require debridement, including resection of devitalized tissue back to bleeding tissue, removal of all foreign bodies including clothing, wadding from shotgun shells or debris from the object that penetrated the chest. Most of these patients will require thoracotomy to treat visceral injuries and to control bleeding from the lung or chest wall.

After the wounds have been thoroughly debrided and irrigated, the size of the defect may necessitate reconstruction. The use of synthetic material such as Marlex to repair large defects in the chest wall has mostly been abandoned. Instead, myocutaneous flaps, such as latissimus dorsi or pectoralis major, have proven efficacy, particularly when cartilage or ribs must be debrided. The flap provides prompt healing and minimizes infection to the ribs or the costal cartilages. If potential muscle flaps have been destroyed by the injury, a temporary dressing can be placed, the patient stabilized in the intensive care unit and returned to the operating room in 24–48 h for a free myocutaneous graft or alternative reconstruction. Complications include wound infection and respiratory insufficiency, the latter usually as a result of associated parenchymal injury. Ventilatory embarrassment can persist secondary to the large defect. If the chest wall becomes infected, debridement, wound care and myocutaneous flaps should be considered.

Tension pneumothorax (pneumo-/haemothorax)

Tension pneumothorax is a common threat to life. The patients may present to the emergency department (ED) either dead or dying. The importance of making the diagnosis is that it is the most easily treatable life-threatening surgical emergency in the ED. ‘Simple’ closed pneumothorax, which is not quite as dramatic, occurs in approximately 20 per cent of all penetrating chest injuries. Haemothorax, in contrast, is present in about 30 per cent of penetrating injuries and haemopneumothorax is found in 40–50 per cent of penetrating injuries. The diagnosis of tension pneumothorax can be difficult in a noisy ED. The classic signs are decreased breath sounds and percussion tympany on the ipsilateral side and tracheal shift to the contralateral side. Diagnosis is clinical. In the patient who is dying there should be no hesitation in performing a tube thoracostomy. Massive haemothorax is equally life-threatening. Approximately 50 per cent of patients with hiliar, great vessel or cardiac wounds expire immediately after injury. Another 25 per cent live for periods of 5–6 min and, in urban centres, some of these patients may arrive alive in the ED after rapid transport. The remaining 25 per cent live for periods of up to 30 min and it is this group of patients who may arrive alive in the ED and require immediate diagnosis and treatment.

The diagnosis of massive haemothorax is invariably made by the presence of shock, ventilatory embarrassment and shift in the mediastinum. A chest radiograph will confirm the extent of blood loss but most of the time tube thoracostomy is done immediately to relieve the threat of ventilatory embarrassment. If a gush of blood is obtained when the chest tube is placed, autotransfusion should be considered. There are simple devices for this that should be in all major trauma resuscitation centres. The only contraindication to autotransfusion is a high suspicion of hollow viscus injury. Lesser forms of haemothorax are usually diagnosed by routine chest radiograph.

The treatment of massive haemothorax is to restore blood volume. Essentially all such patients will require thoracotomy. In about 85 per cent of patients with massive haemothorax, a systemic vessel has been injured such as the intercostal artery or internal mammary artery. In a few patients, there may be injury to the hilum of the lung or the myocardium. In about 15 per cent of instances the bleeding is from deep pulmonary lacerations. These injuries are treated by oversewing the lesion, making sure that bleeding is controlled to the depth of the lesion or, in some instances, tructotomy – resection of a segment or lobe.

Complications of haemothorax or massive haemothorax are almost invariably related to the visceral injuries. Occasionally there is a persistence of undrained blood that may lead to a cortical peel, necessitating thoracoscopy or thoracotomy and removal of this peel.
Aggressive use of two chest tubes should minimize the incidence of this complication.

Tracheobronchial injuries
Penetrating injuries to the tracheobronchial tree are uncommon and constitute less than 2 per cent of all major thoracic injuries. Disruption of the tracheobronchial tree is suggested by massive haemoptysis, airway obstruction, progressive mediastinal air, subcutaneous emphysema, tension pneumothorax and significant persistent air leak after placement of a chest tube. Treatment for tracheobronchial injuries is straightforward. If it is a distal bronchus, there may be a persistent air leak for a few days but it will usually close with chest tube drainage alone. If, however, there is a persistent air leak or the patient has significant loss of minute volume through the chest tube, bronchoscopy is used to detect whether or not this is a proximal bronchus injury, and the involved haemothorax is explored, usually through a posterior lateral thoracotomy. If possible, the bronchus is repaired with monofilament suture. In some instances, a segmentectomy or lobectomy may be required.

Pulmonary contusion
Pulmonary contusions represent bruising of the lung and are usually associated with direct chest trauma, high-velocity missiles and shotgun blasts. The pathophysiology is the result of ventilation-perfusion defects and shunts. The bruise also serves as a source of sepsis.

The treatment of significant pulmonary contusion is straightforward and consists primarily of cardiovascular and ventilatory support as necessary. Adjunctive measures such as steroids and diuretics are no longer used, because it is impossible to dry out a bruise selectively.

Antibiotics are not generally used, because this will simply select out nosocomial, opportunistic and resistant organisms. It is preferable to obtain a daily Gram stain of the sputum and chest radiographs when necessary. If the Gram stain shows presence of a predominant organism with associated increase in polymorphonuclear cells, antibiotics are indicated.

Air embolism
Air embolism is an infrequent event following penetrating trauma. It occurs in 4 per cent of all major thoracic trauma. Sixty-five per cent of the cases are the result of penetrating injuries and the key to diagnosis is to be aware of the possibility. The pathophysiology is a fistula between a bronchus and the pulmonary vein. Those patients who are breathing spontaneously will have a pressure differential from the pulmonary vein to the bronchus that will cause approximately 22 per cent of these patients to have haemoptysis on presentation. If, however, the patient has a Valsalva-type respiration, grunts or is intubated with positive pressure in the bronchus, the pressure differential is from the bronchus to the pulmonary vein, causing systemic air embolism. These patients present in one of three ways: focal or lateralizing neurological signs, sudden cardiovascular collapse and froth when the initial arterial blood specimen is obtained.

Any patient who has obvious chest injury, does not have obvious head injury and yet has focal or lateralizing neurological findings should be assumed to have air embolism. Confirmation occasionally can be obtained by fundoscopic examination, which shows air in the retinal vessels. Patients who are intubated and have a sudden unexplained cardiovascular collapse with absence of vital signs should be immediately assumed to have air embolism to the coronary vessels. Finally, those patients who have a frothy blood sample drawn for initial blood gas determination will have air embolism. When a patient comes into the ED in extremis and an ERT is carried out, air should always be looked for in the coronary vessels. If air is found, the hilum of the offending lung should be clamped immediately to reduce the ingress of air into the vessels.

The treatment of air embolism is immediate thoracotomy, preferably in the operating theatre. In most patients, the left or right chest is opened depending on the side of penetration. If a resuscitative thoracotomy has been carried out and there is no parenchymal injury to the lung on the left, it may be necessary to extend this across the sternum into the opposite chest. Definitive treatment is to oversew the lacerations to the lung, and in some instances to perform a lobectomy and only rarely a pneumonectomy.

Other resuscitative measures in patients who have 'arrested' from air embolism include internal cardiac massage and reaching up and holding the ascending aorta with the thumb and index finger for one or two beats – this will tend to push air out of the coronary vessels and thus establish perfusion. Epinephrine (adrenaline 1:1000) can be injected intravenously or down the endotracheal tube to provide an alpha effect, driving air
out of the systemic microcirculation. It is prudent to
vent the left atrium and ventricle as well as the ascend-
ing aorta to remove all residual air once the lung hilum
has been clamped. This prevents further air embolism
when the patient is moved. Using aggressive diagnosis
and treatment, it is possible to achieve up to a 55 per
cent salvage rate in patients with air embolism sec-
ondary to penetrating trauma.

Cardiac injuries
In urban trauma centres, cardiac injuries are most com-
mon after penetrating trauma and constitute about 5 per
cent of all thoracic injuries.10,11 The diagnosis of cardiac
injury is usually fairly obvious. The patient presents with
exsanguination, cardiac tamponade and, rarely, acute
heart failure. Patients with tamponade caused by pen-
etrating injuries usually have a wound in proximity,
decreased cardiac output, increased central venous pres-
sure, decreased blood pressure, decreased heart sounds,
narrow pulse pressure and occasionally paradoxical
pulse.

Many of these patients do not have the classic Beck’s
triad. Patients presenting with acute failure usually have
injuries of the valves or chordae tendineae, or have sus-
tained interventricular septal defects, but they represent
less than 2 per cent of the total patients with cardiac
injuries. Pericardiocentesis is not a very useful diagnos-
tic technique but may be temporarily therapeutic. In
patients in whom the diagnosis of pericardial tampon-
ade cannot be confirmed on clinical signs, an echocar-
diogram is useful.

The treatment of all cardiac injuries is immediate
thoracotomy, ideally in the operating theatre. In the
patient who is in extremis, thoracotomy in the ED can
be life saving. The great majority of wounds can be
closed with simple sutures or horizontal mattress sutures
of a 3/0 or 4/0 monofilament. Bolstering the suture with
Teflon pledgets occasionally may be required, particu-
larly if there is surrounding contusion, or proximity of
the wound to a coronary artery. If the stab or gunshot
wound is in proximity to the coronary artery, care must
be taken not to suture the vessels. This can be achieved
by passing horizontal mattress sutures beneath the
coronary vessels, avoiding ligation of the vessel. If
the coronary arteries have been transected, two options
exist. Closure can be accomplished in the beating heart
using fine 6/0 or 7/0 Prolene sutures, under magnifica-
tion if necessary. The second option is temporarily to
initiate inflow occlusion and fibrillation. However, both
of these measures have a high risk associated with them.
Heparinization is optimally avoided in the trauma
patient and fibrillation in the presence of shock and
acidosis may be difficult to reverse. Bypass is usually
reserved for patients who have injury to the valves, chor-
dae tendineae or septum. In most instances these injuries
are not immediately life-threatening but become
evident over a few hours or days after the injury.

Complications from myocardial injuries include
recurrent tamponade, mediastinitis and post-cardiotomy
syndrome. The first can be avoided by placing a medi-
stinal chest tube or leaving the pericardium partially
opened after repair. Most cardiac injuries are treated
through a left anterolateral thoracotomy and only occa-
sionally via a median sternotomy. If mediastinitis does
develop, the wound should be opened (including the
sternum), and debridement carried out with secondary
closure in 4–5 days. If this is impossible, myocutaneous
flaps should be considered. Another complication is her-
niation of the heart through the pericardium, which may
occlude venous return and cause sudden death. This is
avoided by loosely approximating the pericardium after
the cardiac injury has been repaired.

Injuries to the great vessels
Injuries to the great vessels from penetrating forces are
infrequently reported. According to Rich, before the
Vietnam War there were fewer than 10 cases in the sur-
gical literature.12,13 The reason for this is that extensive
injury to the great vessels results in immediate exsan-
guination into the chest and most of these patients die
at the scene of injury.

The diagnosis of penetrating great vessel injury is usu-
ally obvious. The patient is in shock and there is an
injury in proximity to the thoracic outlet or posterior
mediastinum. If the patient stabilizes with resuscitation,
an arteriogram should be performed to localize the
injury. Approximately 8 per cent of patients with major
vascular injuries do not have clinical signs, stressing the
need for arteriograms when there is a wound in proxim-
ity. These patients usually have a false aneurysm or ar-
teriovenous fistula. Treatment of penetrating injuries to
the great vessels can almost always be accomplished
using lateral repair, because larger injuries that might
necessitate grafts are usually incompatible with survival
long enough to permit the patient to reach the ED
alive.14,15
Complications of injuries to the great vessels include rebleeding, false aneurysm formation and thrombosis. A devastating complication is paraplegia, which usually occurs after blunt injuries but rarely after penetrating injuries, because of either associated injury to the spinal cord or at the time of surgery ligation of important intercostal arteries. The spinal cord has a segmental blood supply to the anterior spinal artery and every effort should be made to preserve intercostal vessels, particularly those that appear to be larger than normal.

**Oesophageal injuries**

Penetrating injuries to the thoracic oesophagus are quite uncommon. Injuries to the cervical oesophagus are somewhat more frequent and are usually detected at the time of exploration of zone I and II injuries of the neck. In those centres where selective management of neck injuries is practised, the symptoms found are usually related to pain on swallowing and dysphagia. Occasionally, patients may present late with signs of posterior mediastinitis. Injuries to the thoracic oesophagus may present with pain, fever, pneumomediastinum, persistent pneumothorax, in spite of tube thoracostomy, and pleural effusion with extravasation of contrast on Gastrografin swallow.

Treatment of cervical oesophageal injuries is relatively straightforward. As noted above, the injury is usually found during routine exploration of penetrating wounds beneath the platysma.

Once found, a routine closure is performed. In more devitalizing injuries it may be necessary to debride and close using draining to protect the anastomosis. Injuries to the thoracic oesophagus should be repaired if the injury is less than 6 h old, and there is minimal inflammation and devitalized tissue present. A two-layer closure is all that is necessary. Postoperatively, the patient is kept on intravenous support and supplemental nutrition. Antibiotics may be indicated during the 24-h perioperative period. If the wound is more than 6 h and less than 12 h old, a decision will be necessary to determine whether primary closure can be attempted or whether drainage and nutritional support are the optimal management. Almost all injuries older than 24 h will not heal primarily when repaired. Open drainage, antibiotics, nutritional support and consideration of diversion are the optimal management. Complications after oesophageal injuries include wound infection, mediastinitis and empyema.

**Flail chest**

Traditionally, flail chest has been managed by internal splinting (‘internal pneumatic stabilization’). Although this is undoubtedly the method of choice in most instances, there has been increasing interest in open reduction and fixation of multiple rib fractures. In uncontrolled trials, there have been considerable benefits shown, with a shortening of hospital time and improved mobility. A flail chest may be stabilized using pins, plates, wires, rods and, more recently, absorbable plates.

Exposure for the insertion of these can be via either a conventional posterolateral thoracotomy or incisions made over the ribs.

**Diaphragm injuries**

Diaphragmatic injuries occur in approximately 6 per cent of patients with mid-torso injuries from penetrating trauma. The left diaphragm is injured more commonly than the right. The diaphragm normally rises to the fifth intercostal space during normal expiration, so that any patient with a mid-torso injury is at risk for diaphragmatic injury.

The diagnosis of penetrating injury to the diaphragm is less problematic than injury from blunt trauma. Typically, the patient has a wound in proximity and the surgeon’s decision is how best to assess the diaphragm. Thoracoscopy is a good method because it is so easy to visualize the diaphragm from above. Laparoscopy has an additional advantage in that it is possible to assess intraperitoneal organs for injury as well as the diaphragm. However, laparoscopy has not withstood the degree of specificity and sensitivity necessary for it to be the method of choice.

Optimally, all diaphragmatic injuries should be repaired, even small penetrating puncture wounds of no apparent importance. Those injuries that are not repaired will present late, usually with incarceration of the small bowel, colon or omentum into the hernia defect. The preferred closure of diaphragmatic injuries is with interrupted non-absorbable suture. The use of synthetic material to close large defects from high-velocity missile injuries or shotgun blasts is only rarely indicated.

The complications of injuries to the diaphragm are primarily related to late diagnosis with hernia formation and incarceration. Phrenic nerve palsy is another complication but is uncommon after penetrating trauma.
Complications

As noted in the preface, the lung is a target organ for reperfusion injury and any injury to the viscera within the thorax can result in impaired oxygen transport. The lungs are at high risk from aspiration, which can accompany shock or substance abuse, and is often associated with penetrating injuries. Finally, pulmonary sepsis is one of the more common sequelae after a major injury of any kind.

5.1.9 Emergency department thoracotomy

Rapid emergency medical response times and advances in pre-hospital care have led to increased numbers of patients arriving in resuscitation in extremis. Salvage of these patients often demands immediate control of haemorrhage and desperate measures to resuscitate them. It has often been attempted in hopeless situations, after both blunt and penetrating injury, and failure to understand the indications and sequelae will almost inevitably result in the death of the patient. With the increasing financial demands on medical care, and the increasing risk of transmission of communicable diseases, a differentiation must be made between the true ERT and futile care.

In 1874, Schiff described open cardiac massage and, in 1901, Rehn sutured a right ventricle in a patient presenting with cardiac tamponade. The limited success of ERT in most circumstances, however, prohibited the use of the technique for the next 60–70 years. A revival of interest occurred in the 1970s when it was applied as a means of temporary aortic occlusion in exsanguinating abdominal trauma. This was short-lived, and in the 1980s, and subsequently, there has been decreased enthusiasm and a more selective approach, particularly with respect to blunt trauma.

It must be noted that there is an extremely high mortality rate associated with all thoracotomies performed anywhere outside the operating theatre, especially when performed by non-surgeons.

It is also important early on to differentiate the definitions of thoracotomy:

- Thoracotomy performed in the ED (ERT) for patients in extremis:
  - to control haemorrhage
  - to control the aortic outflow (aortic ‘cross-clamping’)
- Planned resuscitative thoracotomy, i.e. in operating theatre or ICU minutes to hours after injury in acutely deteriorating patients:
  - urgent planned control of haemorrhage.

It is also important to differentiate between the following patients:

- Patients with ‘no signs of life’
- Patients with ‘no vital signs’ in whom pupillary activity and/or respiratory effort is still evident.

Obviously, the results of ERT in these two circumstances will differ. This topic concentrates on those thoracotomies performed by the surgeon in those patients who present in extremis in the resuscitation area.

5.1.9.1 Objectives

The primary objectives of ERT in this set of circumstances are to:

- release cardiac tamponade
- control intrathoracic bleeding
- control air embolism or bronchopleural fistula
- permit open cardiac massage
- allow for temporary occlusion of the descending aorta to redistribute blood to the upper body and possibly limit subdiaphragmatic haemorrhage.

Emergency department thoracotomy has been shown to be most productive in life-threatening penetrating cardiac wounds, especially when cardiac tamponade is present. Patients requiring ERT, even in established trauma centres, for anything other than isolated penetrating cardiac injury, rarely survive. Outcome in the field is even worse.

Indications in military practice are essentially the same as in civilian practice.

Emergency department thoracotomy and the necessary rapid use of sharp surgical instruments and exposure to the patient’s blood pose certain risks to the resuscitating surgeon. Contact rates of patient’s blood to the surgeon’s skin approximate 20 per cent. HIV rates among the patient population at the Johannesburg Hospital Trauma Unit have risen from 6 per cent in 1993 to 50 per cent in 2000. There are additional risks from other blood-borne pathogens, such as hepatitis C. Use of universal precautions and the selective use of ERT may minimize this.
5.1.9.2 INDICATIONS AND CONTRAINDICATIONS

There are instances where ERT has been shown to have clear benefit. These indications include:

• Those patients in whom there is a witnessed arrest and high likelihood of isolated intrathoracic injury, especially penetrating cardiac injury (‘salvageable’ post-injury cardiac arrest)
• Those with severe post-injury hypotension (BP < 60 mmHg) as a result of cardiac tamponade, air embolism or thoracic haemorrhage.

Less clear benefit occurs in:

• those patients presenting with moderate post-injury hypotension (BP < 80 mmHg) potentially as a result of intra-abdominal aortic injury (e.g. epigastric gunshot wound)
• major pelvic fractures
• active intra-abdominal haemorrhage.

The first group of patients constitutes those in whom ERT is relatively indicated. One must consider the patient’s age, pre-existing disease, signs of life and injury mechanism as well as proximity of the ED to the operating theatre and personnel available in applying the principles related to ERT. Although optimal benefit of the procedure will be obtained with an experienced surgeon, in cases where a moribund patient presents with a penetrating chest wound, the emergency physician should not hesitate to perform the procedure.

ERT is contraindicated:

• When there has been cardiopulmonary resuscitation (CPR) in the absence of endotracheal tube intubation in excess of 5 min
• When there has been CPR for more than 10 min with or without endotracheal tube intubation
• In cases of blunt trauma, when there have been no signs of life at the scene, or pulseless electrical activity (PEA) only is present in the ED.

5.1.9.3 RESULTS

The results of ERT vary according to injury mechanism and location, and the presence of vital and life signs. Emergency department thoracotomy has been shown to be beneficial in around 50 per cent of patients presenting with signs of life after isolated penetrating cardiac injury and only rarely in those patients presenting without signs of life (< 2 per cent). In non-cardiac penetrating wounds, 25 per cent of patients benefit when signs of life and detectable vital signs are present, compared with 8 per cent of those with signs of life only and 3 per cent of those without signs of life.

Only 1–2 per cent of patients requiring ERT are salvaged after blunt trauma regardless of their clinical status on admission. A decision-making algorithm has been formulated, based on these findings and the four factors found to be most predictive of poor outcome after ERT:

1. Signs of life at scene absent
2. Signs of life in ED absent
3. Cardiac activity at ERT absent
4. Systolic blood pressure < 70 mmHg after aortic occlusion.

At the scene, patients in extremis and without cardiac electrical activity are declared dead. Those with electrical activity are intubated, supported with CPR and transferred to the ED. If blunt injury is present, ERT is embarked on only if PEA is present. (In penetrating trauma, all undergo ERT.) If no blood is present in the pericardial cavity and there is no cardiac activity, the patient is declared dead. All others are treated according to the type of injury as above. Those with intra-abdominal injury who respond to aortic occlusion with a systolic blood pressure > 70 mmHg and all other surviving patients are rapidly transported to the operating theatre for definitive treatment.

5.1.9.4 WHEN TO STOP ERT?

Emergency department thoracotomy is a ‘team event’. It should not be prolonged unduly but should have specific endpoints. If an injury is repaired, and the patient responds, he or she should be moved to the operating theatre for definitive repair or closure.

Emergency department thoracotomy should be terminated if:

• Irreparable cardiac damage has occurred
• The patient is identified as having massive head injuries
• PEA is established
• Systolic BP < 70 mmHg after 20 min
• Asystolic arrest has occurred.

5.1.9.5 CONCLUSION

The success in the management of thoracic injury in those cases requiring surgery lies in rapid access to the
thoracic cavity with good exposure. Thus, good lighting, appropriate instrumentation, functioning suctioning apparatus and a ‘controlled, aggressive but calm frame of mind’ on the part of the surgeon will result in acceptable, uncomplicated survival figures.

5.1.10 Approaches to the thorax

The choice of approach to the injured thorax should be determined by three factors:

1. The hemithorax and its contents
2. The stability of the patient
3. Whether the indication for surgery is acute or chronic (non-acute).

A useful distinction can be made with respect to indications (Table 5.1). It will be noted that the acute indications include all acutely life-threatening situations, whereas the chronic or non-acute indications are essentially late presentations.

The surgical approaches in current use include:

- Anterolateral thoracotomy
- Median sternotomy
- Bilateral thoracotomy (‘clamshell’ incision)
- The ‘trapdoor’ incision
- Posterolateral thoracotomy.

In the unstable patient, the choice of approach will usually be an anterolateral thoracotomy or median sternotomy, depending on the suspected injury. In the case of the stable patient, the choice of approach must be planned after proper evaluation and work-up has clearly identified the nature of the injury.

If time permits in the more stable patient, intubation (or reintubation) with a double-lumen endotracheal tube, to allow selective deflation or ventilation of each lung, can be very helpful and occasionally life saving.

It is seldom necessary to resort to the remaining three approaches in the acute situation. Of these, the bilateral trans-sternal thoracotomy (the ‘clamshell’ incision) and the ‘trapdoor’ incision are complex and somewhat mutilating, with significant postoperative morbidity and difficulty in terms of access and closure.

5.1.11 References


Table 5.1 Indications for surgery in the thorax

<table>
<thead>
<tr>
<th>Chronic indications</th>
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<tbody>
<tr>
<td>Cardiac tamponade</td>
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<td>Acute deterioration</td>
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<tr>
<td>Vascular injury at the thoracic outlet</td>
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<td>Loss of chest wall substance</td>
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<tr>
<td>Endoscopic or radiological evidence of tracheal or oesophagus or great vessel injury</td>
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<tr>
<td>Massive or continuing haemothorax</td>
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<tr>
<td>Bullet embolism to the heart/pulmonary artery</td>
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<tr>
<td>Penetrating mediastinal injury</td>
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<tr>
<td>Unevacuated clotted haemothorax</td>
<td></td>
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<tr>
<td>Chronic traumatic diaphragmatic hernia</td>
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<tr>
<td>Traumatic arteriovenous fistula</td>
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<tr>
<td>Traumatic cardiac septal or valvar lesions</td>
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<tr>
<td>Missed tracheobronchial injury or tracheo-oesophageal fistula</td>
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<tr>
<td>Infected intrapulmonary haematoma</td>
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</table>


5.2 ACCESS TO THE THORAX

5.2.1 Incisions

5.2.1.1 ANTEROLATERAL THORACOTOMY (FIGURE 5.5)

This is the approach of choice in most unstable patients and is used for ERT:

1 This approach allows rapid access to the injured hemithorax and its contents.

2 It is made with the patient in the supine position with no special positioning requirements or instruments.

3 It has the advantages that it:
   – may be extended across the sternum into the contralateral hemithorax (the ‘clamshell’ incision or bilateral thoracotomy)
   – may be extended downwards to create a thoracoabdominal incision
   – may be converted into a ‘trap-door’ incision by creating an additional sternotomy with supraclavicular extension.

This is the approach of choice in injury to any part of the left thorax or an injury above the nipple line in the right thorax. It should be noted that right lower thoracic injuries (i.e. below the nipple line) usually involve bleeding from the liver; the approach in these patients initially should be a midline laparotomy, the chest being entered only if no source of intra-abdominal bleeding is found.

Technique

Slight tilt of the patient to the right is advisable, and this is achieved either by use of a sandbag or other support or by tilt of the table.

Incision is made through the fourth or fifth intercostal space from the costochondral junction anteriorly to the midaxillary line posteriorly, following the upper border of the lower rib in order to avoid damage to the intercostal neurovascular bundle.

The muscle groups are divided (preferably with cautery) down to the peristeum of the lower rib. The muscle groups of the serratus anterior posteriorly, and the intercostals medially and anteriorly, are divided. The trapezius and the pectoralis major are avoided.

Care should be taken at the anterior end of the incision, where the internal mammary artery runs and may be transected.

The peristeum is opened, leaving a cuff of approximately 5 mm for later closure. The parietal pleura is then opened, taking care to avoid the internal mammary artery adjacent to the sternal border. These vessels are ligated if necessary.

A Finochietto retractor is placed with the handle away from the sternum (i.e. laterally placed), the ribs are spread and intrathoracic inspection for identification of injuries is carried out after suctioning. In cases of ongoing bleeding, an autotransfusion suction device is advisable.
Note that it is important to identify the phrenic nerve in its course across the pericardium if this structure is to be opened – the pericardiotomy is made 1 cm anterior and vertical to the nerve trunk in order to avoid damage and subsequent morbidity.

Closure

After definitive manoeuvres, the anterolateral thoracotomy is closed in layers over one or two large-bore intercostal tube drains and after careful haemostasis and copious lavage.

- Ribs and intercostal muscles should be closed with synthetic absorbable sutures.
- Closure of discrete muscle layers reduces both pain and long-term disability.
- Skin is closed routinely.

5.2.1.2 MEDIAN STERNOTOMY (FIGURE 5.6)

This incision is the approach of choice in patients with penetrating injury at the base of the neck (zone 1) and the thoracic outlet. It allows access to the pericardium and heart, the arch of the aorta and the origins of the great vessels. It has the attraction of allowing upward extension into the neck (as Henry’s incision), extension downward into a midline laparotomy or lateral extension into a supraclavicular approach. It has the relative disadvantage of requiring a sternal saw or chisel (of the Lebschke type). In addition, the infrequent but significant complication of sternal sepsis may occur postoperatively, especially in the emergency setting.

Technique

The incision is made with the patient fully supine, in the midline from the suprasternal notch to below the xiphoid cartilage.

Finger sweep is used to open spaces behind the sternum, above and below. Excision of the xiphoid cartilage may be necessary if this is large and intrusive, and can be done with heavy scissors.

Split (bisection) section of the sternum is carried out with a saw (either oscillating or a braided-wire Gigli saw) or a Lebschke knife, commencing from above and moving downwards. This is an important point to avoid inadvertent damage to vascular structures in the mediastinum. In addition, be aware of the possible presence of the large transverse communicating vein, which may be found in the areolar tissue of Burns’ suprasternal space, and must be controlled.

Closure

- The pericardium is usually left open, or only partially closed. It is advisable to close the pericardium with an absorbable suture to avoid adhesion formation.
- Two mediastinal tube drains are brought out through epigastric incisions.
- Closure of the sternotomy is made with horizontal sternal wires or encircling heavy non-absorbable suture (of the Ethiflex or Ethibond type).
- Closure of the linea alba should be by non-absorbable suture.

5.2.1.3 THE ‘CLAMSHELL’ THORACOTOMY

The ‘clamshell’ is essentially a bilateral fourth or fifth interspace thoracotomy, linked by division of the sternum, allowing the chest to be opened very widely, anteriorly. Care should be taken to ligate the internal mammary arteries and ensure haemostasis before closure.

The incision is particularly effective in those situations where it is important to achieve rapid access to the opposite side of the chest, especially posteriorly:

- Transmediastinal injury
- Lung injury
• Injury on the right, where aortic control may be necessary.

5.2.1.4 POSTEROLATERAL THORACOTOMY

This approach requires appropriate positioning of the patient and is usually used in the elective setting for definitive lung and oesophageal surgery. It is not usually employed in the acute setting. It is more time-consuming in approach and closure, because the bulkier muscle groups of the posterolateral thorax are traversed and scapular retraction is necessary.

5.2.1.5 ‘TRAPDOOR’ THORACOTOMY (FIGURE 5.7)

This is a combination of an anterolateral thoracotomy, a partial sternotomy and an infra-or supraclavicular incision with resection or dislocation of the clavicle. The incision is considered obsolete. It has the disadvantages of being relatively more time-consuming and retraction of the bony ‘trapdoor’ created being often difficult, resulting in multiple fractures of the ribs laterally or posteriorly.

Closure too is time-consuming. It may be argued that, in this instance, a median sternotomy with extension of the incision into the neck will provide more rapid and efficient exposure of injury in this region.

5.2.2 Procedures

5.2.2.1 EMERGENCY DEPARTMENT THORACOTOMY

Requirements

The numbers of instruments and types of equipment necessary to perform ERT do not even begin to approach those used for formal thoracotomy in the operating theatre, and really include only the following:

• Scalpel, with a no. 20 or 21 blade
• Forceps
• Suitable retractor such as Finochietto’s chest retractor, or Balfour’s abdominal retractor
• Lebschke’s knife and mallet or Gigli saw for the sternum
• Large vascular clamps such as Satinski’s vascular clamps (large and small)
• Mayo’s scissors
• Metzenbaum’s scissors
• Long needle holders
• Internal defibrillator paddles
• Sutures, swabs, Teflon pledgets
• Sterile skin preparation and drapes, good light

Approach

Two basic incisions are used in ERT, which are applied according to the best incision for the particular suspected injury (based on entrance and exit wounds, trajectory and most likely diagnosis from clinical examination), and may be extended in various ways according to need.

Routine immediate resuscitation protocols as per ATLS® are instituted and, once indications for ERT have been fulfilled, ERT should follow without delay.

Pitfall: if the conditions are not fulfilled, you are embarking upon futile care.

The left anterolateral thoracotomy is the most common site for urgent access. The incision is placed in the fifth intercostal space through muscle, periosteum and parietal pleura, from costochondral junction anteriorly to midaxillary line laterally, following the upper border of rib; care is taken to avoid the internal mammary artery. This incision can be extended as a bilateral incision requiring horizontal division of the sternum and ligation of the internal mammary vessels bilaterally. It affords excellent access to both pleural cavities, to the pericardial cavity and even to the abdominal cavity if
required. The incision may also be extended cranially in the midline by dividing the sternum for penetrating wounds involving mediastinal structures. The same incision may be employed on the right side in hypotensive patients with penetrating right chest trauma, in whom massive blood loss or air embolism is suspected. This, too, may be extended trans-sternally if a cardiac wound is discovered.

The median sternotomy affords the best exposure to the anterior and middle mediastinum, including the heart and great vessels, and is typically advocated for penetrating wounds particularly of the upper chest between the nipples. This can be extended supraclavicularly for access to control subclavian and brachiocephalic vascular injuries.

Applications

Suspected cardiac injury and cardiac tamponade
Access is achieved via either lateral or midline sternotomy, the former allowing more rapid access while the latter approach ensures better exposure. It is important to identify the phrenic nerve before opening the pericardium at least 1 cm anterior to this structure. The pericardial incision is initiated using either a knife or the sharp point of a scissors, and blood and clots are evacuated.

Cardiac bleeding points on the ventricle are initially managed with digital pressure, and those of the atria and great vessels by partially occluding vascular clamps. If the heart is beating, repair should be delayed until initial resuscitation measures have been completed. If not beating, suturing precedes resuscitation.

Foley catheters may be used temporarily to control haemorrhage before definitive repair in the ED or operating theatre. A Foley catheter with a 30 mL balloon is preferable. Once placed, great care should be taken not to exert too much traction on the catheter, because it will easily tear out, making the hole dramatically bigger.

Suturing of the right ventricle requires placement of Teflon pledgets, which are utilized selectively on the left ventricle using a horizontal mattress suture under the coronary vessels to avoid trauma to or occlusion of coronary vessels. Low pressure venous and atrial wounds can be repaired with simple continuous sutures. Posterior wounds are more difficult because they necessitate elevation of the heart before their closure, which might lead to further haemodynamic compromise. With large wounds of the ventricle or inaccessible posterior wounds, temporary digital inflow occlusion might be necessary to facilitate repair.

After initial repair, fluids are best slowed down to limit further bleeding (concept of hypotensive resuscitation), aiming for critical organ perfusion while minimizing additional haemorrhage (i.e. blood pressure of about 85 mmHg). The patient is best transferred to the operating theatre where repair of the injury and closure of the access procedure is done under controlled circumstances with adequate resources.

Pulmonary haemorrhage
Access is best achieved by anterolateral thoracotomy on the appropriate side. With localized bleeding sites, control can be achieved with vascular clamps placed across the affected segment. The affected segment is then dealt with, preferably under controlled circumstances in the operating theatre by local oversewing, segmental resection or pulmonary tractotomy.

Pulmonary tractotomy is a means of controlling tracts that pass through multiple lung segments, where the extent of injury precludes pulmonary resection. It is a means of non-anatomical lung preservation, where linear staplers are passed along both sides of the tract formed and the lung divided to allow blood vessels and airways in the bases to be repaired, and the divided edges then oversewn.

With massive haemorrhage from multiple or indeterminate sites, or widespread destruction of lung parenchyma leaving large areas of non-viable tissue, hilar clamping with a large soft vascular clamp, across the hilar structures occluding the pulmonary artery, pulmonary vein and main stem bronchus, is employed until a definitive surgical procedure can be performed.

Air embolism is controlled by placing a clamp across hilar structures, and air is evacuated by needle aspiration of the elevated left ventricular apex.

Thoracotomy with aortic cross-clamping
This technique is employed to optimize oxygen transport to vital proximal structures (heart and brain), maximize coronary perfusion and possibly limit infradiaphragmatic haemorrhage in both blunt and penetrating trauma.

The thoracic aorta is cross-clamped inferior to the left pulmonary hilum and the area exposed by elevating the left lung anteriorly and superiorly. The mediastinal pleura is dissected under direct vision; the aorta is separated by blunt dissection from the oesophagus anteriorly.
and the prevertebral fascia posteriorly. When properly exposed the aorta is occluded using a large vascular clamp. It is important that aortic cross-clamp time be kept to the absolute minimum, i.e. removed once effective cardiac function and systemic arterial pressure have been achieved, because the metabolic penalty rapidly becomes exponential once beyond 30 min.

Bilateral trans-sternal thoracotomy
This is the thoracic equivalent of the chevron or ‘bucket-handle’ upper abdominal incision, providing wide exposure to both hemithoraces. It is relatively time-consuming in terms of both access and closure. It may be argued that median sternotomy will provide the same degree of exposure with greater ease of access and closure.

It usually will be necessary to use this incision only when it becomes necessary to gain access to both hemithoraces.

It extends, usually, as a fifth intercostal space anterolateral thoracotomy, across the sternum. The sternum is divided using a Gigli saw, chisel or bone-cutting forceps. Care is taken to ligate the internal mammary arteries.

5.2.2.2 DEFINITIVE PROCEDURES

Pericardial tamponade
• Open the pericardium in a cranial-to-caudal direction, anterior to the phrenic nerve.
• It is important to examine the whole heart to localize the source of bleeding.
• Deal with the source of bleeding.
• It is not essential to close the pericardium after the procedure.
• If the pericardium is closed, it should be drained to avoid a recurrent tamponade.

Myocardial laceration
• Wherever possible, initial control of a myocardial laceration should be digital, while the damage is assessed.
• A Foley catheter can also be used.

Pitfall: there is a real danger of extending the damage if the balloon pulls through the laceration. Use a 30 mL balloon, and avoid other than gentle traction.
• Use 3/0 or 4/0 braided sutures (e.g. Ethibond) tied gently to effect the repair. Pledgets may be helpful.
• Care should be exercised near coronary arteries. Although a vertical mattress suture is normally acceptable, it may be necessary to use a horizontal mattress suture under the vessel to avoid occluding it.
• In inexperienced hands, and as a temporizing measure, a skin stapler will allow control of the bleeding, with minimal manipulation of the heart.

Pitfall: staples often tear out eventually, and so the repair should not be regarded as definitive.

Pulmonary haemorrhage
Hilar clamping
• Wide anterolateral or posterolateral thoracotomy is the exposure of choice.
• A soft vascular clamp can be placed across the hilum, occluding the pulmonary artery, vein and main stem bronchus.

Lobectomy or pneumonectomy
• Usually performed to control massive intrapulmonary haemorrhage
• A double-lumen endotracheal tube should be used whenever possible.
• For segmental pneumonectomy, use of the GIA stapler is helpful. The staple line can then be oversewn.

Pulmonary tractotomy
• Used where the injury crosses more than one segment, commonly caused by penetrating injury. Anatomical resection may not be possible.
• Linear staplers can be introduced along both sides of the tract, the tract divided, and then oversewn.
• This procedure is also helpful in ‘damage control’ of the chest.

Aortic injury
• Most patients with these injuries do not survive to reach hospital.
• Cardiopulmonary bypass is preferable in order to avoid paraplegia.

Oesophageal injury
• Surgical repair is mandatory.
• Two-layer repair (mucosal and muscular) is preferable.
• If possible, the repair should be wrapped in autogenous tissue.
• A feeding gastrostomy is preferable to a nasogastric tube through the area of repair, and the stomach should be drained.
• A cervical oesophagostomy may be required.

Tracheobronchial injury
• Flexible bronchoscopy is very helpful in assessment.
• Formal repair should be undertaken under ideal conditions, with removal of devitalized tissue.

5.2.3 Conclusion

The success in the management of thoracic injury in those cases requiring surgery lies in rapid access to the thoracic cavity with good exposure. Thus, good lighting, appropriate instrumentation, functioning suction apparatus and a 'controlled, aggressive but calm frame of mind' on the part of the surgeon will result in acceptable, uncomplicated survival figures.

5.2.4 Recommended reading

6.1 THE ABDOMINAL CAVITY

6.1.1 Overview

6.1.1.1 INTRODUCTION

Delay in the diagnosis and treatment of abdominal injuries is one of the most common causes of preventable death from blunt or penetrating trauma. Approximately 20 per cent of abdominal injuries will require surgery. In the UK, western Europe and Australia, the trauma is predominantly blunt in nature, whereas, in the military context, and in civilian trauma in the USA, South Africa and South America, it is predominantly penetrating.

It is important to appreciate the difference between surgical resuscitation and definitive treatment for abdominal trauma. Surgical resuscitation includes the technique of ‘damage control’, and implies only that the surgical procedure is necessary to save life by stopping bleeding and preventing further contamination or injury.

6.1.1.2 RESUSCITATION

Resuscitation of patients with suspected abdominal injuries should always take place within the ATLS (Advanced Trauma Life Support) context. Attention is paid to adequate resuscitative measures, including adequate pain control. Adequate analgesia (titrated intravenously) will never mask abdominal symptoms, and is much more likely to make abdominal pathology easier to assess, with clearer physical signs and a cooperative patient.

Blood is not initially a peritoneal irritant, and therefore it may be difficult to assess the presence or quantity of blood present in the abdomen.

Bowel sounds may remain present for several hours after abdominal injury, or may disappear soon after trivial trauma. This sign is therefore particularly unreliable.

Investigation and assessment of the abdomen can be based on three groups:

1. The patient with the normal abdomen
2. An equivocal group requiring further investigation
3. The patient with an obvious injury to the abdomen.

6.1.1.3 DIAGNOSIS

The diagnosis of injury after blunt trauma can be difficult and knowledge of the injury mechanism can be helpful. Passenger restraints can themselves cause blunt trauma to the liver and duodenum or pancreas, and rib fractures can cause direct damage to the liver or spleen. Lap belts can cause shearing injury to intestine and mesentery, especially when incorrectly placed above the iliac crest. Virtually all penetrating injury to the abdomen should be addressed promptly, especially in the presence of hypotension.

Diagnostic modalities depend on the nature of the injury:

- Physical examination
- Diagnostic peritoneal lavage (DPL)
- Ultrasonography: focused abdominal sonography for trauma (FAST)
- Computed tomography (CT)
- Diagnostic laparoscopy.

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.

The haemodynamically stable patient

The stable patient, who is not haemodynamically normal, will benefit from investigations aimed at establishing the following:
• 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. Computed tomography is currently the modality of choice, although FAST may also be helpful, though dependent on the operator.

The haemodynamically unstable patient

Efforts must be made to try to define the cavity where bleeding is taking place, e.g. chest, pelvis or abdominal cavity. Diagnostic modalities are of necessity limited, because it may not be possible to move an unstable patient to have a CT scan, even if it were to be readily available. Diagnostic peritoneal lavage remains one of the most common, most sensitive, cheapest and most readily available modalities to assess the presence of blood in the abdomen. Focused abdominal sonography for trauma is similarly useful, but is more operator dependent – key if the individual doing the scan is inexperienced in dealing with trauma patients and interpreting their scans. Importantly, DPL and FAST can be performed without moving the patient from the resuscitation area.

It must be emphasized that a negative DPL carries much greater importance than a positive one, because it gives a very clear indication in the unstable patient that the bleeding is unlikely to be intraperitoneal in nature. This situation also lends itself to FAST examination, which is somewhat quicker than DPL, and haemodynamic instability caused by intraperitoneal haemorrhage is likely to be readily found.

During resuscitation, standard ATLS guidelines should be followed, which should include:

• Nasogastric tube or orogastric tube
• Urinary catheter.

Antibiotics

Routine, single-dose, intravenous antibiotic prophylaxis should be employed. Subsequent antibiotic policy will depend on the intraoperative findings. As a general rule:

• No pathology found: no further antibiotics
• Blood only: no further antibiotics
• Small bowel or gastric contamination: continuation for 24 h only
• Large bowel, gross contamination: copious peritoneal wash-out; 24–72 h of antibiotics.

The antibiotics commonly recommended include a second-generation cephalosporin and amoxicillin/clavulanate. There is some evidence that aminoglycosides should not be used in acute trauma, partly because of the shifts in fluids, which requires substantially higher doses of aminoglycosides to reach the appropriate minimum inhibitory concentration (MIC), and partly because they work best in an alkaline environment (traumatized tissue is acidotic).

The administered dose should be increased two- to threefold, and repeated after every 10 units of blood transfusion until there is no further blood loss. If intra-abdominal bleeding is significant, it may be necessary to give a further dose of antibiotic therapy intraoperatively, as a result of dilution of the preoperative dose.

6.1.1.4 OVERVIEW OF INDIVIDUAL ORGAN SYSTEM INJURIES

Difficult abdominal injury complexes

There are at least four complex abdominal injuries:

1. Major liver injuries: management of hepatic injuries is difficult, and experience in dealing with these injuries can obviously lead to a better outcome. Good judgement comes from experience, but unfortunately experience comes from bad judgement.

2. Pancreaticoduodenal injuries: these are challenging because of the difficulties in diagnosis, and associated retropancreatic vascular injuries, which are difficult to access. Missed injuries lead to a significant morbidity and mortality.

3. Aortic and caval injuries: these are difficult because access to these injuries, and control of haemorrhage from them, are especially problematic.

4. Complex pelvic injuries with associated open pelvic injury: these are particularly difficult to treat and associated with a high mortality.

Damage control approaches to these injuries may dramatically improve survival.

Bowel injuries

The unstable patient

The first priority is haemorrhage control. If the patient is haemodynamically unstable, bowel injuries should be treated using damage control procedures. Small wounds
can be rapidly closed. More extensive injuries should be transected and closed for later repair using staplers, umbilical tape, skin staples, etc. Enterostomies should be avoided at this stage.

**The stable patient**

Small bowel injuries should be closed, with primary repair or resection and primary anastomosis as appropriate. Consider one resection and anastomosis when several wounds are localized close to each other. When there are multiple small and large bowel lacerations, a protective ileostomy can be helpful.

For colonic injuries, indications for colostomy are still debated. Time from injury, haemodynamic status, comorbid conditions and degree of contamination will influence the decision. More primary repairs/primary anastomoses are being performed, with fewer colostomies.

In rectal injuries, primary repair should be considered for intraperitoneal injuries and extraperitoneal injuries that can be mobilized. There is no indication for routine presacral drains or distal wash-out. A proximal diverting colostomy (often a loop sigmoidostomy) is indicated in more extensive rectal injuries and when repair is impossible.

In patients with complex abdominal injuries peritoneal soiling is of secondary importance. When haemorrhage has been controlled, devascularized areas accompanying deep injuries should be removed. In severely injured patients requiring damage control, simple proximal and distal closure of the injured bowel by stapler is the best way to prevent ongoing soiling. The damaged areas can be removed at a later time and the need for colostomy assessed.

Serosal sutures and primary or secondary anastomoses can be secured with adhesives. Covering the anastomosis with collagen fleece can help prevent leakage.

**Splenic injury**

The first aim of successful operative management is to control the active haemorrhage and the second is to achieve surgical repair that preserves as much of the damaged spleen as possible. For splenic preservation the choice of procedure depends not only on the clinical findings but also on the surgeon’s experience in splenic surgery and the equipment available at the particular hospital. In trauma cases conservation of the spleen should not take significantly more time than a splenectomy. The best course with deep lacerations is splenorrhaphy with absorbable mesh; this quickly and effectively arrests bleeding. With this method the first step is identical to splenectomy (i.e. complete mobilization of the spleen and rotation onto the abdominal wall).

If it appears at this point that the organ cannot be saved, a splenectomy can be performed immediately without loss of time. Fast, complete mobilization of the spleen with avoidance of any further damage is of vital importance with regard to the decision whether the organ can be preserved. If the decision is to preserve the spleen, it is already in the correct position for further measures.

**Partial resection**

Injuries involving only one pole of the spleen can be treated with partial resection. Before resection the spleen should be mobilized. Stapler resection makes organ conservation possible in many cases, and it represents a valuable alternative to sutured partial splenectomy or splenorrhaphy. Its greatest advantages are simplicity of use, the practicality of the instrument itself and the reduction in time and blood transfusion.

**Use of compressive mesh**

An appropriately sized piece of mesh (Vicryl) with an absorbable thread is wrapped around the spleen; the string is pulled together on the hilar surface to effect haemostasis without compromising intraparenchymal circulation. It is extremely important that the pouch should be slightly smaller than the spleen, so that the suture lies on the acute and obtuse margin when it has been pulled taut. After using one of the above-described surgical techniques, definitive treatment can be completed by application of adhesives to secure the resected edge or the mesh-covered splenic tissue.

**Hepatic injury**

Repair and resection for treatment of hepatic trauma demand a working knowledge of the anatomy of the liver, including the arterial supply, portal venous supply and hepatic venous drainage. Segmental anatomical resection has been well documented, but is usually not applicable to trauma. Knowledge of the anatomy is important, and its understanding also helps explain some of the patterns of injury after blunt trauma. In addition, there are differences in tissue elasticity, which also determine injury patterns.

The forces from blunt injury are usually direct compressive forces or shear forces. The elastic tissue within
arterial blood vessels makes them less susceptible to tearing than any other structures within the liver. Venous and biliary ductal tissue are moderately resistant to shear forces, whereas the liver parenchyma is the least resistant of all. Thus, fractures within the liver parenchyma tend to occur along segmental fissures or directly in the parenchyma. This causes shearing of branches lateral to the major hepatic and portal veins. With severe deceleration injury, the origin of the short retrohepatic veins may be ripped from the cava, causing devastating haemorrhage. Similarly, the small branches from the caudate lobe entering directly into the cava are at high risk for shearing with linear tears on the caval surface.

Direct compressive forces usually cause tearing between segmental fissures in an anteroposterior orientation. Horizontal fracture lines into the parenchyma give the characteristic burst pattern to such liver injuries. If the fracture lines are parallel, these have been dubbed 'bear-claw'-type injuries and probably represent where the ribs have been compressed directly into the parenchyma. This can cause massive haemorrhage if there is direct extension or continuity with the peritoneal cavity.

Diagnosis of hepatic trauma preoperatively may be difficult. The liver is at risk of damage in any penetrating trauma to the upper abdomen and lower thorax, especially of the right upper quadrant.

The treatment of severe liver injuries begins with temporary control of haemorrhage. This is best achieved in the first instance by direct manual compression of the liver. The goal is to try to restore the normal liver anatomy by manual compression and packing. Most catastrophic bleeding from hepatic injury is venous in nature and can therefore be controlled by direct compression and liver packs. Liver packing can be definitive treatment, particularly when there is bilobar injury, or it can simply buy time if the patient develops a coagulopathy or hypothermia, or there are no blood resources. Liver packing is the method of choice where expertise in more sophisticated techniques is not available. If packing is successful, and the bleeding is controlled, no further action may be needed.

Packing is performed using large flat abdominal packs, placed laterally, below and around the liver. If necessary, the liver can be mobilized by division of the hepatic ligaments. Packs must not be forced into any splits or fractures because this increases the damage and encourages haemorrhage. During the period of time that the packs are placed, it is important to establish more intravenous access lines and other monitoring devices as needed. Hypothermia should be anticipated and corrective measures taken. After haemodynamic stability has been achieved, the packs are removed, and the injury to the liver rapidly assessed. The control of haemorrhage is the first consideration, followed by control of contamination. If in doubt, apply damage control techniques.

If packing does not control bleeding, it is prudent at this time to enter the gastrohepatic ligament using either sharp or blunt dissection, so that a vascular clamp can be placed across the portal triad. With gentle traction on the dome of the liver, a sudden gush of blood should make the surgeon suspicious of injury to the hepatic venous system. If there is bright red blood pouring from the parenchyma, it is then appropriate to apply a vascular clamp to the porta hepatis (Pringle's manoeuvre). If this controls the bleeding, the surgeon should be suspicious of hepatic arterial or possible portal venous injury. Dissection of the porta hepatis should then be carried out and selective clamping of vessels performed to determine the source of haemorrhage. Hepatorrhaphy is then performed by extending the lacerations, getting into the depths of wounds and controlling the bleeding vessels with ligatures.

It may be necessary to perform ‘finger fracture’ through normal live tissue, to get to the injured vessels deep in the parenchyma. The normal capsule is 'scored' using diathermy or a scalpel. Then the normal liver tissue is gently compressed between the thumb and forefinger, rubbing the normal parenchymal tissue away, leaving just the intact vessels for ligation or clipping. Avoid forceful pinching or crushing of the liver tissue, because this may disrupt the hepatic vasculature, increasing the haemorrhage.

The options for hepatic vein and cava injuries include direct compression and extension of the laceration as mentioned above, atriocaval shunt, non-shunt isolation (Heaney’s technique) and venovenous bypass.

Pancreatic injury

Penetrating pancreatic trauma should be obvious because the patient will almost invariably have been explored for an obvious injury. Once the retroperitoneum has been violated in penetrating trauma, it is imperative for the surgeon to do a thorough exploration in the central region. This includes an extended Kocher manoeuvre, taking down the ligament of Treitz, opening
up the lesser sac from gastrohepatic and omentocolic access points, and particularly visualizing the tail of the pancreas as it extends into the splenic hilum. Any parenchymal haematoma of the pancreas should be thoroughly explored, including irrigation of the haematoma. Consideration should be given to pancreatic duct contrast studies, although this may be difficult during laparotomy and should be avoided if possible.

Pancreatic trauma
Parenchymal lacerations that do not involve the pancreatic duct can be sutured when the tissue is not too soft and vulnerable. With or without sutures, a worthwhile option in the treatment of such lacerations is fibrin sealing and collagen tamponade, and adequate drainage is essential.

When ductal injury to the body and/or the tail of the pancreas is suspected, the best and safest treatment is resection. In the case of severe injuries, therapeutic options range from drainage alone to Whipple’s procedure. The latter is a rarely used option with a high incidence of morbidity and mortality. A good, effective and safe option is pyloric exclusion with drainage of the injured area. With concomitant duodenal injuries an additional duodenal tube is necessary. If there is obvious disruption to the pancreatic duct, it should be ligated with distal pancreatic resection.

Diagnosis of blunt pancreatic trauma is much more problematic. As the pancreas is a retroperitoneal organ there may be no anterior peritoneal signs. History can be helpful if information from the paramedics indicates that the steering column was bent or if the patient can give a history of epigastric trauma. The physical examination, as stated above, is often misleading. However, a 'doughy' abdomen should make the clinician suspicious. Amylase and full blood count (FBC) are non-specific. Diagnostic peritoneal lavage and FAST are unhelpful. Gastrograffin swallow has a fair sensitivity, CT is at least 85 per cent accurate and remains the non-operative diagnostic modality of choice for blunt pancreatic injury. Endoscopic retrograde cholangiopancreatography (ERCP) can be helpful in selected patients.

Injuries to the tail and body of the pancreas are either can be drained or, if a strong suspicion for major ductal injury is present, resection can be carried out with good results. The injuries that vex the surgeon most, however, are those to the head of the gland, particularly those juxtaposed with, or also involving, the duodenum. Resection (Whipple’s procedure) is usually reserved for those patients who have destructive injuries or those in whom the blood supply to the duodenum and pancreatic head has been embarrassed. The remainder are usually treated with variations of drainage and pyloric exclusion. This includes extensive closed (suction) drainage around the injury site. Common duct drainage is not indicated.

Aorta and inferior vena caval injuries
Aorta and caval injuries are primarily a problem of access (rapid) and control of haemorrhage. If the surgeon opens the abdomen and there is extensive retroperitoneal bleeding centrally, there are two options:

1. If the bleeding is primarily venous in nature, the right colon should be mobilized to the midline, including the duodenum and head of pancreas. This will expose the infrarenal cava and infrarenal aorta. It will also facilitate access to the portal vein.

2. If the bleeding is primarily arterial in nature, it is best to approach the injury from the left. This includes taking down the left colon and mobilizing the pancreas and spleen to the midline. Access to the posterior aorta includes mobilizing the left kidney. By approaching the aorta from the left lateral position, it is possible to identify the plane of Leriche more rapidly than by approaching it through the lesser sac. The problem is the coeliac and superior mesenteric ganglion, which can be quite dense and hinder dissection around the origins of the coeliac and superior mesenteric artery. Additional exposure can be obtained simply by dividing the left crus of the diaphragm. This will allow proximal control of the abdominal aorta until complete dissection of the visceral vessels can be accomplished. The exception is in the area of the coeliac ganglion, which can contain aortic haemorrhage from significant injuries, and which may require short segmental graft replacement.

Treatment of aortic or caval injuries is usually straightforward. Extensive lacerations are not compatible with survival and it is uncommon to require graft material to repair the aorta. Caval injuries below the renal veins, if extensive, can be ligated, although lateral repair is preferred. Injuries above the renal veins in the cava should be repaired if at all possible, including onlay graft of autogenous tissue.
Retroperitoneum

Injuries to retroperitoneal structures associated with a high mortality are often underestimated or missed. Rapid and efficient access techniques are required to deal with exsanguinating vascular injuries, where large retroperitoneal haematomas often obscure the exact position and extent of the injury.

The retroperitoneum is explored when major abdominal vascular injury is suspected, or there is injury to the kidneys, ureters and renal vessels, pancreas, duodenum and colon. As a result of the high incidence of intraperitoneal and retroperitoneal injuries occurring simultaneously, the retroperitoneum is always approached via a transperitoneal incision. The patient is prepared and draped to expose widely the chest, abdomen, and both groins and thighs. A long midline laparotomy is performed and the peritoneal cavity systematically explored. Major bleeding sites and gastrointestinal soiling are temporarily controlled. In the event of severe hypotension, the aorta can be compressed or occluded at the hiatus, or transthoracic cross-clamping can be done before opening the abdomen (see Section 5.2, Access to the thorax).

The decision to explore a retroperitoneal haematoma is based on its location, the mechanism of injury and whether the haematoma is pulsating or rapidly enlarging.

The retroperitoneum is divided into:

- A central zone (zone 1)
- Two lateral zones (zone 2)
- A pelvic zone (zone 3).

If the haematoma is not expanding, then other abdominal injuries take priority. If the haematoma is expanding, it must be explored. Before the haematoma is opened, it is important to try to gain proximal and distal control of vessels supplying the area. Direct compression with abdominal swabs and digital pressure may help to ‘buy time’ while vascular control is being obtained.

Lateral haematomas need not be explored routinely, unless perforation of the colon is thought to have occurred. The source of bleeding is usually the kidney and, unless expanding, will probably not require surgery.

Pelvic haematomas should not be explored if it can be avoided. It is preferable to perform a combination of external fixation on the pelvis and angiographic embolization. Attempts at tying internal iliac vessels are usually unsuccessful. Expanding pelvic haematomas should be packed. There is some evidence to suggest that extraperitoneal packing might be more effective than intraperitoneal pelvic packing.

Upper midline, central, retroperitoneal haematomas should be explored to rule out underlying duodenal, pancreatic or vascular injuries. It is wise to ensure that proximal and distal control of the aorta and distal control of the inferior vena cava can be rapidly achieved, before the haematoma is explored.

The stomach is grasped and pulled inferiorly, allowing the surgeon to identify the lesser curvature and the pancreas through the lesser sac. Frequently, the coeliac artery and the body of the pancreas can be identified well through this approach. The omentum is then grasped and drawn upward. A window is made in the omentum (via the gastrocolic ligament), and the surgeon’s hand is passed into the lesser sac posterior to the stomach. This allows excellent exposure of the entire body and tail of the pancreas, and the posterior aspect of the proximal part of the first portion of the duodenum and the medial aspect of the second part. Any injuries to the pancreas can be easily identified. If there is a possibility of an injury to the head of the pancreas, then Kocher’s manoeuvre is performed. Better exposure can be achieved using the right medial visceral rotation.

Complex pelvic fractures

Complex pelvic fractures with open pelvic injury can be the most difficult of all injuries to treat. Initially, they can cause devastating haemorrhage and may later be associated with overwhelming pelvic sepsis and distant multiple organ failure.

Diagnosis

For those patients who present with compound pelvic fractures and are haemodynamically stable, diagnostic studies such as plain films of the pelvis, CT and occasionally an arteriogram should be carried out rapidly, particularly if the patient was initially unstable and has been resuscitated, and there is a margin of time to do the arteriogram safely.

Initial management

In all cases there should be some early attempt to reduce the amount of bleeding from the pelvis by binding it together. This can be done with a sheet wrapped tightly around the pelvis at the level of the greater trochanters, or a proprietary pelvic binder. In vertical shear fractures
with dislocation, traction should be applied to the leg on the side of cranial dislocation of the pelvis, before binding the pelvis together. These manoeuvres attempt to align the fractures anatomically, to prevent further movement and reduce further blood loss. More formal fixation can be performed in the operating theatre when time allows.

For patients with haemodynamic instability, urgent external stabilization of the pelvic bones, followed by angiography and embolization, may be required.

Surgery
All open pelvic fractures should be taken to the operating theatre as soon as the necessary diagnostic studies have been carried out. In the case of the patient who is haemodynamically too unstable for angiography and embolization, resuscitation is optimized in the operating theatre. The priorities facing the surgeon are to control the pelvic haemorrhage and rule out other intra-abdominal organ injury with associated haemorrhage. Sometimes it is prudent to perform a rapid laparotomy to rule out additional haemorrhage. Stabilization of pelvic bleeding can be temporarily achieved by packing the wound and then making the decision whether or not to obtain a pelvic arteriogram (which will be positive in 15 per cent of cases) or to move rapidly to external fixation of the anterior pelvis and consideration for posterior stabilization as well. When damage control surgery is necessary, extraperitoneal pelvic packing should be considered. These decisions are made on an individual basis, taking into account the patient’s status, the injury pattern and the surgeon’s experience in dealing with these complex injuries.

Based on the location of the injury, colostomy may be required in order to prevent soilage of the wound in the post-injury period. In general, all injuries involving the perineum and perianal area should have a diverting colostomy. However, in the damage control situation, establishment of a colostomy should be postponed until the patient’s physiology has returned to normal.

All vaginal injuries should be explored under a general anaesthetic. Vaginal lacerations should be managed as follows:

- High lesions should be repaired and closed.
- Lower lesions should be packed.

After the initial haemorrhage has been controlled by external fixation of the pelvis, the patient should be reviewed daily, in either the operating theatre or the intensive care unit (ICU), with wound examination, debridement and removal of packs when possible. A caveat of pack removal is that, the longer the packs are left in, the greater the risk of pelvic sepsis. Acetabular fractures can be addressed later and reconstructive procedures started as soon as pelvic sepsis is controlled.

If adequate orthopaedic experience is unavailable, consideration should be given to early transfer of this patient to an institution with the necessary expertise, as soon as the patient’s condition allows.

6.1.1.5 TISSUE ADHESIVES IN TRAUMA

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

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; the fibrin is applied with a sprayer. The necessary volume of blood (125 mL) can already be drawn in the emergency department and the autologous adhesive is ready within 30 min.

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. Heterologous fibrin is a biological two-component adhesive and has high concentrations of fibrinogen and factor XIII, which, together with thrombin, calcium and aprotinin, 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.

Tachosil 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 use. It is available in most countries in Europe.
Even after surgical haemostasis, deep parenchymal injuries can require a resorbable tamponade; here, collagen fleece is suitable. Collagen fleece is composed of heterologous collagen fibrils obtained from devitalized connective tissue and is fully resorbable, e.g. Tissofleece. 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.

Application
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 is first sprayed alone, followed by short bursts of fibrin. This creates a surface that is free of blood and almost 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 depends on the size and depth of the injury.

Special situations
Hepatic injury
In severe liver injury, after successful surgical treatment including removal of devascularized necrotic tissue and resectional debridement, the liver is packed and the injured area compressed with warm pads. After complete exploration of the abdomen and treatment of other injuries and sources of bleeding, the liver packs are removed and any slight oozing on the surface of the liver can be arrested by sealing with fibrin and collagen fleece as described above. Fibrin glue cannot, however, compensate for inadequate surgical technique.

Splenic injury
When possible, in the stable patient, the surgeon should try to achieve splenic repair that preserves as much of the damaged spleen as possible. For splenic preservation, the choice of procedure depends not only on the clinical findings, but also on the surgeon’s experience of splenic surgery and the equipment available. In trauma cases, conservation of the spleen should not take significantly more time than a splenectomy.

After using one of the above-described surgical techniques, definitive treatment can be completed by application of adhesives to secure the resected edge or the mesh-covered splenic tissue. Fibrin is sprayed on and the collagen fleece pressed on it for a few minutes. After removal of the compressing pad a new layer of fibrin glue can help to ensure the prevention of rebleeding. In cases where mesh is used, the collagen fleece and fibrin are placed directly on the injured splenic surface and then covered with the mesh. Additional fibrin spray may be then added.

Pancreatic injury
When pancreatic injury is suspected, extended exploration of the whole organ is imperative. Parenchymal lacerations that do not involve the pancreatic duct can be sutured when the tissue is not too soft and vulnerable. With or without sutures a worthwhile option in the treatment of such lacerations is fibrin sealing and collagen tamponade, for which adequate drainage is essential.

Bowel injury
Serosal sutures and primary or secondary anastomoses can be secured with adhesives. Covering the anastomosis with collagen fleece can potentially prevent leakage.

Retroperitoneal haematoma
Injuries to the retroperitoneal vessels can cause haematomas of varying size, depending on the calibre of the vessels injured and the severity of the injury. Retroperitoneal haematomas can be treated by packing after surgical control of injured vessels, and followed by catheter embolization.

When the patient is stable, the packs can be removed after 24–48 h. Rebleeding after removal of the packs can necessitate re-packing. Slight bleeding can, however, be stopped effectively by spraying on adhesives.

6.1.2 The trauma laparotomy

Temperature control is fundamental. Preparatory measures should be taken before commencement of the procedure. These include warming of the operating theatre, warming of
all fluids infused, and warming of anaesthetic gases and external warming devices such as a Bair Hugger.

Haemorrhage control

Massive haemoperitoneum must be controlled before proceeding further with a laparotomy. The abdomen must be opened with a full-length incision to allow the best visibility to find any site of bleeding. Preparations must be made for collection of blood and possible autotransfusion if indicated.

1 As soon as the abdomen has been opened, scoop out as much blood as possible into a receiver.

2 Eviscerate the small bowel.

3 Perform a rapid exploration to ascertain whether there is an obvious site of large volume (audible!) bleeding. If so, this should be controlled with direct pressure, or proximal control, e.g. on the aorta.

4 Place large packs:
   – under the left diaphragm
   – in the left paracolic gutter
   – in the pelvis
   – in the right paracolic gutter
   – into the subhepatic pouch
   – above and lateral to the liver
   – directly on any other bleeding area.

5 Allow the anaesthetist to achieve an adequate blood pressure and to establish any lines required.

6 Remove the packs, one at a time, starting in the area least likely to be the site of the bleeding:
   – the packs in the left upper quadrant are removed and, if there is associated bleeding from the spleen, a decision should be made whether the spleen should be preserved or removed. A vascular clamp placed across the hilum will allow temporary haemorrhage control.
   – the packs are removed from the right upper quadrant, and injury to the liver assessed. It is prudent at this time to dissect the gastrohepatic ligament using blunt and sharp dissection so that a vascular clamp can be placed across the portal triad (Pringle’s manoeuvre). If this controls the injury, the surgeon should be suspicious of hepatic arterial or portal injury.
   – dissection of the porta hepatis should then be carried out, and selective clamping of vessels performed to determine the source of the haemorrhage.
   – hepatorrhaphy is then performed to control intrahepatic vessels (see under Section 6.2, Liver).

7 Deal with lesions in order of their lethality:
   – injuries to major blood vessels
   – major haemorrhage from solid abdominal viscera
   – haemorrhage from mesentery and hollow organs
   – contamination.

Be prepared to convert to a damage control procedure, as appropriate.

6.1.2.1 SURGICAL APPROACH

Preoperative antibiotics are given according to the recommendations in the Trauma Practice Management Guidelines of the Eastern Association for the Surgery of Trauma.

The abdomen

Incision

In trauma it is essential to be able to extend the access of the incision if required. All patients should therefore have both thorax and abdomen prepared and draped to allow access to the thorax, abdomen and groins if required (Figure 6.1). Minimization of patient hypothermia by raising the operating theatre temperature to a higher than normal level, and warming the patient with warm air blankets, warmed intravenous fluids and warmed anaesthetic gases, is very important.

All patients undergoing a laparotomy for abdominal trauma should be explored through a long midline incision. The incision is generally placed through, or to the left of, the umbilicus, to avoid the falciform ligament. The incision is made from the xiphisternum to the pubis. If necessary, this can be extended into a sternotomy, or extended right or left as a thoracotomy for access to the liver, diaphragm, etc.

In patients who have had significant previous surgery, and with gross haemodynamic instability, a bilateral subcostal (‘clamshell’ or ‘chevron’) incision, extending from the anterior axillary line on each side, transversely across the midline just superior to the umbilicus, can be used.

A quick exploratory ‘trauma laparotomy’ is performed to identify any other associated injuries.

The ligament of Treitz

The ligament of Treitz suspends the distal duodenum and proximal jejunum from the diaphragm. Access is best from
the left side. Division of this ligament facilitates exposure of these areas of bowel. Care should be taken to identify the inferior mesenteric vein during this dissection.

**Kocher’s manoeuvre**
The hepatic flexure is retracted medially, dividing adhesions along its lateral border down to the caecum (Figure 6.2).

Initially Kocher’s manoeuvre is performed by dividing the lateral peritoneal attachment of the duodenum. The adhesions on the outer border of the duodenum are divided from below, upwards, allowing the medial rotation of the duodenum (Figure 6.3).

The posterior wall of the duodenum can be inspected, together with the right kidney, porta hepatis and inferior vena cava (Figure 6.4). By reflecting the duodenum and pancreas towards the anterior midline, the posterior surface of the head of the pancreas can be completely inspected (Figure 6.5).

**Right medial visceral rotation**
The small bowel mesentery is mobilized, and the right retroperitoneum is also exposed (see Figure 6.4). The small bowel mobilization is undertaken by sharply incising its retroperitoneal attachments from the right lower quadrant, to the ligament of Treitz. The entire ascending colon, and caecum are then reflected superiorly towards the left upper quadrant of the abdomen. This gives excellent exposure to the entire inferior vena cava, the aorta, and the third and fourth parts of the duodenum.

As the dissection is carried further, the inferior border of the entire pancreas can be identified and any injuries inspected. Severe oedema, crepitation or bile staining of the periduodenal tissues implies a duodenal injury until proved otherwise. If the exploration of the duodenum is negative, but there is still strong suspicion of duodenal injury, methylene blue can be instilled through the nasogastric tube. Rapid staining of periduodenal tissues is unmistakable evidence of an intestinal
leak in this area, and the lack of staining has proved reliable in ruling out full-thickness duodenal injury. Mobilization of the whole duodenum is mandatory for exclusion of duodenal injury.

These manoeuvres allow for complete exposure of the first, second, third and fourth parts of the duodenum, along with the head, neck and proximal body of the pancreas (Figure 6.5). Access to the vena cava is also facilitated.

Exposure for repair of the aorta, distal body and tail of pancreas is not ideal with the right medial visceral rotation, and better exposure can be obtained by performing a left medial visceral rotation.

Left medial visceral rotation

The left medial visceral rotation procedure: this entails mobilization of the splenorenal ligament and incision of the peritoneal reflection in the left paracolic gutter, down to the level of the sigmoid colon (Figure 6.6). The left-sided viscera are then bluntly dissected free of the retroperitoneum and mobilized to the right. Care should be taken to remain in a plane anterior to Gerota’s fascia. The entire abdominal aorta and the origins of its branches are exposed by this technique, including the coeliac axis, origin of the superior mesenteric artery, iliac vessels and left renal pedicle. The dense and fibrous superior mesenteric and coeliac nerve plexus overlie the proximal aorta and need to be sharply dissected in order to identify the renal and superior mesenteric arteries.

6.1.2.2 THE RETROPERITONEUM

The stomach is grasped and pulled inferiorly, allowing the operator to identify the lesser curvature and the pancreas through the lesser sac. Frequently, the coeliac artery and the body of the pancreas can be well identified through this approach. The omentum is then grasped and drawn upward. A window is made in the omentum (via the gastrocolic ligament) and the surgeon’s hand is passed into the lesser sac posterior to the stomach. This allows excellent exposure of the entire body and tail of the pancreas, the posterior aspect of the

Figure 6.3 Mobilization of the duodenum (Kocher’s manoeuvre).

Figure 6.4 Reflection of the duodenum and right hemicolon to show the right kidney and inferior vena cava.
proximal part of the first portion of the duodenum and the medial aspect of the second part. Any injuries to the pancreas can be easily identified. If there is a possibility of an injury to the head of the pancreas, Kocher’s manoeuvre is performed. Better exposure can be achieved using the right medial visceral rotation.

Kocher’s manoeuvre is performed by initially dividing the lateral peritoneal attachment of the duodenum. The loose areolar tissue around the duodenum is bluntly dissected, and the entire second and third portions of the duodenum are identified and mobilized medially, with a combination of sharp and blunt dissection. This dissection is carried all the way medial to expose the inferior vena cava and a portion of the aorta.

By deflecting the duodenum and pancreas towards the anterior midline, the posterior surface of the head of the pancreas can be completely inspected. Better inspection of the third part and inspection of the fourth part of the duodenum may be achieved by mobilizing the ligament of Treitz and performing the Cattell and Braasch manoeuvre mentioned earlier. This manoeuvre requires mobilization of the right colon (including the hepatic flexure) from right to left, so the right colon and small intestine can be elevated. The small bowel mobilization is undertaken by sharply incising its retroperitoneal attachments from the lower right quadrant to the ligament of Treitz. The entire ascending colon and caecum are then reflected superiorly towards the left upper quadrant of the abdomen. This gives excellent exposure of the entire vena cava, the aorta, and the third and fourth portions of the duodenum.

As the dissection is carried further, the inferior border of the entire pancreas can be identified and any injuries inspected. These manoeuvres allow for complete exposure of the first, second, third and fourth portions of the duodenum along with the head, neck, body and tail of the pancreas.

Medial rotation of the left side of the abdominal contents can be performed by mobilizing the spleen and descending colon, and by medial rotation of the spleen,
descending colon and sigmoid colon to the right (left medial visceral rotation). This allows inspection of the left kidney, retroperitoneum and tail of the pancreas. If vascular access to the kidney is required, Gerota’s fascia should be divided on the lateral aspect of the kidney, and the kidney rotated medially to allow access to the renal hilum.

Pelvic haematomas should not be explored. It is preferable to perform a combination of external fixation on the pelvis, pelvic packing and angiographic embolization. Attempts at tying internal iliac vessels are usually unsuccessful.

It is important to replace the small intestine in the abdominal cavity with great care at the conclusion of the operation. Iatrogenic volvulus of the mobilized bowel is possible if such care is not taken. Severe oedema, crepitation, or bile staining of the periduodenal tissues, implies a duodenal injury until proved otherwise. If, as with the Cattel and Braasch manoeuvre, the exploration of the duodenum is negative but there is still strong suspicion of duodenal injury, methylene blue can be instilled through the nasogastric tube. Rapid staining of periduodenal tissues is unmistakable evidence of an intestinal leak in this area, and the lack of staining has proved reliable in ruling out full-thickness duodenal injury. Mobilization of the whole duodenum is mandatory for exclusion of duodenal injury.

6.1.3 References


6.1.4 Recommended reading


6.2 THE LIVER AND BILIARY SYSTEM

6.2.1 Overview

Any surgeon who has been confronted with a patient exsanguinating from a liver injury will attest to the sense of helplessness experienced. To improve the outcome of these severely injured patients, the surgeon requires the following:

- A thorough understanding of the pathophysiology of severe hepatic injury
- A knowledge of the liver: both gross and segmental anatomy including the arterial and venous supply
- A complete armamentarium of surgical techniques and ‘tricks of the trade’
- A team approach in the operating and resuscitating environment with all necessary equipment prepared.

Appropriate decision-making is critical to a good outcome. As a general rule, the simplest, quickest technique that can restore haemostasis is the most appropriate. Once the patient is cold, coagulopathic and in irreversible shock, the battle is usually lost.

6.2.1.1 Resuscitation

Haemodynamically stable patients with no signs of peritonitis or other indication for surgery are increasingly being managed non-operatively.

All haemodynamically unstable patients with liver injuries are treated by surgical exploration and repair or haemostasis. The patient in whom a surgical approach is decided upon or who is mandated by haemodynamic instability should be transferred to the operating theatre as rapidly as possible after the following are completed:

- Emergency airway or ventilatory management if necessary
• Establishment of adequate upper limb, large-bore, vascular access and initiation of crystalloid resuscitation
• Ordering 6 units fresh (if available) whole blood or equivalent.

6.2.1.2 DIAGNOSIS

The patient’s surgery should not be delayed by multiple emergency department procedures such as limb radiographs, unnecessary ultrasonography and vascular access procedures. Computed tomography of the brain should be delayed until the patient is stable. The anaesthetist can continue resuscitation in the operating theatre.

In patients with blunt trauma there may be an absence of clear clinical signs such as rigidity, distension or unstable vital signs. Up to 40% per cent of patients with significant haemoperitoneum have no obvious signs. Focused abdominal sonography for trauma may be particularly useful in the setting of blunt injury and haemodynamic instability, because the presence of free fluid in the abdominal cavity will influence the need for operation. With a haemodynamically stable patient, CT is an invaluable diagnostic aid and allows the surgeon to make decisions on the need for embolization or operative management. Diagnostic peritoneal lavage is also quite useful, particularly when CT support services are inadequate.

The purpose of diagnostic investigation in the stable patient is to help identify those patients who can be safely managed non-operatively, assist decision-making in non-operative management and act as a baseline for comparison in future imaging studies. Accurate, good quality, contrast-enhanced CT has enhanced our ability to make an accurate diagnosis of liver injuries.

Penetrating wounds of the liver usually do not present a diagnostic problem, because most surgeons would advocate exploration of any wound in the unstable patient. Peritoneal lavage as a diagnostic tool in penetrating trauma has been misleading. CT scans using contrast are not routinely advocated for penetrating injuries, but can be useful, especially to delineate vascular viability, and assist with the decision of whether to treat the injury non-operatively, with or without embolization, or by conservation or resection.

6.2.1.3 LIVER INJURY SCALE

The American Association for the Surgery of Trauma’s Committee on Organ Injury Scaling has developed a grading system for classifying injuries to the liver (Table 6.1).

Hepatic injuries are graded on a scale of I–VI, with I representing superficial lacerations and small subcapsular haematomas and VI representing avulsion of the liver from the vena cava. Isolated injuries that are not extensive (grades I–III) usually require little or no treatment; however, extensive parenchymal injuries and those involving the juxtahepatic veins (grades IV and V) may require complex manoeuvres for successful treatment, and hepatic avulsion (grade VI) is usually lethal.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Type of injury</th>
<th>Description of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Subcapsular, &lt; 10% surface area</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, &lt; 1 cm parenchymal depth</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Subcapsular, 10–50% surface area: intraparenchymal &lt; 10 cm in diameter</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear 1–3 cm parenchymal depth, &lt; 10 cm in length</td>
</tr>
<tr>
<td>III</td>
<td>Haematoma</td>
<td>Subcapsular, &gt; 50% surface area of ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma &gt; 10 cm or expanding 3 cm parenchymal depth</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Parenchymal disruption involving 25–75% hepatic lobe or one to three Couinaud’s segments</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Parenchymal disruption involving &gt; 75% of hepatic lobe or more than three Couinaud’s segments within a single lobe</td>
</tr>
<tr>
<td>V</td>
<td>Vascular</td>
<td>Juxtahepatic venous injuries, i.e. retrohepatic vena cava/central major hepatic veins</td>
</tr>
<tr>
<td>VI</td>
<td>Vascular</td>
<td>Hepatic avulsion</td>
</tr>
</tbody>
</table>

aAdvance one grade for multiple injuries up to grade IV.

From Moore et al.¹

¹From Moore et al.¹
6.2.1.4 MANAGEMENT

Traditionally, discussion of liver injuries differentiates those arising from blunt trauma from those arising from penetrating trauma. Most stab wounds cause minor liver injuries unless a critical structure, such as the hepatic vein, intrahepatic cava or portal structures, is injured. In contrast, gunshot wounds can be quite devastating, particularly medium-velocity, high-velocity and shotgun blasts. Injuries from severe blunt trauma continue to be the most challenging for the surgeon. Twenty-five per cent of penetrating injuries to the liver can be managed non-operatively.

Richardson2 and co-workers managed approximately 1200 blunt hepatic injuries over a 25-year period. Non-operative management (NOM) was used in up to 80 per cent of cases. Deaths secondary to injury dropped from 8 per cent to 2 per cent.

Non-operative management3

Almost all children and 50–80 per cent of adults with blunt hepatic injuries are treated without laparotomy. This change in approach has been occasioned by the increasing availability of rapid ultrasonography and helical CT, and the development of interventional radiology.

The primary requirement for non-operative therapy is haemodynamic stability. To confirm stability, frequent assessment of vital signs and monitoring of the haematocrit are necessary, in association with CT scans as required. Continued haemorrhage occurs in 1–4 per cent of patients. Hypotension may develop, usually within the first 24 h of hepatic injury, but sometimes several days later.

The presence of extravasation of contrast on CT denotes arterial haemorrhage. There should be a low threshold for performance of diagnostic or therapeutic angiography and embolization. Otherwise, operative intervention will become necessary in these patients.

A persistently falling haematocrit should be treated with packed red blood cell (PRBC) transfusions. If the haematocrit continues to fall after 2 or 3 units of PRBCs, embolization in the interventional radiology suite should be considered. Overall, non-operative treatment obviates laparotomy in more than 95 per cent of cases.

Operative management

Most injuries (approximately 60–70 per cent) are managed simply by evacuating the free intraperitoneal blood, and some will require drainage of the injury because of possible bile leak. Many of these patients can be managed non-surgically. Caution must be exercised because bile within the peritoneal cavity is not always benign.

Twenty-five per cent of liver injuries will require direct control of the bleeding. This is best achieved in the first instance by direct manual compression of the liver. The goal is to try to restore the normal anatomy by manual compression and packing. If this is successful, and the bleeding is controlled, no further action may be required.

If compression and packing are unsuccessful, it is necessary to achieve direct access to the bleeding vessel and direct suture ligation. This will often necessitate extension of the wound to gain access and view the bleeding point. During this direct access, bleeding can be temporarly controlled by direct compression, which requires a capable assistant. Temporary clamping of the porta hepatis (Pringle’s manoeuvre) is also a useful adjunctive measure. Other adjunctive measures include interruption of the venous or arterial inflow to a segment or lobe (< 1 per cent of all liver injuries), haemostatic agents such as crystallized bovine collagen, fibrin adhesives, gelfoam, use of the argon laser and the hot air gun.

Approximately 5 per cent of all liver injuries will require resection or debridement, and subtotal or total lobectomy. In most instances the indication for such surgery will be determined by the injury. Resectional therapy almost invariably requires drainage of some type. A useful adjunctive measure is to use a pedicle of omentum over the cut surface.

Approximately 2 per cent of all liver injuries are complex and represent injuries to major hepatic venous structures – portal triad, intrahepatic cava – or are difficult to control because of hypothermia and coagulopathy. Injuries to the hepatic vein or intrahepatic cava can be approached in the following ways:

- Direct compression and definitive repair
- Intracaval shunt
- Temporary clamping of the porta hepatis, suprarenal cava and suprahepatic cava (vascular isolation)
- Venovenous bypass
- Packing.

Direct compression and control of hepatic venous injuries can be accomplished in some patients. The significant liver injury requires manual compression and simultaneous medial rotation and retraction – a difficult manoeuvre. In such a situation the most senior surgeon
should be the one doing the direct compression and the assistant should do the actual suturing of the hepatic vein or cava.

The intracaval shunt has been maligned because only 25–35 per cent of these patients survive their injury. Usually this is a result of the use of the device late in the course of treatment when the patient has already developed coagulopathy and is premorbid. If the shunt is to be used, this decision should be made early and ideally before massive transfusion.

Hepatic isolation, by clamping of the porta hepatis, suprarenal cava and suprahepatic cava can be done on a temporary basis. This requires considerable experience by the anaesthetist and a surgeon capable of dealing with the problems rapidly.

Venovenous bypass has been used successfully in liver transplant surgery and, with new heparin free pumps and tubing, it is possible to use this in the trauma patient.

In some patients who have bilobar injuries with extensive bleeding, or in patients who have developed coagulopathy secondary to massive transfusions and hypothermia, it may be prudent to institute damage control procedures (temporarily pack the injury and take the patient to the intensive care setting, warm him or her up, correct the coagulopathy, and return to surgery when additional resources such as relatively fresh whole blood have been obtained). Packing may often be used as definitive treatment. Dexon mesh and omental pedicles have also been advocated for controlling severe lacerations.

Injuries to the porta hepatitis can also be exsanguinating. Right and left hepatic arteries can usually be managed by simple ligation, as can injuries to the common hepatic artery.

Injuries to the left or right portal vein can be ligated. Ligation of the portal vein has been reported to be successful; however, repair is recommended whenever possible.

### 6.2.1.5 SUBCAPSULAR HEMATOMA

An uncommon but troublesome hepatic injury is subcapsular haematoma, which arises when the parenchyma of the liver is disrupted by blunt trauma but Glisson’s capsule remains intact. Subcapsular haematomas range in severity from minor blisters on the surface of the liver to ruptured central haematomas accompanied by severe haemorrhage. They may be recognized either at the time of the operation or in the course of CT scanning. Regardless of how the lesion is diagnosed, subsequent decision-making is often difficult. If a grade I or II subcapsular haematoma (i.e. a haematoma involving < 50 per cent of the surface of the liver that is not expanding and is not ruptured) is discovered during an exploratory laparotomy, it should be left alone. If the haematoma is explored, hepatotomy with selective ligation may be required to control bleeding vessels.

Even if hepatotomy with ligation is effective, one must still contend with diffuse haemorrhage from the large denuded surface, and packing may also be required. A haematoma that is expanding during operation (grade III) may have to be explored. Such lesions are often the result of uncontrolled arterial haemorrhage, and packing alone may not be successful. An alternative strategy is to pack the liver to control venous haemorrhage, close the abdomen, and transport the patient to the interventional radiology suite for hepatic arteriography and embolization of the bleeding vessels. Ruptured grade III and IV haematomas are treated with exploration and selective ligation, with or without packing.

### 6.2.1.6 COMPLICATIONS

Overall mortality for patients with hepatic injuries is approximately 10 per cent. The most common cause of death is exsanguination, followed by multisystem organ dysfunction syndrome (MODS) and intracranial injury. Three generalizations may be made with regard to the risk of death and complications:

1. Both increase in proportion to the injury grade and to the complexity of repair
2. Hepatic injuries caused by blunt trauma carry a higher mortality than those caused by penetrating trauma
3. Infectious complications occur more often with penetrating trauma.

Postoperative haemorrhage occurs in a small percentage of patients with hepatic injuries. The source may be either a coagulopathy or a missed vascular injury (usually to an artery). In most instances of persistent postoperative haemorrhage, the patient is best served by being returned to the operating theatre. Arteriography with embolization may be considered in selected patients. If coagulation studies indicate that a coagulopathy is the likely cause of postoperative haemorrhage, then correction of the coagulopathy must be a critical part of the strategy.

Perihepatic infections occur in fewer than 5 per cent of patients with significant hepatic injuries. They
develop more often in patients with penetrating injuries than in patients with blunt injuries, presumably because of the greater frequency of enteric contamination. An elevated temperature and a rising white blood cell count should prompt a search for intra-abdominal infection. In the absence of pneumonia, an infected line or urinary tract infection, an abdominal CT scan with intravenous and upper gastrointestinal contrast should be obtained.

Many perihepatic infections (but not necrotic liver) can be treated with CT or ultrasound-guided drainage. In refractory cases, especially for posterior infections, right twelfth rib resection remains an excellent approach.

Bilomas are loculated collections of bile that may become infected. They are best drained percutaneously under radiological guidance. If a biloma is infected, it should be treated as an abscess; if it is sterile, it will eventually be resorbed.

Biliary ascites is caused by disruption of a major bile duct, and requires reoperation and the establishment of appropriate drainage. Even if the source of the leaking bile can be identified, primary repair of the injured duct can be difficult to achieve. It is best to wait until a firm fistulous communication is established with adequate drainage. Adjunctive, transduodenal drainage by endoscopic retrograde cholangiopancreatography (ERCP) and papillotomy (ductotomy), or stent placement, has recently been shown to be of benefit in selected cases.

Biliary fistulae occur in up to 15 per cent of patients with major hepatic injuries. They are usually of little consequence and generally close without specific treatment. In rare instances, a fistulous communication with intrathoracic structures forms in patients with associated diaphragmatic injuries, resulting in a bronchobiliary or pleurobiliary fistula. As a result of the pressure differential between the biliary tract and the thoracic cavity, most of these fistulas must be closed surgically.

Haemorrhage from hepatic injuries is often treated without identifying and controlling each bleeding vessel individually, and arterial pseudoaneurysms may develop as a consequence. As the pseudoaneurysm enlarges, it may rupture into the parenchyma of the liver, a bile duct or an adjacent branch of the portal vein. Rupture into a bile duct results in haemobilia, which is characterized by intermittent episodes of right upper quadrant pain, upper gastrointestinal haemorrhage and jaundice; rupture into a portal vein may result in portal vein hypertension with bleeding varices. Both of these complications are exceedingly rare and are best managed with hepatic arteriography and embolization.

6.2.1.7 REFERENCES

6.2.2 Access to the liver

6.2.2.1 EXPOSURE

The patient is placed in the supine position. Warming devices are placed around the upper body and lower limbs. The chest and abdomen are surgically prepared and draped. The instruments necessary to extend the incision into a sternotomy or thoracotomy must be available. A generous midline incision from pubis to xiphisternum is the minimum incision required. For the patient in extremis, a combined sternotomy and midline laparotomy approach is recommended from the outset in order to allow access for internal cardiac massage and vena caval vascular control. Supradiaphragmatic intrapericardial inferior vena caval control is often easier than abdominal control adjacent to a severe injury. An Omnittract- or Buckwalter-type automatic retractor greatly facilitates access.

6.2.2.2 INITIAL ACTIONS

Once the abdomen has been opened, intraperitoneal blood evacuated and bleeding controlled, and there is evidence of hepatic bleeding, the liver should be packed and the abdomen rapidly examined to exclude extrahepatic sites of blood loss. Autotransfusion should be considered. Once the anaesthetist has had an opportunity to restore intravascular volume and haemostasis has been achieved for any extrahepatic injury, the liver injury can then be approached. Most injuries require mobilization of the injured lobe to permit repair. Strong upward retraction of the right costal margin allows liver mobilization. The right lobe of the liver is mobilized by dividing the right coronary and right triangular
ligament. This can usually be done under vision but in the larger patient it can be accomplished blindly from the patient’s left side. Care must be taken to avoid injury to the lateral wall of the right hepatic vein or the adrenal gland. The left lobe can be easily mobilized by dividing the left triangular ligament under vision, avoiding injury to the left inferior phrenic vein and the left hepatic vein.

### 6.2.2.3 EXPOSURE OF THE LIVER

Access to the right lobe of the liver is restricted as a result of the right subcostal margin and the posterior attachments. The costal margin should be elevated, initially with a Morris retractor, and then with a Kelly or Deaver retractor. The right triangular and coronary ligaments are divided with scissors. The superior coronary ligament is divided, avoiding the lateral wall of the right hepatic vein. The inferior coronary ligament is divided, taking care not to injure the right adrenal gland (which is vulnerable because it lies directly beneath the peritoneal reflection) or the retrohepatic vena cava.

When the ligaments have been divided, the right lobe of the liver can be rotated medially into the surgical field. Mobilization of the left lobe is done under direct vision. Rotational retraction is combined with manual compression to minimize further injury or bleeding.

In the event of evidence of a retrohepatic haematoma, rotation of the right lobe of the liver should be avoided unless strong indications are present, and adequate expertise is available. Packing and transport to a higher-level centre may be a safer option!

If exposure of the junction of the hepatic veins and the retrohepatic vena cava is necessary, the midline abdominal incision can be extended by means of a median sternotomy. The pericardium and the diaphragm can then be divided in the direction of the inferior vena cava.

### 6.2.2.4 TECHNIQUES FOR TEMPORARY CONTROL OF HAEMORRHAGE

Injuries that have stopped bleeding at the time of exploration in a normotensive patient require no specific treatment, and such injuries should not be interfered with.

During treatment of a major hepatic injury, ongoing haemorrhage may pose an immediate threat to the patient’s life, and temporary control gives the anaesthetist time to restore the circulating volume before further blood loss occurs. Second, multiple bleeding sites are common with both blunt and penetrating trauma and, if the liver is not the highest priority, temporary control of hepatic bleeding allows repair of other injuries without unnecessary blood loss. The following are the most useful techniques for the control of hepatic haemorrhage:

- Perihepatic packing
- Electrocautery or argon beam coagulator
- Pringle’s manoeuvre
- Tourniquet or liver clamp application
- Haemostatic agents and glues
- Hepatic suture
- Finger-fracture hepatotomy and vessel ligation
- Tract tamponade balloons
- Tractotomy and direct suture ligation
- Mesh wrap
- Hepatic artery ligation
- Hepatic vascular isolation
- Techniques to control retrohepatic caval bleeding
- The atrio caval shunt
- Moore–Pilcher balloon
- Venovenous bypass.

**Perihepatic packing**

The philosophy of packing has altered, and packs for a liver wound should not be placed within the wound itself. The packs are used to restore the anatomical relationship of the components.

Perihepatic packing, with careful placement of packs, is capable of controlling haemorrhage from almost all hepatic injuries. The right costal margin is elevated, and packs placed over and around the bleeding site. Additional packs may be placed between the liver and the diaphragm, posteriorly and laterally, and between the liver and the anterior chest wall until the bleeding has been controlled. Several packs may be required to control the haemorrhage from an extensive right lobar injury. The minimum number of packs to achieve haemostasis should be used. Packing is not as effective for injuries of the left lobe, because, with the abdomen open, there is insufficient abdominal and thoracic wall anterior to the left lobe to provide adequate compression. Fortunately, haemorrhage from the left lobe can be controlled by dividing the left triangular and coronary ligaments and compressing the lobe between the hands.

- Consider angiography and embolization with damage control surgery.
• Packs should preferably be removed within 24–72 h.
• The packs should be carefully removed to avoid precipitating further bleeding.
• If there is no bleeding, the packs can be left out, and adequate drainage established.
• Necrotic tissue should be resected where possible.
• Ongoing bleeding mandates repacking or other haemostatic procedure and consideration of embolization.

Two complications may be encountered with the packing of hepatic injuries. First, tight packing compresses the inferior vena cava, decreases venous return, and reduces right ventricular filling; hypovolaemic patients may not tolerate the resultant decrease in cardiac output. Second, perihepatic packing forces the right diaphragm to move superiorly and impairs its motion; this may lead to increased airway pressures and decreased tidal volume.

Pringle’s manoeuvre
Pringle’s manoeuvre is often used as an adjunct to packing for the temporary control of haemorrhage. When encountering life-threatening haemorrhage from the liver, the hepatic pedicle should be compressed manually. The compression of the hepatic pedicle via the foramen of Winslow is known as Pringle’s manoeuvre. The liver should be packed as above. The hepatic pedicle is best clamped from the left side of the patient, by digitally dissecting a small hole in the lesser omentum, near the pedicle, and then placing a soft clamp over the pedicle from the left hand side, through the foramen of Winslow.

The advantage of this approach is the avoidance of injury to the structures within the hepatic pedicle, and the assurance that the clamp will be properly placed the first time. The pedicle can be left clamped for up to an hour. However, this is probably true only in the haemodynamically stable patient. In the shocked patient, Pringle’s manoeuvre should be performed only for about 15 min at a time, for fear of decreasing liver fibrinogen production and other consequences of hepatic ischaemia.

Pringle’s manoeuvre also allows the surgeon to distinguish between haemorrhage from branches of the hepatic artery or the portal vein, which ceases when the clamp is applied, and haemorrhage from the hepatic veins or the retrohepatic vena cava, which does not.

Hepatic tourniquet
Once the bleeding lobe has been mobilized, Penrose tubing can be wrapped around the liver near the anatomical division between the left and right lobes. The tubing is stretched until haemorrhage ceases, and tension is maintained by clamping the drain. Unfortunately, tourniquets are difficult to use, and they tend to slip off or tear through the parenchyma if placed over an injured area. An alternative is the use of a liver clamp; however, the application of such devices is hindered by the variability in the size and shape of the liver.

Haemostatic agents and glues (see Section 6.1.1.5: Tissue adhesives)
Fibrin adhesive has been used in treating both superficial and deep lacerations and appears to be the most effective topical agent. Some are suitable for injection deep into bleeding gunshot and stab wound tracts to prevent extensive dissection and blood loss. Others are more suitable for surface application. Fibrin adhesives are made by mixing concentrated human fibrinogen (cryoprecipitate) with a solution containing bovine thrombin and calcium.

Hepatic suture
Suturing of the hepatic parenchyma is often employed to control persistently bleeding, more superficial lacerations. If, however, the capsule of the liver has been stripped away by the injury, sutures that are tied over the capsule are far less effective.

The liver is usually sutured using a large curved needle with 0 or 2/0 resorbable sutures. The large diameter prevents the suture from pulling through Glisson’s capsule. For shallow lacerations, a simple continuous suture may be used to approximate the edges of the laceration. For deeper lacerations, interrupted horizontal mattress sutures may be placed parallel to the edges and tied over the capsule. The danger of suturing is that sutures tied too tight may cut off the blood supply to viable liver parenchyma, resulting in necrosis.

Most sources of venous haemorrhage can be managed with intraparenchymal sutures. Even injuries to the retrohepatic vena cava and the hepatic veins have been successfully tamponaded by closing the hepatic parenchyma over the bleeding vessels.

An adjunct to parenchymal suturing or hepatotomy is the use of the omentum to fill large defects in the liver and to buttress hepatic sutures. The rationale for this use of the omentum is that it provides an excellent source for macrophages and fills a potential dead space with viable tissue. In addition, the omentum can provide a
little extra support for parenchymal sutures, often enough to prevent them from cutting through Glisson’s capsule.

Tract tamponade balloons
These can be very useful in haemostasis of a tract, from stab or gunshot wounds. The balloon is threaded down the tract and inflated, to tamponade the bleeding. The balloon can be either manufactured by the surgeon using Penrose rubber tubing or use made of a commercially available tube (e.g. a Sengstaken–Blakemore tube for tamponade of oesophageal varices).

Mesh wrap
A technique that may be attempted if packing fails is to wrap the injured portion of the liver with a fine porous material (e.g. polyglycolic acid mesh) after the injured lobe has been mobilized. Using a continuous suture or a linear stapler, the surgeon constructs a tight-fitting stocking that encloses the injured lobe. Blood clots beneath the mesh, which results in tamponade of the hepatic injury. It is best to secure this mesh to the falci-form ligament once full mobilization is complete, in order to keep the mesh wrap from stripping off the liver.

Finger-fracture tractotomy
Finger-fracture tractotomy and direct vessel ligation is suitable for most grade III–V injuries with bleeding that the operator judges will not be controlled by suture alone.

Hepatic resection
In elective circumstances anatomical resection produces good results, but, in the uncontrolled circumstances of trauma, a mortality rate in excess of 50 per cent has been recorded. Resection should be reserved for patients with:

- extensive injuries of the lateral segments of the left lobe where bimanual compression is possible
- delayed lobectomy in patients where packing initially controls the haemorrhage, but where there is a segment of the liver that is non-viable
- almost free segments of liver
- devitalized liver at the time of pack removal.

Hepatic isolation
Hepatic vascular isolation is accomplished by executing Pringle’s manoeuvre, clamping the aorta at the diaphragm and clamping the inferior vena cava above the right kidney (suprarenal) and above the liver (suprarehepatic). The technique is not straightforward, and is best achieved by those experienced in its use. In patients scheduled for elective procedures, this technique has enjoyed almost uniform success but, in trauma patients, the results have been disappointing.

Hepatic shunts
The atriocaval shunt was designed to achieve hepatic vascular isolation while still permitting some venous blood from below the diaphragm to flow through the shunt into the right atrium.

A 9-mm endotracheal tube with an additional side hole cut into it (for return of blood into the right atrium) is introduced into the auricular appendage via a hole surrounded by a purse-string suture. The tube is passed into the inferior vena cava, and caudally so that the end of the tube lies infrahepatically, below infrahepatic liver damage. The cuff is then inflated. Blood passes into the tube from below and exits into the right atrium. The top of the tube is kept clamped (or can be used for additional blood transfusion). The suprahepatic inferior vena cava should be looped so as to prevent back bleeding down the inferior vena cava. Hepatic isolation is then completed with Pringle’s manoeuvre.

Care must be taken to avoid damage to the integral inflation channel for the balloon. An alternative to the atriocaval shunt is the Moore–Pilcher balloon. This device is inserted through the femoral vein and advanced into the retrohepatic vena cava. When the balloon is properly positioned and inflated, it occludes the hepatic veins and the vena cava, thus achieving vascular isolation. The catheter itself is hollow, and appropriately placed holes below the balloon permit blood to flow into the right atrium, in much the same way as the atriocaval shunt. The survival rate for patients with juxtahepatic venous injuries who are treated with this device is similar to that for patients treated with the atriocaval shunt.

Only occasional survivors have been reported.

6.2.2.5 PERIHEPATIC DRAINAGE
Several prospective and retrospective studies have demonstrated that the use of either Penrose or sump drains carries a higher risk of intra-abdominal infection than the use of either closed suction drains or no drains at all. It is clear that, if drains are to be used, closed suction devices are preferred. Patients who are initially
treated with perihepatic packing may also require drainage; however, drainage is not indicated at the initial procedure, given that the patient will be returned to the operating theatre within the next 48 h.

6.2.2.6 RECOMMENDED READING


6.2.3 Injury to the bile ducts and gallbladder

Injuries to the extrahepatic bile ducts, although rare, can be caused by either penetrating or blunt trauma. The diagnosis is usually made by noting the accumulation of bile in the upper quadrant during laparotomy for treatment of associated injuries.

Bile duct injuries can be divided into those below the confluence of the cystic duct and common duct and those above the cystic duct. Treatment of common bile duct (CBD) injuries after external trauma is complicated by the small size and thin wall of the normal duct.

For the lower ductal injuries (those injuries below the cystic duct), when the tissue loss is minimal, the lesion can be closed over a T-tube (as with exploration of the CBD for stones). A choledochoduodenostomy can be performed if the duodenum has not been injured. If the duodenum has been injured, or there is tissue loss, because the common duct is invariably small, a modification of the Carrel patch can be used.

In higher ductal injuries between the confluence of the cystic duct and the common duct and the hepatic parenchyma, a hepatico-jejunostomy with an internal splint is recommended. An adjunctive measure is to bring the roux-en-Y end to the subcutaneous tissue so that access can be gained later if a stricture develops. Percutaneous intubation of the roux-en-Y limb is then possible with dilatation of the anastomosis.

Treatment of injuries to the left or right hepatic duct is even more difficult. If only one hepatic duct is injured, a reasonable approach is to ligate it and deal with any infections or atrophy of the lobe rather than to attempt repair. If both ducts are injured, each should be intubated with a small catheter brought through the abdominal wall. Once the patient has recovered sufficiently, delayed repair is performed under elective conditions with a roux-en-Y hepato-jejunostomy.

6.2.3.1 RECOMMENDED READING


6.3 THE SPLEEN

6.3.1 Overview

The conventional management of splenic injury used to be splenectomy. Over the last 20 years, the importance of splenic preservation has been realized as a means of preventing overwhelming post-splenectomy infection (OPSI). The risk is said to be 0.5–1 per cent in adults, but in infants it is up to 50 per cent. The first aim of successful operative management is to control the active haemorrhage, and the second is to achieve surgical repair that preserves as much of the damaged organ as possible.

6.3.1.1 ANATOMY

The splenic artery, a branch of the coeliac axis, provides the principal blood supply to the spleen. The artery gives rise to a superior polar artery from which the short gastric arteries arise. The splenic artery also gives rise to superior and inferior terminal branches that enter the
splenic hilum. The artery and the splenic vein are embedded in the superior border of the pancreas.

Three avascular splenic suspensory ligaments maintain the intimate association of the spleen and diaphragm (splenophrenic ligament), left kidney (lienorenal/splenorenal ligament) and splenic flexure of the colon (splenocolic ligament). The gastrosplenic ligament contains the short gastric arteries.

These attachments place the spleen at risk of avulsion during rapid deceleration. The spleen is also relatively delicate and can be damaged by impact from overlying ribs.

6.3.1.2 DIAGNOSIS

Clinical
A third of patients complain of left upper quadrant pain. Pain may radiate to the left shoulder, and there may be a palpable mass.

Radiology
The most reliable diagnostic modalities of splenic injury are ultrasonography and CT, because they show whether blood has collected around the spleen. Contrast blush on the CT scan will indicate whether there is still active bleeding. If so, angiography with embolization should be considered.

Ultrasonography
Ultrasonic diagnosis has the great advantage that it can be performed in the emergency department during resuscitation. Focused abdominal sonography for trauma will show blood around the spleen and in the paracolic gutter. It will not show whether active bleeding is taking place. Serial ultrasonic examinations may be necessary.

6.3.1.3 SPLENIC INJURY SCALE

Staging of splenic injury originally evolved from angiographic studies of the spleen, which identified specific injury patterns. This information was further classified to indicate which injuries were likely to require surgical intervention for splenic salvage or splenectomy.

The Organ Injury Scale of the American Association for the Surgery of Trauma (AAST)\(^1\) is based on the most accurate assessment of injury, whether it is by radiological study, laparotomy, laparoscopy or postmortem evaluation (Table 6.2).

6.3.1.4 MANAGEMENT

Non-operative management\(^2\)
Management of hepatic and splenic injury has evolved with an increasing emphasis on NOM. Previously, DPL was an indication for laparotomy because of ongoing haemorrhage. However, stimulated by the success of NOM in children of both hepatic and splenic injury, there has been a similar trend in adults.

The approach of NOM to blunt splenic injuries (or splenic salvage) among children is well described, with a success rate of more than 90 per cent.

Non-operative management is contraindicated if there is a risk that hollow organ abdominal injury could present, requiring surgical intervention. Where a significant brain injury is present, NOM may be contraindicated because there is a risk of secondary brain injury from hypotension.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Type of injury</th>
<th>Description of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Subcapsular &lt; 10% surface area</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear &lt; 1 cm parenchymal depth</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Subcapsular 10–50% surface area; intraparenchymal &lt; 5 cm in diameter</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear 1–3 cm parenchymal depth that does not involve a trabecular vessel</td>
</tr>
<tr>
<td>III</td>
<td>Haematoma</td>
<td>Subcapsular &gt; 50% surface area or expanding; ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma ≥ 5 cm or expanding</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>&gt; 3 cm parenchymal depth or involving trabecular vessels</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Laceration involving segmental or hilar vessels producing major devascularization (&gt; 25% of spleen)</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Completely shattered spleen</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Hilum vascular injury with devascularized spleen</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.*
Indications for urgent open surgical intervention after a trial of NOM include:

- Haemodynamic instability
- Evidence of continued splenic hemorrhage
- Associated intra-abdominal injury requiring surgery
- Replacement of more than 50 per cent of the patient’s blood volume.

The advantages of NOM include the avoidance of non-therapeutic laparotomies (with associated cost and morbidity), fewer intra-abdominal complications and reduced transfusion risk.

After resuscitation and completion of the trauma work-up, haemodynamically stable patients with grade I, II or III splenic injuries, who have no associated intra-abdominal injuries requiring surgical intervention and no co-morbidities to preclude close observation, may be candidates for NOM.

Angiography with embolization is a useful adjunct to NOM. The indications include CT evidence of ongoing bleeding with contrast extravasation outside or within the spleen, and a drop in haemoglobin, tachycardia and haemoperitoneum, as well as formation of pseudoaneurysm.

A large number of publications support NOM in the haemodynamically stable patient. However, there is less evidence to support the use of serial CT scans, without clinical indications, to monitor progress. There is no evidence that bedrest or restricted activity is beneficial.

The risk of delayed rebleeding of the spleen after NOM is acceptably low, reportedly in the range 1–8 per cent. Rebleed is considered more likely if a higher-grade injury (grade IV) has been managed non-operatively.

### 6.3.1.5 COMPLICATIONS

- Left upper quadrant haematoma
- Pancreatitis
- Pleural effusion
- Pulmonary atelectasis
- Pseudoaneurysm of the splenic artery
- Splenic arteriovenous fistula
- Subphrenic abscess
- Overwhelming post-splenectomy sepsis
- Pancreatic injury/fistula/ascites.

### 6.3.1.6 REFERENCES


### 6.3.2 Access to the spleen

Access to the spleen in trauma is best performed via a long midline incision.

The spleen is mobilized under direct vision. Great care and gentle handling are necessary to avoid pulling on the spleen, avulsing the capsule and making a minor injury worse:

- The spleen is gently pulled upwards and medially, and the lienorenal ligaments are divided
- The spleen is then gently pulled downwards and the lienophrenic ligaments are divided with scissors
- The short gastric vessels between the greater curvature of the stomach and the spleen must be divided between ligatures. These vessels must be divided away from the greater curvature, as there is a danger of avascular necrosis of the stomach if they are divided too close to the stomach itself.

The spleen is pulled forward, and several packs can be placed in the splenic bed to hold it forward, so that it can be inspected.

#### 6.3.2.1 SURGICAL TECHNIQUES

In the presence of other major injuries or haemodynamic instability, or if the spleen has sustained damage at the hilum, a routine splenectomy should be carried out. In the stable patient and in the absence of other life-threatening injuries, a partial splenectomy or the use of local haemostyptic agents should be considered (see Section 6.3.2.2).

**Spleen not actively bleeding**

If not actively bleeding, the spleen can be left alone.

**Splenic surface bleed only**

These bleeds will usually stop with a combination of manual compression, packing, diathermy, argon beam or fibrin adhesives in combination with collagen fleece.
Minor lacerations

These may be sutured using absorbable sutures, with or without Teflon pledgets. Suturing is time-consuming and mostly not helpful in trauma patients. The superficial lacerations are best treated with fibrin adhesive and collagen tamponade. These measures should best be taken at the beginning of the operation and the spleen packed. Upon completion of the operation, the pack can be removed without displacing the collagen fleece.

Splenic tears

If the lacerations are deep and involve both the concave and the convex surfaces, the spleen is best and most effectively preserved with a mesh splenorrhaphy. If the lacerations involve only one pole or half of the organ, then the respective vessels should be ligated and a partial splenectomy performed.

Mesh wrap

If the spleen is viable, it can be wrapped in an absorbable mesh to tamponade the bleeding.

The prerequisite for mesh splenorrhaphy is complete mobilization and elevation of the spleen. An absorbable mesh should be chosen (e.g. Vicryl). There are meshes that already include two or three purse-string sutures and that can be used according to the size of the spleen. If one wants to make one’s own purse-string pouch, it is advantageous to make an impression of the spleen and then to stitch in a circle exactly on the edges, using absorbable suture material. The mesh is pulled laterally over the spleen like a headscarf; the suture is tied on the hilar side but without compressing the hilum. Mild bleeding through the holes in the mesh can be stopped with collagen tampons together with fibrin glue or, if possible, autologous fibrin.

Partial splenectomy

Diathermy and/or finger-fracture techniques are employed. The procedure is usually reserved for a demarcated ischaemic pole, after ligating segmental vessels. Division and adequate hemostasis of the splenic parenchyma can also be achieved with mechanical linear stapling devices, which should be applied progressively to avoid excessive crushing. The splenic tissue is compressed on the demarcation line with finger-fracture technique, without tearing the capsule. The resection follows this compression line, using a stapler with large staples (4.8 mm). Control of bleeding from the raw surface can be accomplished with mattress sutures, and a combination of manual compression, packing, diathermy, argon beam, infrared, and ultracision or fibrin glue with collagen fleece. The remnant may require mesh wrap. Complete mobilization of the spleen is necessary.

Splenectomy

After careful mobilization of the spleen, the splenic vessels (artery and vein) must be isolated and tied separately, because there is a small risk of subsequent arteriovenous fistula formation. Access to the splenic pedicle can be anterior or posterior. In the anterior approach, the short gastric vessels must be ligated away from the stomach to avoid the risk of avascular necrosis of the greater curvature of the stomach. The posterior approach, more expedient, entails manual mobilization and rotation of the spleen medially, after opening the peritoneum lateral to the convex surface of the spleen. The tail of the pancreas lies very close to the spleen and should be dissected free.

Splenectomized patients should be informed of the defect in their immune system and be encouraged to keep their pneumococcal and influenza immunizations current. These patients are more susceptible to malaria than the rest of the population.

6.3.2.2 DRAINAGE

The splenic bed is not routinely drained after splenectomy. If the tail of the pancreas has been damaged, then a closed suction drain should be placed in the area affected.

6.4 THE PANCREAS

6.4.1 Overview

6.4.1.1 INTRODUCTION

The pancreas and duodenum are difficult areas for surgical exposure and represent a major challenge for the operating surgeon when these organs are substantially injured. Although the retroperitoneal location of the pancreas means that it is commonly injured, it also contributes to the difficulty in diagnosis, because the organ
is concealed, and often results in delay with an attendant increase in morbidity. The increase, particularly in penetrating injuries, and the increase in wounding energy from gunshots, have made the incidence of pancreatic injury more common. As these organs are in the retroperitoneum, they usually do not present with peritonitis and are usually delayed in their presentation. It requires a high level of suspicion and significant clinical acumen, as well as aggressive radiological imaging, to identify an injury to these organs early in their course.

The management varies from simple drainage to highly challenging procedures depending on the severity, site of the injury and the integrity of the duct. The position of the pancreas makes its access and all procedures on it challenging. To compound this, pancreatic trauma is associated with a high incidence of injury to adjoining organs and major vascular structures, which adds to the high morbidity and mortality.1 Appropriate intraoperative investigation of the pancreatic duct will reduce the incidence of complications and dictate the correct operation.

The surgeon must always be critically aware of the patient’s changing physiological state and be prepared to forgo the technical challenge of definitive repair for life-saving damage control.

6.4.1.2 ANATOMY

The pancreas lies at the level of the pylorus and crosses the first and second lumbar vertebrae. It is about 15 cm long from the duodenum to the hilum of the spleen, 3 cm wide and up to 1.5 cm thick. The head lies within the concavity formed by the duodenum, with which it shares its blood supply through the pancreaticoduodenal arcades.

It has an intimate anatomical relationship with the upper abdominal vessels. It overlies the inferior vena cava, the right renal vessels and the left renal vein. The uncinate process encircles the superior mesenteric artery and vein, whereas the body covers the suprarenal aorta and the left renal vessels. The tail is closely related to the splenic hilum and left kidney and overlies the splenic artery and vein, with the artery marking a tortuous path at the superior border of the pancreas.

There are a number of named arterial branches to the head, body and tail that must be ligated in spleen-sparing procedures. Studies have shown that between 7 and 10 branches of the splenic artery and between 13 and 22 branches of the splenic vein run into the pancreas.

6.4.1.3 MECHANISMS OF INJURY

Blunt trauma

The relatively protected location of the pancreas means that a high-energy force is required to damage it. Most injuries result from motor vehicle accidents in which the energy of the impact is directed to the upper abdomen – epigastrium or hypochondrium – commonly through the steering wheel of an automobile. This force results in crushing of the retroperitoneal structures against the vertebral column, which can lead to a spectrum of injury from contusion to complete transection of the body of the pancreas.

Penetrating trauma

The rising incidence of penetrating trauma has increased the risk of injury to the pancreas. A stab wound damages tissue only along the track of the knife, but in gunshot wounds the passage of the missile and its pressure wave will result in injury to a wider region. Consequently, the pancreas and its duct must be fully assessed for damage in any penetrating wound that approaches the substance of the gland. Injuries to the pancreatic duct occur in 15 per cent of cases of pancreatic trauma, and are usually a consequence of penetrating trauma.2

6.4.1.4 INVESTIGATION

The central retroperitoneal location of the pancreas makes the investigation of pancreatic trauma a diagnostic challenge; in particular, if there are life-threatening vascular and other intra-abdominal organ injuries, the specific diagnosis is often not clear until laparotomy. In recent years there has been debate about the need to assess accurately the integrity of the main pancreatic duct. Bradley et al.3 showed that mortality and morbidity were increased when recognition of ductal injury was delayed. When these results are reviewed together with earlier work4,5 that showed an increase in late complications if ductal injuries were missed, the importance of evaluating the duct becomes evident.

Clinical evaluation

In a patient with an isolated pancreatic injury, even ductal transection may be initially asymptomatic or have only minor signs; this possibility must be kept in mind.
Serum amylase

The level of the serum amylase is not related to pancreatic injury in either blunt or penetrating trauma. A summary of recent work on serum amylase in blunt abdominal trauma by Jurkovich and Bulger showed a positive predictive value of 10 per cent and negative predictive value of 95 per cent for pancreatic injury, although more recent work has suggested that accuracy may be improved when the activity is measured more than 3 hours after injury. At present serum amylase has little value in the initial evaluation of pancreatic injury.

Diagnostic peritoneal lavage

The retroperitoneal location of the pancreas renders DPL inaccurate in the prediction of isolated pancreatic injury. However, the numerous associated injuries that may occur with pancreatic injury may make the lavage diagnostic, and the pancreatic injury is often found intraoperatively.

Ultrasonography

The posterior position of the pancreas almost completely masks it from diagnostic ultrasonography. Together with its location, a post-traumatic ileus with loops of gas-filled bowel will mask it even further and assessment of the pancreas is particularly difficult in obese patients.

Computed tomography

Computed tomography has been advocated as the best investigation for the evaluation of the retroperitoneum. In a haemodynamically stable patient, CT with contrast enhancement has a sensitivity and specificity as high as 80 per cent. However, particularly in the initial phase, CT may miss or underestimate the severity of a pancreatic injury, so normal findings on the initial scan do not exclude appreciable pancreatic injury, and a repeated scan in the light of continuing symptoms may improve its diagnostic ability.

Endoscopic retrograde cholangiopancreatography

There are two phases in the investigation of pancreatic injury in which ERCP may have a role.

Acute phase

Patients with isolated pancreatic trauma occasionally have benign clinical findings initially. It must be stressed that these patients are few, because most patients will not be stable enough and their injuries will not allow positioning for ERCP. However, where appropriate, ERCP will give detailed information about the ductal system.

Post-traumatic or delayed presentation

A small number of patients present with symptoms months to years after the initial injury. ERCP is effective in these patients and in association with CT will allow a reasoned decision to be made about the need for surgical intervention.

Magnetic resonance cholangiopancreatography

New software has opened up investigation of the pancreas and biliary system to MRI. However, to date there has been little work done in pancreatic injuries.

Intraoperative pancreatography

Intraoperative visualization of the pancreatic duct has been advocated in the investigation of the pancreatic duct, particularly when it is not possible to assess the integrity of the duct by examination. An accurate assessment of the degree of injury to the duct will reduce the complication rate, indicate the most appropriate operation and, when no involvement is found, allow a less aggressive procedure to be done. The ductal system can be examined at operation by transduodenal pancreatic duct catheterization, distal cannulation of the duct in the tail or needle cholecystocholangiogram.

Operative evaluation

Operative evaluation of the pancreas necessitates complete exposure of the gland. A central retroperitoneal haematoma must be thoroughly investigated, and intra-abdominal bile staining makes a complete evaluation essential to find the pancreatic or duodenal injury. In this case a ductal injury must be assumed until excluded.

If the sphincter of Oddi and the distal biliary tract are intact, it is wise to attempt to preserve the head and neck of the pancreas. One can survive quite well with 10 per cent of the pancreas without pancreatic insufficiency or diabetes. Major injuries to the body of the pancreas are usually treated by a distal pancreatectomy with splenectomy. If the injury is to the head of the pancreas, involving the duct and sphincter, Whipple’s procedure must be contemplated. Increasingly, there is a move
towards lesser procedures because the mortality from Whipple's procedure continues to be significant in the severely injured trauma patient. These injuries continue to be a major challenge for the trauma surgeon. It is essential to understand the manoeuvres necessary for gaining complete control of the duodenum and pancreas in order completely to explore and identify any injuries.

6.4.1.5 ORGAN INJURY SCALE (TABLE 6.3)

The organ injury scale developed by the American Association for the Surgery of Trauma (AAST)\(^1\) (see Appendix B) has been accepted by most institutions that regularly deal with pancreatic trauma.

6.4.1.6 OPERATIVE MANAGEMENT

Damage control

The origin of the concept of damage control was described by Halsted as the packing of liver injuries, and was reported and repopularized by Stone \(et\) \(al\). in 1908;\(^1\) they advocated early packing and termination of the operation in patients who showed signs of intraoperative coagulopathy. Patients with severe pancreatic or pancreaticoduodenal injury (AAST grades IV and V) are not stable enough to undergo complex reconstruction at the time of initial laparotomy. Damage control with the rapid arrest of haemorrhage and bacterial contamination, and placement of drains and packing, is preferable. It may be helpful to place a tube drain directly into the duct, both for drainage and to allow easier isolation of the duct at the subsequent operation. The damage control laparotomy is followed by a period of intensive care and continued aggressive resuscitation to correct physiological abnormalities and restore reserve before the definitive procedure.

Contusion and parenchymal injuries

Relatively minor pancreatic lacerations and contusions (AAST grades I and II) comprise most injuries to the pancreas. Nowak \(et\) \(al\).\(^1\) showed that these require simple drainage and haemostasis, and this has become standard practice.\(^1\) There is, however, debate about whether the ideal drainage system is a closed suction system or an open pencil drain. Those in favour of suction drainage claim that fewer intra-abdominal abscesses develop and there is less skin excoriation\(^1\) with a closed suction system.

Suturing of parenchymal lesions (AAST grades I and II) in an attempt to gain haemostasis simply leads to necrosis of the pancreatic tissue. Bleeding vessels should be ligated individually and a viable omental plug sutured into the defect to act as a haemostatic agent.

Ductal injuries: tail and distal pancreas

Distal pancreatectomy

In most cases in which there is a major parenchymal injury of the pancreas to the left of the superior mesenteric vessels (AAST grades II or III), a distal pancreatectomy is the procedure of choice, independent of the degree of ductal involvement. Where there is concern over the involvement of the duct, an intraoperative pancreateogram can be done. After mobilization of the pancreas and ligation of the vessels, the pancreatic stump can be closed with sutures and the duct ligated separately, or it can be closed with a stapling device.\(^1\) An external drain should be placed at the site of transection, because there is a postoperative rate of fistula formation of 14 per cent.\(^1\) Suction drains are preferable.

Procedures associated with resection of more than 80 per cent of the pancreatic tissue are associated with a risk of adult-onset diabetes mellitus. Most authors agree that a pancreatectomy to the left of the superior mesenteric

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Table 6.3 Pancreas injury scale (see also Appendix B.7)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Type of injury</th>
<th>Description of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Minor contusion without duct injury</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Superficial laceration without duct injury</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Major contusion without duct injury or tissue loss</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Major laceration without duct injury or tissue loss</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Distal transection or parenchymal injury with duct injury</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Proximal transection or parenchymal injury involving ampulla</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Massive disruption of pancreatic head</td>
</tr>
</tbody>
</table>
vessels usually leaves enough pancreatic tissue to result in an acceptably low rate of type 1 diabetes.\textsuperscript{19}

**Internal drainage of the distal pancreas**

Drainage of the distal pancreas with a roux-en-Y pancreaticojejunostomy has been suggested in cases in which there is not enough proximal tissue for endocrine or exocrine function. Its popularity has greatly declined because of the high reported morbidity and mortality.\textsuperscript{20}

**Splenic salvage in distal pancreatectomy**

Splenic salvage has been advocated in elective distal pancreatectomy, and is possible in some cases of pancreatic trauma. However, this should be saved for the rare occasions when the patient is haemodynamically stable and normothermic, and the injury is limited to the pancreas. The technical problems of dissecting the pancreas free from the splenic vessels and ligating of the numerous tributaries contraindicates the procedure in an unstable patient with multiple associated injuries.\textsuperscript{21}

When this operation is considered, the surgeon must clearly balance the extra time that it takes and the problems associated with lengthy operations in injured patients against the small risk of the development of OPSI postoperatively.

**Ductal injuries: combined injuries of the head of the pancreas and duodenum**

Severe combined pancreaticoduodenal injuries account for less than 10 per cent of injuries to these organs, and are commonly associated with multiple intra-abdominal injuries, particularly of the vena cava.\textsuperscript{22} They are usually the result of penetrating trauma. The integrity of the distal common bile duct and ampulla on cholangiography and the severity of the duodenal injury will dictate the surgical procedure. If the duct and ampulla are intact, simple repair and drainage or repair and pyloric exclusion will suffice.

**Suture and drainage**

In most trauma units, simple suture and drainage is reserved for minor injuries in which the pancreatic duct is not involved, and injuries to both organs are slight.\textsuperscript{22}

**Duodenal diverticulization**

Duodenal diverticulization was developed to deal with the higher mortality of combined injuries to the duodenum and pancreas than to each organ when injured in isolation.\textsuperscript{23} On the premise that diversion of the enteric flow will promote duodenal healing, the procedure incorporates truncal vagotomy, antrectomy with gastrojejunostomy, duodenal closure, tube duodenostomy, drainage of the biliary tract and external drainage. However, the authors reported high complication rates associated with this complex operation and it has fallen from favour.

**Pyloric exclusion**

Pyloric exclusion has been widely reported for the management of severe combined pancreaticoduodenal injuries without major damage to the ampulla or the common bile duct. The technique involves the temporary diversion of enteric flow away from the injured duodenum by closure of the pylorus. This is best achieved with access from the stomach through a gastrostomy and the use of a slowly absorbable suture. The stomach is decompressed with a gastrojejunostomy. Contrast studies have shown that the pylorus reopens within 2–3 weeks in 90–95 per cent of patients, allowing flow through the anatomical channel. Feliciano et al.\textsuperscript{22} reported on this technique in 68 of 129 patients with combined injuries. Their results showed a 26 per cent rate of pancreatic fistula formation and a 6.5 per cent rate of duodenal fistula, but a reduced overall mortality compared with patients who did not have pyloric exclusion. The procedure has been adopted in many institutions for the treatment of grade III and IV combined pancreaticoduodenal injuries.

**T-tube drainage**

Some surgeons advocate closing the injury over a T-tube in combined injuries where the second part of the duodenum is involved. This ensures adequate drainage and allows the formation of a controlled fistula once the track has matured. Our preference in these injuries is, however, primary closure, pyloric exclusion and gastroenterostomy.

**Internal and external drainage**

Although there is little controversy on the importance of external, periduodenal drainage of complex duodenal or pancreaticoduodenal injuries, the role of internal decompression via a naso-gastroduodenal or retrograde jejuno-duodenal tube, or tube duodenostomy, is more controversial. Delay in repairing a duodenal injury often
results in duodenal leaks, emphasizing the importance of adequate external drainage that allows the formation of a controlled fistula once the track has matured. The preferred method of managing complex duodenal injuries is, however, primary closure, pyloric exclusion and gastroenterostomy.

Pancreaticoduodenectomy (Whipple’s procedure)

In only 10 per cent of combined injuries will a pancreaticoduodenectomy, or Whipple’s procedure, be required, and that will be when there is severe injury to the head of the pancreas with unreconstructable injury to the ampulla or pancreatic duct, or destruction of the duodenum and pancreatic head, particularly if it is compromising the blood supply. Whipple’s procedure, as first described for carcinoma of the ampulla, is indicated only in the rare stable patient with this type of injury. The nature and severity of the injury and the coexisting damage to vessels are often accompanied by haemodynamic instability and the surgeon must therefore control the initial damage and delay formal reconstruction until the patient has been stabilized. The results of this operation vary and, when patients with major retroperitoneal vascular injuries are included, the mortality rate can approach 50 per cent.20 Oreskovich and Carrico, however, reported a series of 10 Whipple’s procedures for trauma with no deaths.

Non-operative management

In isolated blunt pancreatic injuries, exclusion of a major pancreatic duct injury with ERCP, followed by expectant NOM, is gaining popularity. Recent reports using early ERCP to identify and sometimes treat blunt pancreatic injuries by transpapillary stent insertion are showing promising results.27,28

6.4.1.7 ADJUNCTS

Somatostatin and its analogues

Somatostatin and its analogue octreotide have been used to reduce pancreatic exocrine secretion in patients with acute pancreatitis. Despite meta-analysis its role has not been clearly defined. Büchler et al.29 reported a slight but not significant reduction in the complication rate in patients with moderate-to-severe pancreatitis, but this was not verified by Imrie’s group in Glasgow30 who found that somatostatin gave no benefit.

After pancreatic surgery, somatostatin can reduce the output from a pancreatic fistula.31 Retrospective work on the role of octreotide in pancreatic trauma differs, however. Somatostatin cannot be recommended in trauma on the current evidence, and a level 1 study is required.

Nutritional support

Whether nutritional support is required should be considered the definitive operation. Major injuries that precipitate prolonged gastric ileus and pancreatic complications may preclude gastric feeding. The creation of a feeding jejunostomy, ideally 15–30 cm distal to the duodenojejunal flexure, should be routine and will allow early enteral feeding. We prefer elemental diets that are less stimulating to the pancreas and have no greater fistula output than total parenteral nutrition (TPN).32 Total parenteral nutrition is far more expensive, but may be used if enteral access distal to the duodenojejunal flexure is impossible.

6.4.1.8 PANCREATIC INJURY IN CHILDREN

The pancreas is injured in up to 10 per cent of cases of blunt abdominal trauma in children, usually as a result of a handlebar injury. Whether they should be operated upon or managed conservatively (the current vogue for management of solid organ injuries in children) is controversial. Shilyansky et al.33 reported that NOM of pancreatic injuries in children was safe for both contusion and pancreatic transection, and Keller et al.34 recommended conservative management if there were no signs of clinical deterioration or major ductal injury. Although pseudocysts are more likely to develop with transection injuries, they tend to respond to percutaneous drainage.34

6.4.1.9 COMPLICATIONS

Pancreatic trauma is associated with up to 19 per cent mortality. Early deaths result from the associated intra-abdominal vascular and other organ injuries, and later deaths from sepsis and the systemic inflammatory response syndrome (SIRS). Pancreatic injuries have postoperative complication rates of up to 42 per cent, and the number rises with increasing severity of injury; with combined injuries and associated injuries the complication rate approaches 62 per cent.5,35
Most complications are treatable or self-limiting, however, and could be avoided by an accurate assessment of whether the pancreatic duct is damaged. Pancreatic complications can be divided into those occurring early and late in the postoperative period.

**Early complications**

*Pancreatitis*

Postoperative pancreatitis may develop in about 7 per cent of patients. It may vary from a transient biochemical leak of amylase to a fulminant haemorrhagic pancreatitis. Fortunately, most cases run a benign course and respond to bowel rest and nutritional support.

*Fistula*

The development of a postoperative pancreatic fistula is the most common complication, with an incidence of 11 per cent; this increases when the duct is involved, and may be as high as 37 per cent in combined injuries. Most fistulas are minor (< 200 mL fluid/day) and self-limiting when there is adequate external drainage. However, high-output fistulas (> 7000 mL/day) may require surgical intervention for closure or prolonged periods of drainage with nutritional support. Management is directed locally at adequate drainage, reduction of pancreatic output with octreotide and (recently) transpapillary pancreatic stenting of confirmed ductal injuries. Systemic treatment includes treatment of the underlying cause (such as sepsis) and early adequate nutrition, preferably with distal enteral feeds through a feeding jejunostomy. If the fistula persists, the underlying cause should be investigated with ERCP, CT and surgery as necessary.

*Abscess formation*

Most abscesses are peripancreatic and associated with injuries to other organs, specifically the liver and intestine. A true pancreatic abscess is uncommon and usually results from inadequate debridement of necrotic tissue. For this reason simple percutaneous drainage is generally not enough and further debridement is required.

**Late complications**

*Pseudocyst*

Accurate diagnosis and surgical treatment of pancreatic injuries should result in a rate of pseudocyst formation of about 2–3 per cent, but Kudsk et al. reported pseudocysts in half their patients who were treated non-surgically for blunt pancreatic trauma. Investigation entails imaging of the ductal system with either ERCP or MRI. Accurate evaluation of the state of the duct will dictate management and, if it is intact, percutaneous drainage is likely to be successful. However, a pseudocyst together with a major ductal disruption will not be cured by percutaneous drainage, which will convert the pseudocyst into a chronic fistula. Current options include cystogastrostomy (open or endoscopic), endoscopic stenting of the duct or resection.

*Exocrine and endocrine deficiency*

Pancreatic resection distal to the mesenteric vessels will usually leave enough tissue for adequate exocrine and endocrine function, as work has shown that a residual 10–20 per cent of pancreatic tissue is usually enough. Patients who have procedures that leave less functioning tissue will require exogenous endocrine and exocrine enzyme replacement.

**6.4.1.10 CONCLUSION**

Pancreatic and combined pancreaticoduodenal injuries remain a dilemma for most surgeons and, despite advances and complex technical solutions, they still carry a high morbidity and mortality. Pancreatic injury must be suspected in all patients with abdominal injuries, even those who initially have few signs. Accurate intraoperative investigation of the pancreatic duct will reduce the incidence of complications and dictate the correct operation. The management varies from simple drainage to highly challenging procedures depending on the severity, the site of the injury and the integrity of the duct. However, the surgeon must always be critically aware of the patient’s changing physiological state and be prepared to forsake the technical challenge of definitive repair for life-saving damage control.

**6.4.1.11 REFERENCES**


6.4.2 Access to the pancreas

Access to the pancreas in trauma is gained via a long midline incision. For the complete evaluation of the gland, it is essential to see the pancreas from both anterior and posterior aspects. To examine the anterior surface of the gland, it is necessary to divide the gastrocolic ligament and open the lesser sac. Kocher’s manoeuvre is required so that the duodenum can be mobilized and an adequate view gained of the pancreatic head, uncinate process and posterior aspect. Injury to the tail requires mobilization of the spleen and left colon to allow medial reflection of the pancreas and access to the splenic vessels. Division of the ligament of Treitz and reflection of the fourth part of the duodenum and duodenojejunal flexure give access to the inferior aspect of the pancreas.

6.4.2.1 ACCESS VIA THE LESSER SAC

The stomach is then grasped and pulled inferiorly, allowing the surgeon to identify the lesser curvature and the pancreas through the lesser sac. Frequently, the coeliac artery and the body of the pancreas can be identified through this approach. The omentum is then grasped and drawn upwards. An ostomy is made in the omentum and the surgeon’s hand is passed into the lesser sac posterior to the stomach. This allows excellent exposure of the entire body and tail of the pancreas. Any injuries to the pancreas can be easily identified.

6.4.2.2 DUODENAL ROTATION (KOCHER’S MANOEUVRE) (SEE SECTION 6.1.2, TRAUMA LAPAROTOMY)

If there is a possibility of an injury to the head of the pancreas, Kocher’s manoeuvre is performed. The loose areolar tissue around the duodenum is bluntly dissected, and the entire second and third portion of the duodenum identified and mobilized medially. This dissection is carried all the way medial to expose the inferior vena cava and a portion of the aorta. By reflecting the duodenum and pancreas towards the anterior midline, the posterior surface of the head of the pancreas can be completely inspected.

6.4.2.3 RIGHT MEDIAL VISCERAL ROTATION (SEE SECTION 6.1.2, TRAUMA LAPAROTOMY)

The inferior border of the proximal portion of the pancreas can be identified by performing a right medial visceral rotation. This is performed by taking down the ascending colon and then mobilizing the caecum, terminal ileum and mesentery towards the midline. The entire ascending colon and caecum are then reflected superiorly towards the left upper quadrant of the abdomen. This gives excellent exposure of the entire vena cava, aorta and third and fourth portions of the duodenum.

6.4.2.4 LEFT MEDIAL VISCERAL ROTATION

The descending colon on the left is mobilized, together with the spleen and the tail of the pancreas. These are rotated medially, allowing inspection of the tail, and posterior and inferior aspects of the pancreas.

These manoeuvres allow for complete exposure of the first, second, third and fourth portions of the duodenum, along with the head, neck, body and tail of the pancreas.

6.5 THE DUODENUM

6.5.1 Overview

6.5.1.1 INTRODUCTION

Duodenal injuries can pose a formidable challenge to the surgeon, and failure to manage them properly can have devastating results. The total amount of fluid passing through the duodenum exceeds 6 L/day and a fistula in this area can cause serious fluid and electrolyte imbalance. The combination of a large amount of activated enzymes liberated into the retroperitoneal space and the peritoneal cavity can be life-threatening.

Both the pancreas and the duodenum are well protected in the superior retroperitoneum deep within the abdomen. As these organs are in the retroperitoneum, they usually do not present with peritonitis and are delayed in their presentation. Therefore, in order to
sustain an injury to either one of them, there must be other associated injuries. If there is an anterior penetrating injury, frequently the stomach, small bowel, transverse colon, liver, spleen or kidneys are also involved. If there is a blunt traumatic injury, there are often fractures of the lower thoracic or upper lumbar vertebrae. It requires a high level of suspicion and significant clinical acumen as well as aggressive radiographic imaging to identify an injury to these organs this early in the presentation.

Preoperative diagnosis of isolated duodenal injury can be very difficult to make and there is no single method of duodenal repair that completely eliminates dehiscence of the duodenal suture line. As a result, the surgeon is frequently confronted with the dilemma of choosing between several preoperative investigations and many surgical procedures. A detailed knowledge of the available operative choices and when each one of them is preferably applied is important for the patient’s benefit.1

6.5.1.2 Mechanism of injury

Penetrating trauma

Penetrating trauma is the leading cause of duodenal injuries in countries with a high incidence of civilian violence. As a result of the retroperitoneal location of the duodenum, and its close proximity to a number of other viscer and major vascular structures, isolated penetrating injuries of the duodenum are rare. The need for abdominal exploration is usually dictated by associated injuries and the diagnosis of duodenal injury is usually made in the operating room.

Blunt trauma

Blunt injuries to the duodenum are both less common and more difficult to diagnose than penetrating injuries, and they can occur in isolation or with pancreatic injury. These usually occur when crushing the duodenum between the spine and a steering wheel or a handlebar, or some other force is applied to the duodenum. These injuries can be associated with flexion/distraction fractures of L1–2 vertebrae – the chance fracture. ‘Stomping’ and striking the mid-epigastrium are common. Less common in deceleration injury patterns are tears at the junction or the third and fourth parts of the duodenum (and, less commonly, first and second parts). These injuries occur at the junction of free (intraperitoneal) parts of the duodenum with fixed (retroperitoneal) parts. A high index of suspicion based on mechanism of injury and physical examination findings may lead to further diagnostic studies.

6.5.1.3 INVESTIGATION

Clinical presentation

The clinical changes in isolated duodenal injuries may be extremely subtle until severe, life-threatening peritonitis develops. In the vast majority of the retroperitoneal perforations there is initially only mild upper abdominal tenderness with progressive rise in temperature, tachycardia and occasionally vomiting. After several hours the duodenal contents extravasate into the peritoneal cavity, with development of peritonitis. If the duodenal contents spill into the lesser sac, they are usually ‘walled off’ and localized, although occasionally they can leak into the general peritoneal cavity via the foramen of Winslow, with resultant generalized peritonitis.2 Diagnostic difficulties do not arise in the cases where the blunt injury causes intraperitoneal perforations.

Serum amylase

Theoretically, duodenal perforations are associated with a leak of amylase and other digestive enzymes and it has been suggested that determination of the serum amylase concentration may be helpful in the diagnosis of blunt duodenal injury.1,4 However, the test lacks sensitivity.5,6 The duodenum is retroperitoneal, the concentration of amylase in the fluid that leaks is variable, and amylase concentrations often take hours to days to increase after injury. Although serial determinations of serum amylase are better than a single, isolated determination on admission, sensitivity is still poor, and necessary delays are inherent in serial determinations. If serum amylase is elevated on admission, a diligent search for duodenal rupture is warranted. The presence of a normal amylase level, however, does not exclude duodenal injury.7

Diagnostic peritoneal lavage

Although virtually all patients with blunt duodenal injury will eventually have increased white blood cell and amylase levels in DPL fluid, DPL has a low sensitivity for duodenal perforations.8

Radiological investigation

Radiological studies may be helpful in the diagnosis. Plain radiographs of the abdomen are useful when gas
bubbles are present in the retroperitoneum adjacent to the right psoas muscle, around the right kidney or anterior to the upper lumbar spine. They can also show free intraperitoneal air and, although rarely seen, air in the biliary tree has also been described. Obliteration of the right psoas muscle shadow or fractures of the transverse processes in the lumbar vertebrae are indicative of forceful retroperitoneal trauma and serve as a predictor of duodenal trauma.

An upper gastrointestinal (GI) series using water-soluble contrast material can provide positive results in 50 per cent of patients with duodenal perforations. Meglumine (Gastrograffin) should be infused via the nasogastric tube and then swallowed, and the study should be done under fluoroscopic control with the patient in the right lateral position. If no leak is observed, the investigation continues with the patient in the supine and left lateral position. If the Gastrograffin study is negative, it should be followed by administration of barium to allow detection of small perforations more readily. Upper GI studies with contrast are also indicated in patients with a suspected haematoma of the duodenum, because they may demonstrate the classic 'coiled-spring' appearance of complete obstruction by the haematoma.

Computed tomography has been added to the diagnostic tests used for investigation of subtle duodenal injuries. It is very sensitive to the presence of small amounts of retroperitoneal air, blood or extravasated contrast from the injured duodenum, especially in children. Its reliability in adults is more controversial. The presence of peri-duodenal wall thickening or haematoma without extravasation of contrast material should be investigated with a GI study with Gastrograffin. If normal it should be followed by a barium study contrast, if the patient's condition allows this.

Diagnostic laparoscopy

Unfortunately, diagnostic laparoscopy does not confer any improvement over more traditional methods in the investigation of the duodenum. In fact, because of its anatomical position, diagnostic laparoscopy is a poor modality to determine organ injury in these cases.

Exploratory laparotomy

Exploratory laparotomy remains the ultimate diagnostic test if a high degree of suspicion of duodenal injury continues in the face of absent or equivocal radiographic signs.

### Table 6.4 Duodenum injury scale (see also Appendix B.7)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Type of injury</th>
<th>Description of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Involving single portion of duodenum</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness, no perforation</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Involving more than one portion</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Disruption &lt; 50% of circumference</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Disruption 50–75% of circumference of D2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Disruption 50–100% of circumference of D1, D3, D4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Disruption &gt; 75% of circumference of D2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Involving ampulla or distal common bile duct</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Massive disruption of duodenopancreatic complex</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Devascularization of duodenum</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III. D1, first portion of duodenum; D2, second portion of duodenum; D3, third portion of duodenum; D4, fourth portion of duodenum.

#### 6.5.1.4 Duodenal injury scale

Grading systems have been devised to characterize duodenal injuries (Table 6.4).

#### 6.5.1.5 SURGICAL MANAGEMENT OF DUODENAL INJURIES

Although useful for research purposes, the specifics of the grading systems are less important than several simple aspects of the duodenal injuries:

- The anatomical relationship to the ampulla of Vater
- The characteristics of the injury (simple laceration versus destruction of duodenal wall)
- The involved circumference of the duodenum
- Associated injuries to the biliary tract, pancreas or major vascular injuries.

Timing of the operation is also very important because the mortality rate rises from 11 to 40 per cent if the time interval between injury and operation is more than 24 h.

From a practical point of view, the duodenum can be divided into one ‘upper’ portion that includes the first and second part and another ‘lower’ portion that includes the third and fourth part. The ‘upper’ portion has complex anatomical structures within it (common bile duct and the sphincter) and the pylorus. It requires distinct manoeuvres to diagnose injury (cholangiogram,
direct visual inspection) and complex techniques to repair them. The first and second parts of the duodenum are densely adherent and dependent for their blood supply on the head of the pancreas, so diagnosis and management of any injury are complex, and resection, unless involving the entire ‘C’ loop and pancreatic head, is impossible. The ‘lower’ portion involving the third and fourth part of the duodenum can generally be treated like the small bowel and injury diagnosis and management are relatively simple, including debridement, closure, resection and re-anastomosis.

**Intramural haematoma**

This is a rare injury of the duodenum specific to patients with blunt trauma. It is most common in children with isolated force to the upper abdomen, possibly because of the relatively flexible and pliable musculature of the child's abdominal wall, and half of the cases can be attributed to child abuse. The haematoma develops in the submucosal or subserosal layers of the duodenum. The duodenum is not perforated. Such haematomas can lead to obstruction. The symptoms of gastric outlet obstruction can take up to 48 h to present. This is a result of the gradual increase of the size of a haematoma as the breakdown of the haemoglobin makes it hyperosmotic, with resultant fluid shifts into it. The diagnosis can be made by double-contrast CT or upper GI contrast studies that show the ‘coiled spring’ or ‘stacked coin’ sign.\(^{10}\)

The injury is usually considered non-surgical and best results are obtained by conservative treatment, if associated injuries can be ruled out.\(^{14}\) After 3 weeks of conservative management with nasogastric aspiration and TPN, the patient is re-evaluated. If there is no improvement, the patient undergoes laparotomy to rule out the presence of duodenal perforation or injury of the head of the pancreas, which may be an alternative cause of duodenal obstruction.

The treatment of an intramural haematoma that is found at early laparotomy is controversial. One option is to open the serosa, evacuate the haematoma without violation of the mucosa and carefully repair the wall of the bowel. The concern is that this may convert a partial tear to a full-thickness tear of the duodenal wall. Another option is carefully to explore the duodenum to exclude a perforation, leaving the intramural haematoma intact and planning nasogastric decompression postoperatively.

**Duodenal laceration**

The great majority of duodenal perforations and lacerations can be managed with simple surgical procedures. This is particularly true with penetrating injuries when the time interval between injury and operation is normally short. On the other hand, a minority are ‘high risk’, e.g. with increased risk of dehiscence of the duodenal repair, with increased morbidity and sometimes mortality. These injuries are related to associated pancreatic injury, blunt or missile injury, involvement of more than 75 per cent of the duodenal wall, injury of the first or second part of the duodenum, time interval of more than 24 h between injury and repair and associated common bile duct injury. In these high-risk injuries, in an attempt to reduce the incidence of dehiscence of the duodenal suture line, several adjunctive operative procedures have been proposed. The methods of repair of the duodenal trauma as well as the ‘supportive’ procedures against dehiscence are described.

**Repair of the perforation**

Most injuries of the duodenum can be repaired by primary closure in one or two layers. The closure should be oriented transversely, if possible, to avoid luminal compromise. Excessive inversion should be avoided.

Longitudinal duodenotomies can usually be closed transversely if the length of the duodenal injury is less than 50 per cent of the circumference of the duodenum. If primary closure would compromise the lumen of the duodenum, several alternatives have been recommended. Pedicled mucosal graft, as a method of closing large duodenal defects, has been suggested using a segment of jejunum or a gastric island flap from the body of the stomach. An alternative to that is the use of a jejunal serosal patch to close the duodenal defect.\(^{15}\) The serosa of the loop of the jejunum is sutured to the edges of the duodenal defect. Although encouraging in experimental studies, the clinical application of both methods has been limited and suture line leaks have been reported.\(^{16}\) Laying a loop of jejunum on to the area of the injury so that the serosa of the jejunum buttresses the duodenal repair has also been suggested,\(^{17}\) although no beneficial results have been reported from this technique.\(^{18}\)

**Complete transection of the duodenum**

The preferred method of repair is usually primary anastomosis of the two ends after appropriate debridement
and mobilization of the duodenum. This is frequently the case with injuries of the first, third and fourth parts of the duodenum where mobilization is technically not difficult. However, if a large amount of tissue is lost, approximation of the duodenum may not be possible without producing undue tension to the suture line. If this is the case and complete transection occurs in the first part of the duodenum, it is advised to perform an antrectomy with closure of the duodenal stump and a Bilroth II gastrojejunostomy. When such injury occurs distal to the ampulla of Vater, closure of the distal duodenum and roux-en-Y duodenojejunal anastomosis is appropriate.\(^1^9\) Mobilization of the second part of the duodenum is limited by its shared blood supply with the head of the pancreas. A direct anastomosis to a roux-en-Y loop sutured over the duodenal defect in an end-to-side fashion is the procedure of choice. This can also be applied as an alternative method of operative management of extensive defects to the other parts of the duodenum when primary anastomosis is not feasible.

External drainage should be provided in all duodenal injuries because it affords early detection and control of the duodenal fistulas. The drain is preferably a simple, soft silicon rubber, closed system placed adjacent to the repair.

Duodenal diversion

In high-risk duodenal injuries duodenal repair is followed by a high incidence of suture line dehiscence. To protect the duodenal repair, the gastrointestinal contents – with their proteolytic enzymes – can be diverted, a practice that would also make the management of a potential duodenal fistula easier.

‘Tube decompression’ was the first technique used for decompression of the duodenum and diversion of its contents in an attempt to preserve the integrity of the duodenorrhaphy. It was first described in 1954 as a method of management of a precarious closure of the duodenal stump after a gastrectomy.\(^2^0\) In trauma, the technique was introduced by Stone and Garoni as a ‘triple ostomy’.\(^2^1\) This consists of a gastrostomy tube to decompress the stomach, a retrograde jejunosotomy to decompress the duodenum and an anterograde jejunosotomy to feed the patient. The initial favourable reports on the efficacy of this technique to decrease the incidence of dehiscence of the duodenorrhaphy have not been supported by more recent reports.\(^2^2\) The drawbacks of this technique are that several new perforations are made in the GI tract, the inefficiency of the jejunosotomy tube to decompress the duodenum properly and the common scenario of finding that the drains fell out or were removed by the patient. The fashioning of a feeding jejunosotomy at the initial laparotomy in patients with duodenal injury and extensive abdominal trauma (abdominal trauma index > 25) is highly recommended.

Duodenal diverticulation

This includes a distal Bilroth II gastrectomy, closure of the duodenal wound, placement of a decompressive catheter into the duodenum and generous drainage of the duodenal repair.\(^2^1\) Truncal vagotomy and biliary drainage could be added. The disadvantage of duodenal diverticulation is that it is an extensive procedure totally inappropriate for the haemodynamically unstable trauma patient, or the patient with multiple injuries. Resection of a normal distal stomach cannot be beneficial to the patient and should not be considered unless there is a large amount of destruction and tissue loss and no other course is possible.

Pyloric exclusion

Pyloric exclusion was devised as an alternative to this extensive procedure in order to shorten the operative time and make the procedure reversible. After primary repair of the duodenum, a gastrotomy is made at the antrum along the greater curvature. The pyloric ring is grasped and invaginated outside the stomach through the gastrotomy, and closed with a large running suture or stapled. The closed pyloric ring is returned into the stomach and the gastrojejunostomy is fashioned at the gastrotomy site (Figure 6.7).

The closure of the pylorus breaks down after several weeks and the GI continuity is re-established. This occurs regardless of whether the pylorus was closed with

Figure 6.7 Pyloric exclusion and gastric bypass.
absorbable suture, non-absorbable suture or staples. Major concern has been expressed about the ulcerogenic potential of the pyloric exclusion, as marginal ulceration has been reported in up to 10 per cent of patients. The long-term incidence of marginal ulceration in patients who underwent pyloric exclusion is probably underestimated because it is notoriously difficult to obtain long-term follow-up in the trauma population. We do not practise vagotomy in our patients with pyloric exclusion. Ginzburg et al. question the need to perform routine gastrojejunostomy after pyloric exclusion, taking into consideration that the continuity of the GI tract in 90 per cent of patients will be re-established within 3 weeks. A duodenal fistula can still occur with pyloric exclusion, and there is concern that spontaneous opening of the pyloric sphincter will negatively influence the closure of the fistula. This has been shown not to be a clinically relevant problem. Pyloric exclusion is a technically easier, less radical and quicker operation than diverticulization of the duodenum and appears to be equally effective in the protection of the duodenal repair.

The use of octreotide in the protection of the suture line in pancreaticojejunostomies after pancreaticoduodenectomies has been shown to be beneficial. The principle is attractive, but further experience is required before sound conclusions can be drawn.

Pancreaticoduodenectomy

This is a major procedure to be practised in trauma only if no alternative is available. Damage control, with control of bleeding and bowel contamination, and ligation of the common bile and pancreatic ducts should be the rule. Reconstruction should take place within the next 48 h when the patient is stable. Indications for considering pancreaticoduodenectomies are massive disruption of the pancreaticoduodenal complex, devascularization of the duodenum and sometimes extensive duodenal injuries of the second part of the duodenum involving the ampulla or distal common bile duct.

The role of pancreaticoduodenectomy in trauma is best summarized by Walt:

Finally, to Whipple or not to Whipple, that is the question. In the massively destructive lesions involving the pancreas, duodenum and common bile duct, the decision to do a pancreaticoduodenectomy is unavoidable; and, in fact, much of the dissection may have been done by the wounding force. In a few patients, when the call is of necessity close, the overall physiologic status of the patient and the extent of damage become the determining factors in the decision. Though few in gross numbers, more patients are eventually salvaged by drainage, TPN and meticulous overall care than by a desperate pancreaticoduodenectomy in a marginal patient.

Specific injuries

Simple combined injuries of the pancreas and duodenum should be managed separately. More severe injuries require more complex procedures. Feliciano et al. reported by far the largest experience of combined pancreaticoduodenal injuries and suggested the following:

• Simple duodenal injuries with no ductal pancreatic injury (grades I and II) should be managed with primary repair and drainage.
• Grade III duodenal and pancreatic injuries are best treated with repair or resection of both organs as indicated, pyloric exclusion, gastrojejunostomy and closure.
• Grades IV and V duodenal and pancreatic injuries are best treated by pancreaticoduodenectomy.

Extensive local damage of the intraduodenal or intrapancreatic bile duct injuries frequently necessitates a staged pancreaticoduodenectomy. Less extensive local injuries can be managed by intraluminal stenting, sphincteroplasty or reimplantation of the ampulla of Vater.

6.5.1.6 CONCLUSION

In conclusion, upper GI radiological studies and CT can lead to the diagnosis of blunt duodenal trauma, but exploratory laparotomy remains the ultimate diagnostic test if a high suspicion of duodenal injury continues in the face of absent or equivocal radiographic signs. Most duodenal injuries can be managed by simple repair. More complicated injuries require more sophisticated techniques. ‘High-risk’ duodenal injuries are followed by a high incidence of suture line dehiscence and their treatment should include duodenal diversion. Pancreaticoduodenectomy is practised only if no alternative is available. ‘Damage control’ should precede the definitive reconstruction. The detailed knowledge of available operative choices and the situation for which each one of them is preferably applied is important for the patient’s benefit.
6.5.1.7 REFERENCES


6.5.2 Access to the duodenum

See Section 6.4.2, ‘Access to the pancreas’.

6.6 ABDOMINAL VASCULAR INJURY

6.6.1 Overview

6.6.1.1 IMPORTANT CONSIDERATIONS

Abdominal vascular injury presents a serious threat to life where preparedness and anticipation are vital to a successful outcome. Consideration of both the possible injuries and the surgical approach to manage them is crucial. Adequate preparation is essential and an adequate incision will be needed.

*It is helpful to have all the apparatus available for massive transfusion, with adequate warming of all intravenous fluids. Autotransfusion should be considered in all cases.*

Major vessel injuries within the abdominal cavity primarily present as haemorrhagic shock that does not respond to resuscitation; thus, immediate surgery becomes a part of the resuscitative effort. In penetrating injury this may necessitate an emergency department/room thoracotomy (ERT) and aortic cross-clamp.

*However, the ERT is not indicated in the severely shocked patient with blunt abdominal trauma, because the survival rate is close to zero.*

Direct or proximal control of the vessel is mandatory for success. Injuries above the pelvic brim can be approached from the right side if the injury is thought to be below the renal artery and from the left side for injuries between the renal artery and the hiatus. Vascular injuries in the pelvis following blunt trauma are best managed by arteriogram. This will determine whether a direct surgical approach or interventional radiology is appropriate.

6.6.1.2 CENTRAL HAEMATOMA IN THE UPPER ABDOMEN

To expose the potential sites of arterial bleeding in the upper midline region of the retroperitoneum, medial visceral rotation is performed by mobilizing not only the left colon, but also the spleen, pancreas and stomach.

The lienorenal and lienophrenic ligaments are divided, followed by an incision down the left paracolic gutter and a blunt dissection to free the organs from the retroperitoneum towards the centre of the abdomen. An extended reflection of the abdominal structures from the left to the right will reflect the spleen, colon, tail of pancreas and fundus of the stomach towards the midline. This provides access to the aorta, coeliac axis, superior mesenteric artery, splenic artery and vein, and left renal artery and vein. To reach the posterior wall of the aorta, the kidney should be mobilized as well, and rotated medially on its pedicle, taking great care not to cause further injury.

6.6.1.3 LATERAL HAEMATOMAS

If these are not expanding or pulsatile, blunt injuries are best left alone, as the damage is usually renal. Renal injuries can generally be managed non-operatively including the use of selective embolization. However, with penetrating injury, because of the risk of damage to adjacent structures such as the ureter, it is safer to explore lateral haematomas, even if not expanding. The surgeon must also be confident that there is no perforation of the posterior part of the colon in the paracolic gutters on either side.

6.6.1.4 PELVIC HAEMATOMA

If the patient is stable, contrast-enhanced CT in the emergency situation may demonstrate a large pelvic haematoma with a vascular ‘blush’, indicating ongoing arterial bleeding. In this case, it may be more appropriate to transfer the patient for immediate embolization.

*This surgery is fraught with hazard, and exploration of such haematomas should be a last resort. Wherever possible, angiographic visualization and embolization of any arterial bleeding must be tried before surgery is started if the patient is sufficiently stable. However, rapidly expanding or pulsating haematomas in this region may need exploration.*

The first concern should be to apply a binder to reduce pelvic volume. Stabilization of the pelvis using external fixators or a C-clamp in the emergency situation can be considered, but this does not always provide adequate posterior fixation, and may interfere with subsequent visualization of vessels for embolization. If the patient is too unstable for angiography, damage control surgery with packing of the pelvis should provide initial control. The peritoneum is incised over the distal aorta or
the iliac vessels, in order to control the arterial inflow, before attention is directed to the actual injury. However, it is best to leave pelvic haematomas undisturbed if they are not rapidly expanding or pulsatile, because they are most probably the result of pelvic venous damage. These veins are notoriously fragile and unforgiving to any attempt at repair.

There is recent literature to support extraperitoneal pelvic packing as the most efficient damage control technique to control this type of bleeding (see Section 7.6, Pelvic packing and associated references). After packing the patient should be sent directly to the angiographic suite from the operating theatre, for embolization, without further exploration.

### 6.6.2 Access

#### 6.6.2.1 Incision

The patient must be prepared ‘from sternal notch to knee’. It is critical to gain proximal and distal control, and the patient preparation should include the need to extend to a left lateral thoracotomy to gain access to the thoracic aorta, a median sternotomy to control the intracardiac inferior vena cava and groin incisions to gain control of the iliac vessels.

#### 6.6.2.2 Aorta

Control of the aorta can be achieved at several different levels depending on the site of injury (Figure 6.8). The supraceliac aorta can be exposed by incising the gastrohepatic ligament and retracting the left lobe of the liver superiorly and the stomach inferiorly. A window is then made in the lesser omentum and the peritoneum overlying the crura of the diaphragm is divided. The fibres of the crura are separated by sharp or blunt dissection. This is often difficult, but is essential for proper exposure in this area. The oesophagus is mobilized to the left in order to reach the abdominal aorta at the diaphragmatic hiatus. The aorta can be clamped or compressed at this point.

Exposure of the suprarenal aorta is not ideal with this anterior approach, and better exposure can be obtained by performing a left medial visceral rotation procedure. This entails mobilization of the splenorenal ligament and incision of the peritoneal reflection in the left paracolic gutter, down to the level of the sigmoid colon. The left-sided viscera are then bluntly dissected free of the retroperitoneum and mobilized to the right. Care should be taken to remain in a plane anterior to Gerota’s fascia. The entire abdominal aorta and the origins of its branches are exposed by this technique. This includes the coeliac axis, the origin of the superior mesenteric artery, the iliac vessels and the left renal pedicle. The dense and fibrous superior mesenteric and coeliac nerve plexus, however, overlie the proximal aorta and need to be sharply dissected in order to identify the renal and superior mesenteric arteries.

The distal aorta can be approached transperitoneally by retracting the small bowel to the right, the transverse colon superiorly and the descending colon to the left. The aorta below the left renal vein can be accessed by incising the peritoneum over it and mobilizing the third and fourth parts of the duodenum superiorly. Both iliac vessels can be exposed by distal continuation of the dissection. The ureters should be identified and carefully preserved, especially in the region of the bifurcation of the iliac vessels.

#### 6.6.2.3 Coeliac Axis

The left colon is reflected to the right, together with the spleen and tail of pancreas, to display the aorta and its
branches. The coeliac trunk lies behind and inferior to the gastro-oesophageal junction. Injuries to this area are commonly missed, particularly in patients with stab wounds. Major vascular injury is particularly likely if there is a central retroperitoneal haematoma. In this situation, proximal vascular control before entering the haematoma is essential, either locally or via a left lateral thoracotomy. Division of the left triangular ligament and mobilization of the lateral segment of the left lobe of the liver are also helpful.

It is difficult to repair the coeliac axis. The artery can be tied off, provided that the tie is proximal to its main branches and the superior mesenteric artery is intact. The left gastric and splenic arteries can be tied. The common hepatic artery can be safely tied provided that the injury is proximal to the gastroduodenal artery.

6.6.2.4 SUPERIOR MESENTERIC ARTERY

The superior mesenteric artery is a vital artery for the viability of the small bowel, and should always be repaired, using conventional techniques. Proximally, the artery is accessible from the aorta at the level of the renal arteries, and is best approached with a left medial visceral rotation. More distally, the artery is accessed at the root of the small bowel mesentery.

If a period of ischaemia has elapsed, the artery should be shunted, using a plastic vascular shunt (e.g. Javed shunt), until repair can be effected.

If repair is not possible, and replacement of the artery with a graft is required, it is best to place the graft on the infrarenal aorta, away from the pancreas and areas of potential leak. Placement of the proximal end of the graft too high can result in kinking and subsequent occlusion of the graft when the bowel is returned to the abdominal cavity. The graft must be tailored so that there is no tension and the aortic suture line must be covered to prevent aortoenteric fistula.

The survival rate with penetrating injuries of the superior mesenteric artery is approximately 58 per cent, falling to 22 per cent if a complex repair is required.

6.6.2.5 INFERIOR MESENTERIC ARTERY

Injuries to the inferior mesenteric artery are uncommon, and the artery can generally be tied off. The viability of the colon should be checked before closure, with planned reoperation to evaluate viability of the colon.

6.6.2.6 RENAL ARTERIES

Preliminary vascular control is best obtained by accessing the renal arteries on the aorta using a standard infrarenal aortic approach. Access can also be obtained by mobilizing the viscera medially.

Repair is done using standard vascular techniques. However, the kidney tolerates warm ischaemia poorly, and its viability after 45 min is in doubt. Therefore, if there has been complete transection of the artery, and the kidney is of doubtful viability, preservation may not be in the best interest of the patient.

6.6.2.7 Iliac Vessels

Proximal and distal control may be required, and distal control via a separate groin incision should be considered.

The iliac vessels are exposed by lifting the small bowel upwards, out of the pelvis. On the left, the sigmoid colon and its mesentery can be mobilized and, on the right, division of the peritoneal attachments over the caecum and mobilization of the caecum to the midline will aid exposure of the vessels.

The ureters must be formally identified as they cross the iliac bifurcation.

The common iliac veins are often adherent to the back wall of the common iliac artery, and attempts to mobilize the veins for control may result in torrential bleeding. A vascular clamp, applied proximally from above to the hypogastric and iliac veins, may be preferable to direct control.

6.6.2.8 Inferior Vena Cava

Suprahepatic inferior vena cava

Access to the suprahepatic inferior vena cava (IVC) can be obtained by incising the central tendon of the diaphragm or by performing a median sternotomy and opening the pericardium.

Infrahepatic IVC

The infrahepatic IVC can be exposed by means of a right medial visceral rotation procedure (see Right medial visceral rotation, in Section 6.1.2). The right colon is mobilized by taking down the hepatic flexure and incising the peritoneal reflection down the length of the right paracolic gutter. The colon is then reflected medially in a
plane anterior to Gerota’s fascia. If more exposure is required, the root of the mesentery can be mobilized by dividing the inferior mesenteric vein. Performance of Kocher’s manoeuvre and medial mobilization of the duodenum and head of the pancreas will reveal the segment of vena cava immediately below the liver, and provide excellent exposure of the right renovascular pedicle as well (Figures 6.9 and 6.10).

Control is best achieved by direct pressure on the IVC above and below the injury, using swabs, or controlling inflow and outflow using Rummell tourniquets (Figure 6.11).

Injuries to the posterior part of the IVC should always be expected with penetrating injury to the anterior part of the IVC. Not all bleeding posterior wounds need to be repaired.

It is very difficult to ‘roll’ the IVC to approach it posteriorly, as a result of multiple lumbar veins, so all injuries should be approached transcavally. Not all non-bleeding posterior wounds require repair.

Figure 6.9 Access to the inferior vena cava.

Figure 6.10 Access to the inferior vena cava.

Figure 6.11 Control of inferior vena cava with a combination of vascular clamps and a Rumel tourniquet.
Provided that it is infrarenal, ligation of the IVC is acceptable.

6.6.2.9 PORTAL VEIN

The portal vein lies in the free edge of lesser omentum, together with the common bile duct and hepatic artery (Figure 6.12). It can generally be controlled with Pringle’s manoeuvre. If the injury is more proximal, it may be necessary to reflect the duodenum medially or divide the pancreas.

The portal vein should be shunted early to avoid venous hypertension of the bowel, which will make access to the area increasingly difficult. The stent can be left in place as part of a damage control procedure, or repaired. Portocaval shunt is a possibility, and ligation as a last resort, however, it carries a high mortality.

6.6.2.10 REFERENCES


6.7 THE UROGENITAL SYSTEM

6.7.1 Overview

Urogenital trauma refers to injuries to the kidneys, ureters, bladder, urethra, the female reproductive organs in the pregnant and non-pregnant state, and the penis, scrotum and testes.

Death from penetrating bladder trauma was mentioned in Homer’s Iliad, as well as by Hippocrates and Galen, while Evans and Fowler in 1905 demonstrated that the mortality rate from penetrating intraperitoneal bladder injuries could be reduced from 100 per cent to 28 per cent with laparotomy and bladder repair. Ambroise Paré observed death after a gunshot wound to the kidney, with haematuria and sepsis, and it was only in 1884 that nephrectomy became the recommended treatment for renal injury.

Haematuria is the hallmark of urological injury but may be absent even in severe trauma, and a high index of suspicion is then needed, based on the mechanism of injury and presence of abdominal and pelvic injury.

6.7.2 Renal injuries

6.7.2.1 INVESTIGATION

The first investigation is to look for gross haematuria, followed by urinalysis to check for microscopic haematuria.

Up to 30 per cent of patients with serious renal trauma will have no haematuria whatsoever, although the majority of patients with significant abdominal trauma will have microscopic haematuria, often in the absence of relevant renal injury.

The haemodynamic status of the patient will then determine the subsequent steps, in both blunt and penetrating trauma.

Unstable patient

The investigation of choice in unstable patients is immediate surgery.

Stable patient

The investigation of choice is the multiphase, double- or triple-contrast CT scan; however, it can misgrade the renal injury. More commonly, however, it does allow
grading of renal injuries and forms the basis for non-operative treatment, possibly up to, and inclusive of, non-vascular grade IV injuries and blunt renal artery thrombosis.

It has been shown that the size of the haematoma can be related to the grade of renal injury, which is a useful correlation in suboptimal studies and where older machines are used.

Contrast-enhanced ultrasonography can allow visualization of active intrarenal bleeds and duplex Doppler ultrasonography can also allow visualization of arteriovenous fistulas and active intrarenal bleeds.

**Penetrating trauma**

The individual trauma centre's accepted method of evaluation of penetrating torso trauma must be used, the renal visualization being provided by intravenous urogram (IVU)/tomogram, followed by angiography if suspicious, or multi-phase contrast-enhanced CT scan of the abdomen as a stand-alone investigation.

**Blunt trauma**

Investigations as above are reserved for children irrespective of urinalysis, and adults with frank haematuria, or blood pressure < 90 mmHg.

### 6.7.2.2 MANAGEMENT

**Unstable patient**

At laparotomy, it will become apparent whether or not the kidneys are the source of the shock. Should a large retroperitoneal haematoma be present in the region of the kidney, the options are to leave the kidney alone at first and perform a single shot on-table IVU, to assess the functionality of both kidneys, explore the injured kidney immediately or pack the area around the kidney and get out, if in a damage control situation.

**Stable patient**

**Penetrating trauma**

The procedure of choice is to explore the kidney, unless it is obvious from observation or on-table IVU that the injury to the kidney is not the cause of the instability and there is no expanding haematoma, or damage control is required.

Up to half of renal stab injuries and up to a third of gunshot injuries in one series can be treated non-operatively, as long as excellent diagnostic methods can visualize the injuries.

**Blunt trauma**

An on-table IVU should be obtained even in the absence of a large haematoma to exclude renal artery thrombosis, followed by exploration with repair or nephrectomy in the persistently unstable patient.

### 6.7.2.3 POSTOPERATIVE CARE

Urinomas, infected urinomas, perinephric abscesses and delayed bleeding are the most common complications of conservative management and are often amenable to imaging and percutaneous, transureteric or angiographic management. Even when the kidney appears to be shattered into several pieces, drainage of the surrounding urinomas seems to encourage healing and avoid sepsis.

Hypertension is a rare late complication.

### 6.7.2.4 ACCESS

Access should be by midline laparotomy, even if isolated renal injury is suspected, because there is always the likelihood of other injuries.

The kidneys are usually explored after dealing with the intra-abdominal emergencies. Ideally, control of the renal pedicle should be obtained before opening Gerota's fascia.

A direct approach to suspected peripheral penetrating injuries is advocated, by some, as faster and equally safe. A left medial visceral rotation on the left, including division of Gerota's fascia and medial rotation of the left kidney, or extended Kocher's manoeuvre on the right, can also afford good control of the aorta, IVC and renal vessels, if required (see Figures 6.8–6.10 pages 131 and 133).

The peritoneum over the aorta is opened, and the anterior wall of the aorta followed up to the left renal vein (Figure 6.14). After exposing the retroperitoneum from the right or left, the left renal artery is identified by dissecting upwards on the lateral aspect of the aorta above the inferior mesenteric vein. The left renal vein crosses the aorta just below the level of the origin of the renal arteries. Access to the left renal artery may be improved by one of two manoeuvres. Ligation of the adrenal, gonadal and lumbar tributaries of the left renal vein will enhance the mobilization of the vein to expose the renal
artery (Figure 6.15). The second method involves ligation of the distal renal vein at the IVC to improve exposure of the origin of the renal artery. The collateral drainage via the lumbar gonadal and adrenal vessels will be sufficient to deal with the venous drainage on the left.

The right renal artery can be found by dissecting posteriorly between the aorta and the IVC. Dissection lateral to the IVC may lead to inadvertent isolation of a segmental branch of the right renal artery. The vessels are then controlled by loops to allow rapid occlusion should bleeding occur on opening Gerota’s fascia. The right renal vein is easily controllable after reflection of the right colon and duodenum, and has to be mobilized to expose the artery. It should always be repaired if possible, because of the lack of collateral venous drainage.

After control of the renal pedicle has been obtained, Gerota’s fascia can be opened or debrided as necessary. Care must be taken not to strip the renal capsule from the underlying parenchyma, because this may bleed profusely. The mobilized kidney can now be examined, debrided, trimmed and sutured with drainage.

The kidney tolerates a single ischaemic event much better than repeated ischaemic times. However, the maximum warm ischaemic time that the kidney will tolerate is less than 1 hour, although it can be prolonged by ice packing.

With complete vascular isolation for up to 30 min, Gerota’s fascia is then opened, and the injured kidney debrided by sharp dissection and sutured, or partially

Figure 6.13 Access to the right kidney.

Figure 6.14 Access to the left kidney.

Figure 6.15 Mobilization of the renal vessels on the left.
amputated Figure 6.16). The renal pelvis collecting system should be closed with a running absorbable suture to provide a water-tight seal. Nephrectomy will be required in less than 10 per cent of stable patients.

Cover can then be effected using the renal capsule, omentum, meshes, etc., replacing the kidney within Gerota’s fascia, and draining the area with a silicone or suction drain until it is draining minimally and collections have been excluded.

Nephrostomy tubes or ureteric stents can be used either immediately or at some later time in cases of major renal trauma with extravasation.

6.7.2.5 RECOMMENDED READING


6.7.3 **Ureteric injuries**

6.7.3.1 **INVESTIGATION**

Significant ureteric injuries are often missed or not picked up until late, after the onset of complications or deterioration in renal function.

They present without even microscopic haematuria in up to 50 per cent of cases, and are mostly associated with penetrating trauma, although ureteric avulsion and rupture can happen in blunt trauma, especially among children. Ureteric injuries may even be missed by high-dose IVU.

6.7.3.2 **MANAGEMENT**

**Unstable patients**

Unstable patients require immediate surgery and exploration of the ureter after life-threatening injuries have been dealt with, and ideally preceded by one-shot, on-table IVU.

If the patient requires an abbreviated laparotomy, the ureteric injury can be safely left alone, stented or ligated until the patient returns to the operating theatre for definitive procedures; indeed, successful repair has frequently been affected after delayed or missed presentation. Percutaneous nephrostomy can be used as a postoperative adjunct for the ligated ureter.

In unstable patients with associated colonic injuries, especially those requiring colectomy, even nephrectomy could be justified.

**Stable patients**

Stable patients with fresh injuries between the pelvi-ureteric junction (PUJ) and the pelvic brim are treated by end-uretero-ureterostomy with spatulation (Figure 6.17) and interrupted suturing over a double J-stent. The stent can be safely left in situ for 4–6 weeks. It has been suggested that stents can be omitted in injuries requiring minimal debridement, such as stab wounds, but not in gunshot wounds where stenting results in significantly fewer leaks.

Injuries to the PUJ and its vicinity are treated in the same fashion, but a tube nephrostomy should be added.

Injuries around the pelvic brim are best treated by uretero-neocystostomy, with an anti-reflux reimplantation.

More advanced repair methods include the retrocolic transuretero-ureterostomy, and the creation of a Boari flap with attached uretero-neocystostomy.

In the case of loss of long segments where anastomosis to the contralateral ureter is not possible, an end-ureterostomy could be brought out, or nephrectomy done in rare cases of serious associated injuries in the area.

After surgery, the bladder is drained transurethrally, or ideally suprapubically, and closed suction drains can be placed retroperitoneally in proximity to the repaired ureter and can be expected to drain for a number of days.

6.7.3.3 **COMPLICATIONS**

Complications comprise stricture with hydronephrosis, leakage from the anastomosis and infected urinomas, especially in late diagnosis, most of which are amenable to percutaneous management.

![Figure 6.17 Repair of ureter.](image-url)
6.7.3.4 ACCESS

The procedures described for access to the retroperitoneal great vessels also allow perfect exposure to both ureters. Ureteric injuries are rare, usually as a result of penetrating trauma, so that local exploration and mobilization of part of the ascending or descending colon alone may be sufficient, depending on the site of injury. Minimal dissection of the periureteric tissues should take place, except at the precise level of injury, so as to preserve the delicate blood supply. Ureteric injuries close to the kidney are accessed by left or right medial visceral rotation as described above. Ureteric injuries near the bladder may be accessed by opening the peritoneal layer in the region and mobilizing the bladder.

6.7.3.5 RECOMMENDED READING


6.7.4 Bladder injuries

Bladder injuries are mainly the result of blunt trauma and found in about 8 per cent of pelvic fractures. Penetrating trauma is caused by gunshot, stabs, impalement or iatrogenic injuries, mostly in relation to orthopaedic pelvic fixation.

6.7.4.1 INVESTIGATION

Signs and symptoms vary from inability to void and frank haematuria, to vague abdominal or suprapubic tenderness without haematuria in a small percentage of cases. Intraperitoneal injuries may be associated with a higher serum creatinine and urea, and low sodium, but this biochemical derangement takes some time to develop.

Ultrasonography and CT can be of use to demonstrate free fluid in the abdomen, the presence of clots in the bladder and a change in bladder filling and shape (with sonar probe compression). CT cystogram can be done as part of an abdominal CT study, and can differentiate between intra- and extraperitoneal bladder injuries.

Retrograde cystography is the method of choice in the emergency department, because it is very accurate as long as a large enough volume of contrast (about 7 mL/kg) is instilled, and at least two separate projections (anteroposterior and lateral views) are obtained. Post-micturition films are essential.

Contrast extravasation will delineate loops of bowel and the peritoneal contours in intraperitoneal ruptures, although it will be tracking along the pelvic bones, scrotum, obturator areas, etc. in extraperitoneal ruptures.

6.7.4.2 MANAGEMENT

Urgent operative treatment is indicated in all intraperitoneal, and some types of extraperitoneal injuries, while others require delayed surgery upon failure of non-operative methods. The majority of penetrating injuries require immediate surgery.

Non-operative management

Urethral or suprapubic catheter drainage with a large bore catheter, for up to 2 weeks, will allow most extraperitoneal injuries from blunt trauma to heal; surgery will be needed only if a cystogram at that stage shows ongoing leakage. Contraindications to non-operative management are bladder neck injury, presence of bony fragments through the bladder wall, infected urine and associated female genital injuries.

Extraperitoneal bladder repair during a laparotomy for other trauma is often easily accomplished, but may be dangerous if requiring opening into a tamponaded pelvic haematoma, and inappropriate in the context of damage control.

Operative management

Bladders can be repaired easily and with few complications with absorbable sutures.

All repairs should be carried out through an intraperitoneal approach, from within the lumen of the bladder, after performing an adequate longitudinal incision on the anterior surface in order to avoid entering lateral pelvic haematomas.

The presence and patency of both ureteric orifices must be confirmed in all cases. If suturing in the vicinity, these should be cannulated with a size 5 feeding tube or ureteric catheter.
In cases of gunshot wounds, wounds to the bladder must be both sought and identified. In some situations, it will be necessary to open the bladder widely, explore and repair from within.

Single layer mass suturing is indicated in extraperitoneal ruptures but for intraperitoneal ruptures closure should be in separate layers.

A large-bore transurethral or suprapubic catheter, or both, can be used, the latter being fed extraperitoneally into the bladder, and a drain left in Retzius’s space. A cystogram will be done in most cases after 10 days to 2 weeks, followed by removal of the suprapubic catheter.

6.7.5 Urethral injuries

Urethral injuries can have the most disastrous consequences of all genitourinary trauma, such as incontinence, long lasting impotence and strictures.

6.7.5.1 INVESTIGATION

Mechanism of injury, pelvic fracture and blood at the meatus must alert the surgeon to the possibility of a urethral rupture, mainly of the posterior urethra from blunt trauma.

Rectal examination is mandatory before urethral catheter insertion, and a high riding prostate will suggest that the urethra is disrupted. Rupture of the female urethra is, fortunately, very uncommon.

Once rupture is suspected, two completely different approaches are practised and acceptable: retrograde urethrography is performed by placing a small Foley catheter in the fossa navicularis, with the patient in the oblique position.

The preferable approach is, however, not to intervene with an emergency procedure at all. This is particularly important if a pelvic haematoma is present as a result of the risk of effectively causing a compound injury. It is preferable to place a suprapubic catheter (it will be necessary to allow the bladder to fill until it is palpable) before its insertion. A cystogram and, if necessary, cystoscopy can be done under controlled circumstances at a later stage.

6.7.5.2 MANAGEMENT

Suprapubic cystostomy

The mainstay of immediate treatment is the placement of a suprapubic catheter for urinary drainage. This can be done as an isolated open procedure or an open procedure during a laparotomy, or with a percutaneous method. Isolated open method requires a lower midline laparotomy incision and an intraperitoneal approach to the bladder to avoid entering a pelvic haematoma. Suprapubic placement during a laparotomy done for other reasons follows the same principles.

Percutaneous placement is done using specifically designed trochar and catheter kits. It requires a full bladder, as identified clinically or on ultrasonography. If this is not the case, and the patient is not in a condition to produce a lot of urine, a small intravenous catheter can be placed under ultrasonic guidance using the Seldinger technique, and the bladder is then distended with saline until a standard percutaneous method can be used.

6.7.5.3 RUPTURED URETHRA

Urethral repair

Immediate surgical intervention is recommended for the following conditions:

- All penetrating injuries of the posterior urethra and most of the anterior urethra
- Posterior urethral injuries associated with rectal injuries and bladder neck injuries
- Where there is wide separation of the ends of the urethra.
- Penile fracture.

Accurate approximation and end-to-end anastomosis is recommended for injuries to the anterior urethra, whereas, for membranous urethra injuries, realignment and stenting over a Foley catheter for 3 or 4 weeks may be sufficient. This can be achieved by an open lower midline laparotomy and passage of Foley catheters from above and below, with ultimate passage into the bladder, or via flexible cystoscopy and manipulation.

Patients managed with a suprapubic catheter alone should have their definitive urethral repair after about 3 months from the injury.
Primary realignment may have better results than delayed repair, but delayed primary repair (days 8–10) is recommended when there is a large haematoma.

6.7.6 Injury to the scrotum

6.7.6.1 Diagnosis

Ultrasonography of the scrotum is indicated in the evaluation of blunt trauma to the testicle, and can differentiate torsion, disruption and haematoma.

6.7.6.2 Management

Blood supply is so good that penetrating trauma can usually be treated by debridement and suturing.

If the tunica vaginalis of the testis is disrupted, the extruding seminiferous tubules should be trimmed off and the capsule closed as soon as possible, in order to minimize host reaction against the testis.

Loss of scrotal skin with exposed testicle, a well-described occurrence after burns and other trauma, can often be remedied by the creation of pouches in the proximal thigh skin, and subsequent approximation, with little effect on the testicles.

6.7.6.3 Recommended Reading


6.7.7 Gynaecological injury or sexual assault

Any evidence of gynaecological injury requires external and internal examination using a speculum, and exclusion of associated urethral and anorectal injuries. If rape is suspected or reported, the official sexual assault evidence collection kit should be used and detailed clinical notes made. The patient must be counselled and informed consent must be obtained for examinations where possible.

Reporting of all cases of sexual assault should be carried out by the treating physician, in order to minimize under-reporting by the already traumatized patient.

6.7.7.1 Management

Lacerations of the external genitalia and vagina can be sutured under local or general anaesthesia, and a vaginal pack left in for 24 h to minimize swelling.

Intrapelvic organs are dealt with at laparotomy by suturing, hysterectomy or oophorectomy. Oxytocin is used to minimize uterine bleeding and colostomy to avoid soiling.

Additional supportive care for the psychological effects of sexual assault should be made available.

Anti-retroviral treatment is more effective if instituted within 3 h of injury and sexually transmitted infection and pregnancy prophylaxis should be given according to standard protocols. Baseline blood tests required include HIV, hepatitis B, FBC, and liver and renal functions, and follow-up arrangements must be made to monitor medication and HIV status.

6.7.8 Injury of the pregnant uterus

Aggressive resuscitation of the mother and the fetus must be carried out in keeping with ATLS® recommendations. Midline laparotomy should always be used when surgery is necessary, but simple intrauterine death is best managed by induced labour at a later stage.
INTRODUCTION

Fractured pelvis is a surgical problem, as 65 per cent of patients with a fractured pelvis suffer associated injuries, and mortality is largely a result of haemorrhage and infections in pelvic soft tissues. Both can lead to multiple organ failure. General surgeons are trained to resuscitate and care for patients as a whole, and rely on other disciplines for system-specific input. Severe single system trauma is best managed by the appropriate discipline after surgical assessment.

Pelvic fractures should be easily identified if Advanced Trauma Life Support (ATLS®) guidelines are followed (i.e. routine chest radiograph, and cervical spine and pelvis radiographs for any blunt injury in a patient unable to walk). The first problem to deal with is hypovolaemic shock, of which the unstable pelvic ring is one of the major possible causes. One-third of trauma victims with pelvic ring fractures sustain circulatory instability on arrival. Localized soft tissue injuries such as perineal and urethral injuries are inherently hidden and more frequently missed. Evidence of perineal injury or haematuria mandates radiological evaluation of the urinary tract from below upward (retrograde urethrogram followed by cystogram or computed tomography [CT] cystogram, followed by excretory urogram as appropriate). The presence of blood per rectum in a patient with pelvic injury indicates anorectal injury, which should be identified and definitively managed.

ANATOMY

The surgical anatomy of the pelvis is a key to the pathogenesis of pelvic injuries:

- The pelvis has a rich collateral blood supply, especially across the sacrum and posterior part of the ileum. The cancellous bone of the pelvis also has an excellent blood supply. Most pelvic haemorrhage emanates from venous injury and fracture sites.
- Postmortem examination has shown that the pelvic peritoneum, which 'should' tamponade pelvic haematomas, can accommodate more than 3000 mL.
- The pelvic inlet is circular, a structure that routinely gives way at more than one point, should sufficient pressure be applied to it.
- All iliac vessels, the sciatic nerve roots, including the lumbosacral nerve, and the ureters cross the sacroiliac joint. Disruption of this joint may cause severe haemorrhage and sometimes causes arterial obstruction of the iliac artery and nerve palsy. Fortunately, injuries to the ureters are rare.
- The sacroiliac joint is covered by the psoas muscle, which is embedded in a tight fascia, also enclosing the femoral nerve. Swelling within this fascia may cause a compartment syndrome and possibly quadriiceps muscle paralysis.
- The pelvic viscera are suspended from the bony pelvis by condensations of the endopelvic fascia. Shear forces acting on the pelvis will transmit these to pelvic viscera, leading to avulsion and shearing injuries.
- The pelvis also features the acetabulum, a major structure in weight transfer to the leg. Inappropriate treatment will lead to severe disability.

CLINICAL EXAMINATION

Clinical examination starts with gentle bimanual lateral and anteroposterior (AP) compression (not distraction!) of the pelvis. Any instability felt indicates the presence of major pelvic instability, associated with life-threatening blood loss, which requires appropriate measures. The absence of clinical instability does not, however, preclude an unstable pelvic fracture. An early pelvic radiograph is part of the initial assessment of any severe trauma patient.
Inspection of the skin may reveal lacerations in the groin, perineum or sacral area, indicating a compound pelvic fracture, the result of gross deformation. Inspection of the urethral meatus may reveal a drop of blood, indicating urethral rupture.

Inspection of the anus may reveal lacerations of the sphincter mechanism. Diligent rectal examination may reveal blood in the rectum and/or discontinuity of the rectal wall, indicating a rectal laceration. In men, the prostate is palpated; a high riding prostate indicates a complete urethral avulsion.

A full neurological examination is performed of the perineal area, sphincter mechanism, and femoral and sciatic nerves.

### 7.4 CLASSIFICATION

Pelvic ring fractures can be classified into three types, using the Tile classification,\(^1\)\(^2\) based on their severity.

**Type A**

Isolated fracture of iliac wing, or pubic rami, mostly caused by direct compression. These are stable fractures, to be treated conservatively.

**Type B**

These are subdivided into the following types.

**TYPE B1**

This occurs less commonly, and is a lateral compression-type injury, which results in an intrinsically stable fracture of the pelvic ring, with an impression of the posterior complex in the sacral bone and mostly a fracture of the pubic arch. Perforation of the bladder can be caused by the anterior fracture, but also hypovolaemic shock as a result of severe disruption of the soft tissues of the pelvic diaphragm and organ injury to the lower urogenital tract and rectum can be seen in these cases.

**TYPE B2**

This is the most common type of fracture, also known as an ‘open-book’ fracture. There is horizontal (rotational) instability as a result of an anterior lesion (disruption of the symphysis and/or fracture of superior and inferior pubic rami), combined with a posterior disruption of the anterior or posterior ligaments of the sacroiliac joint. It can result in bleeding in an enlarged lesser pelvic cavity. Injury of the lower urogenital tract, rectum, vagina and severe soft tissue damage as a result of the rotation is often seen in these patients. Internal or external stabilization is required.

**TYPE C**

Complete horizontal and vertical instability, as a result of anterior and posterior fractures and/or disruptions (complete sacroiliac disruption, displaced vertical sacral fracture). A fall from height, but also AP shearing forces in a dashboard impact in a motor vehicle accident result in this type of fracture. Type C fracture is the result of major mechanical forces, and is associated with major blood loss and related injuries within the pelvis (bladder, urethra, rectum, vagina, sciatic and femoral nerve, iliac vein and artery). The most extreme case with complete neurovascular threat to one leg might qualify as a traumatic hemipelvectomy – fortunately a very rare situation.

In both types B and C pelvic injuries there is a high risk of associated abdominal injuries (bowel perforation) and laceration of the diaphragm.

### 7.5 RESUSCITATION

The priorities for resuscitating patients with pelvic fractures are no different from the standard. These injuries produce a real threat to the circulation and management is geared towards controlling this threat. The patient is haemodynamically normal, haemodynamically stable (maintaining output as a result of ongoing resuscitation) or haemodynamically unstable. Management is based on haemodynamic status:

**HAEMODYNAMICALLY NORMAL PATIENTS**

This is usually an isolated injury, possibly requiring external or internal (open) reduction and fixation to limit future instability and disability. The management is not critically urgent.

**HAEMODYNAMICALLY STABLE PATIENTS**

As a result of the capacity of the pelvis to bleed, these patients require urgent control of haemorrhage.
Traditional external fixation cannot provide complete stability or compression. A force applied to a segment of a circle cannot stabilize defects outside that segment; it can do so only in one dimension and will aggravate disruption outside the segment across which it is applied. Pelvic C-clamps are applied close to the maximum diameter of the pelvis and should be more effective in providing pelvic compression. Their application may be more difficult.

- If facilities allow and time permits, performing angiographic embolization provides better control of haemorrhage than external fixation.
- Apply an external fixator with anterior compression if a type B injury.
- Apply a pelvic clamp in a type C injury at the level of the sacroiliac joint with posterior compression.

HAEMODYNAMICALLY UNSTABLE

As a result of the capacity of the pelvis to bleed, these patients require urgent control of any haemorrhage. These patients tend to exsanguinate rapidly and immediate measures are required to control bleeding. Applying a sheet at the level of the greater trochanters is often effective and less time-consuming than the external fixator and the C-clamp, immediately followed by bleeding control. In a type C fracture, any vertical dislocation should be reduced by applying traction to the leg on the side of the cranially dislocated hemipelvis, before binding the pelvis together.

- If facilities allow and the patient's haemodynamics permit, angiographic embolization should be performed.
- Consider application of external fixator with anterior compression in a type B injury. Traditional external fixation cannot provide complete stability or compression. A force applied to a segment of a circle cannot stabilize defects outside that segment; it can do so only in one dimension and will aggravate disruption outside the segment across which it is applied.
- Consider application of pelvic clamp in a type C injury at the level of the sacroiliac joint with posterior compression. Pelvic C-clamps are applied close to the maximum diameter of the pelvis and should be more effective in providing pelvic compression. Their application may be more difficult.
- In the more stable patient, if bleeding persists, explore the pelvis and tamponade the area with suturing or ligature of lacerations of major blood vessels; repair the anatomical structures (bladder, rectum) where possible; and use cystostomy and/or colostomy with rectal wash-out as required. If required, and possible at this stage, internally fix the pelvic ring.
- In the exsanguinating patient, immediate control of haemorrhage can be achieved by aortic clamping. Other sources of intra-abdominal bleeding must be excluded. Consider damage control surgery (DCS) with just packing of the pelvis (consider extraperitoneal pelvic packing). If bleeding persists, direct exploration with suturing or ligature of lacerations of major blood vessels should be performed. Angiography should be performed after DCS for control of remaining pelvic bleeding by embolization of the bleeding vessels.
- Consider damage control, with just packing of the pelvis and external fixator in type II fractures. Repair anatomical structures (bladder, rectum), and use cystostomy and/or colostomy with rectal wash-out as required.
- Tamponade pelvic area for any residual capillary bleeding. Leave appropriate drains.
- Temporary closure of abdominal wall, using damage control techniques and leaving skin and subcutaneous tissue open.

Requirements for blood average 15 units for compound pelvic fractures. To avoid dilutional coagulopathy, massive transfusion protocols should be instituted. Volume replacement is ultimately only an adjunct for the treatment of haemorrhagic shock – stopping the bleeding. Hypotension is particularly dangerous in the presence of associated head injury.

7.6 PELVIC PACKING

The strategy for dealing with major pelvic haemorrhage remains controversial. Currently, the cornerstones of management of haemorrhage from unstable pelvic fractures revolve around the following:

- Early identification of the problem
- Resuscitation
- Control of pelvic bleeding with a combination of pelvic binders and external fixation
- Angiographic embolization of bleeders.

If this is unsuccessful, transabdominal packing is used, with attempts at direct control, packing and ligation of internal iliac arteries.
Of pelvic bleeding, 85 per cent is venous, arising from the multiple venous plexus around the pelvis. This bleeding is not controllable by embolization.

Extraperitoneal packing was first described in 1985 by Pohleman et al., and the technique was further described by Ertel et al. in 2001 and Smith et al. in 2005. The original technique was more aggressive, but the current technique stops below/medial to the external iliac vessels at the pelvic brim.

### 7.6.1 Technique of retroperitoneal packing

The patient is positioned supine and, if necessary, an external fixator or C-clamp is applied. An 8 cm midline suprapubic incision is made and the fascia anterior to the rectus muscle exposed. The fascia is divided in the midline, protecting against urinary bladder damage. The fascia is then dissected away from the pelvic brim, as far posteriorly as possible at the level of the pelvic brim. The bladder is held to one side. Three laparotomy sponges are then placed deep to the brim: the first is placed posteriorly just below the sacroiliac joint; the second more anteriorly, at a point corresponding to the middle of the pelvic brim; and the third in the retropubic space just deep and lateral to the bladder. The procedure is repeated on the opposite side.

The outer fascia is closed with a single running suture, and the skin closed. If laparotomy is required, it should follow the packing procedure. As in the abdomen, the packs should be removed after 24–48 h.

### 7.7 ASSOCIATED CONDITIONS

Associated injuries can be managed only once the patient is haemodynamically stable. Procedures for damage control may be the only available option:

- Head injuries are the most commonly associated major injuries. It is worthwhile remembering that ‘C’ precedes ‘D’ during resuscitation and management. Computed tomography and neurosurgical procedures have to wait for haemodynamic stability, which may be achieved only after DCS.
- Intra-abdominal injuries are frequently masked by pelvic pain. Retroperitoneal haematomas may break through into the peritoneal cavity, causing a false-positive diagnostic peritoneal lavage (DPL). In the presence of a pelvic fracture, CT is the diagnostic modality of choice in the stable patient. In all other patients, diagnostic ultrasonography is preferred. Open DPL is preferred to exclude intraperitoneal haemorrhage. If open DPL is performed, the entry point should be above the umbilicus to avoid entering extraperitoneal haematomas tracking up the anterior abdominal wall. A low threshold should be maintained for laparotomy because of associated intraperitoneal injury.
- Urethral injuries should be managed conservatively. Primary urethral repair by cystoperineal traction sutures results in minimal disability in the hands of experts, when performed immediately in stable patients. For the majority, suprapubic cystostomy and delayed urethral repair are required.
- Anorectal injuries are managed according to the degree of damage to the sphincters and anorectal mucosa. Injuries superficial to these require only debridement and dressings. Deep injuries require colostomy and drainage (presacral drainage via a coccygectomy is not required). There is doubt about the benefit of prograde wash-out of the rectum as a result of the risk of pelvic infection introduced by washing faeces into the pelvic cavity. Careful mechanical cleansing of the rectum, wash-out via a wide-bore tube after gentle anal dilatation and adequate debridement performed in a stable patient makes common sense. Sphincter repair is best left for the experts but repeated debridement and early approximation of mucosa to skin should limit infection and scarring.

In summary, a haemodynamically normal patient can be safely transferred for stabilization of unstable fractures within hours of injury and after control of the associated damage.

Associated injuries can be managed only once the patient is haemodynamically stable. Procedures for damage control may be the only option available.

### 7.8 REFERENCES


### 7.9 RECOMMENDED READING


8.1 OVERVIEW

This chapter should be read in conjunction with Chapter 13 (Skeletal trauma), which focuses on the orthopaedic aspects of extremity injury.

8.1.1 Introduction

Injuries of the limbs rarely threaten life – but it happens, and the possibility must be borne in mind when there are multiple long bone fractures associated with vascular damage and ongoing bleeding. Where this is the case, a timely and appropriately applied tourniquet may buy time to stabilize the patient and treat other life-threatening injuries.

Extremity injuries often look dramatic and occur in 85 per cent of patients who sustain blunt trauma, but rarely cause a threat to life or limb. However, in some circumstances, the relevance of such injuries assumes major importance.

8.1.2 Massive limb trauma: life versus limb

Certain skeletal injuries by their nature indicate significant forces sustained by the body and should prompt the treating surgeon to look for other associated injuries. Other limb injuries, presenting with crush injury with extensive soft tissue damage, concomitant vascular or nerve injury, and major bony disruption, pose other threats to either life or limb and it is on these that this section concentrates.

Despite huge advances in the management of these injuries, and the resultant decrease in amputation rates associated with them, there remains a small group of patients who present with ‘mangled limbs’, produced by mechanisms of high-energy transfer or crush in which there is vascular disruption in combination with severe, open, comminuted fractures and moderate loss of soft tissue. These injuries most frequently affect healthy individuals during their prime years of gainful employment and can result in varying degrees of functional and emotional disability.

There are many ways to classify major limb injuries and their complications, and these scoring systems can be found in the tables in this chapter.

During the past two decades, better understanding of the individual injuries, and technical advances in diagnostic evaluation and surgery (allowing revascularization of the extremity, stabilization of the complex fracture and reconstruction of the soft tissues), medicine and rehabilitation have led to an increased frequency of attempts at limb salvage. In some of these patients, however, limb salvage may have subsequent deleterious results, being associated with a high morbidity and a poor prognosis, and often requiring late amputation (27–70 per cent) despite initial success. In these, early or primary ablation might even be beneficial.

Thus the management of the mangled limb remains a vexing problem and should be multidisciplinary and involve the combined skills of the orthopaedic, vascular, and plastic and reconstructive surgeons. Poorly coordinated management often results in more complications, increased duration of treatment and a less favourable outcome for the patient. Ultimately, the decision to amputate or repair is often a difficult one and best shared, if possible, with a senior colleague. The cost of rehabilitation is often less – and the time shorter – if a primary amputation is performed, than if lengthy and repeated operations are undertaken and persistent painful debility or an insensate or flail limb is still the outcome. A successful limb salvage is defined by the overall function and satisfaction of the patient.
8.1.3 Management

It is important to remember that a fracture is not a separate entity from the soft tissue damage that accompanies it – it is simply an extension of the soft tissue injury that involves bone, and the principles of management are the same.

8.1.3.1 Vascular Injury

Vascular injuries are present in 25–35 per cent of all penetrating trauma to the extremities. More recently, duplex scanning may play a useful screening role. Except for inconsequential intimal injuries and distal arteries, most extremity vascular injuries should be repaired.

Signs of vascular injury include an expanding or pulsating haematoma, to-and-fro murmurs, a false aneurysm, continuous murmurs of arteriovenous fistulas, loss of pulses, progressive swelling of an extremity, unexplained ischaemia or dysfunction, and unilateral cool or pale extremities. A significant percentage of these patients have no physical findings to suggest vascular trauma; routine further investigation has been advocated.

The most common cause of peripheral vascular injury is penetrating trauma, which includes the spectrum from simple puncture wounds to wounds resulting from high-velocity missiles. Normal pulses do not rule out vascular injuries; 10 per cent of significant and major vascular injuries have no physical findings. Penetrating trauma also includesiatrogenic injuries such as those following percutaneous catheterization of peripheral arteries for diagnostic procedures or access for monitoring. When a needle or catheter dislodges an arteriosclerotic plaque or elevates the intima, a vessel may thrombose, leading to acute ischaemia in a limb. The key, therefore, is to maintain a high index of suspicion based on the mechanism of injury and the proximity of vascular structures.

Recently, duplex scanning of blood vessels has been shown to be a useful adjunct in determining whether an arteriogram is indicated. A positive duplex scan is valuable, but a negative one does not exclude vascular injury for duplex scanning. A positive duplex scan, or an ankle brachial index (ABI) < 0.9 in a distal pulse, is a mandatory indication for an arteriogram and possible surgery.

The gold standard for confirming a suspected vascular injury remains the arteriogram. However, arteriography should not be performed in a patient who is unstable and needs emergency laparotomy or thoracotomy. An arteriogram should be delayed until after resuscitation and treatment of the life-threatening emergency.

If doubt exists, an angiogram should be obtained!

Blunt trauma may also cause peripheral vascular injuries, with shear injuries as the most common cause. Contusions or crushing injuries may produce transmural or partial disruption of arteries, resulting in elevation of the intima and formation of intramural haematomas. Blunt trauma, such as posterior dislocation of the knee, may cause total disruption of a major vessel. Blunt trauma may also indirectly contribute to vascular occlusion by creating large haematomas in proximity to the vessel. This haematoma may lead to arterial spasm, distortion or compartment syndromes that interfere with arterial flow.

In principle, it is wise to fix the bony skeleton before embarking on vascular repair. However, this can be catastrophic if ischaemia is present. The following protocol should be used:

- Initial assessment for ischaemia
- Exploration of the vessels
- Fasciotomy if required
- Temporary stenting of the vein and artery
- Orthopaedic fixation of the skeletal damage
- Definitive repair of the vascular damage.

Damage control of the extremity injury should take place in the same fashion as in the abdomen. If there is doubt regarding viability, the wound should not be closed.

There are five options open to the surgeon when vascular damage is encountered: vessels may be repaired, replaced (grafted), ligated (and bypassed), stented or shunted.

Intraluminal shunts may be manufactured out of intravenous tubing, nasogastric tubing, biliary ‘T’ tubes or even chest drain tubing, depending on the size of the vessel to be shunted. Commercially made shunts are on the market (as used routinely in carotid surgery) and others are now being made specifically for trauma. Essentially, the shunt is tied into the damaged vessel and ligated securely proximally and distally – there is no need for heparinization – and this allows time for other damage control procedures to take precedence while maintaining perfusion of the limb. Where possible, both artery and vein should be shunted if both are damaged. If not possible, the vein should be tied off. The shunts may safely be left in place for 24 h and probably longer – there are no controlled trials.

Some injury complexes should raise a specific suspicion of vascular damage: the supracondylar fracture of
the humerus or femur, and posterior dislocation of the knee. The presence of palpable pulses does not exclude arterial injury, and a difference of 10 per cent in the measured Doppler pressure compared with the opposite uninjured limb mandates urgent angiography. This is not hard to do and the technique is well described elsewhere. An absent pulse mandates exploration if the level of injury is known, and angiography if not.

Repairs, and particularly graft replacements of injured vessels, should be attempted only by those competent to do them, and only in limbs where the viability of the soft tissues is not in doubt (i.e. after fasciotomy). Ligation may be done as a measure of desperation in the exsanguinating patient and limb survival is often surprising. Claudication pain may be dealt with at a later date. Extra-anatomical bypass has no place in the setting of damage control and trauma surgery. Endovascular stenting is rapidly becoming a procedure of choice in some areas (e.g. traumatic aortic rupture), but requires facilities and expertise that may not always be available.

8.1.3.2 CHEMICAL VASCULAR INJURIES

The frequency of chemical injury to blood vessels has increased secondary to iatrogenic injury and intra-arterial injection of illicit drugs. These agents may cause intense vasospasm or direct damage to the vessel wall, often associated with intense pain and distal ischaemia.

Chemical vascular injuries may be treated with intra-arterial or intravenous administration of 10 000 units heparin to prevent distal thrombosis. Reserpine (0.5 mg) has also been recommended, although its only effect experimentally has been to protect against the release of catecholamines from the vessel walls. Other vasodilators and thrombolytic enzymes have been tried with variable results. A reliable combination is 5000 units heparin in 500 mL Hartmann’s solution (Ringer’s lactate) to which is added 80 mg papaverine to combat arterial spasm. This is administered in boluses of 20–30 mL, intra-arterially every 30 min, or intravenously at the rate of 1000 units heparin/h.

8.1.3.3 CRUSH SYNDROME

Badly injured limbs will all have an element of crush syndrome associated with them, unless dealing with a traumatic amputation by a sharp instrument such as a chain saw or machete. As such, a watch must be kept for the development of a compartment syndrome and/or myoglobinuria.

8.1.3.4 COMPARTMENT SYNDROME

Compartment syndrome occurs relatively commonly, after trauma or ischaemia to an extremity. It is important to emphasize that reperfusion probably plays a major role. As such, the classic clinical findings may be absent before vascular repair. Once the diagnosis of compartment syndrome has been made, urgent fasciotomy is indicated.

The measurement of intracompartment pressure is invaluable when doubt exists about the diagnosis. It must be emphasized that a pulse may still be palpable, or recordable on Doppler, even though a compartment syndrome exists.

Should there be doubt as to whether the compartment syndrome is significant, then a fasciotomy should be performed.

8.1.3.5 INFECTION CONTROL

Sepsis is a constant threat to the healing of open fractures and the main risk factors include the severity of the injury, the delay from injury to surgical care, failure to use prophylactic antibiotics and inappropriate wound closure. In fractures classified using Gustilo grading (see Appendix B), grades I and II may be given agents effective against *Staphylococcus aureus*, but for grade III injuries broader Gram-negative cover is advisable.

8.1.3.6 VENOUS THROMBOEMBOLISM PROPHYLAXIS

Deep venous thrombosis prophylaxis remains an integral part of management of patients with severe limb injury.

8.1.3.7 TIMING OF FIXATION

The timing of skeletal fixation can prove challenging because the limb injury may only be part of a multisystem insult with associated head, chest and other injuries. Hypoxia, hypotension and tissue injury provide an initial ‘hit’ to prime the patient’s inflammatory response; surgical treatment of fractures constitutes a modifiable secondary insult. In addition, fat embolism has been implicated in the respiratory compromise that appears after orthopaedic injury. Nevertheless, most comparative studies have shown a reduction in the risk of post-traumatic respiratory compromise after early, definitive fixation of fractures (within 48 h) both for isolated injuries and for multisystem trauma. There is also evidence of a reduction in mortality, duration of mechanical ventilation, thromboembolic
events and cost in favour of early fixation. There is no evidence that early fixation alters the outcome in those with concomitant head injury.

8.1.4 Complications (Table 8.1)

In a review of 53 mangled lower extremities, Bondurant and associates\(^1\) compared primary with delayed amputation in terms of morbidity and cost. Patients undergoing delayed ablation had longer periods of hospitalization (22.3 vs 53.4 days), more surgical procedures (1.6 vs 6.9) and at greater cost ($US28 964 vs $US53 462). Six patients with delayed amputation developed sepsis from the injured lower extremity and died, whereas no patient with a primary amputation developed sepsis or died. Georgiadis et al.\(^2\) indicated that patients with limb salvage at an average of 3 years follow-up had more complications, more procedures and a longer hospital stay than patients with early below-the-knee amputations. Slow recuperation and decreased motivation towards gainful employment were also noted. The cost of initial hospitalization was lower with early amputation.

The decision to amputate primarily is difficult. At the initial examination, the extent of the eventual loss of soft tissue can never be fully appreciated, distal perfusion is also difficult to assess (many are shocked) and the neurological evaluation is often unreliable (associated head injury or ischaemia and soft tissue disruption). Any thoughts of limb salvage should take account of ATLS\(^\circ\) (Advanced Trauma Life Support) protocols, always maintaining the priority of life over limb, and thus minimizing systemic complications and missed injuries. In an attempt to facilitate this early decision-making, a number of systems have been devised providing objective criteria that use a grading score to predict which injuries might eventually require amputation.

8.2 SCORING SYSTEMS

8.2.1 Mangled Extremity Syndrome Index (Table 8.2)

Gregory et al.\(^3\) retrospectively reviewed 17 patients with severe injuries (12 of the lower extremity) and proposed a mangled extremity syndrome index (Table 8.2). The injury was categorized according to the integument, nerve, vessel and bone injury. A point system quantified injury severity, delay in revascularization, ischaemia, age of the patient, pre-existing disease and whether the patient was in shock.

<table>
<thead>
<tr>
<th>Table 8.1 Complications of fractures</th>
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<tbody>
<tr>
<td><strong>Skin and tissue loss, wound slough, coverage failure</strong></td>
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<tr>
<td>Bone and fracture site</td>
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<td>Nerve</td>
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<tr>
<td>Joint motion</td>
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<td>Secondary</td>
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<td></td>
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<tr>
<td>Cosmesis</td>
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</table>
8.2.2 Predictive salvage index system (Table 8.3)

Howe et al.\(^4\) reviewed 21 patients with pelvic or lower-extremity trauma with vascular injuries, and proposed a predictive index incorporating the level of the arterial injury, degree of bony injury, degree of muscle injury and interval for warm ischaemia time. Variables such as additional injuries and the presence of shock were not felt to be predictive of amputation: 43 per cent were amputated; the infrapopliteal injuries had the highest amputation rate (80 per cent).

The Mangled Extremity Severity Score (MESS – Table 8.4), which characterizes the skeletal and soft tissue injury, warm ischaemia time, shock, and age of the patient, has also been proposed as a means of solving this dilemma.

8.2.3 Mangled Extremity Severity Score (Table 8.4)

Johansen et al.\(^6\) described the Mangled Extremity Severity Score (MESS) which characterizes the skeletal and soft tissue injury, warm ischaemia time, presence of shock and age of the patient, as a means of solving the dilemma of which patient needed amputation. A MESS value $\geq 7$ predicted amputation.

8.2.4 NISSSA scoring system (Table 8.5)

McNamara and others\(^6\) have, subsequently, retrospectively evaluated the MESS in 24 patients with severely injured tibias. Attempts have been made to address
criticisms of the MESS by including nerve injury in the scoring systems and by separating soft-tissue and skeletal injury components of the MESS. The result is the NISSSA (nerve injury, ischaemia, soft-tissue injury/contamination, skeletal injury, shock/blood pressure, age) scoring system, which is thus more sensitive and more specific than the MESS.

Scoring systems clearly have their limitations when the resuscitating surgeon is faced with an unstable polytrauma patient. Thus, these scoring systems are not universally accepted. They have shortcomings with respect to reproducibility, prognostic value and treatment planning in this context. These factors can lead to inappropriate attempts at limb salvage when associated life- and limb-threatening injuries might be overlooked if attention is focused mainly on salvage of the mangled limb, or to an amputation when salvage may have been possible. Although experience with these scoring systems is generally limited, they may provide some objective parameters on which clinicians can base difficult decisions regarding salvage of life or limb, but it must be stressed that any recommendations derived from them must be judged in terms of available technology and expertise.

In summary the decision whether to amputate primarily or to embark on limb salvage and continue with planned repetitive surgeries is complex. Prolonged salvage attempts that are unlikely to be successful should be avoided, especially in patients with insensitive limbs and predictable functional failures. Scoring systems should be used only as a guide for decision-making. The relative importance of each of the associated trauma parameters (with the exception of prolonged, warm ischaemia time or risking the life of a patient with severe, multiple organ trauma) is still of questionable predictive value. A good understanding of the potential complications facilitates the decision-making process in limb salvage versus amputation.
8.2.5 **Gustilo–Anderson classification**

(Table 8.6)

The Gustilo–Anderson classification describes soft tissue injury, but does not necessarily describe fracture comminution.

8.3 **FASCIOTOMY**

Two techniques have been described:

1. Fibulectomy
2. Two-incision, four-compartment fasciotomy.

The skin must be opened widely, in order to allow a good view of the underlying fascia. It is critical that the fascia be split over its entire length, and this can be done only under direct vision. Care must be taken not to damage the saphenous veins, which may constitute the major system of venous return in such an injured leg.

*In trauma there is no place for subcutaneous fasciotomy.*

8.3.1 **Four-compartment fasciotomy**

Two long incisions are made:

1. A long incision, anterolaterally, 2 cm anterior to the shaft of the fibula. The anterior and lateral fascial compartments are opened separately.
2. A long posteromedial incision is made 2 cm posterior to the medial border of the tibia. The subcutaneous tissue is pushed away by blunt dissection, and the superficial and deep posterior compartments are opened separately.

Fasciotomy must be performed before arterial exploration when an obvious arterial injury exists, or where there is a suspicion of high intracompartmental pressures.

8.3.2 **Fibulectomy**

This is a difficult procedure, leading to extensive blood oozing, and may well result in damage to the peroneal artery.

*It should not be practised in the trauma situation.*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Wound &lt; 1 cm with minimal soft tissue injury</td>
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<tr>
<td></td>
<td>Wound bed is clean</td>
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<td></td>
<td>Bone injury is simple with minimal comminution</td>
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<td></td>
<td>With intramedullary nailing, average time to union is 21–28 weeks</td>
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<tr>
<td>II</td>
<td>Wound is &gt; 1 cm with moderate soft tissue injury</td>
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<tr>
<td></td>
<td>Wound bed is moderately contaminated</td>
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<tr>
<td></td>
<td>Fracture contains moderate comminution</td>
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<td></td>
<td>With intramedullary nailing, average time to union is 26–28 weeks</td>
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<tr>
<td>III</td>
<td>After fracture automatically results in classification as type III:</td>
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<tr>
<td></td>
<td>Segmental fracture with displacement</td>
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<td></td>
<td>Fracture with diaphyseal segmental loss</td>
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<tr>
<td></td>
<td>Fracture with associated vascular injury requiring repair</td>
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<tr>
<td></td>
<td>Farmyard injuries or highly contaminated wounds</td>
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<td></td>
<td>High-velocity gunshot wound GSW</td>
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<td></td>
<td>Fracture caused by crushing force from fast-moving vehicle</td>
</tr>
<tr>
<td>IIIA</td>
<td>Wound &gt; 10 cm with crushed tissue and contamination</td>
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<tr>
<td></td>
<td>Soft tissue coverage of bone is usually possible</td>
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<td></td>
<td>With intramedullary nailing, average time to union is 30–35 weeks</td>
</tr>
<tr>
<td>IIIB</td>
<td>Wound &gt; 10 cm with crushed tissue and contamination</td>
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<td></td>
<td>Soft tissue is inadequate and requires regional or free flap</td>
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<td></td>
<td>With intramedullary nailing, average time to union is 30–35 weeks</td>
</tr>
<tr>
<td>IIIC</td>
<td>A fracture in which there is a major vascular injury requiring repair for limb salvage</td>
</tr>
<tr>
<td></td>
<td>Fractures can be classified using the MESS</td>
</tr>
<tr>
<td></td>
<td>In some cases it will be necessary to consider below-knee amputation</td>
</tr>
</tbody>
</table>

From Wheeless' Textbook of Orthopaedics.

8.4 **REFERENCES**


8.5 **RECOMMENDED READING**


Part 5

Additional (Optional) Modules
9.1 INTRODUCTION

Most trauma mortality in the intensive care unit (ICU) occurs during the first few days of admission, primarily as a result of closed head injury, respiratory failure or refractory haemorrhagic shock, all of which are largely non-preventable deaths. The remainder, many of which may be preventable, occur late and are caused by multiple organ failure, infection or both.

9.2 GOALS OF TRAUMA ICU CARE

The fundamental goals of trauma ICU care are early restoration and maintenance of tissue oxygenation, diagnosis and treatment of occult injuries, and prevention and treatment of infection and multiple organ failure. Trauma ICU care is best provided by a multidisciplinary team focused on resuscitation, monitoring and life support. In the ICU, those who take care of a patient admitted with lethal brain injury play a vital role in the support and maintenance of potential organ donors.

9.3 PHASES OF ICU CARE

9.3.1 Resuscitative phase (first 24 h post-injury)

During this phase, management is focused on fluid resuscitation and the goal of treatment is the maintenance of adequate tissue oxygenation. At the same time, occult life- or limb-threatening injuries are sought.

The recognition and treatment of inadequate tissue oxygenation are required. Deficient tissue oxygen delivery in the acutely traumatized patient is usually caused by impaired perfusion or severe hypoxaemia. Although several different types of shock can be present, inadequate resuscitation from hypovolaemia and blood loss is most common.

After major trauma, some patients experience considerable delay before organ perfusion is fully restored, despite apparently adequate systolic blood pressure and apparently normal urine output. This phenomenon has been called ‘occult hypoperfusion’.1 A clear association has been identified between occult hypoperfusion after major trauma and increased rates of infections, length of stay, days in surgical/trauma ICU, hospital charges and mortality. Equally, early identification and aggressive resuscitation aimed at correcting occult hypoperfusion have been shown to improve survival and reduce complications in severely injured trauma patients.2 Several studies have shown that patients who demonstrate ‘supranormal’ (or optimal) haemodynamic values after resuscitation are more likely to survive than those who do not.3

9.3.1.1 ENDPOINTS OF RESUSCITATION

These generally include the following:

- Clinical examination: cold and clammy, blood pressure, central venous pressure (CVP), heart rate, PaO₂, etc. may not identify occult hypoperfusion
- Base deficit and lactic acidosis
- Pulmonary artery catheter measurements may be used to derive measures of cardiac index and oxygen delivery
- Gastric tonometry
- Tissue oximetry.

9.3.1.2 POST-TRAUMATIC RESPIRATORY FAILURE

Aetiology

- Chest trauma
- Fluid overload
- Shock
• Aspiration
• Post-traumatic adult respiratory distress syndrome (ARDS)
• Spinal cord injury
• Fat embolism syndrome
• Pre-existing respiratory disease.

9.3.1.3 RESPIRATORY ASSESSMENT AND MONITORING

Work of breathing
• Respiratory rate
• Arterial blood gases
• Oxygen delivery and consumption
• Bronchoscopy.

Ventilatory support should be instituted earlier rather than later; select a mode of ventilation tailored to the patient’s need:
• Volume cycled
• Pressure support ventilation (PSV)
• Non-invasive ventilatory support.

9.3.1.4 RECOGNITION AND TREATMENT OF HYPOTHERMIA

Hypothermia is present in those injured patients who have suffered exposure to shock, undergone massive fluid and blood resuscitation, or experienced prolonged operative courses, especially with an open body cavity (chest or abdomen), and in patients undergoing ‘damage control’ operations.

Complications of hypothermia
• Coagulopathy
• Altered metabolism:
  – metabolic acidosis
  – raised serum lactate.

Management of hypothermia
• Increase ambient room temperature
• Foil blankets to reduce convectional heat loss
• Warmed ventilator circuits (airway warming)
• Warmed fluids
• External warming blankets
• Radiator heaters
• Pleural and peritoneal lavage
• Arterial and venous warming circuits
• Cardiopulmonary bypass (CPR).

9.3.2 Early life support phase (24–72 h post-injury)

During this phase, treatment is focused on the management of post-traumatic respiratory failure and progressive intracranial hypertension in patients with severe head injury. Usually, the diagnostic evaluation for occult injuries is now complete. Evidence of early multiple organ failure may become apparent during this time.

Problems that may develop at this time include intracranial hypertension, systemic inflammatory response syndrome (SIRS), early multisystem organ dysfunction syndrome (MODS) and continued respiratory insufficiency. The main priorities of the early life support phase are the maintenance of tissue oxygenation, control of intracranial pressure (ICP), ongoing search for occult injuries, and the institution of nutritional support and withdrawal or replacement of trauma resuscitation lines or devices that may have been placed in less than ideal conditions. Further establishment of the medical history or events of the injury is also completed.

9.3.2.1 PRIORITIES
• Gas exchange and ventilatory support
• ICP monitoring and control
• Fluid and electrolyte balance
• Haematological parameters
• Occult injuries
• Delayed intracranial haematoma formation:
  – follow-up computed tomography (CT) of the head
• Intra-abdominal injuries:
  – follow-up CT or ultrasonography of the abdomen
• Cervical spine injury:
  – completion of the radiological survey and clinical examination if possible
• Thoracic and lumbar spine injury
• Extremity injury: hands and feet
• Nerve injuries.

9.3.3 Prolonged life support (> 72 h post-injury)

Duration of the prolonged life support phase depends on the severity of the injury and associated complications. Many of those who are critically injured can be successfully weaned from life support, although more seriously injured individuals enter a phase in which ongoing life
support is necessary to prevent organ system failure. Predominant clinical concerns that arise include infectious complications that may lead to the development of late multiple organ failure or death.

The main objective of the management of patients developing MODS is to provide support for failing organ systems while attempts are made to isolate and eliminate inflammatory foci that could be perpetuating the organ system failure. In addition, prolonged immobility can cause problems with muscle wasting, joint contractures and compromise of skin pressure areas. Physiotherapy should be commenced early, with the proper use of splints, early exercise and ambulation when possible.

9.3.3.1 RESPIRATORY FAILURE
- Unexplained respiratory failure – look for occult infection or necrotic tissue
- Tracheotomy – early.

9.3.3.2 INFECTIOUS COMPLICATIONS
- Nosocomial pneumonia:
  – Gram stain of sputum and microbiological culture
- Lung abscess and empyema
- Surgical infection:
  – superficial (wound)
  – deep (intra-abdominal abscess)
  – intravenous catheter-related sepsis
- Urinary tract infection (UTI)
- Acalculous cholecystitis
- Sinusitis and otitis media
- Ventriculitis and meningitis
- Endovascular infection.

Antibiotic therapy should ideally be of limited spectrum and directed towards cultures. Remember the risk of antibiotic-associated colitis.

9.3.3.3 NON-INFECTIONOUS CAUSES OF FEVER
- Drugs
- Pulmonary embolism
- Deep vein thrombosis (DVT).

9.3.3.4 PERCUTANEOUS TRACHEOSTOMY
Percutaneous tracheostomy has been shown to have fewer perioperative and postoperative complications compared with conventional tracheostomy and is now the technique of choice in critically ill patients.

Various techniques are described, with dilatation by forceps, or multiple or single dilators. Patient selection is important, and percutaneous tracheostomy should not be attempted if the procedure is non-elective, the landmarks are obscure in the neck or the patient has a coagulopathy. Confirmation of correct placement by fibreoptic bronchoscopy is valuable. Percutaneous tracheostomy is not suitable for children.

9.3.3.5 WEANING FROM VENTILATORY SUPPORT
During the recovery phase, the most important transition made is that from mechanical ventilation to unassisted breathing, known as weaning. Weaning begins when the causes of respiratory failure have resolved.

When signs of infection, respiratory failure or multisystem failure abate, recovery from critical illness requiring prolonged ICU care is imminent.

9.3.3.6 EXTUBATION CRITERIA (‘SOA2P’)

S – secretions – minimal
O – oxygenation – good
A – alert
A – airway: without injury or compromise
P – pressures or parameters: measurements of tidal volume, vital capacity, negative inspiratory force, etc.

9.3.4 Recovery phase (separation from the ICU)

During the recovery phase, full ventilatory support is weaned until the patient is breathing spontaneously and invasive monitoring devices can be removed. The patient and family are prepared for the transition from the ICU to a general patient or intermediate care unit and plans for further convalescence and rehabilitation are developed.

9.4 MULTISYSTEM ORGAN DYSFUNCTION SYNDROME

Multisystem organ dysfunction syndrome (MODS) is a clinical syndrome characterized by the progressive
failure of multiple and interdependent organs. It occurs along a continuum of progressive organ failure, rather than absolute failure. The lungs, liver and kidneys are the principal target organs; however, failure of the cardiovascular and central nervous system may be prominent as well. The main inciting factors in trauma patients are haemorrhagic shock and infection. As life support and resuscitation techniques have improved, the incidence of MODS has increased. The early development of MODS (<3 days after injury) is usually a consequence of shock or inadequate resuscitation, whereas late onset is usually a result of severe infection.

Multisystem organ dysfunction syndrome develops as a consequence of uncontrolled or inappropriate systemic inflammatory response to inciting factors such as severe tissue injury (e.g. brain, lung, soft tissue), hypoperfusion or infection. Two basic models have emerged; the ‘one-hit’ model involves a single insult that initiates an SIRS, which may result in progressive MODS. The ‘two-hit’ model involves sequential insults that may lead to MODS. The initial insult may prime the inflammatory response such that a second insult (even a modest one) results in an exaggerated inflammatory response and subsequent organ dysfunction.

Early factors that increase the risk for MODS include persistent and refractory shock with lactic acidosis and elevated base deficit, high injury severity score (ISS) and the need for multiple blood transfusions. Advanced age may also increase a patient’s risk of developing MODS because of co-morbid disease or decreased organ reserves secondary to normal ageing.

Specific therapy for MODS is currently limited, apart from providing adequate and full resuscitation, treatment of infection and general ICU supportive care. Strategies to prevent MODS include vigorous fluid resuscitation that is effective in establishing and maintaining tissue oxygenation, debridement of devitalized tissue, early fracture fixation and stabilization, early enteral nutritional support when possible, prevention and treatment of nosocomial infections, and early mobility and resumption of exercise.

9.5 COAGULOPATHY OF MAJOR TRAUMA

Coagulopathy is common after very severe injury and resuscitation; four factors contribute to the coagulopathy of major trauma:

1. Haemodilution: dilutional thrombocytopenia is the most common coagulation abnormality in trauma patients
2. Consumption of clotting factors
3. Hypothermia: causes platelet dysfunction and reduction in the rate of the enzymatic clotting cascade
4. Acidosis: metabolic derangements (especially acidosis) also interfere with the clotting mechanism.

Disseminated intravascular coagulopathy (DIC) may be precipitated by massive trauma. In DIC, a variety of insults may cause release of free thrombin into the circulation. Widespread microvascular thrombosis produces tissue ischaemia and organ damage. In an attempt to maintain vascular patency, excess plasmin is generated so that systemic fibrinogenolysis as well as local fibrinolysis occurs. Free thrombin and plasmin within the circulation lead to the thrombotic and haemorrhagic manifestations of DIC.

Platelet survival is so short that severe thrombocytopenia is common. There is a consumptive deficiency of coagulation factors.

Excess plasmin generation is reflected by elevated plasma levels of fibrin and fibrinogen degradation products (FDPs), with abnormal concentrations being found in 85 per cent of patients.

9.5.1 Management

The management of diffuse bleeding after trauma relies on haemorrhage control, active re-warming and replacement of blood products. Empirical transfusion of platelets, fresh frozen plasma and cryoprecipitate is recommended in patients with major injuries (i.e. the damage control group).

Clinically, it is difficult to identify DIC as a separate entity from the coagulopathy of major trauma described above. The distinction is, however, largely academic; the key step in management of DIC is resolution of the condition predisposing to the coagulopathy. The condition will not resolve until the underlying cause is corrected; while this is achieved component therapy is indicated.

9.5.2 Suggested massive transfusion guidelines (Table 9.1)
9.5.3 Patients at risk of massive transfusion

- Hypovolaemic shock:
  - BP < 80 mmHg in adult
  - BP between 80 and 90 mmHg unresponsive to 2 L crystalloid bolus
  - BP < 60 mmHg in child < 12 years
- Major obvious blood loss:
  - presence of an unstable pelvic fracture
- Massive haemothorax.

9.6 RECOGNITION AND TREATMENT OF RAISED ICP

Early mortality in blunt trauma patients in the ICU is often caused by head injury. The primary goal in ICU management of the patient with a severe head injury is the prevention of secondary neuronal injury. One important factor that can contribute to secondary brain injury is increased ICP. Consequently, monitoring and controlling ICP and cerebral perfusion pressure is a high priority in this phase of ICU care. Other conditions that worsen brain injury include:

- hypotension
- hypoxia
- hyperglycaemia
- hyperthermia
- hypercapnia.

9.7 RECOGNITION OF ACUTE RENAL FAILURE

Although the frequency of acute renal failure (ARF) is relatively low, injured patients are at high risk for its development because of tissue damage and necrosis, hypotension, rhabdomyolysis, the use of iodinated contrast for diagnostic tests and pre-existing conditions such as diabetes. The development of ARF complicates the ICU management of a patient, increases the length of stay and is associated with a mortality rate of about 60 per cent.

Approximately a third of post-traumatic ARF cases are caused by inadequate resuscitation, while the remainder seem to develop as part of MODS. Look for and manage these common causes:

- Hypovolaemia
- Rhabdomyolysis
- Abdominal compartment syndrome
- Obstructive uropathy
  Avoid nephrotoxic dyes and drugs.

9.8 EVALUATION OF METABOLIC DISTURBANCES

Disturbances in acid–base and electrolyte balance can be anticipated in patients in shock, those who have received massive transfusions and elderly people with co-morbid conditions.
Typical abnormalities may include:

- acid–base disorders
- electrolyte disorders
- hypokalaemia
- hyperkalaemia
- hypocalcaemia
- hypomagnesaemia.

In acid–base disorders one must identify and correct the aetiology of the disturbance, e.g. metabolic acidosis caused by hypoperfusion secondary to occult pericardial tamponade.

### 9.9 PAIN CONTROL

A number of adverse consequences result when pain is inadequately treated. These include increased O₂ consumption, increased minute volume demands, psychic stress, sleep deprivation and impaired lung mechanics with associated pulmonary complications. Subjective pain assessment is best documented objectively and, after initiation of treatment, requires serial re-evaluation. Inadequate pain relief can be determined objectively by the failure of the patient to achieve adequate volumes on incentive spirometry, persistently small radiological lung volumes, or reluctance to cough and cooperate with chest physiotherapy. If the patient can cooperate, visual analogue pain scores may be helpful. Early pain control in the ICU is primarily achieved through the use of intravenous opiates. Other techniques are employed and tailored to the individual patient and injury:

- Bolus opiates: morphine titrated intravenously
- Patient-controlled analgesia (PCA)
- Epidural analgesia
- Intrapleural anaesthesia
- Extrapleural analgesia
- Intercostal nerve blocks.

### 9.10 FAMILY CONTACT AND SUPPORT

It is very important to establish early contact with family members to explain the injuries, clinical condition and prognosis of the patient. This provides family members with essential information and establishes a relationship between the ICU care team and the family. Administrative facts, such as ICU procedures, visiting hours and available services, should also be explained. With elderly people, determination of living wills or other predetermination documents is important.

### 9.11 ICU TERTIARY SURVEY

The tertiary survey is a complete re-examination of the patient, plus a review of the history and all available results and imaging. Missed injuries are a potent cause of morbidity and most will be identified by a thorough tertiary survey.

#### 9.11.1 Evaluation for occult injuries

Factors predisposing to missed injuries

- Mechanism of injury; re-verify the events surrounding the injury.

High priority occult injuries

- Brain, spinal cord and peripheral nerve injury
- Thoracic aortic injury
- Intra-abdominal or pelvic injury
- Vascular injuries to the extremities
- Cerebrovascular injuries: occult carotid/vertebral artery injury
- Cardiac injuries
- Aerodigestive tract injuries – ruptured bowel
- Occult pneumothorax
- Compartment syndrome – foreleg, thigh, buttock, arm
- Eye injuries (remember to remove contact lenses)
- Other occult injuries: hands, feet, digits, joint dislocations
- Vaginal tampons.

#### 9.11.2 Assessment of co-morbid conditions

- Medical history (including drugs and alcohol)
- Contact personal physicians
- Check pharmacy records.

### 9.12 NUTRITIONAL SUPPORT

Trauma patients are hypermetabolic and have increased nutritional needs as a result of the immunological response
to trauma and the requirement for accelerated protein synthesis for wound healing. Early enteral feeding has been shown to reduce postoperative septic morbidity after trauma. A meta-analysis of a number of randomized trials demonstrated a twofold decrease in infectious complications in patients treated with early enteral nutrition compared with total parenteral nutrition.

Head-injured patients appear to have similar outcomes whether fed enterally or parenterally. A Cochrane review has confirmed that early (either parenteral or enteral) feeding is associated with a trend towards better outcomes in terms of survival and disability compared with later feeding. However, the superior results of enteral feeding over parenteral nutrition are not so clear for patients with brain injury. Parenteral and enteral nutritional support for brain-injured patients has been compared in a randomized prospective study. The patients receiving parenteral support had a better outcome at 3, 6 and 12 months. Furthermore, the enterally fed group had a higher septic complication rate ($p < 0.008$), which was felt to be a result of lower total protein intake, cumulative caloric balance and negative nitrogen balance.

Enteral nutrition should be used when the gut is accessible and functioning. Enteral nutrition is not invariably safer and better than parenteral nutrition but a mix of the two modalities can be used safely. 'Immunonutrition' holds promise for the future. Patients at risk include those with major trauma and burns. It is critical to:

- determine energy and protein requirements
- determine and establish a route of administration
- set a time to begin nutritional support.

9.12.1 Access for enteral nutrition

9.12.1.1 SIMPLE

- Nasogastric tube
- Nasoduodenal tube
- Nasojejunal tube.

Most critically ill trauma patients should be started on early enteral nutrition. Most do not require prolonged feeding (beyond 10–14 days) and simple nasoenteric tube feeding is all that is required. For patients who have prolonged tube-feeding requirements nasoenteric tubes are inconvenient, because they tend to dislodge, worsen aspiration and are uncomfortable.

9.12.1.2 MORE COMPLICATED

- Percutaneous endoscopic gastrostomy (PEG): does not interfere with swallowing, is easy to nurse and target feeding rates are more likely to be achieved compared with nasoenteric tubes. However, it is an invasive procedure with some risk.
- Jejunostomy can be placed endoscopically or during laparotomy. Rates of major complications should be less than 5 per cent.

9.13 PREVENTIVE MEASURES IN THE ICU

9.13.1 Stress ulceration

Stress ulceration and the associated upper gastrointestinal bleeding have been on the decline in most ICUs for the past decade. This is, in great part, a result of the improved resuscitation efforts in the pre-hospital environment, emergency departments and operating theatres. In addition, the use of acid-blocking and cytoprotective therapies has become commonplace. Those patients at greatest risk for stress ulcer development are those with a previous history of ulcer disease, those requiring mechanical ventilation and those with coagulopathy, regardless of whether it is intrinsic or chemically induced. Burn patients have also been labelled as high risk in historical studies.

Cytoprotective agents (e.g. sucralfate) as preventive measures have been shown to be the most cost-effective by statistical analysis in several trials, although there are fewer cases of stress ulcer bleeding in the H2-receptor blockade arm of these trials. However, the marked decrease in the rate of development of ventilator-associated pneumonia seen in the sucralfate population does make this therapeutic option quite attractive.

Intravenous H2-receptor blockade therapy (e.g. ranitidine) blocks production of stomach acid to some degree. Most studies demonstrating its efficacy in stress ulcer prevention do not attempt to neutralize gastric pH. Newer intravenous proton pump inhibitors may well replace H2-receptor blockade as the mainstay of therapy.

Perhaps the simplest and safest method of stress ulcer prevention is adequate resuscitation and early intragastric enteric nutrition. During the early resuscitative phase, and while vasoactive drugs for elevation of blood
pressure are in use, it is not always prudent to provide nutrition enterally. It is in these circumstances that use of acid blockade, cytoprotective agents or both is necessary.

9.13.2 Deep venous thrombosis and pulmonary embolus

A pulmonary embolus (PE) from DVT continues to be a leading preventable cause of death in the injured patient. Recognizing the risk factors for DVT development and instituting an aggressive management regimen can reduce this risk from DVT in the ICU with little added morbidity. Trauma patients at highest risk for fatal PE include those with spinal cord injuries, weight-bearing pelvis fracture and combined long bone fracture/traumatic brain injury (TBI) and long bone/pelvis fracture.

A high index of suspicion in these severely injured patients should result in preventive therapy and diagnostic screening measures being taken in the ICU. Unless hemorrhagic TBI or spinal cord epidural haematoma precludes the use of subcutaneous heparin therapy, these patients should all receive fractionated low-molecular-weight subcutaneous heparin. Unfractionated heparin does not appear to be nearly as effective in this severely injured population. Similarly, unless extremity injury precludes their use, graded pneumatic compression devices should be used on all such patients. Foot pumps may be of some benefit as well.

Screening for the presence of DVT which, if present, would necessitate more aggressive anticoagulant therapy, should also be implemented in these patients. The easiest and safest screening tool is venous Doppler ultrasonography or duplex. This is portable, readily available, repeatable and cost-effective, with no side effects for the patient. These modalities are operator dependent and can fail to diagnose DVT in deep pelvic veins, but contrast ultrasound trials to overcome this weakness are now being conducted. This screening should be performed whenever clinical suspicion of DVT arises, within 48 h of admission and each 5–7 days thereafter as long as the patient remains in the ICU.

In the highest-risk patients previously mentioned, every consideration for prophylactic placement of an inferior vena caval (IVC) filter should be made. The lifetime risk of the filter appears to be quite low in several studies, with an obvious significant benefit in prevention of death. An additional subgroup of patients to be considered for prophylactic IVC filter placement would be in those patients with significant injuries who have also either a contraindication to full anticoagulation (PE treatment) or severe lung disease (long-standing or acutely acquired, i.e. ARDS), which could result in death even from a small PE. The combination of aggressive prevention measures, screening by duplex and prophylactic IVC filters can result in a fatal PE rate of significantly less than 1 per cent of the trauma ICU population.

9.13.3 Infection

In patients with any open wounds from trauma, it is imperative that the tetanus immunization status of the patient be addressed. For those patients immunized within the previous 5 years, no additional treatment is generally needed, although booster tetanus toxoid should be administered to those who have previously received the initial tetanus series but have not been reimmunized in the preceding 5–10 years. Tetanus immune globulin should be administered to those patients who lack any history of immunization.

Patients undergoing splenectomy require immunization for *Haemophilus influenzae* b, meningococci and pneumococci. Debate continues with regard to the timing of administration of these vaccines in trauma patients, but it is clear that adult patients do not benefit from the antibacterial chemoprophylaxis needed in children post-splenectomy. As a result of the multiple strains of each organism, the immunizations are not foolproof in preventing overwhelming post-splenectomy infection (OPSI). Therefore, patients must be carefully counselled to seek medical attention immediately for high fevers, and healthcare providers must be aggressive in the use of empirical antibiotics in patients who may have OPSI upon presentation in the outpatient setting. Currently, booster immunization with Pneumovac is indicated every 5 years for these splenectomized patients.

Adequate wound debridement and irrigation are necessary to eliminate non-viable tissue and debris from all traumatic wounds, in order to limit infection of these wounds. Whenever possible, these wounds should be thoroughly prepared as above and closed primarily. If skin coverage is lacking or > 6 h have elapsed since injury, then moist dressings (to prevent tissue dessication...
and further non-viable wound tissue) should be applied and changed twice a day, further wound debridement performed as indicated, and skin grafts or flap coverage performed once the health of the wound can be assured. Special attention must be given to difficult wounds of the perineum (consider faecal diversion), complex fractures with soft tissue injury and contamination (osteomyelitis), and on the back and occiput (pressure may cause additional wound necrosis).

Thrombophlebitis and sepsis from intravenous cannulae are significant considerations because these intravenous lines are frequently placed under less than optimal circumstances and technique in the field and in resuscitation areas. Removal and replacement of all such lines as early as possible, but in every instance in less than 24 h, are paramount to avoid these infectious complications.

9.14 ANTIBIOTICS

The goal of antibiotic treatment is to improve survival; however, prevention of the emergence of antibiotic resistance is also important.

There is good evidence to support the limited use of antibiotics in the critically ill trauma patient. Many institutions will administer a single dose of a cephalosporin in the emergency area in all patients with open injury, irrespective of origin. There is no evidence to support this unless surgical operation is required.14 There is conflicting evidence with regard to the needs for routine antibiotics with tube thoracostomy.

For thoracoabdominal injuries requiring surgery, a single dose of broad-spectrum antibiotics is indicated. Prolonged courses of antibiotics, extending beyond 24 h, are not currently indicated in these patients.

Patients with open fractures are frequently treated with both Gram-negative and Gram-positive prophylaxis for long periods. There is no evidence for this practice or whether the correct management should be any different to that for torso injury.15

Patients in the ICU on mechanical ventilation, with or without known aspiration, have no indication for antibiotics to prevent pneumonia. In fact, this practice has hastened the onset of antibiotic resistance worldwide.

According to the Centers for Disease Control in the USA, diagnosis of pneumonia must meet the following criteria:

- Râles or dullness to percussion AND any of the following:
  - new purulent sputum or change in sputum
  - culture growth of an organism from blood or tracheal aspirate, bronchial brushing or biopsy.
- Radiological evidence of new or progressive infiltrate, consolidation, cavitation or effusion AND any of the following:
  - isolation of virus or detection of viral antigen in respiratory secretions
  - diagnostic antibody titres for pathogen
  - histopathological evidence of pneumonia.

Empirical antibiotic treatment for ventilator-associated pneumonia (VAP) may not be adequate and a recent international consensus supports a policy based on early initiation of high-dose broad-spectrum antibiotics with 'de-escalation of treatment' to narrow-spectrum antibiotics based on microbiological results.

Antibiotic guidelines should be formulated locally and the empirical antibiotic regimen rotated.

In every case, the simplest, shortest antibiotic regimen should be chosen, and wherever possible it should be based on culture results, or institutional resistance patterns.

9.15 RESPIRATION

- ‘Gentle’ mechanical ventilation; high tidal volumes and pressures can damage the lungs
- Prevention of aspiration
- Early tracheostomy
- Pulmonary toilet and pain control in rib fracture patients
- Pressure control ventilation and high positive end-expiratory pressure (PEEP) for ARDS
- Prone ventilation may improve oxygenation in patients with ARDS or severe sepsis
- VAP is the most common hospital-acquired infection in the ICU.16

9.16 ORGAN DONATION

Identification of potential organ donors in brain-dead patients is an important role in every critical care department. It is difficult to balance the requirements of the organ transplant teams with a sympathetic and
understanding approach to grieving relatives. Specific training is VITAL.

9.17 REFERENCES

1 Claridge JA, Crabtree TD, Pelletier SJ, Butler K, Sawyer RG, Young JS. Persistent occult hypoperfusion is associated with a significant increase in infection rate and mortality in major trauma patients. J Trauma 2000;48:8–14.

9.18 RECOMMENDED READING

Operating in austere and military environments

10.1 INTRODUCTION

Austere: severely simple, morally strict, harsh
Harsh: unpleasantly rough or sharp, severe, cruel

Before the surgeon picks up a scalpel, or the anaesthetist picks up a syringe, he or she must really understand the environment in which he or she is deployed.

Modern surgery and anaesthesia require infrastructure and resources. Infrastructure includes shelter and this needs to provide power for lighting and temperature control, and water for hygiene purposes. Waste needs to be disposed of properly.

Resources include medical gases, drugs, fluids and other consumables. Fluids include blood and blood products, and these need a reliable cold chain.

In the civilian setting these materials and resources are provided by a series of complex supply chains.

To provide a similar standard of care in a deployment requires similar supply chains. After a natural disaster, or in a conflict or post-conflict environment, supply chains and systems will be disrupted and vulnerable. In a conflict they may be deliberately targeted.

So, all members of a surgical team need to understand this and have a realistic expectation of what they can, or cannot, provide in the situation in which they find themselves.

10.2 INJURY PATTERNS

Injury patterns sustained on the modern battlefield are likely to be modified by the type of engagement and modern protective equipment. During a recent conflict in an urban setting in Somalia, casualty distribution was similar to that of the Vietnam War: 11 per cent died on the battlefield, 3 per cent died after reaching a medical facility, 47 per cent were evacuated and 39 per cent returned to duty. The proportion of penetrating injuries caused by bullets or fragments will depend on the nature of the battle, with blunt injury and burns likely to comprise a significant component of the injuries. Most fatal penetrating injuries are likely to be caused by missiles entering through areas not protected by body armour, such as the face and junctional areas in the neck, groin and buttocks.

It must be borne in mind by military medical planners that microbiological, biological and chemical warfare may be deployed. In addition, troops may be exposed to radiation through battlefield weapons such as depleted uranium munitions. It is imperative that military medical personnel become familiar with the medical consequences of toxin exposure, the illnesses caused by these agents and the measures required to protect military healthcare providers.

Military medical practitioners have been described as ‘working at the interface of two dynamic technologies, warfare and trauma management’. In addition to the problems of dispersed battlefields, highly mobile frontlines, extended lines of logistics and delay in evacuation, the modern military doctor is likely to be called upon to treat civilians including pregnant women and children, as well as service personnel, with a requirement to offer immediate care well away from their specialty; problems in ophthalmology, ear, nose and throat, paediatrics, gynaecology, tropical medicine or even public health may fall under the remit of the military surgeon.

10.3 EMERGENCY MEDICAL SERVICES SYSTEMS

The patient presented to the surgical team in a civilian hospital has, him- or herself, been part of a ‘supply chain’. Consider a victim of a road traffic collision. Summoning help implies an intact telephone or radio system, appropriately trained individuals arriving in suitably equipped vehicles and an unimpeded journey to a hospital.
In the deployed environment this ‘chain’ is vulnerable in the same way that others are. Patients may experience delays of hours or days getting to care, which will in turn influence how they present to the surgical team.

Deployed military medical systems usually consist of a series of ‘echelons’ or ‘roles’. Close to the point of injury a casualty either applies self aid or receives ‘buddy aid’. The next stage is care by a ‘medic’, then by a doctor or nurse at an aid post. Usually by the time casualties reach a surgical team they have received some treatment (analgesia and antibiotics) and resuscitation (to various fluid protocols). The situation becomes complicated when casualties move between systems (e.g. military to host nation or non-governmental organization [NGO] to military), having received surgery in the other one. As standards of care can vary enormously between systems, these casualties need a thorough examination and re-evaluation.

10.3.1 Incident management and multiple casualties

At incidents in which bombs are involved or secondary devices suspected, the ‘4 Cs’ must be adopted:

Confirm
Clear
Cordon
Control.

CONFIRM

Incident commanders must be clear about what is happening and the risk and position of further hazards. Factors that must be considered are clearance priorities, cordon locations, safe areas, access and egress routes, and rendezvous points.

CLEAR

The scene should be cleared to a safe distance – the distance varying depending on the terrain. The method and urgency of clearance will depend on the incident.

CORDON

Cordons establish the area in which the rescue effort is taking place and define safe zones and tiers of command. An outer cordon should be established as a physical barrier preventing accidental or unauthorized access to the site. An inner cordon may be set up around wreckage, especially if hazards still exist.

CONTROL

Once set up, the control of the cordons and scene is maintained by clear rendezvous and access points. Once the ‘4 Cs’ have been established, medical management and support can begin, the principles of which are:

• Command and control
• Safety
• Communication
• Assessment
• Triage
• Treatment
• Transport.

COMMAND AND CONTROL

This is the paramount principle. If good command and control are not established, the initial chaos will continue and the injured will suffer regardless of how well some individual casualties are treated.

SAFETY

Healthcare workers must remember that their own safety is paramount and that they must not become casualties themselves.

COMMUNICATION

Every major incident enquiry has identified failings in communications. Without good communication, command and control are impossible.

ASSESSMENT

This is a constant process. Commanders must always be considering what the current situation is, what resources are required and where these can be obtained.

TRIAGE

In any situation when there is more than one casualty, a system of triage must be used (Table 10.1 and see Section 10.4, Triage).
TREATMENT

At the scene of any ballistic incident treatment teams may be faced with casualties with multiple serious injuries. Treatment must follow the ‘C’ ABC paradigm (see Section 10.7, Resuscitation).

TRANSPORT

Not every patient needs to travel in an ambulance; buses or other multi-passenger vehicles should be used to move the walking wounded.

It is recognized that speed of evacuation from point of wounding to first surgical intervention is a critical determinant of outcome. The Korean War saw the introduction of helicopter evacuation of wounded from frontline to Mobile Army Surgical Hospitals (MASH) with onward transport by fixed-wing aircraft to base hospitals. During the Vietnam conflict, the average pre-hospital time for combat casualties treated at the Da Nang US Navy Hospital was 80 min. Limited provision of aircraft in a combat setting has meant that medical evacuation has used assets earmarked for other purposes. During Operations Desert Shield/Desert Storm in 1991, many patients were successfully airlifted using converted cargo aircraft.

10.4 TRIAGE

Effective triage is crucial in an efficient military healthcare system – first described by Napoleon’s surgeon, Dominique Jean Larrey, who introduced a system of sorting casualties as they presented to field dressing stations. His priority, and the aim of the system, was to identify those soldiers who had minor wounds and therefore with minor treatment could return to the battle. Although we might now call this reverse triage, he had introduced a formal system of prioritizing casualties. Triage remains a fundamental principle in modern military medicine. It is dynamic, and must be applied at all levels of medical care, from point of wounding to definitive surgical care.

The system for ‘surgical triage’ may be slightly different to the triage system used in resuscitation, but the same principles apply. Those requiring life-saving surgical intervention take priority over patients requiring limb-saving surgery in the forward locations and, taking into account all the other factors, the key question will be whether they need to go on the table at all. Over-triage is a feature of all mass casualty situations; however a rate of over-triage is acceptable to avoid missing patients who really did require an intervention. In a series of 1350 laparotomies from the Vietnam War, based on clinical assessment of wounded soldiers, the rate of negative laparotomy was 19.2 per cent.1 In a modern military setting, an accurate screening tool (perhaps focused abdominal sonography for trauma) would be very useful.

Effective triage is crucial in an efficient military healthcare system. Triage is ‘sorting’ casualties into priorities for care.

Transfer time will also dictate who requires life-saving interventions at that point, and who can wait until they reach the next echelon of medical care. Equipment will always be in limited supply in these forward locations, and must be used appropriately, because re-supply will take time.

The flow of casualties in a fast-moving battle will also influence how many and what type of casualties should be operated on. The prospect of in-coming serious casualties will change triage decisions for wounded already at the medical facility. If there is only one surgical table available forward, ‘Who goes on first?’ and ‘Do they need to go on at all?’ may be simple questions to ask, but the answers are anything but straightforward. The need for effective triage poses difficult questions. A patient with extensive multiple injuries, who would get maximum effort and resources in a civilian trauma centre, may need to be labelled ‘expectant’ if several other patients face a better chance of survival given early access to the limited equipment and expertise available.

10.4.1 Forward surgical teams and triage

Many military healthcare teams around the globe are now incorporating far-forward resuscitative surgery capabilities into doctrine and mission planning. Although

<table>
<thead>
<tr>
<th>Priority</th>
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<tr>
<td>T1</td>
<td>Red</td>
<td>Immediate</td>
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<tr>
<td>T2</td>
<td>Yellow</td>
<td>Urgent</td>
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<tr>
<td>T3</td>
<td>Green</td>
<td>Delayed</td>
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<tr>
<td>Dead</td>
<td>White or black</td>
<td>Dead</td>
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<tr>
<td>T4</td>
<td>Blue (not standard)</td>
<td>Expectant</td>
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these forward surgical teams provide only a limited trauma surgical capability, they aim to provide life- and limb-saving surgery to the select group of potentially salvageable patients who would otherwise die or suffer permanent disability as a result of delays in evacuation from the fast-moving modern battlefield.

These teams should be capable of providing life-saving thoracoabdominal haemorrhage control, control of contamination within body cavities, temporary limb revascularization and stabilization of fractures, and evacuation of major intracranial haematomas. Each nation has a slightly different balance and skill mix within this forward surgical capability, but most will normally provide the three main tenets of forward care: a resuscitation capability, one or more surgical tables and a critical care capability. Most nations ‘mission tailor’ their teams to the specific operational environment, and sizes range from as few as six men to larger teams of 30 plus. As part of the casualty estimate, military planners need to decide on the number of surgical tables required and the speed at which they can empty their ‘back door’. The size and sophistication of the attached critical care element will be determined by the capability of tactical aeromedical evacuation to move patients to the next echelon of medical care.

There are several factors that will influence decision-making within the triage process at the forward surgery level. Forward surgical teams must be light, mobile and rapidly deployable, to allow them to respond in an uncertain battlefield. Restrictions and constraints within these teams are many, and include limitations of space and equipment, poor lighting and the need to perform under extremes of climatic conditions. Some re-sterilization of surgical tools may be possible, but disposable equipment, water and oxygen will all be limited. Human factors of physical and emotional fatigue will also affect how long the surgical team can endure the challenges of operating in austere environments without reinforcement or re-supply. The teams will often have to function independently, but may also deploy as augmentation of an existing medical facility during a casualty surge.

There is a difference between a well-equipped relatively static ‘field’ hospital and a ‘forward surgical team’. As mentioned earlier the raison d’être of the team is delivery of the life- and limb-saving surgery as far forward as possible to the select group of potentially salvageable patients.

Triage is challenging, it requires difficult decisions to be made, but it remains crucial to effective use and efficiency of the forward surgical teams.

### 10.5 MASS CASUALTIES

One of the fundamental planning parameters for medical support is an estimate of the numbers and types of casualties expected. Casualty estimates are major resource drivers and will determine what capabilities are required, and at what level. The medical support for a specific operation will therefore be force packaged as a direct result of the perceived threat.

Mass casualties may, however, occur for many reasons, and the cause of the major incident may not have been identified as one of the known ‘threats’.

Multiple motor vehicle accidents, downed helicopters, floods and even earthquakes have recently produced mass casualty situations or major incidents for military forces around the world. All these incidents produced an unexpected surge in casualties, far greater than the casualty estimate that each operation had declared. The key in all these events was that the medical facilities were overwhelmed, and available resources could not meet the required demand. When major incidents produce mass casualties in civilian situations, e.g. rail crashes, there are often a number of receiving hospitals to choose from to spread the load of casualties. This luxury is rarely available in the military environment. In some situations, other nations’ medical facilities may be available, but often the only available ‘receiving hospital’ will be the forward surgical team. Triage remains the key to effective medical management of a mass casualty event, especially when large numbers of wounded arrive at the location in a short space of time. Equipment, personnel and transport will be in short supply, so sound training and adhering to the principles of triage should ensure effective use of the limited resources available.

After triage and treatment, transport remains the third key element of medical support in a mass casualty event. Unlike in a civilian environment, when there will be many options for both ground and air transport, in the military mass casualty situation transport is likely to be very limited. Regular and effective triage will determine who is transported first, and by what means, to ensure that the right patient arrives at the right time at the next level of medical care.

### 10.6 EVACUATION

It is recognized that speed of evacuation from point of wounding to first surgical intervention is a critical
determinant of outcome. In the earlier section on transport some details were given for types of transport made use of. The concept of using converted cargo aircraft, used during Operations Desert Shield/Desert Storm, was originally validated in the Second World War and is still in vogue today. Improvements in civilian aeromedical transport, using specially equipped aircraft and enhanced flight medic training and standards, have led to calls for dedicated aeromedical capability in the military.

10.7 RESUSCITATION

Military casualties can occur from blunt injury, penetrating injury, blast injury or a combination of all of these. Bombing victims may also have burns. Injury patterns sustained on the modern battlefield are likely to be modified by the type of engagement and modern protective equipment (see Section 10.2, Injury patterns).

The treatment and resuscitation available will alter with each echelon of care; generally resources and complexity of care increase as the casualty moves away from the battlefield.

Hypovolaemia remains the most common cause of death among those killed in action during military conflicts. Although the principles remain the same, resuscitation of wounded combatants remains a formidable challenge on the battlefield and there are some crucial differences to consider in this environment. Unlike in the urban setting, the military must consider the weight and therefore the quantity of supplies that can be transported into austere locations. Large volumes of fluids at any stage in the resuscitation process are therefore not a realistic option. There is no consensus on which is the most appropriate fluid in the military trauma casualty. The Advanced Trauma Life Support (ATLS) standard of 2 L infused crystalloid for the acutely injured hypotensive patient is not feasible in the far forward environment because of logistical constraints, but is also likely to be detrimental to the survival of the patient with uncontrolled haemorrhagic shock (UHS) in whom surgical intervention is not immediately available. For patients with UHS, the goal of maintaining a systolic arterial pressure of 70–80 mmHg is now accepted by most practitioners treating wounded forward of the first surgical capability. The UK military are teaching the approach of ‘ABC for ballistic injury,’ where ‘C’ stands for ‘catastrophic haemorrhage’ (Table 10.2). This is because of the high incidence of severe (but potentially survivable) injuries to limbs and junctional areas in military casualties.

Blood is the gold standard fluid of choice in casualties with UHS, particularly those in profound shock, and is now carried by a few military resuscitation teams forward of the first surgical teams. However, in most military healthcare systems, blood will not be available forward of the surgical teams, and other fluids need to be carried. Options available to resuscitation teams include isotonic crystalloids, colloids, hypertonic saline and hypertonic saline plus colloid. The choice of fluid remains unresolved, and may in fact be less important than the quantity and rate of fluid infused in patients with uncontrolled haemorrhage.

The temperature of any fluid given to trauma patients, particularly in a military or austere environment, is crucial. Any fluid used in resuscitation must be warmed to avoid further cooling of a haemorrhagic casualty. The actual process of warming the fluids remains a considerable challenge and in most cases requires improvisation on behalf of the provider.

Difficulty in obtaining vascular access can be experienced in austere conditions, when hypotension, low ambient temperature and tactical considerations, such as the presence of mass casualties or operating light restriction, can conspire to frustrate attempts at vascular access; intraosseous access is an attractive option in these scenarios.

10.8 BATTLEFIELD ANALGESIA

Relief of pain is an important consideration for both the wounded and the military care giver. Provision of effective analgesia is humane but also attenuates the adverse pathophysiological responses to pain, and is likely to aid evacuation from the battlefield and maintain morale. Analgesia may be given at self- and buddy-aid levels; protocols to guide medical and paramedical staff in the provision of safe and effective analgesia are available.

Analgesia methods used in recent conflicts include:

- Simple non-pharmacological:
  - reassurance
  - splinting of fractures
  - cooling of burns
- Oral analgesics: such as non-steroidal anti-inflammatory drugs or paracetamol
Nerve blocks and infiltration of local anaesthesia
• Intramuscular and intravenous opiates
• Fentanyl ‘suckers’.

Methods under development include intranasal ketamine.

10.9 BATTLEFIELD ANAESTHESIA

Surgery requires both adequate analgesia and anaesthesia. No single agent can provide an appropriate level of both anaesthesia and analgesia, so a combination of drugs and
techniques is required. The choices of anaesthetic are narrowed in austere conditions; these are limited to general anaesthesia (either intravenous or inhalational), regional anaesthesia or none at all. For surgical exploration of body cavities, general anaesthesia is most frequently chosen, whereas a regional aesthetic may be more appropriate for injuries of the extremities or perineum. In the field, rapid sequence induction (RSI) is the norm, using fast-acting hypnotic and neuromuscular blocking agents to facilitate rapid airway control. In the absence or limitation of supplemental oxygen supplies, RSI becomes even more crucial as preoxygenation of the patient’s lungs is often not possible. There are several RSI cocktails used in the prehospital setting, most using a combination of induction agent, paralysing agent and analgesia. Sedation, amnesia and analgesia can then be maintained with intravenous agents such as ketamine, benzodiazepines and opiates.

For long procedures or surgical sites involving the abdomen or thorax, a combination anaesthetic that includes an inhalational agent such as isoflurane may be used. British surgical teams use a portable ‘tri-service apparatus’ that does not require a compressed gas source, and have gained much experience with this technique of field anaesthesia. This ‘draw-over’ type vaporizer is currently also in use by US forces in austere settings.

Regional anaesthesia remains an important option in battlefield anaesthesia, because it provides both patient comfort and surgical analgesia, while maintaining patient consciousness and spontaneous ventilation. With the relatively large number of extremity wounds in modern conflicts, and certainly in the mass casualty setting with a limited anaesthesia capability, regional aesthetic techniques should not be overlooked. Continuous infusion nerve blocks have provided excellent analgesia for postoperative casualties evacuated from Iraq.

Battlefield anaesthesia presents many challenges; including the need to maintain airway control, hypothermia of the casualty, restricted drug availability, lack of supplementary oxygen and the possible requirement for prolonged postoperative mechanical ventilation. Mass casualty situations are also a constant possibility in the military arena.

### 10.10 Damage Control Surgery in the Military Setting

Anaesthesia for ‘damage control’ procedures and major cavity injury is really a fusion of continuing resuscitation and critical care. This requires optimization of haemodynamic status, re-warming of the casualty and pain relief. One of the biggest challenges will be reversing the hypothermia that is almost universal in haemorrhagic patients in these conditions. As well as warming all intravenous fluids and ventilator circuits, an active re-warming device will be required. If a return to the operating theatre for more definitive surgery is not planned in the forward location, critical care must be maintained throughout the aeromedical evacuation. The ability to ‘empty the back door’ of the forward surgical facility by specialist critical care transfer teams at the earliest opportunity will reduce the critical care capacity necessary to deliver such care within the forward surgical team.

The typical civilian damage control patient is likely to require the direct attention of at least two surgeons and one nurse during the first 6 h, full invasive monitoring, multiple operations, massive transfusion of blood and products, and require prolonged intensive care unit (ICU) stay with a high mortality.

The utility of this damage control philosophy is untested in mass casualty, military wounding situations. Indeed, damage control has been labelled as ‘impractical for common use in a forward military unit during times of war’. However, some military surgeons, while grudgingly accepting that patients who undergo damage control surgery in the civilian setting ‘might not even be candidates (for surgery) in a field setting’, have defended the concept of ‘minimally acceptable care’ with rapid procedures and minimal objectives as a time-honoured principle of forward surgery, even if the name ‘damage control’ has been ‘hijacked’ by the civilians.

In the far-forward, highly mobile, austere military environment, it is quite likely that the surgeon will not have the luxury of being able to perform definitive surgery on every casualty. Short, focused surgical interventions can be used on peripheral vascular injuries, extensive bone and soft tissue injuries and thoracoabdominal penetrations in patients with favourable physiology, instead of definitive surgery for every injured soldier. This may conserve precious resources such as time, operating table space and blood. Instead of applying these temporary abbreviated surgical control (TASC) manoeuvres to patients about to exhaust their physiological reserve, as in classic damage control, TASC would be applied when the limitations of reserve exist outside the patient.

This philosophy relies heavily on the military medical system, with postoperative care and evacuation to the
‘resource-replete environment’ a priority. In the military, the key would seem to be triage, i.e. patient selection. The philosophy for the military surgeon exposed to numbers of casualties in the setting of limited resources remains to do the best for the most, rather than expend resources on limited numbers of critically wounded.

10.11 CRITICAL CARE

If TASC is going to be the norm for the far-forward surgeon, then a critical care capability must be a part of the forward surgical team structure. The priorities will therefore be optimization of haemodynamic status, re-warming of the casualty, pain relief and preparation for return to theatre, or evacuation depending on the situation. The healthcare providers looking after the critical care of a patient in these surroundings face many of the problems already identified for the anaesthetic provider. One of the biggest challenges will be reversing the hypothermia that is almost universal in haemorrhagic patients in these conditions. As well as warming all intravenous fluids and ventilator circuits, an active re-warming device will be required. If a return to the operating theatre for more definitive surgery is not planned in the forward location, critical care must be maintained throughout the aeromedical evacuation.

10.12 CONCLUSION

These are exciting times to be a military medical practitioner. Dramatic changes in the global world order have shifted the priorities for military planners. Surgical doctrines also have to adapt to the likely scenarios of future conflict. Low-density dispersed battlefields, highly mobile operations, extended lines of evacuation and logistic supply, civilian wounded and the possibility of chemical, biological and nuclear attack all mean that military doctors will have to demonstrate their adaptability and resourcefulness, as well as their surgical skills. Whether labelled ‘TASC’ or ‘damage control’, limited initial surgery is likely to be part of the surgeon’s armamentarium.

10.13 REFERENCE


10.14 RECOMMENDED READING

10.14.1 Immediate management of ballistic casualties


10.14.2 War surgery


10.14.3 Ballistics: history, mechanisms, ballistic protection and casualty management


10.14.4 General reading


11.1 FOCUSED ABDOMINAL SONOGRAPHY FOR TRAUMA (FAST)

Four areas of the torso are scanned for the detection of free fluid (blood):

1. Perihepatic
2. Perisplenic
3. Pericardial
4. Pelvic.

Focused abdominal sonography for trauma (FAST) is not organ specific, but detection of free fluid in any of the four views is regarded as a positive examination; however, if any of the views is not clearly seen, then the examination is deemed incomplete and an alternative means of investigation is required, or FAST must be repeated at frequent intervals.

Focused abdominal sonography for trauma is non-invasive and repeatable. Repeated scans have been shown to increase sensitivity. It is as accurate when used by appropriately trained surgeons as when used by radiologists.

Focused abdominal sonography for trauma relies on detection of free intraperitoneal fluid. In the hands of most operators, ultrasonography will detect a minimum of 200 mL fluid, so false-negative examinations may occur (e.g. hollow viscus injury).

The retroperitoneum is not well visualized.

A positive FAST after penetrating injury is a strong predictor of significant injury but, if negative, additional diagnostic studies may be required to rule out occult injury.

BLUNT TRAUMA

In blunt abdominal trauma, FAST has a sensitivity of about 86 per cent and a specificity of about 98 per cent for detection of intra-abdominal injuries. Positive predictive value is about 87 per cent and negative predictive value 98 per cent.

An International Consensus Meeting concluded that a negative FAST examination should be followed up by a period of observation of at least 6 h and a follow-up FAST. The alternative is to use diagnostic intraperitoneal lavage (DPL) and computed tomography (CT) to confirm the ultrasound findings.

11.2 OTHER APPLICATIONS OF ULTRASONOGRAPHY IN TRAUMA

11.2.1 Ultrasonography in abdominal trauma

False-negative rates have been high in FAST after penetrating abdominal injury.

A positive FAST after penetrating injury is a strong predictor of significant injury but, if negative, additional diagnostic studies may be required to rule out occult injury.

BLUNT TRAUMA

In blunt abdominal trauma, FAST has a sensitivity of about 86 per cent and a specificity of about 98 per cent for detection of intra-abdominal injuries. Positive predictive value is about 87 per cent and negative predictive value 98 per cent.

An International Consensus Meeting concluded that a negative FAST examination should be followed up by a period of observation of at least 6 h and a follow-up FAST. The alternative is to use diagnostic intraperitoneal lavage (DPL) and computed tomography (CT) to confirm the ultrasound findings.

11.2.2 Ultrasonography in thoracic trauma

The evaluation of fluid in the pericardium is a standard part of the FAST assessment after blunt trauma, although the presence of a left-sided pneumothorax may create technical difficulties in visualization of the pericardial space.

A prospective, multicentre evaluation of 261 patients with a penetrating precordial or transthoracic wound suspicious for cardiac injury demonstrated an accuracy of 97.3 per cent.

Ultrasonography can be valuable in the detection of haemothorax, with sensitivity and specificity similar to those for the portable chest radiographs; however, the performance time for ultrasonography is significantly shorter.

Ultrasonography has been used to detect pneumothorax with 95 per cent sensitivity.
11.3 CONCLUSION

In haemodynamically stable patients with blunt abdominal injury, clinical findings may be used to select those who may be safely observed. This is safe only if the patient is alert, cooperative and sober, and does not have significant distracting injuries.

- In the absence of a reliable physical examination, FAST is a good initial screening tool for blunt abdominal injury.
- CT can be used to delineate injury patterns in stable patients with equivocal FAST.
- A single negative FAST examination should be supported by a period of observation, repeated FAST or other diagnostic modalities.
- Haemodynamically unstable patients with blunt abdominal injury should be initially evaluated with FAST or DPL.
- Ultrasonography is useful in detecting thoracic injuries, particularly cardiac tamponade.

11.4 RECOMMENDED READING


Minimally invasive techniques have yet to be enthusiastically adopted by trauma surgeons, unlike their general surgical colleagues. However, selective indications for the use of these techniques are emerging rapidly in both adult and paediatric fields. Physiological instability and severe head injury are a contraindication to the creation of the pneumoperitoneum associated with minimally invasive techniques.

12.1 THORACIC INJURY

Persistent, non-exsanguinating haemorrhage can be investigated and occasionally treated by video-assisted thoracoscopic surgery (VATS):

- VATS can accomplish evacuation of a clotted haemothorax successfully.
- VATS allows direct visualization and stapling of persistent air leaks, with aspiration of associated haemothorax.
- Injuries to the thoracic duct are rare after chest trauma; however, thoracoscopic ligation may be successful when conservative medical management fails to reduce chyle leakage.

12.2 DIAPHRAGMATIC INJURY

In asymptomatic patients with anterior or flank stab wounds of the lower chest or upper abdomen, the risk of an occult diaphragmatic injury is about 7 per cent. Specifically, in patients with left-sided thoracoabdominal stab wounds, the risk is 17 per cent. Laparoscopy is sufficient to exclude occult diaphragmatic injury after penetrating abdominal trauma. A diaphragmatic injury can be repaired thoracoscopically or laparoscopically.

Suspected diaphragmatic injury caused by both blunt and penetrating injury can be accurately evaluated by VATS. In the largest series of patients evaluated using VATS for suspected penetrating diaphragmatic injury, 171 stable patients with penetrating chest injury and no separate indication for either thoracotomy or laparotomy were investigated with VATS. Sixty patients (35 per cent) had a diaphragmatic injury and most (93 per cent) were repaired using a laparotomy. Of the patients with diaphragmatic injury, 47 of 60 (78 per cent) had an associated intra-abdominal injury. Other than to repair the diaphragm, no therapeutic intervention was required at laparotomy in 36 per cent.

Diaphragmatic injury can be repaired thoracoscopically; however, there are dangers in that intraperitoneal injuries may be missed.

In a series of patients with suspected tamponade, thoracoscopic pericardial windows have been performed, with no significant complications, and found to be accurate in 97 per cent of cases.

12.3 ABDOMINAL INJURY

12.3.1 Screening for intra-abdominal injury

Caution is warranted when the laparoscopic approach is used because of the risk of tension pneumothorax.

Laparoscopy appears to be a poor screening tool after blunt trauma. In one series, a 16 per cent incidence of missed intra-abdominal injuries was found in patients evaluated laparoscopically after blunt trauma. In recent studies, the sensitivity and specificity of laparoscopy in detecting blunt small bowel injuries have improved considerably from the earlier studies. However, in abdominal stab wounds, diagnostic laparoscopy prevents an unnecessary laparotomy in 54–87 per cent of patients.
Laparoscopy has been used successfully in an attempt to minimize unnecessary laparotomy to assess whether or not an equivocal wound has breached the peritoneal cavity. However, sensitivity is poor for hollow viscus injury. Laparoscopy has also been advocated to rule out diaphragmatic injury when non-operative management of penetrating thoracoabdominal injuries is envisaged.

12.3.2 Splenic injury

Laparoscopic splenic preservation or partial splenectomy has been reported after trauma using fibrin glue or argon beam coagulator and by splenic wrapping with mesh. Successful autotransfusion of haemoperitoneum aspirated from the peritoneum has also been reported. Laparoscopy is an excellent approach for secondary treatment of post-traumatic, localized, splenic infarcts or pseudocysts.

12.3.3 Liver injury

Patients failing a trial of non-operative management for hepatic injury have been managed successfully using minimally invasive surgery, including laparoscopic application of fibrin glue as a haemostatic agent.

Haemoperitoneum may be drained and biliary leaks, with or without peritonitis, can be controlled via the laparoscope, usually combined with endoscopic retrograde cholangiopancreatography (ERCP).

Reports of successful laparoscopic treatment for intra-abdominal organs other than the liver or spleen are increasing.

12.3.4 Colonic injury

Laparoscopy has been used to raise a colostomy to defunction the lower intestinal tract in colorectal injuries.

12.4 CONCLUSION

To date, minimally invasive surgery has played only a small role in trauma surgery. Surgeons should be encouraged to incorporate laparoscopy and VATS into their protocols, and gain familiarity and expertise with their use.

2.5 REFERENCES


12.6 RECOMMENDED READING


Morales CH, Salinas CM, Henao CA, Patino PA, Munoz CM.


Fractures of the bony skeleton may occur in isolation, or as part of multiple injuries. Although not usually life-threatening in isolation, poorly managed extremity trauma can lead to significant disability.

13.1 MANAGEMENT OF SEVERE INJURY TO THE EXTREMITY

The primary survey and resuscitation must take priority:
- Rapidly assess limb injuries, making careful note of distal perfusion
- Involve orthopaedic and plastic surgeons early
- Restore impaired circulation
- Cover open wounds with sterile dressing; give tetanus and antibiotic prophylaxis
- Debride non-viable tissue
- Restore skeletal stability
- Achieve wound closure and commence rehabilitation.

13.2 KEY ISSUES

13.2.1 Management of open fractures

Sepsis is a constant threat to the healing of open fractures. The risk factors for infection are:
- Severity of injury (especially the injury to the soft tissue envelope of a limb)
- Delay from injury to surgical care (< 6 h)
- Failure to use prophylactic antibiotics
- Inappropriate wound closure.

13.2.2 Severity of injury (Gustilo classification)\(^1\)

- Grade I: open fracture with a skin wound < 1 cm long and clean
- Grade II: open fracture with a laceration > 1 cm long without extensive soft tissue damage
- Grade III: an open segmental fracture, an open fracture with extensive soft tissue damage or a traumatic amputation.

Gustilo et al.\(^1\) further stratified grade III wounds:
- Grade IIIA: adequate soft tissue coverage of a fractured bone despite extensive soft tissue laceration or flaps, or high-energy trauma, irrespective of the size of the wound (wound sepsis rate: 4 per cent)
- Grade IIIB: extensive soft tissue injury loss with periosteal stripping and bone exposure, usually associated with massive contamination (wound sepsis rate: 52 per cent)
- Grade IIIC: open fractures associated with arterial injury requiring repair. Major soft tissue injury not necessarily significant (wound sepsis rate: 42 per cent).

13.2.3 Antibiotics\(^2\)

The early use of prophylactic antibiotics is important, but it must be recognized that antibiotics are an adjunct to appropriate wound care. The introduction of the Thomas splint and improved understanding of the need for surgical wound care are credited with reducing the mortality rate for open fractures of the femur from 80
per cent to 16 per cent during the First World War. During the Spanish Civil War, Trueta\(^3\) reported a septic mortality rate of 0.6 per cent in 1069 open fractures with a policy of wound excision and debridement, reduction of the fracture, stabilization with plaster and leaving the traumatic wound open.

Recent consensus guidelines (EAST Guidelines\(^3\)) recommend antibiotics be discontinued 24 h after wound closure for grade I and II fractures. For grade III wounds, the antibiotics should be continued for only 72 h after the time of injury or not more than 24 h after soft tissue coverage of the wound has been achieved, whichever occurs first. Agents effective against *Staphylococcus aureus* appear to be adequate for grade I and II fractures; however, the addition of broader Gram-negative coverage may be beneficial for grade III injuries.

### 13.2.4 Timing of skeletal fixation in polytrauma patients\(^6\)

#### 13.2.4.1 RESPIRATORY INSUFFICIENCY

Episodes of respiratory insufficiency often occur after orthopaedic injury. Extremity injury may occur as part of a multisystem insult, with associated head, chest and other injuries. Hypoxia, hypotension and tissue injury provide an initial ‘hit’ to prime the patient’s inflammatory response; operative treatment of fractures constitutes a modifiable secondary insult. In addition, post-traumatic fat embolism has been implicated in the respiratory compromise that appears after orthopaedic injury.

Nevertheless, most comparative studies have shown a reduction in risk of post-traumatic respiratory compromise after early, definitive fixation of fractures (i.e. within 48 h), for both isolated injuries and multisystem trauma. There is also evidence of reduction in mortality, duration of mechanical ventilation, thromboembolic events and cost in favour of early fixation.

#### 13.2.4.2 HEAD INJURY

In approximately 5 per cent of long bone fractures of the leg, the patient is physiologically unstable after initial resuscitation as a result of haemodynamic instability, raised intracranial pressure or other problems. Temporary methods of fixation are attractive in this setting. Although some studies have suggested that early nailing of a femoral fracture may be harmful in patients with a concomitant head injury, there is no compelling evidence that early long bone stabilization in mildly, moderately or severely brain-injured patients either enhances or worsens outcome.

### 13.3 AMPUTATE OR PRESERVE A SEVERELY DAMAGED LIMB?

Salvage of severe lower-extremity fractures can be extremely challenging. Even if successful in preserving the limb, the functional result may be unsatisfactory because of residual effects of injuries to muscle and nerve, bone loss and the presence of chronic infection. Failed efforts at limb salvage consume resources and are associated with increased patient mortality and high hospital costs.

Many lower-extremity injury-severity scoring systems have been developed to assist the surgical team with the initial decision to amputate or salvage a limb. Scores such as the Mangled Extremity Severity Score (MESS) can be used to facilitate identification of the irretrievably injured lower extremity. Recent prospective studies have, however, sounded a note of caution about relying exclusively on a scoring system to make these important decisions.

### 13.4 COMPARTMENT SYNDROME

Compartment syndrome may occur after extremity injury, with or without vascular trauma. Increasing pressure within the closed fascial space of a limb compromises the blood supply of muscle. Early clinical diagnosis and treatment are important to prevent significant morbidity.

### 13.5 CONCLUSION

It seems preferable to perform early, definitive, long bone stabilization in polytrauma patients. Recent consensus guidelines suggest that, for patients with dominant head or chest injuries, the timing of long bone stabilization should be individualized according to the patient’s clinical condition.
13.6 REFERENCES


13.7 RECOMMENDED READING


In the western world, the most common cause of death after trauma is severe brain injury, contributing significantly to half of all deaths from trauma.

The severely brain-injured individual also has the highest mean length of stay in hospital and the highest mean hospital costs.

Head injury is a major cause of morbidity in survivors, disability may occur whatever the initial severity of the head injury, and surviving patients with brain injury are more impaired than patients with injuries to other regions.

An understanding of the concept of secondary brain injury, caused by hypotension and hypoxia, is fundamental and the treatment of a head-injured patient should emphasize early control of the airway (while immobilizing the cervical spine), ensuring adequate ventilation and oxygenation, correcting hypovolaemia and prompt imaging by computed tomography (CT). Recent guidelines have been produced in an attempt to improve outcome after severe traumatic brain injury.

14.1 INJURY PATTERNS

There are two major categories of brain injury: focal injuries and diffuse injuries.

Focal brain injuries, which are usually caused by direct blows to the head, comprise contusions, brain lacerations and haemorrhage, leading to the formation of haematoma in the extradural, subarachnoid, subdural or intracerebral compartments within the head. The availability of CT has been shown to reduce mortality for patients with acute extradural haematoma, because the time taken to diagnose and evacuate an intracerebral haematoma is critical in determining outcome. However, most patients with brain injury do not have a lesion suitable for neurosurgical intervention.

Diffuse brain injuries, which are usually caused by a sudden movement of the head, cause failure of certain axons. The distal segment of the axon undergoes degeneration, with subsequent deafferentation of its target structure. Profound deficits may result from this diffuse axonal injury.

Associated injury: all patients sustaining a major mechanism of injury should be suspected of having a cervical spine injury. Avoidance of hypotension and hypoxia are critical in the management of head-injured patients.

14.2 DEPRESSED SKULL FRACTURES

Traditional wisdom suggests that all open, depressed skull fractures should be surgically treated and that closed, depressed fractures should be elevated when the depth of the depression meets or exceeds the thickness of the adjacent skull table to alleviate compression of the underlying cortex.

The dura under the fracture must always be repaired, if damaged.

14.3 PENETRATING INJURY

Patients with a penetrating craniocerebral injury require emergency craniotomy if there is significant mass effect from a haematoma or bullet track.

Removal of fragments of the projectile or in-driven bone fragments should not be pursued at the expense of damaging normal brain tissue.

Patients with penetrating craniocerebral gunshot injuries with a Glasgow Coma Scale (GCS) score of ≤ 5 after resuscitation, or a GCS of ≤ 8 with CT findings of transventricular or bihemispheric injury, have a particularly poor outcome and conservative treatment may be indicated.
14.4 ADJUNCTS TO CARE

14.4.1 Antibiotics

Broad-spectrum antibiotic prophylaxis is recommended for both military and civilian penetrating craniocerebral injuries, including those resulting from sports or recreational injuries. Generally, a cephalosporin or amoxicillin/clavulanate is recommended.

14.4.2 Anticonvulsants

Seizure activity in the early post-traumatic period following head injury may cause secondary brain damage as a result of increased metabolic demands, raised intracranial pressure and excess neurotransmitter release.

For patients who have had a seizure after a head injury, anticonvulsants are indicated and are usually continued for 6 months to 1 year.

Many neurosurgeons give prophylactic anticonvulsants to all patients with significant head injury for at least the first few days after injury; however, the exact duration and role of these drugs are unclear.

Schierhout and Roberts recently reviewed the available evidence and concluded that, although prophylactic antiepileptics are effective in reducing early seizures, there is no evidence that treatment with prophylactic antiepileptics reduces the occurrence of late seizures, or has any effect on death and neurological disability.

14.5 BURR HOLES

Patients with closed head injury and expanding extradural or subdural haematomas require urgent craniotomy for decompression and control of haemorrhage.

In remote areas where neurosurgeons are not available, non-neurosurgeons may occasionally need to intervene to avert progressive neurological injury and death. Surgeons in remote, rural hospitals in the USA have shown that emergency craniotomy can be undertaken with good results where there are clear indications.

14.6 REFERENCE


14.7 RECOMMENDED READING


15.1 **PAEDIATRICS**

15.1.1 **Introduction**

An understanding of the different anatomy, physiology and injury patterns of the injured child is essential for a successful outcome. Many simple, familiar procedures that are taken for granted in the adult patient need to be practised with children before they can be safely performed in the stress of a resuscitation situation. The need for referral should be considered as soon as the patient will tolerate safe transfer to an appropriate facility.

15.1.2 **Pre-hospital**

Pre-hospital interventions should be limited to basic life support with airway and ventilatory support, securing haemostasis of external bleeding and basic attempts to secure vascular access. No child should have the transfer to hospital delayed as a result of dogged determination to achieve certain steps of resuscitation just because these are routine in adults. The younger the child and the more unstable, the greater the tendency should be to ‘scoop and run’ to the nearest appropriate facility. Extensive unsuccessful roadside resuscitative procedures are a common cause of morbidity and mortality.

15.1.3 **Resuscitation room**

15.1.3.1 **AIRWAY**

The indications for airway control are identical to those for the adult patient. The clinical assessment of cervical spine injury is less reliable in the fearful, uncooperative child and, in most cases of blunt polytrauma, radiological assessment is necessary. The routine administration of oxygen, the stepwise system of management according to severity of airway compromise and the avoidance of surgical cricothyroidotomy are the cardinal features of paediatric airway management.

Intubation is accomplished using a non-cuffed endotracheal tube. A surgical airway is seldom performed; if required a tracheotomy should be performed. Be aware of tube dislodgement. The greatest pitfalls are incorrect endotracheal tube placement, too small an endotracheal tube and tube dislodgement as a result of inadequate securing of the tube. The placement of an endotracheal tube in a small child requires no force, otherwise bothersome or even dangerous post-extubation stridor can ensue from a traumatic intubation. The airway of the obligate nasal breather (the neonate or infant) must not be compromised with a nasogastric tube.

15.1.3.2 **VENTILATION**

Hypoventilation is a prominent cause of hypoxia in the injured child. As the child depends primarily on diaphragmatic breathing, one must be particularly cautious of conditions that impair diaphragmatic movement (tension pneumothoraces, diaphragmatic rupture and severe gastric dilatation) and treat expeditiously. Once a controlled and monitored situation has been obtained, one should avoid both barotrauma and volume trauma by providing about 6 ml/kg body weight tidal volume at the lowest pressure. Usually it is safer to permit mild-to-moderate hypercapnia (permissive hypercapnia) than to cause acute lung injury from hyperventilation.

15.1.3.3 **CIRCULATION**

Frequent assessment of circulatory status is important. The small child has effective compensatory mechanisms for hypovolaemic shock, dependent predominantly on an adequate heart rate. Tachycardia, peripheral vasoconstriction and signs of inadequate central nervous
system (CNS) perfusion predominate. Hypotension is a late sign of blood loss. The practitioner must recognize and treat shock aggressively. Most patients have hypovolaemic shock. Other forms of shock are rarer in children than in adult patients. The primary management of bleeding is surgical haemostasis. Most children respond rapidly to crystalloid resuscitation. Rapid vascular access is obtained tailored to the severity of the child’s shock and practitioner experience. Evaluate the effect of crystalloid fluid resuscitation (e.g. Ringer’s lactate 20 ml/kg as a bolus can be repeated once or twice). If the patient still remains haemodynamically unstable, transfuse with packed red blood cells. Hypothermia is critical and must be avoided. The urinary output is an invaluable aid to determine adequacy of resuscitation.

As mentioned the small child has an effective compensation mechanism for hypovolaemic shock, but a tendency to very rapid and calamitous decompensation. A heart rate of 60–80 beats/min in an infant represents a life-threatening bradycardia and requires external cardiac massage.

Do not delay the transfer of the unstable child to the operating theatre – establish good access and resuscitate in theatre while the surgeon stops the bleeding. Here the anaesthetist is in charge!

15.1.3.4 VASCULAR ACCESS

Rapid vascular access is obtained tailored to the severity of the child’s shock and practitioner experience. Central lines are reserved for the larger child and the more experienced operator.

15.1.3.5 CARDIAC ARREST

In children cardiac arrest is not usually caused by ventricular fibrillation and is often heralded by bradycardia, pulseless electrical activity (PEA) or asystole. The primary objective of resuscitation should be to correct the underlying cause (such as tension pneumothorax, hypovolaemia, hypothermia or hypoxia) and to provide cardiac massage and ventilatory support.

15.1.3.6 RESUSCITATIVE THORACOTOMY

Resuscitative thoracotomy is usually futile and not recommended for blunt trauma although it should be considered for children with witnessed cardiac arrest and penetrating thoracic injury.

15.1.3.7 NEUROLOGICAL ASSESSMENT

In general, children have a lower incidence of intracranial mass lesions requiring surgical drainage after blunt injury, compared with adults. The child with no localizing neurological signs, even with a severe head injury, and who is haemodynamically unstable, should not have surgery delayed in order to obtain computed tomography of the brain. Signs of transtentorial hemiation – a unilateral fixed dilated pupil, contralateral muscle weakness in the lower extremity from anterior cerebral artery compression or deteriorating level of consciousness – require an urgent computed tomography (CT) scan and prompt neurosurgical management, which supersedes all other priorities except the management of the airway and treatment of shock. It may be necessary for the neurosurgeons and abdominal or orthopaedic surgeons to operate in two teams.

15.1.4 Recognition of injury patterns

Certain injury patterns of paediatric trauma are becoming apparent, so it is important to take an accurate history of the mechanism of injury in order to detect associated injuries during the resuscitation stage:

- Lap belt complex
- Pedestrian vehicle accident (PVA) complex
- Forward-facing infant complex
- The common cycle scenarios: the fall astride and the handlebars in epigastrium
- Non-accidental injury complex.

15.1.5 Specific organ injury

Abdominal blunt trauma is a lower priority in the stable patient or the unstable patient with other emergency requirements.

Pelvic fracture is a rare cause for exsanguination.

Severe limb fracture, particularly with extensive soft tissue damage, is an immediate priority.

The head-injured patient with no localizing signs who is haemodynamically unstable or actively bleeding must not have definitive haemostasis delayed for a brain CT scan.

Angiography in small children may be difficult and carries a higher complication rate. Its use must be considered carefully.
15.1.6 Analgesia

Appropriate doses of morphine – 0.1 mg/kg 4-hourly or when required – greatly facilitates resuscitation and assessment, and does not mask important clinical signs but rather improves the patient’s cooperation.

15.2 ELDERLY PEOPLE

15.2.1 Definition

Particularly in the more developed world, the population is ageing. In the USA in 1990, those aged over 65 accounted for 12.5 per cent of the population, whereas, in 2040, it is expected that this will have risen to > 20 per cent of the population.

The definition of ‘elderly’ varies. Although conventionally, the term is used to describe an age of 65 years or more, in trauma scoring systems the break-point for elderly people is 55 years of age. In the USA, the 12.5 per cent of the population over the age of 65 accounts for almost a third of all deaths from injury.

15.2.2 Physiology

15.2.2.1 Respiratory System
- Decreased lung elasticity with decreased pulmonary compliance
- Coalescence of alveoli
- Decrease in surface area for gas exchange
- Atrophy of bronchial epithelium leading to a decrease in clearance of particulate foreign matter
- Chronic bacterial colonization of the upper airway.

15.2.2.2 Cardiovascular System
- Diminished pump function and lower cardiac output
- Inability to mount an appropriate response to both intrinsic and extrinsic catecholamines, and consequent inability to augment cardiac output
- Reduced flow to vital organs
- Coexisting, commonly prescribed medication can blunt normal physiological responses.

15.2.2.3 Nervous System
- Progressive atrophy of the brain
- Deterioration on cerebral and cognitive functions
- Cognition
- Hearing
- Eyesight
- Proprioception.

15.2.2.4 Renal
- Decline in renal mass
- Normal serum creatinine does not imply renal function
- Increased vulnerability to nephrotoxic agents (e.g. non-steroidal anti-inflammatory medication).

15.2.2.5 Musculoskeletal
- Osteoporosis causing fracture in the presence of minimal energy transfer
- Diminution of vertebral body height
- Decrease in muscle mass.

15.2.3 Influence of co-morbid conditions

In addition to the typical changes listed above, the development of disease states commonly associated with elderly people can have a significant impact on the response to injury, and can include the following:
- Cardiac disease including hypertension
- Metabolic disease
- Diabetes mellitus
- Obesity (body mass index or BMI > 35)
- Liver disease
- Malignancy
- Pulmonary disease
- Renal disease
- Neurological or spinal disease.

15.2.4 Outcome

Mortality rates are higher for comparable injuries, compared with younger patients. The following guidelines have been recommended:
- Accept potential for decreased physiological reserve
- Suspect co-morbid disease
- Suspect the use of medication
- Look for subtle signs of organ dysfunction by aggressive monitoring
- Assume that any alteration in mental status is
associated with brain injury, and accept only age-related deterioration after exclusion of injury
• Be aware of poorer outcomes and sudden physiological deterioration
• Be aware of the distinction between aggressive care and futile care.

15.2.5 Recommended reading


15.3 FUTILE CARE

In every environment there are circumstances where provision of adequate healthcare may not alter the outcome. In doing so, there may be a significant drain on resources available, and denial to others of adequate care as a result. This ‘rationing’ of healthcare may be the result of operating theatres being in use, and consequently not available, inadequate numbers of intensive care unit (ICU) beds or financial restrictions.

All patients are entitled to aggressive initial resuscitation and careful comprehensive diagnosis. The magnitude of their injuries should be assessed and the appropriateness and aggression required in their care fully discussed with associated staff and family.

It is essential to be humane, and not to prolong life without definite therapeutic goals and realistic expectations.

There should be full involvement, depending on the circumstances, of ethical and social support staff, the family and the medical team.

Our primary aim as physicians is to relieve suffering.
16.1 INTRODUCTION

In a large number of patients interventional radiology, with arterial embolization (AE), stent or stent graft placement, has become either the first line of treatment or an important adjunct to open surgery. Clinical evaluation, however, determines the course of treatment. Patients who are haemodynamically stable are evaluated with computed tomography (CT) for non-operative management (NOM) with or without interventional radiology. Patients who are haemodynamically unstable despite resuscitation are diagnosed with chest and pelvic radiograph, focused abdominal sonography for trauma (FAST) and/or diagnostic peritoneal lavage (DPL), aimed at determining the most compelling bleeding source, and are then directed to the operating theatre for immediate operative treatment without additional imaging.

16.2 PELVIC FRACTURES

Severe pelvic fractures, particularly with disruption of the sacroiliac joints, are associated with a high risk of severe arterial and venous bleeding. The application of a sheet or external fixation may control the venous bleeding. However, arterial bleeding often requires AE, which has become the first line of treatment in patients stable enough to reach angiography. Established indications are CT evidence of ongoing bleeding, such as contrast extravasation, and pelvic haematoma with bladder compression and ongoing transfusion requirements without evidence of other extrapelvic bleeding sources.

There is also a possibility in this subgroup of patients for severe venous bleeding. The patient in shock refractory to resuscitation should therefore be considered for DCS with (extraperitoneal) pelvic packing before AE. Care should be taken to assess the external iliac veins because these are less amenable to packing.

Arterial embolization is carried out after performing abdominal aortography, followed by selective catheterization of the internal iliac arteries. When contrast extravasation is demonstrated, the bleeding vessels are catheterized superselectively and embolized with coils, or a combination of coils and gelfoam particles. If this is not possible as a result of spasm or uncontrolled bleeding, a central embolization of the internal iliac arteries is performed using coils. If the patient deteriorates haemodynamically during angiography an occlusion balloon may be placed in the infrarenal aorta to achieve haemodynamic control.

16.3 BLUNT SPLENIC INJURIES

Non-operative management of blunt splenic injuries has become the treatment of choice in haemodynamically stable patients, regardless of injury grade and grade of haemoperitoneum, in the absence of other intra-abdominal injuries requiring laparotomy. It has been strongly motivated by the wish to preserve the spleen in order to avoid overwhelming post-splenectomy infections (OPSIs) and laparotomy-associated morbidity. This can be achieved by using splenic AE in selected patients.

The indications for AE include CT evidence of ongoing bleeding with contrast extravasation outside or within the spleen, a drop in haemoglobin, tachycardia and haemoperitoneum, as well as formation of pseudoaneurysm.

Selective catheterization of the splenic artery is performed, followed by superselective catheterization of the bleeding arteries or feeders to the pseudoaneurysm. Embolization is then performed using micro-coils (can be combined with gelfoam particles or microspheres). In this way infarctions caused by embolization are limited to small areas.

If there are multiple bleeding arteries or selective catheterization is impossible as a result of spasm, a
central embolization of the splenic artery may be performed using micro-coils. Such an embolization often contributes to decreasing the perfusion pressure and is often enough to stop the bleeding, while at the same time preserving the circulation to the spleen through collaterals that exist in this area. By applying such selection criteria and technique, NOM of blunt splenic injuries may be successful in up to 85–95 per cent of the patients.

16.4 LIVER INJURIES

Non-operative management of blunt liver injuries in haemodynamically stable or stabilized patients has become standard practice. The introduction of AE has been reported to increase the success rate of NOM to well over 80 per cent.

Surgical treatment of liver injuries, even in experienced hands, still carries a high mortality and morbidity risk. Arterial embolization seems to be a valuable adjunct to surgical management because most patients are haemodynamically abnormal at the end of a damage control laparotomy, and ongoing arterial bleeding is difficult to rule out clinically.

As for blunt splenic injuries, the indications for AE should include CT evidence of ongoing bleeding with contrast extravasation outside or within the liver, a drop in haemoglobin, tachycardia and haemoperitoneum, as well as formation of pseudoaneurysm. The risk of bleeding with NOM in organ injury scale (OIS) grade 4 and 5 liver injuries is significant, and angiography in these patients should offer no controversy. In addition, angiography should be performed after damage control surgery with packing of the liver.

Angiography is performed via femoral artery puncture. Embolization is performed as peripherally as possible by the placement of micro-coils, or in combination with gelfoam particles. A completion angiogram should be performed to confirm haemostasis of the embolized vessel.

16.6 RECOMMENDED READING

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Part 6

Appendices
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Care of the injured patient has been fundamental to the practice of medicine since recorded history. The word ‘trauma’ derives from the Greek meaning ‘bodily injury’. The first trauma centres were used to care for wounded soldiers in Napoleon’s armies. The lessons learned in successive military conflicts have advanced our knowledge of the care of the injured patient. The Korean conflict and the Vietnam War established the concept of minimizing the time from injury to definitive care. The extension of this concept to the management of civilian trauma led to the evolution from the 1970s onward of today’s trauma systems.

In principle, a hospital that provides acute care for the severely injured patient (a trauma centre) should be a key component of a system that encompasses all aspects and phases of care, from prevention and education, to pre-hospital care, to acute care, to rehabilitation. The initial trauma systems did not take into consideration the non-trauma centre hospitals, even though they cared for the majority of patients – those less severely injured. Instead, these systems were driven by the major or severely injured trauma patient who required immediate treatment, optimally at a trauma centre.

A system must be fully integrated into the emergency medical services (EMS) system and must meet the needs of all the patients requiring acute care for injury, regardless of severity of injury, geographical location or population density (Figure A.1). The trauma centre remains an essential component, but the system recognizes the necessity of other healthcare facilities. The goal is to match the facility’s resources with the needs of the patient.

A.1 INCLUSIVE TRAUMA SYSTEM

The structure of a trauma care system involves a number of components and providers, each of which must be adapted to a specific environment. The components and providers are:

- Administrative components:
  - leadership
  - system development
  - legislation
  - finances
- Operational and clinical components
- Injury prevention and control
- Human resources: work force resources
- Education
- Pre-hospital care: EMS
- Ambulance and non-transporting guidelines:
  - communications systems
  - emergency disaster preparedness plan
- Definitive care facilities:
  - trauma care facilities
  - interfacility transfer
  - medical rehabilitation
- Information systems
- Evaluation
- Research.

This can be represented graphically as shown in Figure A.2.
A.1.1 Administration

The system requires administrative leadership, authority, planning and development, legislation and finances. Together, these components form an outer sphere of stability that is vital for the continuation of activities directly related to patient care. The diversity of the population as defined by the environment (urban or rural) or by special segments of the population (young people or elderly people) must be addressed by the system.

A.1.2 Prevention

Prevention reduces the actual incidence of injury, and is cost-efficient for both the system and society. Injury prevention is achieved through public education, legislation and environmental modification.

A.1.3 Public education

Public education leads to a change in behaviour, and thus minimizes injury exposure. Education includes the proper recognition of injury and efficient access to the EMS system. These components stimulate the necessary political and legislative activity to establish legal authority, leadership and system changes.

The development of a system is a major challenge for any community. The concept of centralizing trauma care creates potential political and economic problems, because the normal flow of patients might be altered by trauma triage protocols. Trauma systems, by their nature, will direct the care of the most critically injured patients to a limited number of designated 'trauma centres'. The trauma system will succeed only if all parties are involved in the initial planning, development and implementation.

It is crucial that doctors, and especially surgeons, are involved in the system-planning process. They should help establish standards of care for all clinical components, and participate in planning, verification, performance improvement and system evaluation.

A.2 INJURED PATIENT MANAGEMENT WITHIN A SYSTEM

Once the injury has been identified, the system must ensure easy access and an appropriate response to the
scene of injury. The system must assign responsibility and authority for care and triage decisions made before trauma centre access. Triage guidelines must be accepted by all providers and used to determine which patients require access to trauma centre care. This coordination requires direct communication among pre-hospital care providers, medical direction and the trauma facility.

The trauma centre, which serves as the definitive specialized care facility, is a key component of the system, and is different from other hospitals within the system, in that it guarantees immediate availability of all the specialties necessary for the assessment and management of the patient with multiple injuries. These centres need to be integrated into the other components of the system to allow the best match of resources with the patients’ needs. The system coordinates care among all levels of the facility, so that prompt and efficient integration of hospital and resources can take place according to patient need.

Access to rehabilitation services, first in the acute care hospital and then in more specialized rehabilitation facilities, is an integral part of the total management of the patient. It is important that the patients be returned to their communities when appropriate.

A.3 STEPS IN ORGANIZING A SYSTEM

A.3.1 Public support

Public support is necessary for enabling the necessary legislation. The process takes place as follows:

- Identification of the need
- Establishment of a patient database to assist with need and resource assessment
- Analysis to determine resources available
- Resource assessment formulated to identify current capabilities of the system
- Deficiencies highlighted and solutions formulated.

A.3.2 Legal authority

This is established once the need for a system has been demonstrated. Legislation will be required to establish a lead agency with a strong oversight or advisory body composed of healthcare, public and medical representatives. This agency will develop the criteria for the system, regulate and direct pre-hospital care, establish pre-hospital triage, ensure medical direction, designate the proper facilities to render care, establish a trauma registry and establish performance improvement programmes.

A.3.3 Establish criteria for optimal care

These must be established by the lead authority in conjunction with health and medical professionals. The adoption of system-wide standards is integral to the success of any system.

A.3.4 Designation of trauma centres

This takes place through a public process directed by the lead agency. Consideration must be given to the role of all acute care facilities within the particular region. Representatives from all these facilities must be involved in the planning process.

The number of trauma centres should be limited to the number required (based on the established need) for the patient population at risk from major injury. Having too many trauma centres may weaken the system by diluting the workload and reducing the experience for training, and will unnecessarily consume resources that are not fully utilized.

Development of a system requires that all the principal players be involved from the beginning. There must be agreement about the minimal data that will be contributed by all acute care facilities. Without the data from the hospitals managing less severely injured individuals, the data will be incomplete and skewed towards major injury.

A.3.5 System evaluation

Trauma systems are complex organizational structures with evolving methods and standards of care. It is necessary to have a mechanism for ongoing evaluation, based on:

- self-monitoring
- external evaluation.

A.4 RESULTS AND STUDIES

The Skamania Conference was held in July 1998. The purpose of the conference was to evaluate the evidence
regarding the efficacy of trauma systems. During the conference the evidence was divided into three categories, resulting from panel studies, registry comparisons and population-based research.

A.4.1 Panel review

An overview of panel studies was presented at the Skamania Conference. The critique of panel reviews is that they vary widely and inter-rater reliability has been very low in some studies. Furthermore, postmortem results alone are inadequate and panel studies vary with regard to the process of review and the rules used to come to a final judgement. In general, all panel studies were classified as weak class III evidence. Nevertheless, MacKenzie\(^1\) came to the conclusion that, when all panel studies are considered collectively, they do provide some face validity and support of the hypothesis that treatment at a trauma centre versus treatment at a non-trauma centre is associated with fewer inappropriate deaths and possibly disabilities.

A.4.2 Registry study

Jurkovich and Mock\(^2\) reported on the evidence provided by trauma registries in assessing overall effectiveness. They concluded fairly emphatically that this was not class I evidence but that it was probably better than a panel study. Their critique of trauma registries included the following six items:

1. Data are often missing
2. Miscodings occur
3. There may be inter-rater reliability factors
4. The national norms are not population based
5. There is less detail about the causes of death
6. They do not take into account pre-hospital deaths.

A consensus of the participants at the Skamania Conference concluded that registry studies were better than panel studies but not as good as population studies.

A.4.3 Population-based studies

Populated-based studies probably also fall into class II evidence. They are not prospective randomized trials, but, because of the nature of the population-based evidence, they cover all aspects of trauma care including pre-hospital, hospital and rehabilitation care. A critique of the population-based studies pointed out that there are a limited number of clinical variables, and it is difficult to adjust for severity of injury and physiological dysfunction. There are other problems, although they probably apply to all studies, including secular trends, observational issues and problems with longitudinal population mortality studies.

A.5 SUMMARY

Although there are difficulties with all three types of studies, each may also offer advantages to various communities and regions. All three studies may influence health policy and all can be used pre- and post-trauma system start-up. There was consensus at the Skamania Conference that we need to extend our evaluation of trauma systems to include an economic evaluation and assessment of quality-adjusted life years. Finally, we need to expand trauma systems to include all regions of the USA.

A.6 REFERENCES


A.7 RECOMMENDED READING


Appendix B

Trauma scores and scoring systems

B.1 INTRODUCTION

Estimates of the severity of injury or illness are fundamental to the practice of medicine. The earliest known medical text, the *Smith Papyrus*, classified injuries into three grades: treatable, contentious and untreatable.

Modern trauma scoring methodology uses a combination of an assessment of the severity of anatomical injury with a quantification of the degree of physiological derangement to arrive at scores that correlate with clinical outcomes.

Trauma scoring systems are designed to facilitate pre-hospital triage, identify trauma patients suitable for quality assurance audit, allow accurate comparison of different trauma populations, and organize and improve trauma systems.

In principle, scoring systems can be divided into:

- Physiological scoring systems, based on the body’s response to injury
- Anatomical scoring systems, based on the physical injury that has occurred
- Outcome analysis systems, based on the result after recovery.

B.2 PHYSIOLOGICAL SCORING SYSTEMS

B.2.1 Glasgow Coma Scale

The Glasgow Coma Scale (GCS), developed in 1974, was one of the first numerical scoring systems (Table B.1). The GCS has been incorporated into many later scoring systems, emphasizing the importance of head injury as a triage and prognostic indicator.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td>Nil</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Spontaneously</td>
<td>4</td>
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<tr>
<td>Motor response</td>
<td>Nil</td>
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</tr>
<tr>
<td></td>
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<tr>
<td></td>
<td>Flexor</td>
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<td></td>
<td>Withdrawal</td>
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<tr>
<td></td>
<td>Localizing</td>
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</tr>
<tr>
<td></td>
<td>Obey command</td>
<td>6</td>
</tr>
<tr>
<td>Verbal response</td>
<td>Nil</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Groans</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Orientated</td>
<td>5</td>
</tr>
</tbody>
</table>

B.2.2 Revised Trauma Score

Introduced by Champion et al., the Revised Trauma score (RTS) evaluates blood pressure, GCS and respiratory rate to provide a scored physiological assessment of the patient.

The RTS can be used for field triage and enables pre-hospital and emergency care personnel to decide which patients should receive the specialized care of a trauma unit. An RTS \( \leq 11 \) is suggested as the triage point for patients requiring at least level 2 trauma centre status (surgical facilities, 24-hour radiograph, etc.). An RTS \( \leq 10 \) carries a mortality rate of up to 30 per cent and these patients should be moved to a level 1 institution.

The difference between RTS on arrival and best RTS after resuscitation will give a reasonably clear picture of prognosis. By convention the RTS on admission is the one documented.

The RTS (non-triage) is designed for retrospective outcome analysis. Weighted coefficients are used, which are derived from trauma patient populations and provide...
more accurate outcome prediction than the raw RTS (Table B.2). As a severe head injury carries a poorer prognosis than a severe respiratory injury, the weighting is therefore heavier. The RTS thus varies from 0 (worst) to 7.8408 (best). The RTS is the most widely used physiological scoring system in the trauma literature.

### B.2.3 Paediatric Trauma Score

The Paediatric Trauma Score (PTS)\(^1\) (Table B.3) has been designed to facilitate triage of children. The PTS is the sum of six scores, and values range from –6 to +12, with a PTS of ≤ 8 being recommended as a trigger to send to a trauma centre. The PTS has been shown to predict risk accurately for severe injury or mortality, but is not significantly more accurate than the RTS and is a great deal more difficult to measure.

### B.3 anatomical scoring systems

#### B.3.1 Abbreviated Injury Scale

The Abbreviated Injury Scale\(^4\) (AIS) is an anatomically based, consensus-derived, global, severity-based scoring system, which classifies each injury by body region according to its relative importance on 6-point ordinal scale.

The AIS was developed in 1971 as a system to describe the severity of injury throughout the body. The AIS has been periodically upgraded and AIS-2005 is currently in use.

In AIS-2005, each injury is assigned a 6-digit unique numerical identifier (UNI), to the left of the decimal point. This in known as the ‘pre-dot’ code. There is an additional single digit to the right of the code (the ‘post-dot’ code) which is the AIS severity code. The AIS grades each injury by severity from 1 (least severe) to 5 (critical: survival uncertain). A score of 6 is given to certain injuries termed 'maximal (currently untreatable)'.

The AIS Manual\(^4\) is divided, for ease of reference, into nine different sections based on anatomy. All injuries therefore carry a unique code that can be used for classification, indexing in trauma registry data bases and severity.

#### B.3.2 Injury Severity Score

In 1974, Baker et al. created the Injury Severity Score\(^5\) (ISS) to relate AIS scores to patient outcomes. The ISS body regions are listed in Table B.4.
The ISS is calculated by summing the square of the highest AIS scores in the three most severely injured regions. ISS scores range from 1 to 75 (as the highest AIS score for any region is 5). By convention, an AIS score of 6 (defined as a non-survivable injury) for any region becomes an ISS of 75.

The ISS considers only the single, most serious injury in each region, ignoring the contribution of injury to other organs within the same region. Diverse injuries may have identical ISSs but markedly different survival probabilities (ISS of 25 may be obtained with isolated severe head injury or by a combination of lesser injuries across different regions). Also, ISS does not have the power to discriminate between the impact of similarly scored injuries to different organs, and therefore cannot identify, for example, the different impact of cerebral injury over injury to other organ systems. In response to these limitations, in 1997, the ISS was modified to become the New Injury Severity Score (NISS) as the simple sum of the squares of the three highest AIS scores regardless of body region. NISS is able to predict survival outcomes better than ISS. Although the proponents of NISS proclaim its superiority, it is not in widespread use to date.

### B.3.3 Anatomic Profile

The Anatomic Profile (AP) was introduced in 1990 to overcome some of the limitations of the ISS. In contrast to the ISS, the AP allows the inclusion of more than one serious body injury per region, and takes into account the primacy of central nervous system (CNS) and torso injury over other injuries. AIS scoring is used, but four values are used for injury characterization, roughly weighting the body regions. Serious trauma to the brain and spinal cord, anterior neck and chest, and all remaining injuries constitute three of the four values.

The fourth value is a summary of all remaining non-serious injuries. The AP score is the square root of the sum of the squares of all the AIS scores in a region, thus enabling the impact of multiple injuries within that region to be recognized. Component values for the four regions are summed to constitute the AP score.

A modified Anatomic Profile (mAP) has recently been introduced, which is a 4-number characterization of injury. The four component scores are the maximum AIS score and the square root of the sum of the squares of all AIS values for serious injury (AIS ≥ 3) in specified body regions (Table B.5). This leads to an Anatomic Profile Score, the weighted sum of the four mAP components. The coefficients are derived from logistic regression analysis of admissions to four level 1 trauma centres (the ‘controlled sites’) in the Major Trauma Outcome Study.

### Table B.4 Injury Severity Score body regions

<table>
<thead>
<tr>
<th>Number</th>
<th>Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Head and neck</td>
</tr>
<tr>
<td>2</td>
<td>Face</td>
</tr>
<tr>
<td>3</td>
<td>Thorax</td>
</tr>
<tr>
<td>4</td>
<td>Abdomen/pelvic contents</td>
</tr>
<tr>
<td>5</td>
<td>Extremities</td>
</tr>
<tr>
<td>6</td>
<td>External/skin/general</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Component</th>
<th>Body region</th>
<th>AIS severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>mA</td>
<td>Head/brain</td>
<td>3–6</td>
</tr>
<tr>
<td>mB</td>
<td>Spinal cord</td>
<td>3–6</td>
</tr>
<tr>
<td>mC</td>
<td>Thorax</td>
<td>3–6</td>
</tr>
<tr>
<td>mD</td>
<td>Front of neck</td>
<td>3–6</td>
</tr>
<tr>
<td>mE</td>
<td>All other</td>
<td>3–6</td>
</tr>
</tbody>
</table>

mA, mB, mC scores are derived by taking the square root of the sum of the squares for all injuries defined by each component.

AIS, Abbreviated Injury Scale.

A limitation of the use of AIS-derived scores is their cost. The International Classification of Disease (ICD) taxonomy is a standard used by most hospitals and other healthcare providers to classify clinical diagnoses. Computerized mapping of ICD-9CM rubrics into AIS body regions and severity values has been used to compute ISS, AP and NISS scores. Despite limitations, ICD-AIS conversion has been useful in population-based evaluation when AIS scoring from medical records is not possible. Outside North America, the ICD-10 is most commonly used.

Severity scoring systems have also been directly derived from ICD-coded discharge diagnoses. Most recently, the ICD-9 Severity Score (ICISS) has been proposed, which is derived by multiplying survival risk ratios associated with individual ICD diagnoses. Neural networking has been employed to improve ICISS accuracy further. ICISS has been shown to be better than ISS and to outperform the Trauma and Injury Severity
Score (TRISS) in identifying outcomes and resource utilization. However, modified-AP scores, AP and NISS appear to outperform ICISS in predicting hospital mortality.

There is some confusion as to which anatomical scoring system should be used; however, currently, NISS probably should be the system of choice for AIS-based scoring.

**B.3.4 Organ Injury Scaling system**

Organ Injury Scaling (OIS) is a scale of anatomical injury within an organ system or body structure. The goal of OIS is to provide a common language between trauma surgeons and to facilitate research and continuing quality improvement. It is not designed to correlate with patient outcomes. The OIS tables can be found on the American Association for the Surgery of Trauma (AAST) website, or at the end of this chapter.

**B.3.5 Penetrating Abdominal Trauma Index**

Moore and colleagues facilitated identification of the patient at high risk of postoperative complications when they developed the Penetrating Abdominal Trauma Index (PATI) scoring system for patients whose only source of injury was penetrating abdominal trauma. A complication risk factor was assigned to each organ system involved, and then multiplied by a severity of injury estimate. Each factor was given a value ranging from 1 to 5. The complication risk designation for each organ was based on the reported incidence of postoperative morbidity associated with the particular injury.

The severity of injury was estimated by a simple modification to the Abbreviated Injury Scale, where 1 = minimal injury to 5 = maximal injury. The sum of the individual organ score multiplied by the risk factor comprised the final PATI. If the PATI is ≤ 25, the risk of complications is reduced (and where it is ≤ 10, there are no complications), whereas if it is ≥ 25, the risks are much higher.

In a group of 114 patients with gunshot wounds to the abdomen, Moore et al. showed that a PATI score > 25 dramatically increased the risk of postoperative complications (46 per cent of patients with a PATI score > 25 developed serious postoperative complications compared with 7 per cent of patients with a PATI < 25). Further studies have validated the PATI scoring system.

**B.4 OUTCOME ANALYSIS**

**B.4.1 Glasgow Outcome Scale**

For head-injured patients, the level of coma on admission or within 24 hours expressed by the Glasgow Coma Scale was found to correlate with outcome. The Glasgow Outcome Scale was an attempt to quantify outcome parameters (Table B.6) for head-injured patients.

<table>
<thead>
<tr>
<th>Outcome parameters</th>
<th>GR</th>
<th>MD</th>
<th>SD</th>
<th>PVS</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good recovery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate disability</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe disability</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent vegetative state</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The grading of depth of coma and neurological signs was found to correlate strongly with outcome, but the low accuracy of individual signs limits their use in predicting outcomes for individuals (Table B.7).

<table>
<thead>
<tr>
<th>Outcome related to signs in the first 24 hours of coma after injury (outcome scale as described by Glasgow group)</th>
<th>Dead or vegetative (%)</th>
<th>Moderate disability or good recovery (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pupils:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reacting</td>
<td>39</td>
<td>50</td>
</tr>
<tr>
<td>Non-reacting</td>
<td>91</td>
<td>4</td>
</tr>
<tr>
<td>Eye movements:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intact</td>
<td>33</td>
<td>56</td>
</tr>
<tr>
<td>Absent/bad</td>
<td>90</td>
<td>5</td>
</tr>
<tr>
<td>Motor response:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>36</td>
<td>54</td>
</tr>
<tr>
<td>Abnormal</td>
<td>74</td>
<td>16</td>
</tr>
</tbody>
</table>

**B.4.2 Major Trauma Outcome Study**

In 1982, the American College of Surgeons Committee on Trauma began the ongoing Major Trauma Outcome
Study (MTOS), a retrospective, multicentre study of trauma epidemiology and outcomes.

The MTOS uses TRISS methodology\(^{12}\) to estimate the probability of survival, or \(P(s)\), for a given trauma patient. \(P(s)\) is derived according to the formula:

\[
P(s) = \frac{1}{1 + e^{-b}}
\]

where \(e\) is a constant (approximately 2.718282) and \(b = b_0 + b_1(RTS) + b_2(ISS) + b_3(age\ factor)\). The \(b\) coefficients are derived by regression analysis from the MTOS database (Table B.8).

The \(P(s)\) values range from zero (survival not expected) to 1.000 for a patient with a 100 per cent expectation of survival. Each patient’s values can be plotted on a graph with ISS and RTS axes (Figure B.1).

In addition to analysing individual patient outcomes, TRISS allows comparison of a study population with the huge MTOS database. The ‘\(Z\)-statistic’ identifies whether study group outcomes are significantly different from expected outcomes as predicted from MTOS.

\[
Z = \frac{(A - E)}{S}
\]

where \(A\) is the actual number of survivors, \(E\) the expected number of survivors and \(S\) a scale factor that accounts for statistical variation. \(Z\) may be positive or negative, depending on whether the survival rate is greater or less than predicted by TRISS. Absolute values of \(Z > 1.96\) or \(< -0.96\) are statistically significant (\(p < 0.05\)).

The so-called \(M\)-statistic is an injury severity match allowing comparison of the range of injury severity in the sample population with that of the main database (i.e. the baseline group). The closer \(M\) is to 1, the better the match, the greater the disparity and the more biased \(Z\) will be. This bias can be misleading, e.g. an institution with a large number of patients with low-severity injuries can falsely appear to provide a better standard of care than another institution that treats a higher number of more severely injured patients.

The ‘\(W\)-statistic’ calculates the actual numbers of survivors greater (or fewer) than predicted by MTOS, per 100 trauma patients treated. The Relative Outcome Score (ROS) can be used to compare \(W\)-values against a ‘perfect outcome’ of 100 per cent survival. The ROS may then be used to monitor improvements in trauma care delivery over time.

TRISS has been used in numerous studies. Its value as a predictor of survival or death has been shown to be from 75 per cent to 90 per cent as good as a perfect index, depending on the patient data set used.

![Figure B.1 PRE chart. Survivors (L) and Nonsurvivors (D) are plotted on a graph, using the weighted Revised Trauma Score (RTS) and Injury Severity Score (ISS) of each. The S50 isobar denotes a probability of survival of 0.50.](image-url)
B.4.3 **A Severity Characterization of Trauma**

A Severity Characterization of Trauma\textsuperscript{13,14} (ASCOT), introduced by Champion et al. in 1990, is a scoring system that uses the AP to characterize injury in place of ISS. Different coefficients are used for blunt and penetrating injury and the ASCOT score is derived from the formula:

\[ P(s) = \frac{1}{1 + e^{-k}} \]

The ASCOT model coefficients are shown in Table B.9. ASCOT has been shown to outperform TRISS, particularly for penetrating injury.

<table>
<thead>
<tr>
<th>( k ) coefficients</th>
<th>Type of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blunt</td>
</tr>
<tr>
<td>( K_1 ) (RTS GCS value)</td>
<td>-1.157</td>
</tr>
<tr>
<td>( K_2 ) (RTS SBP value)</td>
<td>0.7705</td>
</tr>
<tr>
<td>( K_3 ) (RTS RR value)</td>
<td>0.6583</td>
</tr>
<tr>
<td>( K_4 ) (AP head region value)</td>
<td>0.281</td>
</tr>
<tr>
<td>( K_5 ) (AP thoracic region value)</td>
<td>-0.3002</td>
</tr>
<tr>
<td>( K_6 ) (AP other serious injury value)</td>
<td>-0.1961</td>
</tr>
<tr>
<td>( K_7 ) (age factor)</td>
<td>-0.2086</td>
</tr>
<tr>
<td>( K_8 ) (age factor)</td>
<td>-0.6355</td>
</tr>
</tbody>
</table>

AP, Anatomic Profile; ASCOT, A Severity Characterization of Trauma; GCS, Glasgow Coma Scale; MTOS, Multiple Trauma Outcome Study; RR, respiratory rate; RTS, Revised Trauma Score; SBP, systolic blood pressure.

B.5 **SUMMARY**

Trauma scoring systems and allied methods of analysing outcomes after trauma are steadily evolving and have become increasingly sophisticated over recent years.

Trauma scoring systems are designed to facilitate pre-hospital triage, identify trauma patients whose outcomes are statistically unexpected for quality assurance analysis, allow accurate comparison of different trauma populations, and organize and improve trauma systems. They are vital for the scientific study of the epidemiology and the treatment of trauma and may even be used to define resource allocation and reimbursement in the future.

Trauma scoring systems that measure outcome solely in terms of death or survival are at best blunt instruments. Despite the existence of several scales (Quality of Well-being Scale, Sickness Impact Profile, etc.), further efforts are needed to develop outcome measures that are able to evaluate the multiplicity of outcomes across the full range of diverse trauma populations.

Despite the profusion of acronyms, scoring systems are a vital component of trauma care delivery systems. The effectiveness of well-organized, centralized, multidisciplinary trauma centres in reducing the mortality and morbidity of injured patients is well documented. Further improvement and expansion of trauma care can occur only if developments are subjected to scientifically rigorous evaluation. Thus, trauma-scoring systems play a central role in the provision of trauma care today and for the future.

B.6 **REFERENCES**


B.7 SCALING SYSTEM FOR ORGAN-SPECIFIC INJURIES

Table 1 Cervical vascular organ injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Thyroid vein</td>
<td>900.8</td>
<td>$15.8</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Common facial vein</td>
<td>900.8</td>
<td>$15.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>External jugular vein</td>
<td>900.81</td>
<td>$15.2</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Non-named arterial/venous branches</td>
<td>900.9</td>
<td>$15.9</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>External carotid arterial branches (ascending pharyngeal, superior thyroid, lingual, facial, maxillary, occipital, posterior auricular)</td>
<td>900.8</td>
<td>$15.0</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Thyrocervical trunk or primary branches</td>
<td>900.8</td>
<td>$15.8</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Internal jugular vein</td>
<td>900.1</td>
<td>$15.3</td>
<td>1–3</td>
</tr>
<tr>
<td>III</td>
<td>External carotid artery</td>
<td>900.02</td>
<td>$15.0</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Subclavian vein</td>
<td>901.3</td>
<td>$25.3</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Vertebral artery</td>
<td>900.8</td>
<td>$15.1</td>
<td>2–4</td>
</tr>
<tr>
<td>IV</td>
<td>Common carotid artery</td>
<td>900.01</td>
<td>$15.0</td>
<td>3–5</td>
</tr>
<tr>
<td></td>
<td>Subclavian artery</td>
<td>901.1</td>
<td>$25.1</td>
<td>3–4</td>
</tr>
<tr>
<td>V</td>
<td>Internal carotid artery (extracranial)</td>
<td>900.03</td>
<td>$15.0</td>
<td>3–5</td>
</tr>
</tbody>
</table>

Increase one grade for multiple grade III or IV injuries involving more than 50 per cent vessel circumference.
Decrease one grade for less than 25 per cent vessel circumference disruption for grade IV or V.
From Moore et al.1 with permission.

Table 2 Chest wall injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Any size</td>
<td>911.0/922.1</td>
<td>$20.2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Skin and subcutaneous</td>
<td>875.0</td>
<td>$20.4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>Fewer than three ribs, closed</td>
<td>807.01/807.02</td>
<td>$22.3</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-displaced clavicle closed</td>
<td>810.00/810.03</td>
<td>$42.0</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Skin, subcutaneous and muscle</td>
<td>875.1</td>
<td>$20.4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>Three or more adjacent ribs, closed</td>
<td>807.03/807.08</td>
<td>$22.4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Open or displaced clavicle</td>
<td>810.10/810.13</td>
<td>$42.0</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-displaced sternum, closed</td>
<td>807.2</td>
<td>$22.2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Scapular body, open or closed</td>
<td>811.00/811.18</td>
<td>$42.1</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Full thickness including pleural penetration</td>
<td>862.29</td>
<td>$21.9</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>Open or displaced sternum</td>
<td>807.2</td>
<td>$22.2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flail sternum</td>
<td>807.3</td>
<td>$22.2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Unilateral flail segment (fewer than three ribs)</td>
<td>807.4</td>
<td>$22.5</td>
<td>3–4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Avulsion of chest wall tissues with underlying rib fractures</td>
<td>807.10/807.18</td>
<td>$22.8</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>Unilateral flail chest (≥ 3 ribs)</td>
<td>807.4</td>
<td>$22.5</td>
<td>3–4</td>
</tr>
<tr>
<td>V</td>
<td>Fracture</td>
<td>Bilateral flail chest (≥ 3 ribs on both sides)</td>
<td>807.4</td>
<td>$22.5</td>
<td>5</td>
</tr>
</tbody>
</table>

This scale is confined to the chest wall alone and does not reflect associated internal or abdominal injuries. Therefore, further delineation of upper versus lower or anterior versus posterior chest wall was not considered, and a grade VI was not warranted. Specifically, thoracic crush was not used as a descriptive term; instead, the geography and extent of fractures and soft tissue injury were used to define the grade.
Upgrade by one grade for bilateral injuries.
From Moore et al.2 with permission.
### Table 3 Heart injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Blunt cardiac injury with minor ECG abnormality (non-specific ST or T-wave changes, premature arterial or ventricular contraction or persistent sinus tachycardia)</td>
<td>861.01</td>
<td>$26.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Blunt or penetrating pericardial wound without cardiac injury, cardiac tamponade or cardiac herniation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Blunt cardiac injury with heart block (right or left bundle branch, left anterior fascicular or atrioventricular) or ischaemic changes (ST depression or T-wave inversion) without cardiac failure</td>
<td>861.01</td>
<td>$26.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Penetrating tangential myocardial wound up to, but not extending through, endocardium, without tamponade</td>
<td>861.12</td>
<td>$26.0</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Blunt cardiac injury with sustained (&gt; 6 beats/min) or multifocal ventricular contractions</td>
<td>861.01</td>
<td>$26.0</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Blunt or penetrating cardiac injury with septal rupture, pulmonary or tricuspid valvular incompetence, papillary muscle dysfunction or distal coronary arterial occlusion without cardiac failure</td>
<td>861.01</td>
<td>$26.0</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Blunt pericardial laceration with cardiac herniation</td>
<td>861.01</td>
<td>$26.0</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Blunt cardiac injury with cardiac failure</td>
<td>861.01</td>
<td>$26.0</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Penetrating tangential myocardial wound up to, but extending through, endocardium, with tamponade</td>
<td>861.12</td>
<td>$26.0</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Blunt or penetrating cardiac injury with septal rupture, pulmonary or tricuspid valvular incompetence, papillary muscle dysfunction, or distal coronary arterial occlusion producing cardiac failure</td>
<td>861.12</td>
<td>$26.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Blunt or penetrating cardiac injury with aortic mitral valve incompetence</td>
<td>861.03</td>
<td>$26.0</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Blunt or penetrating cardiac injury with the right ventricle, right atrium or left atrium</td>
<td>861.03</td>
<td>$26.0</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Blunt or penetrating left ventricular perforation</td>
<td>861.13</td>
<td>$26.0</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Stellate wound with &lt; 50% tissue loss of the right ventricle, right atrium or left atrium</td>
<td>861.03</td>
<td>$26.0</td>
<td>5</td>
</tr>
<tr>
<td>VI</td>
<td>Blunt avulsion of the heart; penetrating wound producing &gt; 50% tissue loss of a chamber</td>
<td>861.13</td>
<td>$26.0</td>
<td>6</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple wounds to a single chamber or multiple chamber involvement.*

*From Moore et al., with permission.*

*With ICD-10 use supplementary character: 0 without open wound into thoracic cavity; 1 with open wound into thoracic cavity.*

### Table 4 Lung injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Unilateral, more than one lobe</td>
<td>861.12/861.31</td>
<td>$27.3</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>Contusion</td>
<td>Unilateral, single lobe</td>
<td>861.20/861.30</td>
<td>$27.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Simple pneumothorax</td>
<td>860.0/1/4/5</td>
<td>$27.0</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Contusion</td>
<td>Unilateral, more than one lobe</td>
<td>861.20/861.30</td>
<td>$27.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Persistent (&gt; 72 h) air leak from distal airway</td>
<td>860.0/1/4/5</td>
<td>$27.3</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Haematoma</td>
<td>Non-expanding intraparenchymal</td>
<td>862.0/861.30</td>
<td>$27.3</td>
<td>3–4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Major (segmental or lobar) air leak</td>
<td>862.21/861.31</td>
<td>$27.4</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td>Haematoma</td>
<td>Expanding intraparenchymal</td>
<td></td>
<td>$25.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Primary branch intrapulmonary vessel disruption</td>
<td>901.40</td>
<td>$25.4</td>
<td>3–5</td>
</tr>
<tr>
<td>V</td>
<td>Vascular</td>
<td>Hilar vessel disruption</td>
<td>901.41/901.42</td>
<td>$25.4</td>
<td>4</td>
</tr>
<tr>
<td>VI</td>
<td>Vascular</td>
<td>Total uncontained transection of pulmonary hilum</td>
<td>901.41/901.42</td>
<td>$25.4</td>
<td>4</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III.*

*Haemothorax is scored under thoracic vascular injury scale.*

*From Moore et al., with permission.*

*With ICD-10 use supplementary character: 0 without open wound into thoracic cavity; 1 with open wound into thoracic cavity.*
Table 5  Thoracic vascular injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Intercostal artery/vein</td>
<td>901.81</td>
<td>S25.5</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Internal mammary artery/vein</td>
<td>901.82</td>
<td>S25.8</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Bronchial artery/vein</td>
<td>901.89</td>
<td>S25.4</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Oesophageal artery/vein</td>
<td>901.9</td>
<td>S25.8</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Hemiazygos vein</td>
<td>901.89</td>
<td>S25.8</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Unnamed artery/vein</td>
<td>901.9</td>
<td>S25.9</td>
<td>2–3</td>
</tr>
<tr>
<td>II</td>
<td>Azygos vein</td>
<td>901.89</td>
<td>S25.8</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Internal jugular vein</td>
<td>900.1</td>
<td>S15.3</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Subclavian vein</td>
<td>901.3</td>
<td>S25.3</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Innominate vein</td>
<td>901.3</td>
<td>S25.3</td>
<td>3–4</td>
</tr>
<tr>
<td>III</td>
<td>Carotid artery</td>
<td>900.01</td>
<td>S15.0</td>
<td>3–5</td>
</tr>
<tr>
<td></td>
<td>Innominate artery</td>
<td>901.1</td>
<td>S25.1</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Subclavian artery</td>
<td>901.1</td>
<td>S25.1</td>
<td>3–4</td>
</tr>
<tr>
<td>IV</td>
<td>Thoracic aorta, descending</td>
<td>901.0</td>
<td>S25.0</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td>Inferior vena cava (intrathoracic)</td>
<td>902.10</td>
<td>S35.1</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Pulmonary artery, primary intraparenchymal branch</td>
<td>901.41</td>
<td>S25.4</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Pulmonary vein, primary intraparenchymal branch</td>
<td>901.42</td>
<td>S25.4</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Thoracic aorta, ascending and arch</td>
<td>901.0</td>
<td>S25.0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Superior vena cava</td>
<td>901.2</td>
<td>S25.2</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Pulmonary artery, main trunk</td>
<td>901.41</td>
<td>S25.4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Pulmonary vein, main trunk</td>
<td>901.42</td>
<td>S25.4</td>
<td>4</td>
</tr>
<tr>
<td>VI</td>
<td>Uncontained total transection of thoracic aorta or pulmonary hilum</td>
<td>901.0</td>
<td>S25.0</td>
<td>5</td>
</tr>
</tbody>
</table>

Increase one grade for multiple grade III or IV injuries if more than 50 per cent circumference.
Decrease one grade for grade IV injuries if less than 25 per cent circumference.
From Moore et al., with permission.

Table 6  Diaphragm injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>862.0</td>
<td>S27.8</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration &lt; 2cm</td>
<td>862.1</td>
<td>S27.8</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration 2–10 cm</td>
<td>862.1</td>
<td>S27.8</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration &gt; 10 cm with tissue loss ≤ 25 cm²</td>
<td>862.1</td>
<td>S27.8</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Laceration with tissue loss &gt; 25 cm²</td>
<td>862.1</td>
<td>S27.8</td>
<td>3</td>
</tr>
</tbody>
</table>

Advance one grade for bilateral injuries up to grade III.
From Moore et al., with permission.
Table 7  Spleen injury scale (1994 revision)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Subcapsular, &lt; 10% surface area</td>
<td>865.01/865.11</td>
<td>$36.0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, &lt; 1 cm parenchymal depth</td>
<td>865.02/865.12</td>
<td>$36.0</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Subcapsular, 10–50% surface area; intraparenchymal, &lt; 5 cm in diameter</td>
<td>865.01/865.11</td>
<td>$36.0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, 1–3 cm parenchymal depth not involving a trabecular vessel</td>
<td>865.02/865.12</td>
<td>$36.0</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Haematoma</td>
<td>Subcapsular, &gt; 50% surface area or expanding; ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma ≥ 5 cm or expanding</td>
<td>865.03</td>
<td>$36.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>&gt; 3 cm parenchymal depth or involving trabecular vessels</td>
<td>865.03</td>
<td>$36.0</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Laceration involving segmental or hilar vessels producing major devascularization (&gt; 25% of spleen)</td>
<td>865.13</td>
<td>$36.0</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Completely shattered spleen</td>
<td>865.04</td>
<td>$36.0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Hilar vascular injury with devascularized spleen</td>
<td>865.14</td>
<td>$36.0</td>
<td>5</td>
</tr>
</tbody>
</table>

*aAdvance one grade for multiple injuries up to grade III.
From Moore et al., with permission.
With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

Table 8  Liver injury scale (1994 revision)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Subcapsular, &lt; 10% surface area</td>
<td>864.01/864.11</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, &lt; 1 cm parenchymal depth</td>
<td>864.02/864.12</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Subcapsular, 10–50% surface area; intraparenchymal &lt; 10 cm in diameter</td>
<td>864.01/864.11</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear 1–3 cm parenchymal depth, &lt; 10 cm in length</td>
<td>864.03/864.13</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Haematoma</td>
<td>Subcapsular, &gt; 50% surface area of ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma &gt; 10 cm or expanding</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>3 cm parenchymal depth</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Parenchymal disruption involving 25–75% hepatic lobe or one to three Couinaud's segments within a single lobe</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Parenchymal disruption involving &gt; 75% of hepatic lobe or more than three Couinaud's segments within a single lobe</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Vascular</td>
<td>Juxtahepatic venous injuries, i.e. retrohepatic vena cava/central major hepatic veins</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>5</td>
</tr>
<tr>
<td>VI</td>
<td>Vascular</td>
<td>Hepatic avulsion</td>
<td>864.04/864.14</td>
<td>$36.1</td>
<td>5</td>
</tr>
</tbody>
</table>

*aAdvance one grade for multiple injuries up to grade III.
From Moore et al., with permission.
With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

Table 9  Extrahepatic biliary tree injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Gallbladder contusion/haematoma</td>
<td>868.02</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Portal triad contusion</td>
<td>868.02</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Partial gallbladder avulsion from liver bed; cystic duct intact</td>
<td>868.02</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration or perforation of the gallbladder</td>
<td>868.12</td>
<td>$36.1</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Complete gallbladder avulsion from liver bed</td>
<td>868.02</td>
<td>$36.1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Cystic duct laceration</td>
<td>868.12</td>
<td>$36.1</td>
<td>3</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV</td>
<td>Partial or complete right hepatic duct laceration</td>
<td>868.12</td>
<td>S36.1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Partial or complete left hepatic duct laceration</td>
<td>868.12</td>
<td>S36.1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Partial common hepatic duct laceration (&lt; 50%)</td>
<td>868.12</td>
<td>S36.1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Partial common bile duct laceration (&lt; 50%)</td>
<td>868.12</td>
<td>S36.1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>&gt; 50% transection of common hepatic duct</td>
<td>868.12</td>
<td>S36.1</td>
<td>3–4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt; 50% transection of common bile duct</td>
<td>868.12</td>
<td>S36.1</td>
<td>3–4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Combined right and left hepatic duct injuries</td>
<td>868.12</td>
<td>S36.1</td>
<td>3–4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intraduodenal or intrapancreatic bile duct injuries</td>
<td>868.12</td>
<td>S36.1</td>
<td>3–4</td>
<td></td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al.,5 with permission.
With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

<table>
<thead>
<tr>
<th>Grade*</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Minor contusion without duct injury</td>
<td>863.81/863.84</td>
<td>S36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Superficial laceration without duct injury</td>
<td>S36.1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Major contusion without duct injury or tissue loss</td>
<td>863.81/863.84</td>
<td>S36.1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Major laceration without duct injury or tissue loss</td>
<td>863.81/863.84</td>
<td>S36.1</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Distal transection or parenchymal injury with duct injury</td>
<td>863.92/863.94</td>
<td>S36.1</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Proximal transection or parenchymal injury involving ampulla</td>
<td>863.91</td>
<td>S36.1</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Massive disruption of pancreatic head</td>
<td>863.91</td>
<td>S36.1</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
863.51, 863.91 – head; 863.99,862.92 – body; 863.83, 863.93 – tail.
Proximal pancreas is to the patient’s right of the superior mesenteric vein.
From Moore et al.,6 with permission.
With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

<table>
<thead>
<tr>
<th>Grade*</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Contusion/haematoma</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness laceration</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Laceration &lt; 50% circumference</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>4</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Laceration &gt; 50% circumference</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>Tissue loss</td>
<td>Segmental loss or devascularization &lt; 2 cm</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Tissue loss</td>
<td>Segmental loss or devascularization &gt; 2 cm</td>
<td>862.22/826.32</td>
<td>S10.0/S27.8/S36.8</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple lesions up to grade III.
From Moore et al.,5 with permission.
With ICD-10, cervical oesophagus S10.0; thoracic oesophagus S27.8; abdominal oesophagus S36.8.
With ICD-10 use supplementary character: 0 without open wound into abdominal or thoracic cavity; 1 with open wound into abdominal or thoracic cavity.
Table 12  Stomach injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Contusion/haematoma</td>
<td>863.0</td>
<td>S36.3</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness laceration</td>
<td>863.0</td>
<td>S36.3</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>&lt; 2 cm in gastro-oesophageal junction or pylorus</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 5 cm in proximal third of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 10 cm in distal two-thirds of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>&gt; 2 cm in gastro-oesophageal junction or pylorus</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 5 cm in proximal third of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 10 cm in distal two-thirds of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Tissue loss</td>
<td>Tissue loss or devascularization of less than two-thirds of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Tissue loss</td>
<td>Tissue loss or devascularization of more than two-thirds of stomach</td>
<td>863.0</td>
<td>S36.3</td>
<td>4</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple lesions up to grade III.

From Moore et al.,5 with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

Table 13  Duodenum injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Involving single portion of duodenum</td>
<td>863.2</td>
<td>S36.4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness, no perforation</td>
<td>863.2</td>
<td>S36.4</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Involving more than one portion</td>
<td>863.2</td>
<td>S36.4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Disruption &lt; 50% of circumference</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Disruption 50–75% of circumference of D2</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Disruption 50–100% of circumference of D1, D3, D4</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Disruption &gt; 75% of circumference of D2</td>
<td>863.3</td>
<td>S36.4</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Involving ampulla or distal common bile duct</td>
<td>863.3</td>
<td>S36.4</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Massive disruption of duodenopancreatic complex</td>
<td>863.3</td>
<td>S36.4</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Devascularization of duodenum</td>
<td>863.3</td>
<td>S36.4</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.

D1, first position of duodenum; D2, second portion of duodenum; D3, third portion of duodenum; D4, fourth portion of duodenum.

From Moore et al.,6 with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

Table 14  Small bowel injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Contusion or haematoma without devascularization</td>
<td>863.2</td>
<td>S36.4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness, no perforation</td>
<td>863.2</td>
<td>S36.4</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Laceration &lt; 50% of circumference</td>
<td>863.3</td>
<td>S36.4</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Laceration ≥ 50% of circumference without transection</td>
<td>863.3</td>
<td>S36.4</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Transection of the small bowel</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Transection of the small bowel with segmental tissue loss</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Devascularized segment</td>
<td>863.3</td>
<td>S36.4</td>
<td>4</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.

From Moore et al.,7 with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.
Table 15 Colon injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Contusion or haematoma without devascularization</td>
<td>863.40–863.44</td>
<td>S36.5</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness, no perforation</td>
<td>863.40–863.44</td>
<td>S36.5</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Laceration &lt; 50% of circumference</td>
<td>863.50–863.54</td>
<td>S36.5</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Laceration ≥ 50% of circumference without transection</td>
<td>863.50–863.54</td>
<td>S36.5</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Transection of the colon</td>
<td>863.50–863.54</td>
<td>S36.5</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Transection of the colon with segmental tissue loss</td>
<td>863.50–863.54</td>
<td>S36.5</td>
<td>4</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al., with permission.

With ICD-9 $863.40/863.50 = $non-specific site in colon; $863.41/863.51 = $ascending; $863.42/863.52 = $transverse; $863.43/863.53 = $descending; $863.44/863.54 = $sigmoid.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavities.

Table 16 Rectum injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Contusion or haematoma without devascularization</td>
<td>863.45</td>
<td>S36.6</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial-thickness laceration</td>
<td>863.45</td>
<td>S36.6</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Laceration &lt; 50% of circumference</td>
<td>863.55</td>
<td>S36.6</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Laceration ≥ 50% of circumference</td>
<td>863.55</td>
<td>S36.6</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Full-thickness laceration with extension into the perineum</td>
<td>863.55</td>
<td>S36.6</td>
<td>5</td>
</tr>
<tr>
<td>V</td>
<td>Vascular</td>
<td>Devascularized segment</td>
<td>863.55</td>
<td>S36.6</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

Table 17 Abdominal vascular injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Non-named superior mesenteric artery or superior mesenteric vein branches</td>
<td>902.20/.39</td>
<td>S35.2</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Non-named inferior mesenteric artery or inferior mesenteric vein branches</td>
<td>902.27/.32</td>
<td>S35.2</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Phrenic artery or vein</td>
<td>902.89</td>
<td>S35.8</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Lumbar artery or vein</td>
<td>902.89</td>
<td>S35.8</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Gonadal artery or vein</td>
<td>902.89</td>
<td>S35.8</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Ovarian artery or vein</td>
<td>902.81/902.82</td>
<td>S35.8</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Other non-named small arterial or venous structures requiring ligation</td>
<td>902.80</td>
<td>S35.9</td>
<td>NS</td>
</tr>
<tr>
<td>II</td>
<td>Right, left, or common hepatic artery</td>
<td>902.22</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Splenic artery or vein</td>
<td>902.23/902.34</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Right or left gastric arteries</td>
<td>902.21</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Gastroduodenal artery</td>
<td>902.24</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Inferior mesenteric artery/trunk or inferior mesenteric vein/trunk</td>
<td>902.27/902.32</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Primary named branches of mesenteric artery (e.g. ileocolic artery) or mesenteric vein</td>
<td>902.26/902.31</td>
<td>S35.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Other named abdominal vessels requiring ligation or repair</td>
<td>902.89</td>
<td>S35.8</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Superior mesenteric vein, trunk and primary subdivisions</td>
<td>902.31</td>
<td>S35.3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Renal artery or vein</td>
<td>902.41/902.42</td>
<td>S35.4</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Iliac artery or vein</td>
<td>902.53/902.54</td>
<td>S35.5</td>
<td>3</td>
</tr>
</tbody>
</table>

*continued*
### Adrenal organ injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>868.01/.11</td>
<td>S37.9</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Laceration involving only cortex (&lt; 2 cm)</td>
<td>868.01/.11</td>
<td>S37.8</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Laceration extending into medulla (&gt; 2 cm)</td>
<td>868.01/.11</td>
<td>S37.8</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>&gt; 50% parenchymal destruction</td>
<td>868.01/.11</td>
<td>S37.8</td>
<td>2</td>
</tr>
<tr>
<td>V</td>
<td>Total parenchymal destruction (including massive intraparenchymal haemorrhage)</td>
<td>868.01/.11</td>
<td>S37.8</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Avulsion from blood supply</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral lesions up to grade V.

From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

### Kidney injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Microscopic or gross haematuria, urological studies normal</td>
<td>866.01</td>
<td>$37.0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Haematoma</td>
<td>Subcapsular, non-expanding without parenchymal laceration</td>
<td>866.01</td>
<td>$37.0</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Haematoma</td>
<td>Non-expanding perirenal haematoma confined to renal retroperitoneum</td>
<td>866.01</td>
<td>$37.0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>&lt; 1.0 cm parenchymal depth of renal cortex without urinary extravasation</td>
<td>866.11</td>
<td>$37.0</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>&gt; 1.0 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation</td>
<td>866.11</td>
<td>$37.0</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Parenchymal laceration extending through renal cortex, medulla and collecting system</td>
<td>866.02/866.12</td>
<td>$37.0</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Main renal artery or vein injury with contained haemorrhage</td>
<td>866.03/866.13</td>
<td>$37.0</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Completely shattered kidney</td>
<td>866.04/866.14</td>
<td>$37.0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Avulsion of renal hilum which devascularizes kidney</td>
<td>866.13</td>
<td>$37.0</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III.

From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.
### Table 20 Ureter injury scale

<table>
<thead>
<tr>
<th>Grade&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Contusion or haematoma without devascularization</td>
<td>867.2/867.3</td>
<td>S37.1</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>&lt; 50% transection</td>
<td>867.2/867.3</td>
<td>S37.1</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>≥ 50% transection</td>
<td>867.2/867.3</td>
<td>S37.1</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Complete transection with &lt; 2 cm devascularization</td>
<td>867.2/867.3</td>
<td>S37.1</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Avulsion with &gt; 2 cm of devascularization</td>
<td>867.2/867.3</td>
<td>S37.1</td>
<td>3</td>
</tr>
</tbody>
</table>

<sup>a</sup>Advance one grade for bilateral up to grade III.

From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal cavity; 1 with open wound into abdominal cavity.

### Table 21 Bladder injury scale

<table>
<thead>
<tr>
<th>Grade&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma</td>
<td>Contusion, intramural haematoma</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Partial thickness</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>Laceration</td>
<td>Extraperitoneal bladder wall laceration &lt; 2 cm</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>4</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
<td>Extraperitoneal (≥ 2 cm) or intraperitoneal (&lt; 2 cm) bladder wall laceration</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Intraperitoneal bladder wall laceration ≥ 2 cm</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Intraperitoneal or extraperitoneal bladder wall laceration extending into the bladder neck or ureteral orifice (trigone)</td>
<td>867.0/867.1</td>
<td>S37.2</td>
<td>4</td>
</tr>
</tbody>
</table>

<sup>a</sup>Advance one grade for multiple lesions up to grade III.

From Moore et al.,<sup>1</sup> with permission.

With ICD-10 use supplementary character: 0 without open wound into pelvic cavity; 1 with open wound into pelvic cavity.

### Table 22 Urethra injury scale

<table>
<thead>
<tr>
<th>Grade&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Injury type</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>Blood at urethral meatus; urethrography normal</td>
<td>867.0/867.1</td>
<td>S37.3</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Stretch injury</td>
<td>Elongation of urethra without extravasation on urethrography</td>
<td>867.0/867.1</td>
<td>S37.3</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Partial</td>
<td>Extravasation of urethrography contrast at injury site with visualization in the bladder</td>
<td>867.0/867.1</td>
<td>S37.3</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Complete</td>
<td>Extravasation of urethrography contrast at injury site without visualization in the bladder; &lt; 2 cm of urethra separation</td>
<td>867.0/867.1</td>
<td>S37.3</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Complete</td>
<td>Complete transaction with ≥ 2 cm urethral separation, or extension into the prostate or vagina</td>
<td>867.0/867.1</td>
<td>S37.3</td>
<td>4</td>
</tr>
</tbody>
</table>

<sup>a</sup>Advance one grade for bilateral injuries up to grade III.

From Moore et al.,<sup>1</sup> with permission.

With ICD-10 use supplementary character: 0 without open wound into pelvic cavity; 1 with open wound into pelvic cavity.
Table 23 Uterus (non-pregnant) injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion/haematoma</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Superficial laceration (&lt; 1 cm)</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Deep laceration (≥ 1 cm)</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration involving uterine artery</td>
<td>902.55</td>
<td>S37.6</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Avulsion/devascularization</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>3</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al., with permission.
With ICD-10 use supplementary character: 0 without open wound into pelvic cavity; 1 with open wound into pelvic cavity.

Table 24 Uterus (pregnant) injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion or haematoma (without placental abruption)</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Superficial laceration (&lt; 1 cm) or partial placental abruption &lt; 25%</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>Deep laceration (≥ 1 cm) occurring in second trimester or placental abruption &gt; 25% but &lt; 50%</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Deep laceration (≥ 1 cm) in third trimester</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration involving uterine artery</td>
<td>902.55</td>
<td>S37.6</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>Uterine rupture:</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>• second trimester</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• third trimester</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Complete placental abruption</td>
<td>867.4/867.5</td>
<td>S37.6</td>
<td>4–5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al., with permission.
With ICD-10 use supplementary character: 0 without open wound into pelvic cavity; 1 with open wound into pelvic cavity.

Table 25 Fallopian tube injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma or contusion</td>
<td>867.6/867.7</td>
<td>S37.5</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>Laceration &lt; 50% circumference</td>
<td>867.6/867.7</td>
<td>S37.5</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Laceration ≥ 50% circumference</td>
<td>867.6/867.7</td>
<td>S37.5</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Transection</td>
<td>867.6/867.7</td>
<td>S37.5</td>
<td>2</td>
</tr>
<tr>
<td>V</td>
<td>Vascular injury; devascularized segment</td>
<td>902.89</td>
<td>S37.5</td>
<td>2</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III.
From Moore et al., with permission.
With ICD-10 use supplementary character: 0 without open wound into abdominal or pelvic cavity; 1 with open wound into abdominal pelvic cavity.
### Table 26 Ovary injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion or haematoma</td>
<td>867.6/7.7</td>
<td>S37.4</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Superficial laceration (depth &lt; 0.5 cm)</td>
<td>867.6/7.7</td>
<td>S37.4</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>Deep laceration (depth ≥ 0.5 cm)</td>
<td>867.8/7.7</td>
<td>S37.4</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Partial disruption or blood supply</td>
<td>902.81</td>
<td>S37.4</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Avulsion or complete parenchymal destruction</td>
<td>902.81</td>
<td>S37.4</td>
<td>3</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III.

From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal or pelvic cavity; 1 with open wound into abdominal pelvic cavity.

### Table 27 Vagina injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion or haematoma</td>
<td>922.4</td>
<td>S30.2</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Laceration, superficial (mucosa only)</td>
<td>878.6</td>
<td>S31.4</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Laceration, deep into fat or muscle</td>
<td>878.6</td>
<td>S31.4</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration, complex, into cervix or peritoneum</td>
<td>868.7</td>
<td>S31.4</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Injury into adjacent organs (anus, rectum, urethra, bladder)</td>
<td>878.7</td>
<td>S39.7</td>
<td>3</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III.

From Moore et al., with permission.

With ICD-10 use supplementary character: 0 without open wound into abdominal or pelvic cavity; 1 with open wound into abdominal pelvic cavity.

### Table 28 Vulva injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion or haematoma</td>
<td>922.4</td>
<td>S30.2</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Laceration, superficial (skin only)</td>
<td>878.4</td>
<td>S31.4</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Laceration, deep (into fat or muscle)</td>
<td>878.4</td>
<td>S31.4</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Avulsion: skin, fat or muscle</td>
<td>878.5</td>
<td>S38.2</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Injury into adjacent organs (anus, rectum, urethra, bladder)</td>
<td>878.5</td>
<td>S39.7</td>
<td>3</td>
</tr>
</tbody>
</table>

Advance one grade for multiple injuries up to grade III.

From Moore et al., with permission.

### Table 29 Testis injury scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion/haematoma</td>
<td>911.0–22.4</td>
<td>S30.2</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Subclinical laceration of tunica albuginea</td>
<td>922.4</td>
<td>S31.3</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Laceration of tunica albuginea with &lt; 50% parenchymal loss</td>
<td>878.2</td>
<td>S31.3</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Major laceration of tunica albuginea with ≥ 50% parenchymal loss</td>
<td>878.3</td>
<td>S31.3</td>
<td>2</td>
</tr>
<tr>
<td>V</td>
<td>Total testicular destruction or avulsion</td>
<td>878.3</td>
<td>S38.2</td>
<td>2</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral lesions up to grade V.

From Moore et al., with permission.
<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
<td>922.4</td>
<td>S30.2</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Laceration &lt; 25% of scrotal diameter</td>
<td>878.2</td>
<td>S31.2</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Laceration &gt; 25% of scrotal diameter</td>
<td>878.3</td>
<td>S31.3</td>
<td>2</td>
</tr>
<tr>
<td>IV</td>
<td>Avulsion &lt; 50%</td>
<td>878.3</td>
<td>S38.2</td>
<td>2</td>
</tr>
<tr>
<td>V</td>
<td>Avulsion &gt; 50%</td>
<td>878.3</td>
<td>S38.2</td>
<td>2</td>
</tr>
</tbody>
</table>

From Moore et al.,1 with permission.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Cutaneous laceration/contusion</td>
<td>911.0/922.4</td>
<td>S30.2/31/2</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Buck's fascia (cavernosum) laceration without tissue loss</td>
<td>878.0</td>
<td>S37.8</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>Cutaneous avulsion Laceration through glans/meatus Cavernosal or urethral defect &lt; 2 cm</td>
<td>878.1</td>
<td>S38.2</td>
<td>3</td>
</tr>
<tr>
<td>IV</td>
<td>Partial penectomy Cavernosal or urethral defect ≥ 2 cm</td>
<td>878.1</td>
<td>S38.2</td>
<td>3</td>
</tr>
<tr>
<td>V</td>
<td>Total penectomy</td>
<td>876.1</td>
<td>S38.2</td>
<td>3</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
From Moore et al.,1 with permission.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of injury</th>
<th>ICD-9</th>
<th>ICD-10</th>
<th>AIS-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Digital artery/vein</td>
<td>903.5</td>
<td>S65.5</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Palmar artery/vein</td>
<td>903.4</td>
<td>S65.3</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Deep palmar artery/vein</td>
<td>904.6</td>
<td>S65.3</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Dorsalis pedis artery</td>
<td>904.7</td>
<td>S95.0</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Plantar artery/vein</td>
<td>904.5</td>
<td>S95.1</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Non-named arterial/venous branches</td>
<td>903.8/904.7</td>
<td>S55.9/S85.9</td>
<td>1–3</td>
</tr>
<tr>
<td>II</td>
<td>Basilic/cephalic vein</td>
<td>903.8</td>
<td>S45.8/S55.8</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Saphenous vein</td>
<td>904.3</td>
<td>S75.2</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Radial artery</td>
<td>903.2</td>
<td>S55.1</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Ulnar artery</td>
<td>903.3</td>
<td>S55.0</td>
<td>1–3</td>
</tr>
<tr>
<td>III</td>
<td>Axillary vein</td>
<td>903.02</td>
<td>S45.1</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Superficial/deep femoral vein</td>
<td>903.02</td>
<td>S75.1</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Popliteal vein</td>
<td>904.42</td>
<td>S85.5</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Brachial artery</td>
<td>903.1</td>
<td>S45.1</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Anterior tibial artery</td>
<td>904.51/904.52</td>
<td>S85.1</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Posterior tibial artery</td>
<td>904.53/904.54</td>
<td>S85.1</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Peroneal artery</td>
<td>904.7</td>
<td>S85.2</td>
<td>1–3</td>
</tr>
<tr>
<td></td>
<td>Tibioperoneal trunk</td>
<td>904.7</td>
<td>S85.2</td>
<td>2–3</td>
</tr>
<tr>
<td>IV</td>
<td>Superficial/deep femoral artery</td>
<td>904.1/904.7</td>
<td>S75.0</td>
<td>3–4</td>
</tr>
<tr>
<td></td>
<td>Popliteal artery</td>
<td>904.41</td>
<td>S85.0</td>
<td>2–3</td>
</tr>
<tr>
<td>V</td>
<td>Axillary artery</td>
<td>903.01</td>
<td>S45.0</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td>Common femoral artery</td>
<td>904.0</td>
<td>S75.0</td>
<td>3–4</td>
</tr>
</tbody>
</table>

*Increase one grade for multiple grade III or IV injuries involving > 50 per cent vessel circumference. Decrease one grade for < 25 per cent vessel circumference disruption for grade IV or V.
From Moore et al.,1 with permission.
B.6.1 References


Injury (trauma) remains a major healthcare problem throughout the world. In addition to improving awareness of trauma prevention and management, improved application of surgical skills is expected to save further lives and contribute to minimizing disability. It is widely recognized that training of surgeons in the management of trauma is substantially deficient because of:

- limited exposure within individual training programmes to the types of patients required to develop the appropriate level of skills
- traditional trauma surgical training that has been organ specific.

Consequently surgeons can finish training with suboptimal skills in this field where there is often little time to contemplate an appropriate course of action.

Through the early 1990s, it became apparent to a number of surgeons familiar with trauma management around the world that there was a specific need for surgical training in the technical aspects of operative care of the trauma patient, with particular emphasis on those who were close to, or had recently completed, their training. This course had its origins in a meeting of Howard Champion (USA), David Mulder (Canada), Donald Trunkey (USA), Stephen Deane (Australia) and Abe Fingerhut (France) in October 1993.

This postgraduate surgical course, developed in collaboration with professional educators, assumes competence with assessment and resuscitative measures that have become standardized through the ATLS® (Advanced Trauma Life Support) Course of the American College of Surgeons. It draws on the specialist surgical training of all course participants and reviews, strengthens and organizes the performance of established and new procedures specially required in trauma surgery. It is expected that the course will have special relevance for surgeons in countries where major trauma rates are high and to rapidly mobilized medical units in areas of conflict. It is also likely to be valuable in developing countries where education and physical resources are limited.

### C.2 COURSE DEVELOPMENT AND TESTING

There have been many attempts to test the concept:

- Dr Fingerhut’s laparoscopic trauma surgical training course, which he had run for two previous years at the European Surgical Education Centre in Paris, was modified to incorporate these concepts. This 2-day course consisted of didactic sessions in the morning and animal laboratory sessions in the afternoon. Evaluation responses were excellent.
- The Uniformed Services University of Health Sciences, Bethesda, USA started a similar course in August 1994. Dr Don Jenkins has now put over 100 military surgeons through the course in the USA. He is working on bringing the course to Chile at present.
- There was a Swedish Trauma Surgery Course that Drs Trunkey, Fingerhut and Champion attended in Sweden in November 1994. This was run by Dr Sten Lennquist. The course was 4 days of didactic teaching and one day of practical work.
• In Sydney in May 1996 a very successful pilot course was organized at Prince Henry Hospital. The international faculty at that course included Don Trunkey, Abe Fingerhut and Howard Champion. The course was a tremendous success and successful courses have been held worldwide.
• From 1999, following courses in Australia, Austria and South Africa, a standardized manual and slide set were developed.

C.3 COURSE DETAILS

C3.1 Ownership

The DSTC™ Course is a Registered Trademark of the International Association for Surgery and Intensive Care (IATSIC). IATSIC is an Integrated Society of the International Society of Surgery/Société Internationale de Chirurgie (ISS–SIC) based in Pratteln, Switzerland.

Only courses recognized by IATSIC may be called DSTC™ Courses.

C3.2 Mission statement

The DSTC™ Course is designed to train participants in the techniques required for the surgical care of the trauma patient. This is done by a combination of lectures, demonstrations, case discussions and practical sessions, utilizing animal and human (cadaver or prosected) tissue if available.

C3.3 Application to hold a course

Application can be made to IATSIC for recognition of a course. Provided that the minimum requirements for the course have been met, as laid down below, IATSIC will recognize the course, which will then be entitled to be called a DSTC™ Course, and carry the IATSIC logo. The Course to be presented will be the course prescribed by IATSIC, and no changes may be made to the course material or syllabus.

C3.4 Eligibility to present

C3.4.1 LOCAL ORGANIZATIONS

The DSTC™ Course can be presented by any tertiary academic institution or recognized surgical organization.

C3.4.2 NATIONAL ORGANIZATIONS

National organizations can present the course in their own country on behalf of IATSIC. A Memorandum of Understanding (MOU) will be signed with IATSIC. Following the presentation of the first two courses, the national organization shall have the right to modify the course to enhance its relevance to local conditions.

C3.5 Course materials and overview

The course is over 2–3 days with the following course materials:
• The content of the course will, as a minimum, contain the core curriculum, as laid down in the IATSIC DSTC™ manual (see Appendix D). Additional material and modules may be included at the discretion of the local organizers, provided that such material is not in conflict with the core curriculum.
• Additional 'add-on' modules may be presented at the discretion of the local organizers.
• The course will use a specific set of slides and the DSTC™ Course manual.

IATSIC is able to furnish the IATSIC DSTC™ Course Manual, and course materials (including slides on PowerPoint) if requested, at a substantial discount. However, provided that the minimum core syllabus is adhered to, a local course manual and material can be used.

C3.6 Course director

In addition to the requirements below, the Course Director must be a full, current member of IATSIC.
• For an inaugural course, the Course Director will be a member of the IATSIC Executive Committee.

C3.7 Course faculty

• Course Faculty will be divided into:
  – Local Faculty
  – International Faculty
  – Guest Lecturers.
• Course Faculty members must have themselves attended a DSTC™ Course.
• Course Faculty members must have completed an ATLS® Instructor Course, Royal College of Surgeons’ ‘Train the Trainers’ Course or an equivalent Instructor training course.
• Course International Faculty must be members of IATSIC.
• Additional Guest Lecturers, with particular expertise in a subject, are permitted.
• Details of all Faculty with confirmation of the above must be lodged with IATSIC before the start of the Course.
• The recommended student/instructor ratio should ideally be 4/1, but may not be larger than 6/1, not including the Course Director.

C3.8 Course participants

• All course participants must be licensed medical practitioners.
• Attendance at the entire course is mandatory.
• The level of applicants can be decided locally, provided that the participants are licensed medical practitioners, and are actively involved in the surgical decision-making and surgery of the trauma patient.
• An entrance examination can be used if needed. An exit examination is not mandatory.

C3.9 Practical skill stations

Practical skills may take place on different material, depending on local constraints. The practical component of the course must include an animal laboratory. However, the use of cadavers is optional and dependent on local conditions. Full local ethical committee certificates of approval for all animal and other tissue work, and any other legal necessary approvals, must be obtained and must be submitted to IATSIC before a course can be approved or held.

C3.10 Course syllabus

In order for IATSIC to recognize the course as a valid DSTC™ Course, the Course must meet or exceed the minimum requirements of the core curriculum. The core curriculum and ‘modules’ are contained in this manual and the course consists of:
• Core knowledge
• Surgical skills (see Appendix D)
• Additional ‘modules’ that may be added as required, at the discretion of the local organizing committee and as required for local needs.

C3.11 Course certification

• Participants are required to attend the entire course.
• Certification of attendance and completion of the course can be issued.
• The certificates of the courses will be numbered.
• Details of the course, final Faculty, participants and a course evaluation must be submitted to IATSIC after the course.

C.4 IATSIC RECOGNITION

Application for recognition of individual courses should be made to IATSIC. IATSIC-recognized courses may carry the endorsement logos of IATSIC and the International Society of Surgery, and will be entitled to be called DSTC™ Courses.

The DSTC™ Course is the intellectual property and a registered trademark of the IATSIC, which is an Integrated Society of the International Society of Surgery (ISS) based in Pratteln, Switzerland. Although it may carry the endorsement (support) of other bodies, this does not imply that other organizations may operate or control the DSTC™ Course in any way.

The DSTC™ Course is designed to train medical practitioners in the techniques required for the definitive surgical care of the trauma patient. This is done by a combination of lectures, demonstrations, case discussions and practical sessions.

The registration and control of the DSTC™ Courses will be controlled by the DSTC™ Sub-committee on behalf of IATSIC. Although it is desirable that national courses be controlled by a national organization, there will be no restriction on local courses provided that international DSTC™ criteria are met. Application to hold a course must be made through IATSIC.

Only courses recognized by IATSIC may be called DSTC™ Courses.

C.5 COURSE INFORMATION

Obtainable from IATSIC via the International Society of Surgery: iatsic@iss-sic.ch.
Appendix D

DSTC™ course – core surgical skills

D.1 THE NECK

1 Standard neck (pre-sternomastoid) incision
2 Control and repair of carotid vessels
   2.1 Zone II
   2.2 Extension into zone III
   2.3 Division of digastric muscle and subluxation or division of mandible
   2.4 Extension into zone I
3 Extension by supraclavicular incision
   3.1 Ligation of proximal internal carotid artery
   3.2 Repair with divided external carotid artery
4 Access to, control of and ligation of internal jugular vein
5 Access to and repair of the trachea
6 Access to and repair of the cervical oesophagus
7 Lung wounds
   5.1 Oversewing
   5.2 Stapling
   5.3 Partial lung resection
   5.4 Tractectomy
   5.5 Lobectomy
8 Access to, and repair of, the thoracic oesophagus
9 Access to, and repair of, the diaphragm
10 Compression of the left subclavian vessels from below
11 Left anterior thoracotomy
12 Access to, and repair of, the thoracic oesophagus

D.2 THE CHEST

1 Incisions
   1.1 Anterolateral thoracotomy
   1.2 Sternotomy
2 Thoracotomy
   2.1 Exploration of thorax
   2.2 Ligation of intercostal and internal mammary vessels
   2.3 Emergency department (resuscitative) thoracotomy
      2.3.1 Supradiaphragmatic control of the aorta
      2.3.2 Control of the pulmonary hilum
      2.3.3 Internal cardiac massage
3 Pericardiotomy
   3.1 Preservation of phrenic nerve
   3.2 Access to the pulmonary veins
4 Access to and repair of the thoracic aorta
5 Access to, and repair of, the thoracic oesophagus
6 Access to, and repair of, the diaphragm
7 Compression of the left subclavian vessels from below
8 Left anterior thoracotomy
9 Access to, and repair of, the thoracic oesophagus
10 Heart repair
   10.1 Finger control
   10.2 Involvement of coronary vessels
11 Insertion of shunt

D.3 THE ABDOMINAL CAVITY

1 Midline laparotomy
   1.1 How to explore (priorities)
   1.2 Packing
   1.3 Localization of retroperitoneal haematomas – when to explore?
   1.4 Damage control
      1.4.1 Skin closure
   1.5 Extension of laparotomy incision
      1.5.1 Lateral extension
      1.5.2 Sternotomy
   1.6 Cross-clamping of the aorta at diaphragm
      (division at left crus)
2 Left visceral medial rotation
3 Right visceral medial rotation
4 Abdominal oesophagus
   4.1 Mobilization
4.2 Repair
   4.2.1 Simple
   4.2.2 Mobilization of fundus to reinforce sutures

5 Stomach
   5.1 Mobilization
   5.2 Access to vascular control
   5.3 Repair of anterior and posterior wounds
   5.4 Pyloric exclusion
   5.5 Distal gastrectomy

6 Bowel
   6.1 Resection
   6.2 Small and large bowel anastomosis
   6.3 Staple colostomy
   6.4 Collagen fleece technique of anastomosis protection
   6.5 Ileostomy technique

D.4 THE LIVER

1 Mobilization (falciform, suspensory, triangular and coronary ligaments)
2 Liver packing
3 Hepatic isolation
   3.1 Control of infrahepatic inferior vena cava
   3.2 Control of suprahepatic superior vena cava
   3.3 Pringle's manoeuvre
4 Repair of parenchymal laceration
5 Technique of finger fracture
6 Tractotomy
7 Packing for injury to hepatic veins
8 Hepatic resection
9 Non-anatomical partial resection
10 Use of tissue adhesives
11 Tamponade for penetrating injury (Foley/Penrose drains/Sengstaken tube)

D.5 THE SPLEEN

1 Mobilization
2 Suture
3 Mesh wrap
4 Use of tissue adhesives
5 Partial splenectomy
   5.1 Sutures
   5.2 Staples
6 Total splenectomy

D.6 THE PANCREAS

1 Mobilization of the tail of the pancreas
2 Mobilization of the head of the pancreas
3 Localization of the main duct and its repair
4 Distal pancreatic resection
   4.1 Stapler
   4.2 Oversewing
5 Use of tissue adhesives
6 Diverticulization
7 Access to mesenteric vessels (division of pancreas)

D.7 THE DUODENUM

1 Mobilization of the duodenum
   1.1 Kocher's manoeuvre (rotation of duodenum)
   1.2 Cattel's anabranch manoeuvre (medial visceral rotation)
   1.3 Division of ligament of Treitz
   1.4 Repair of duodenum

D.8 GENITOURINARY SYSTEM

1 Kidney
   1.1 Mobilization
   1.2 Vascular control
   1.3 Repair
   1.4 Partial nephrectomy
   1.5 Nephrectomy
2 Ureter
   2.1 Mobilization
   2.2 Stenting
   2.3 Repair
3 Bladder
   3.1 Repair of intraperitoneal rupture
   3.2 Repair of extraperitoneal rupture

D.9 ABDOMINAL VASCULAR INJURIES

1 Exposure and control
   1.1 Aorta
      1.1.1 Exposure
      1.1.2 Repair
   1.2 Inferior vena cava (IVC)
      1.2.1 Suprahepatic IVC
      1.2.2 Infrahepatic IVC
1.2.3 Control of haemorrhage with swabs
1.2.4 Repair through anterior wound

2 Pelvis
2.1 Control of pelvic vessels
   2.1.1 Packing
   2.1.2 Suture of artery and vein
   2.1.3 Ligation of artery and vein
   2.1.4 Packing/anchor ligation of sacral vessels

D.10 PERIPHERAL VASCULAR INJURIES

1 Extremities: vascular access
   1.1 Axillary
   1.2 Brachial
   1.3 Femoral
   1.4 Popliteal

2 Fasciotomy
   2.1 Upper limb
   2.2 Lower limb
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