

Manual of Definitive Surgical Trauma Care



Edited by
Kenneth D. Boffard



IATSIC

International
Association for
the Surgery
of Trauma
and Surgical
Intensive Care

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Kenneth D Boffard

**International Association for the Surgery
of Trauma and Surgical Intensive Care**

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Preface

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 aspects can be particularly challenging, and may be infrequently required, yet rapid access to and control of sites of haemorrhage following trauma can constitute 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 needed.

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 responses required in the management of the injured patient.

Planning the response requires an understanding of:

- 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 inherent 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 Société Internationale de

Chirurgie (SIC) and the International Association for the Surgery of Trauma and Surgical Intensive Care (IATSIC), met in San Francisco during the Meeting of the American College of Surgeons. It was apparent that there was a specific need for further surgical training in the technical aspects of the surgical care of the trauma patient, and that routine surgical training was too organ specific or area specific to allow the development of the appropriate judgement and decision-making skills required for traumatized patients with multiple injuries. Particular attention needed to be directed towards those who were senior trainees or had completed their training.

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

The curriculum has been refined and the course has been adapted to the needs of host countries. However, the concept of the DSTC™ Course has remained the same. Courses have been delivered in Austria, Australia, Greece, the Netherlands, Scandinavia, South Africa, Turkey, the UK and the Yemen.

At International Surgical Week in Vienna in 1999, IATSIC's members approved a core curriculum, which forms the basis of the DSTC™ Course. DSTC™ will be produced under the supervision of IATSIC, and may be presented wherever there is a need.

This manual covers the core curriculum and forms the basis for the DSTC™ Course. It is designed to support those who, whether through choice or necessity, must deal with major surgical injury.

The manual is divided along the following lines.

- The theory of injury:
 - detailed physiology of injury,
 - resuscitation controversies and endpoints,
 - critical care controversies and endpoints.
- Surgical decision making.
- Individual organ systems:
 - theoretical background (with references),
 - surgical access to organs and organ systems (with selected readings),
 - skills required for the DSTC™ Course.
- A list of optional modules (overview and selected readings):
 - overview of additional modules that can be added to the core curriculum to enhance the

course and tailor it to the particular needs of the country in which it is being presented.

- Appendices:
 - trauma systems,
 - trauma scoring systems and injury scaling,
 - an overview of the DSTC™ Course,
 - the surgical skills taught on the DSTC™ Course.

An Editorial Board, made up of those who had taught the DSTC™ Course, was formed. I would like to thank them for their very great efforts put into the preparation of this manual, its editing, dissection, re-dissection and assembly.

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

Ken Boffard, Editor
Johannesburg
South Africa

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 'super-specialties' 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 (DSTC™) COURSE

The ATLS® programme is probably the most widely accepted trauma programme in the world, with over 37 national programmes taking place at present.

Yuen reports that over 180 physicians in Hong Kong have been trained in ATLS®.¹

Kobayoshi states that many of the surgeons in Japan have a high standard of surgical skills before entering traumatology, but that 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-operatively, resulting in insufficient operative exposure for the training of young trauma surgeons.²

Barach reported that the ATLS® was introduced to Israel in 1990, and that more than 4000 physicians have been trained. In 1994, the Israeli Medical Association Scientific Board accredited the ATLS®

programme and mandated that all surgery residents become ATLS® certified.³ There is also a combat trauma life support course for active military doctors.⁴

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.⁵

Arreola-Risa 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.⁶

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.⁷

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. Whereas prevention of injury will undoubtedly play one of the major roles in the reduction of mortality and morbidity due to 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 – in the period *after* ATLS® is complete.

In the United States, trauma affects both the young and the elderly. It is the third leading cause of death for all ages and the leading cause of death from age 1 to 44.⁸ Persons under the age of 45

account for 61 per cent of all injury fatalities and for 65 per cent of hospital admissions. However, persons 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 due to massive haemorrhage or neurological injury. Autopsy data have demonstrated that central nervous system injuries account for 40–50 per cent of all injury deaths and that haemorrhage accounts for 30–35 per cent. Motor vehicles and firearms accounted for 29 per cent and 24 per cent of all injury deaths in 1995, respectively.^{9,10}

In South Africa, there is a high murder rate (66 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 caused by motor vehicles, falls, recreational pursuits and affecting the elderly. The relatively limited exposure of surgeons to major trauma is mandating a requirement for designated trauma hospitals and for specific skill development in the management of major trauma.

Further, there are multiple areas of the developing world – in the West Indies, South America and Africa – where general surgical training may not necessarily include extensive operative education and psychomotor technical expertise in 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 definitively control thoracic haemorrhage may not have had the required techniques incorporated into formal surgical training. Various techniques for the 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 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 to 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, are 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 for 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 pre-requisite of the 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 2-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 an overview of best access to organ systems.
- *Cadaver sessions*: use is made of fresh or preserved human cadavers and dissected tissue. These sessions are used to reinforce the vital knowledge of human anatomy related to access in major trauma.
- *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 to improve psychomotor skills and 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. Other alternatives are available if local custom or legislation does not permit the use of such laboratories.
- *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 dealing with life-threatening penetrating and blunt injury, irrespective of whether it is in the military or civilian arena.

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Part I

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 ebb and flow phases 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.

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.

In the flow phase, the body is hypermetabolic, cardiac output and oxygen consumption are increased, and there is increased glucose production. Lactic acid may be normal.

2.1.2 Initiating factors

The magnitude of the metabolic response depends on the degree of trauma and the concomitant contributory factors such as drugs, sepsis and underlying systematic 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 the elderly and in premenopausal women. It is also modified by starvation and nutritional depletion: patients with poor nutritional status have a reduced metabolic response to trauma compared to 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, it is critical to try 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 be broadly divided as follows.

- Hypovolaemia:
 - decrease in circulating volume of blood,
 - increase in alimentary loss of fluid,
 - loss of interstitial volume,
 - extracellular fluid shift.
- Afferent impulses:
 - somatic,
 - autonomic.

- Wound factors – inflammatory and cellular:
 - eicosanoids,
 - prostanoids,
 - leucotrienes,
 - macrophages,
 - interleukin-1 (IL-1),
 - proteolysis-inducing factor (PIF).
- Toxins/sepsis:
 - endotoxins,
 - exotoxins.
- Oxygen free radicals.

2.1.2.1 HYPOVOLAEMIA

It is said that hypovolaemia, specifically involving tissue hypoperfusion, is the most potent precipitator of the metabolic response. Hypovolaemia can also be due to 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. Class III or Class IV shock is severe and, unless treated as a matter of urgency, will make the situation much worse (see Section 2.2).

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

2.1.2.2 AFFERENT IMPULSES

Hormonal responses are initiated by pain and anxiety. The metabolic response may be modified by the 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.

2.1.2.3 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 humoral and cell-derived activation products play a role in the pathophysiology of organ dysfunction.⁴ 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.

Eicosanoids

These compounds, derived from eicosapolyenoic fatty acids, comprise the prostanoids and leucotrienes. Eicosanoids are synthesized from arachidonic acid, which itself has been synthesized from phospholipids of damaged cell walls, white blood cells and platelets by the action of phospholipase A₂. The leucotrienes and prostanoids derived from the arachidonic acid cascade play an important role.

Prostanoids

Cyclo-oxygenase converts arachidonic acid to prostanoids, the precursors of prostaglandin (PG), prostacyclins (PGI) and thromboxanes (TX). The term prostaglandins is used loosely to include all prostanoids.

The prostanoids (prostaglandins of the E and F series) PGI₂ and TX synthesized from arachidonic acid by cyclo-oxygenase (in TXA₂), endothelial cells, white cells, and platelets not only cause vasoconstriction (TXA₂ and PGF₁), but also vasodilatation (PGI₂, PGE₁ and PGE₂). TXA₂ 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.

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 the polymorphonuclear leucocytes and the macrophages. Normal phagocytosis commences with chemotaxis, which is the primary activation of the metabolic response, via the activation of complement.⁵

The classical pathway of complement activation involves an interaction between the initial antibody and the initial trimer of complement components C₁, C₄ and C₂. In the classical pathway, this interaction then cleaves the complement products C₃ and C₅ via proteolysis to produce the very powerful chemotactic factors C_{3a} and C_{5a}.

The so-called alternative pathway seems to be the main route following trauma. It is activated by properdin and proteins D or B to activate C₃ convertase, which generates the anaphylotoxins C_{3a} and C_{5a}. 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).⁶ Measurement of C_{3a} is the most useful because the other products are more rapidly cleared from the circulation. The C_{3a}:C₃ ratio has been shown to correlate positively with outcome in patients after septic shock.⁷

The short-lived fragments of the complement cascade, C_{3a} and C_{5a}, stimulate macrophages to secrete IL-1 and its active circulating cleavage product PIF. These cause proteolysis and lipolysis with fever. IL-1 activates T₄ helper cells to produce IL-2, which enhances cell-mediated immunity. IL-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, α 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 bradikinin from

bradikininogen, 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. It causes vascular margination and sequestration of leucocytes, 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 tumour necrosis factor (TNF) in the macrophages.

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

2.1.3.4 OXYGEN FREE RADICALS

Oxygen radical formation by white cells is a normal host defence mechanism. Changes after injury may lead to excessive production of oxygen free radicals, with deleterious effects on organ function.

2.1.4 Hormonal mediators

During trauma, several hormones are altered. Adrenaline, noradrenaline, cortisol and glucagon are increased, and 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 due to adrenergic and catecholamine effects, and catecholamines are increased after injury.⁸

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. Adrenocorticotrophic 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, β -endorphin and pro-opiomelanocortin (precursor of the ACTH molecule), which modifies the response to pain and reinforces the catecholamine effects. The β -endorphin has little effect, but serves as a marker for anterior pituitary secretion.

Hypotension, hypovolaemia in the form of a decrease in left ventricular pressure, and hyponatraemia stimulate secretion of vasopressin, antidiuretic hormone (ADH) from the supra-optic nuclei in the anterior hypothalamus, aldosterone from the adrenal cortex, and renin from the juxtaglomerular apparatus of the kidney.

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. ACTH stimulates the adrenal cortex to secrete cortisol and aldosterone. Changes in glucose concentration influence the release of insulin from the β cells of the pancreas, and high amino-acid levels influence 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 following 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.

In 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 following injury, primarily stimulated by pain, fear and baroreceptor stimulation.

2.1.4.3 PANCREATIC HORMONES

There is a rise in the blood sugar following trauma. The insulin response to glucose in normal individuals is reduced substantially with alpha-adrenergic stimulation, and enhanced with beta-adrenergic stimulation.⁹

2.1.4.4 RENAL HORMONES

Aldosterone secretion is increased by several mechanisms. The renin-angiotensin mechanism is the most important. When the glomerular arteriolar inflow pressure falls, the juxtaglomerular apparatus of the kidney secretes renin, which acts with angiotensinogen to form angiotensin I. This is converted to angiotensin II, a substance that stimulates production of aldosterone by the adrenal cortex. Reduction in sodium concentration stimulates the macula densa (a specialized area in the tubular epithelium adjacent to the juxtaglomerular apparatus) to activate renin release. An increase in plasma potassium concentration also stimulates aldosterone release. Volume 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.¹⁰ ANF 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.

Prior to the discovery of ANF, it was suggested that a hormone – a third factor – was secreted following 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. ANF has also emphasized the heart's function as an endocrine organ. ANF 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

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

The cardiac index may exceed $4.5 \text{ L min}^{-1} \text{ m}^{-2}$ 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 $2.5 \text{ L min}^{-1} \text{ m}^{-2}$, oxygen consumption may fall to values of less than $100 \text{ mL min}^{-1} \text{ m}^{-2}$ (normal = $120\text{--}160 \text{ mL min}^{-1} \text{ m}^{-2}$). Endotoxins and anoxia may injure cells and limit their ability to utilize oxygen for oxidative phosphorylation.

The amount of adenosine triphosphate (ATP) synthesized by an adult is considerable. However, there is no reservoir of ATP or creatinine phosphate and therefore cellular injury and lack of oxygen result in rapid deterioration of processes requiring energy, and lactate is produced. Because of anaerobic glycolysis, only two ATP equivalents instead of 34 are produced from 1 mol of glucose in the Krebs' cycle.

Lactate is formed from pyruvate, which is the end product of glycolysis. It is normally reconverted to glucose in the Cori cycle in the liver. However, in shock, the oxidation reduction (redox) potential declines and conversion of pyruvate to acetyl co-enzyme A for entry into the Krebs' cycle is inhibited. Lactate therefore accumulates because of impaired hepatic gluconeogenesis, causing a severe metabolic acidosis.

A persistent lactic acidosis in the first 3 days after injury not only correlates well with the ISS, but also confirms the predictive value of lactic acidosis in subsequent adult respiratory distress syndrome (ARDS).¹¹

Accompanying the above changes is an increase in oxygen delivery to the microcirculation. Total body oxygen consumption (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 supra-optic nuclei in the anterior hypothalamus is stimulated by volume reduction and increased osmolality. The latter is due mainly to 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. ADH acts mainly on the connecting tubules of the kidney, but also on the distal tubules to promote reabsorption of water.

Aldosterone acts mainly on the distal renal tubules to promote reabsorption of sodium and bicarbonate and increased excretion of potassium and hydrogen ions. Aldosterone also modifies the effects of catecholamines on cells, thus affecting the exchange of sodium and potassium across all cell membranes. The release of large quantities of intracellular potassium into the extracellular fluid may cause a significant rise in serum potassium, especially if renal function is impaired. Retention of sodium and bicarbonate may produce metabolic alkalosis, with impairment of the delivery of oxygen to the tissues. After injury, urinary sodium excretion may fall to $10\text{--}25 \text{ mmol/24 h}$ and potassium excretion may rise to $100\text{--}200 \text{ mmol/24 h}$.

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 as a result of both increased mobilization and decreased uptake of glucose by the tissues.¹² 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 only 12–18 hours. 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 β cells of the pancreas. Thereafter, gluconeogenesis is stimulated by corticosteroids and glucagon. The suppressed insulin favours the release of amino acids from muscle, and these are

then available for gluconeogenesis. Growth hormone inhibits the effect of insulin on glucose metabolism.

Thyroxine also accelerates gluconeogenesis, but T_3 and 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 characterize the catabolic phase of the metabolic response to trauma.

The glucose level following trauma should be carefully monitored. 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 following 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 following trauma is adipose tissue. Lipids stored as triglycerides in adipose tissue are mobilized when insulin falls below 25 units/mL. Because of the suppression of insulin release by the catecholamine response after trauma, as much as 200–500 g of fat may be broken down daily after severe trauma.¹³ TNF and possibly IL-1 play a role in the mobilization of fat stores.

Catecholamines and glucagon activate adenyl cyclase in the fat cells to produce cyclic adenosine 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 tissues except the blood cells and the central nervous system. Ketones are water soluble and will pass the blood–brain barrier freely, permitting rapid central nervous system adaptation to ketone oxidation.

Free fatty acids provide energy for all tissues and for hepatic gluconeogenesis. Canitine, 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 $\text{kg}^{-1} \text{ day}^{-1}$. This is equivalent to 13–20 g of nitrogen per day. In the absence of an exogenous source of protein, amino acids are principally derived from the breakdown of skeletal muscle protein. Following trauma or sepsis, the release rate of amino acids increases by three to four times. 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 utilized 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 the maintenance of cellular function.

Certain amino acids, such as glutamic acid, asparagine and aspartate, can be oxidized to pyruvate, producing alanine, or to α -ketoglutarate, producing glutamine. The others must first be deaminated before they can be utilized. 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. Since 1 g of urea nitrogen is derived from 6.25 g degraded amino acids, this protein wastage is up to 125 g/day.

One gram of muscle protein represents 5 g wet muscle mass. The patient in this example would be losing 625 g of muscle mass per day. A loss of 40 per cent of body protein is usually fatal, because failing immunocompetence leads to overwhelming infection. Cuthbertson³ showed that nitrogen excretion and hypermetabolism peak 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.¹⁴ 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 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 due to 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 multisystem failure after severe trauma.¹⁵ The extent of bacterial translocation in trauma has not been defined.¹⁶ 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 hours) 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 that contribute to anabolism are growth hormones, androgens and 17-ketosteroids. The utility of growth hormone and also, more recently, of insulin-like growth factor (IGF-1) in reversing catabolism following injury is critically dependent on adequate caloric intake.¹⁷

2.1.7 Clinical and therapeutic relevance

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

Hypovolaemia due to 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 factors 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.

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2.2 SHOCK

2.2.1 Definition

Shock is defined as inadequate circulation of blood to the tissues, resulting in cellular hypoxia. This at first leads to reversible ischaemic-induced cellular injury. If the process is sufficiently severe or protracted, it ultimately results in irreversible cellular and organ injury and dysfunction. The precise mechanisms responsible for the transition from reversible to irreversible injury and the 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

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 are recognized, based on a distinction not only in the pathophysiology but also in the management of the patients:

- 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 $\times$ heart rate,
- blood pressure $\propto$ cardiac output $\times$ total peripheral resistance,
- stroke volume is determined by the preload and the contractility of the myocardium, as well as by the afterload.

2.2.2.1 HYPOVOLAEMIC SHOCK

Hypovolaemic shock is caused by a decrease in the intravascular volume. 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 (Table 2.1).

Initially, the body compensates for shock, and Class I and Class II shock is 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 vaso-

constriction, sweating, cyanosis, hyperventilation, confusion and an oliguria. Cardiac function can be depressed without gross clinical haemodynamic manifestations. The heart shares in the total body ischaemic insult. Systemic arterial hypotension increases coronary ischaemia, causing rhythm disturbances and decreased myocardial performance. As the heart fails, left ventricular end-diastolic pressure rises, ultimately causing pulmonary oedema.

Hyperventilation may maintain arterial PaO_2 at near-normal levels, but the $PaCO_2$ falls to 20–30 mmHg (2.7–4 kPa). Later, pulmonary insufficiency may supervene from alveolar collapse and pulmonary oedema, resulting from damaged pulmonary capillaries, cardiac failure or inappropriate fluid therapy.

Renal function is also critically dependent on renal perfusion. Oliguria is an inevitable feature of hypovolaemia. During volume loss, renal blood flow falls correspondingly with the blood pressure. Anuria sets in when the systolic blood pressure falls to 50 mmHg. Urine output is a good indicator of peripheral perfusion.

2.2.2.2 CARDIOGENIC SHOCK

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

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, myocardial lesions from high circulatory levels of catecholamines, angiotensin and possibly a myocardial depressant factor.

The reduced cardiac output can be a result of:

Table 2.1 Physiological effects of classes of shock based on ATLS® principles

Class	Blood loss (%)	Volume (mL)	Pulse rate (beats/min)	Blood pressure	Pulse pressure	Respiration rate (breaths/min)
Class I	15	<750	<100	Normal	Normal	14–20
Class II	30	750–1500	>100	Normal	Increased	20–30
Class III	40	2000	>120	Decreased	Narrowed	30–40
Class IV	>40	>2000	>140	Decreased	Narrowed	>35

- 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 clinical examples in which the patient may have a nearly normal resting cardiac output but cannot raise the cardiac output under circumstances of stress, because of poor myocardial reserves or an inability to mobilize those myocardial reserves due to pharmacologic β -adrenergic blockade, for example propranolol 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 X-rays.

The 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 less than 90 mmHg, decreased cardiac output (usually less than $1.8 \text{ L m}^{-2} \text{ min}^{-1}$), and a pulmonary arterial wedge pressure (PAWP) of greater than 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 at which there is a risk of pulmonary oedema and 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 COMPRESSIVE SHOCK

Impaired diastolic filling occurs because of restriction of the motions and filling of the heart as a result of pericardial tamponade. The consequence of this compression is an increase in right atrial pressure without an increase in volume, impeding venous return and provoking hypotension.

Clinical presentation

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 less than 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 ($>25 \text{ mL}$) of blood may be sufficient to cause decompensation.

2.2.2.4 INFLAMMATORY SHOCK (CELLULAR DEFECT 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 $6 \text{ L m}^{-2} \text{ min}^{-1}$. 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:

- hypovolaemia due to fluid translocation from the blood into interstitial spaces,
- elevated pulmonary vascular resistance due to adult respiratory distress syndrome (ARDS),
- bioventricular myocardial depression manifested by reduced contractility and an inability to increase stroke work.

The ultimate cause of death in septic shock is failure of energy production at the cellular level, as reflected by a decline in oxygen consumption. It is not only the circulatory insufficiency that is responsible for this, but also the impairment of cellular oxidative phosphorylation by endotoxin or endogenously produced superoxides. There is a narrowing of arterial–mixed venous oxygen difference as an indication of reduced oxygen extraction, which often precedes the fall of cardiac output. Anaerobic glycogenolysis and a severe metabolic acidosis due to lactacidaemia result. The mechanisms responsible for the phenomena observed in sepsis and endotoxic shock are discussed above.

2.2.2.5 NEUROGENIC SHOCK

Neurogenic shock is a hypotensive syndrome in which there is loss of α -adrenergic tone and dilatation of the arterial and venous vessels. The cardiac output is normal, or may even be elevated, but because the total peripheral resistance is reduced, the patient is hypotensive. The consequence is 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 central nervous system.

Clinical presentation

The patient may have weakly palpable peripheral pulses, warm extremities and brisk capillary fill-

ing, and may be alert. 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 only be made 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

Obstructive shock is impaired diastolic filling because of restriction of the venous return to the heart, usually caused by tension pneumothorax. Because of the decreased venous return, the atrial filling is reduced, with consequent hypotension.

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. 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 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, and

- 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 indirectly determine adequate nutrient flow to a visceral organ is the urine output.

The kidney responds to decreased nutrient flow with several compensatory changes to protect its own perfusion.⁴ Over a range of blood pressures, the kidneys maintain a nearly constant blood flow. If

the blood pressure decreases, the kidney's autoregulation of resistance results in dilatation of the vascular bed. It keeps nutrient flow constant by lowering the resistance even though the pressure has decreased. This allows selective shunting of blood to the renal bed.

If the blood pressure falls further and a true decrease in flow across the glomeruli occurs, the rennin-angiotensin mechanism is triggered. Renin from the juxtaglomerular apparatus acts upon angiotensin from the liver. The peptide is cleaved by renin and a decapeptide 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.⁵ 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. Because 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

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.

Thus, placement of a central venous line will allow measurement of the hydrostatic pressure of the right atrium. 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 below 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, either as a result of a full circulation or due to pump failure.

As a general rule, if a patient in shock has both systemic arterial hypotension and central venous hypotension, the shock is due to volume depletion. On the other hand, if central venous pressure is high though arterial pressure is low, shock is not due to volume depletion and is more likely to be due to 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 utilized by anaesthesiologists. It provides ease of access, especially under operative conditions. However, the ability to occlude the site, particularly in the awake patient in the intensive care unit (ICU), is more limited and there is greater discomfort for the patient.

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 hours.

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 the systolic and diastolic blood pressures.

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.⁷ 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.

The arterial Doppler 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 cannulation vary. The radial artery is the most common site; it is usually safe to use, provided 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^{8,9}

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 arterial pressure. The wedged pulmonary arterial pressure is a useful approximation of left ventricular end-diastolic pressure (LVEDP). LVEDP 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 route¹⁰

EQUIPMENT

- Lignocaine.
- Swann–Ganz catheter set: commercial pack.
- Calibrated pressure transducer with continuous heparin flush and connecting tubing.
- Visible oscilloscope screen showing both electrocardiogram (ECG) and pressure tracings.
- A dedicated assistant (e.g. a nurse).

TECHNIQUE

- 1 Prepare all supplies at the bedside.
- 2 Calibrate the transducer for a pressure range of 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. The patient should be on oxygen, and 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 the sternomastoid muscle.
- 8 Insert a 16G needle beneath the anterior border of the 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 (Figure 2.1).
- 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 X-ray.

Cardiac output

Cardiac output can be measured with the thermodilution technique.¹¹ 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

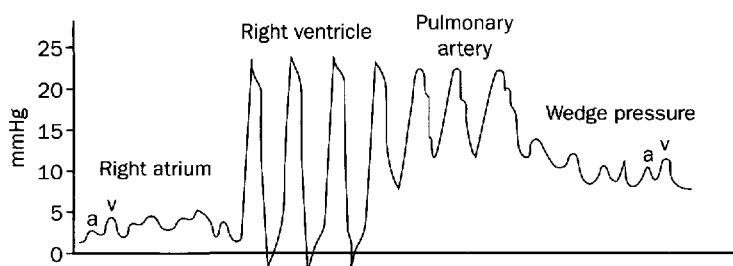


Figure 2.1 Pressures.

output) can be calculated. By estimating oxygen saturation in the pulmonary artery, blood oxygen extraction can be determined.

2.2.4 Metabolism in shock¹²

The ultimate measurement of the impact of shock must be at the cellular level. The most convenient measurement is a determination of the blood gases. Measurement of PaO_2 , $PaCO_2$, pH and arterial lactate will supply information on oxygen delivery and utilization of energy substrates. Both PaO_2 and $PaCO_2$ are concentrations – partial pressure of oxygen and carbon dioxide in arterial blood. If the $PaCO_2$ is normal, there is adequate alveolar ventilation. 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 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 that can indicate what the cells are doing metabolically, which is the most important reflection of the adequacy of their nutrient flow. The pH is the hydrogen ion concentration, which can be determined easily and quickly. 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 diffusible by-products – carbon dioxide and water – both of which leave the body by excretion through the lung and the kidney. Aerobic metabolism is efficient; therefore, there is no accumulation of any products of this catabolism, and a high yield of adenosine triphosphate (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. 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 hydrogen ions increase. However, there is a significant toxicity of hydrogen ions as well. Despite the salutary effect on oxyhaemoglobin dissociation, the hydrogen ion has a negative effect on oxygen delivery. Catecholamines speed up the heart's rate and increase its contractile force, and the product of this inotropic and chronotropic effect is an increase in cardiac output. However, catecholamines are physiologically effective at alkaline or neutral pH. Therefore, an acid pH inactivates this catecholamine method of compensation for decreased nutrient flow. For example, 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 ions, lactate and pyruvate. When the circulation dilates, this sequestered oxygen debt is dumped into the central circulation and the drop in pH inactivates the catecholamine's circulatory improvement as effectively as if the infusion of the agent had been interrupted.

2.2.5 Post-shock sequence and multiple organ failure syndromes¹³

Although the consequences of sepsis following 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 following shock to enable logical discussion of the management of shock.

The ultimate cause of death in shock is failure of energy production, as reflected by a decline in oxygen consumption (VO_2) to less than $100 \text{ mL m}^{-2} \text{ min}^{-1}$. 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 Krebs' cycle is inhibited. Lactic acid accumulates and the oxidation–reduction potential falls, although lactate is normally used by the liver via the Cori cycle to synthesize glucose. Hepatic gluconeogenesis may fail in hypovolaemic and septic shock because of hepatocyte injury and inadequate circulation. The lactacidaemia 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:betahydroxybutyrate ratio in the plasma.

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

2.2.6.1 OXYGENATION¹⁴

The traumatized hypovolaemic or septic patient has an oxygen demand that often exceeds twice the normal. Under these circumstances, hyperventilation would provide an effective means of increasing oxygen delivery. The traumatized shocked patient usually cannot exert this additional effort and, therefore, often develops respiratory failure followed by respiratory acidosis.

In some patients, an oxygen mask may be enough to maintain efficient oxygen delivery. 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 $<5 \text{ mL/kg}$.
- Breathing indications for *ventilation*:
 - inability to oxygenate adequately,
 - $\text{PaO}_2 < 60 \text{ mmHg}$ on 60 per cent oxygen, or
 - $\text{SaO}_2 < 90$ per cent on 60 per cent oxygen,
 - a respiration rate of 30 breaths or more per minute, and
 - excessive ventilatory effort,
 - a $\text{PaCO}_2 > 45 \text{ mmHg}$ with metabolic acidosis, or $> 50 \text{ mmHg}$ with normal bicarbonate levels.
- Circulation indication for *intubation*:
 - systolic blood pressure $> 75 \text{ mmHg}$ despite resuscitation.

- Disability indications for *intubation*:
 - spinal injury with inability to breathe,
 - coma (Glasgow Coma Score <8/15).
- Environmental indication for *intubation*:
 - core temperature <32 °C.

If ventilatory support is instituted, the goals are relatively specific.

The respiratory rate should be adjusted to ensure a PaCO_2 of between 35 and 40 mmHg. This will avoid respiratory alkalosis and a consequential shift of the oxyhaemoglobin dissociation curve to the left. This results in an increased affinity for oxygen and significantly decreases oxygen availability to tissues, which will require increased cardiac output to maintain tissue oxygenation.

The arterial PaO_2 should be maintained between 80 and 100 mmHg with the lowest possible oxygen concentration.

It has been shown that respiratory muscles require a disproportionate share of the total cardiac output, 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 EXPANSION¹⁵⁻¹⁷

Considerable controversy exists regarding the type of fluid to be administered for volume expansion in hypovolaemic shock. Despite many studies, there is minimal convincing evidence favouring any specific fluid regimen. Balanced salt solutions (BSSs) are effective volume expanders for the initial resuscitation of patients with shock. For most patients, Ringer's lactate solution is the preferred crystalloid solution. The lactate acts as a buffer and is eventually metabolized to carbon dioxide 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. In adults, a bolus dose of 2000 mL of BSS (e.g. Ringer's lactate) 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 cardiac output and thus maintain tissue oxygenation. This increased cardiac output 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 these 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. BSS is said to have a half-life in the circulation of 20 minutes; Gelufusine® has a half-life of 4–6 hours. However, additional considerations relate to the 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 to 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.¹⁸

Resuscitation versus no resuscitation

In 1994, Bickell et al.¹⁹ 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.²⁰ 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^{21,22}

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 in all the trials, the patients did best when the hypertonic saline was given as the initial therapy, and those patients most likely to benefit were those with head injuries. Hypertonic saline may be more effective when mixed with a small amount of an oncologically active molecule such as dextran. The combination of hypertonic saline and a colloid is most likely to be beneficial when definitive resuscitation may be delayed.

Blood substitutes

Blood substitutes, including haemoglobin-based oxygen carriers (HBOC) and perfluorocarbons, have several potential advantages: no cross-matching is necessary, disease transmission is not an issue, and shelf life is extended.

All the above at present remain experimental for human use.

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 are reduced (Table 2.2):

- 14G via peripheral cannula: full flow,
- 14G via 30-cm central line: 33 per cent reduction in flow,
- 14G 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.

Technique of subclavian line insertion²³

- 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 lignocaine 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 of 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** Hold the needle and syringe parallel to the frontal plane.
- 7** Direct the needle medially, slightly cephalad, and posteriorly, behind the clavicle, towards the posterior, superior 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 guide wire while monitoring the ECG for abnormalities.
- 11** Insert the catheter over the guide wire 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 X-ray to confirm the position.

Table 2.2 Intravenous flow rates related to cannula size

Cannula size	Flow rates (mL/min)	
	Crystallloid	Colloid
8.5FG	1000	600
14G	125	90
16G	85	65
18G	60	35
20G	40	17

Technique of femoral line insertion²⁴

- 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 lignocaine 1 per cent.
- 5 Introduce a large-calibre needle, attached to a 10 mL-syringe containing 1 mL of saline. The needle, directed towards the patient's head, should enter the skin directly over the femoral vein.
- 6 Hold the needle and syringe 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 the syringe. Occlude the needle to avoid any chance of air embolism.
- 9 Insert the catheter over the guide wire 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 X-ray to confirm the position.

2.2.6.4 PHARMACOLOGIC SUPPORT OF BLOOD PRESSURE²⁵⁻²⁸

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 cardiac output when systemic pressures or cardiac filling pressures are normal or elevated.

Adrenaline (Epinephrine)

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

- increased systemic vascular resistance,
- increased systolic and diastolic blood pressures,

- increased electrical activity in the myocardium,
- increased coronary and cerebral blood flow,
- increased strength of myocardial contraction,
- increased myocardial oxygen requirement.

The primary beneficial effect of adrenaline is peripheral vasoconstriction, with improved coronary and cerebral blood flow. It works as a chronotropic and inotropic agent. The initial dose is $0.03 \mu\text{g kg}^{-1} \text{ min}^{-1}$, 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, or noradrenaline, that stimulates dopaminergic, β_1 -adrenergic and α -adrenergic receptors in a dose-dependent fashion. Low doses of dopamine ($<3 \mu\text{g kg}^{-1} \text{ min}^{-1}$) 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 $10 \mu\text{g kg}^{-1} \text{ min}^{-1}$, the α -adrenergic effects predominate. This results in marked increases in systemic vascular resistance and pulmonary resistance, and increases in preload due to marked arterial, splanchnic and venous constriction. It increases systolic blood pressure without increasing diastolic blood pressure or heart rate.

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

Dobutamine

Dobutamine is a synthetic sympathomimetic amine that has potent inotropic effects by stimulating β_1 -adrenergic and α_1 -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 $10 \mu\text{g kg}^{-1} \text{ min}^{-1}$, dobutamine is less likely to induce tachycardia than either adrenaline 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 adrenaline.

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

Dobutamine and dopamine have been used together. The combination of moderate doses of both ($7.5 \mu\text{g kg}^{-1} \text{min}^{-1}$) 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 the adverse effects on arterial blood pressure.²⁹ 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 intensive care situation, digoxin is usually reserved for the treatment of atrial flutter and supraventricular tachycardias.

2.2.6.5 METABOLIC MANIPULATIONS¹⁴

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, as elevations in this substance can be demonstrated at the time these physiological changes take place.

Naloxone, an opiate antagonist, has been shown to elevate blood pressure and cardiac output, and to significantly improve survival 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. There is experimental evidence that cyclo-oxygenase inhibitors such as indomethacin 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 physiologic 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.

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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 is used in emergency resuscitation.

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 as a decrease in circulating red cell mass to below 24 mL/kg in females and 26 mL/kg in males. Anaemia will result in an increase in cardiac output at Hb 4.5–7 g/dL. Oxygen extraction (O₂ER) increases as O₂ delivery (DO₂) falls, ensuring a constant O₂ uptake (VO₂) by tissues. Threshold for O₂ delivery is at a haematocrit of 10 per cent and Hb of 3 g/dL, breathing 100 per cent O₂ and with a 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 – 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)

Platelet transfusion

- Prophylaxis: platelet count $<15\,000\text{ mm}^{-3}$.
- Pre-surgery: platelet count $<70\,000\text{ mm}^{-3}$.
- Active bleeding: platelet count $<100\,000\text{ mm}^{-3}$.
- 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 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,
- micro-aggregates (platelet/leucocyte/fibrin thrombi) in buffy coat.

2.3.2.2 EFFECTS OF MICRO-AGGREGATES

- Impaired pulmonary gas exchange, adult respiratory distress syndrome (ARDS).
- Reticulo-endothelial 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.

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 hours 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, as these are generally spun off, and whole blood has lost most of its platelets after 3 days of storage. Spontaneous bleeding rarely occurs if the platelet count is greater than $30\,000\text{ mm}^{-3}$. 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 micro-aggregates 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.
- Human immunodeficiency virus (HIV) window period.
- Cytomegalovirus (CMV).
- Atypical mononucleosis and swinging temperature 7–10 days post-transfusion.
- Malaria.
- Brucellosis.
- Yersinia.
- Syphilis.

2.3.3.2 HAEMOLYTIC TRANSFUSION REACTIONS

- Incompatibility: ABO, Rh (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' due to 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 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, 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

due to 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 LABORATORY TESTS

- Fibrinogen degradation products (FDPs).
- International Normalized Ratio (INR) extrinsic.
- Partial thromboplastin time (PTT) intrinsic.
- D-dimer (fibrin deposition).

- 1 Treat the cause, i.e. urgent surgery to stop bleeding, avoid hypothermia and acidosis.
- 2 Packed cells for O₂-carrying capacity. There is no level-1 evidence indicating the ideal trigger for transfusion in trauma patients. In general, uncompromised normovolaemic patients should be transfused at Hb <7 g%, and patients with preceding cardiovascular, cerebrovascular or pulmonary compromise should be transfused at Hb <10 g%. A recent level-1 study indicated improved survival for Hb 8–10 g% vs Hb 10–12 g% for surgical intensive care unit (ICU) admissions.
- 3 Treat deficiencies and complications as they arise. There is no evidence to support prophylactic therapy (Ca⁺, fresh frozen plasma (FFP), platelets etc.).
- 4 Develop a capacity for salvage.

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 hours,
- the administration of 50 per cent of the patient's blood volume in 1 hour.

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.

2.3.5.2 REDUCING UNNECESSARY TRANSFUSION AND ALTERNATIVES TO BLOOD

- Blood is a scarce (and expensive) resource and is also not universally safe.

2.3.6 Autotransfusion

Autotransfusion eliminates the risk of incompatibility and the need for cross-matching; 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 the collection of blood shed into wounds, body cavities and drains.

Autotransfusion is generally contraindicated in the presence of bacterial or malignant cell contamination (e.g. open bowel, infected vascular prostheses etc.) 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.

- 1 The blood is collected, mixed with an anticoagulant, typically citrate, and returned.
- 2 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.

Re-infusion 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 haemoglobin and procoagulants are also re-infused. 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. In-line filters are absolutely essential when auto-

transfusion devices are used. These filters remove gross particles and macro-aggregates during collection and re-infusion, thus minimizing micro-embolization.

Cell washing and centrifugation techniques require a machine and (usually) a technician to be the sole operator. The latter requirement can limit the utility of the devices in everyday practice. The cell-washing cycle produces red cells suspended in saline with a haematocrit of 55–60 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 therefore washing does not entirely 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 needed to clarify the indications further.

2.3.7 Transfusion: red blood cell substitutes

The ideal blood substitute is cheap, has a long shelf life, is universally compatible and well tolerated and has an O₂-delivery profile identical to that of blood. Significant effort has been made to find a suitable substitute, which, essentially, should be treated as an artificial O₂ carrier.

Artificial O₂ carriers can be grouped into perfluorocarbon (PFC) emulsions and modified Hb solutions. The native Hb molecule needs to be modified in order to decrease O₂ 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 Hb, they exhibit a linear relationship with O₂; therefore their efficacy relies on maintaining a high PaO₂. However, PFCs unload O₂ well. They do not expand the intravascular volume and can only be given in small volumes as they overload the reticuloendothelial system. Once thought to hold potential, to date they have not been found to confer additional

benefit compared to crystalloid solutions, especially because there is a significant incidence of side effects.

2.3.7.2 HAEMOGLOBIN SOLUTIONS

Liposomal haemoglobin solutions

These are based on the encapsulation of Hb in liposomes. The mixing of phospholipid and cholesterol in the presence of Hb yields a sphere with Hb at its centre. These liposomes have O₂-dissociation curves similar to that of red cells, with low viscosity, and their administration can transiently produce high circulating levels of Hb.

Problems associated with Hb-based O₂ carriers relate to effects on vasomotor tone, which appears to be modulated by the carriers' interaction with nitric oxide, causing significant vasoconstriction.

Clinical trials are ongoing, and a safe and efficacious product is likely to appear in the future.

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

Although free Hb can transport O₂ 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 Hbs, either with a di-aspirin link or by other means. Both human and bovine Hbs 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®) have produced some dramatic improvement. Currently, the product is licensed for veterinary use only.

No level-1 evidence has yet appeared to support the use of Hb substitutes instead of blood.

The O₂ transport characteristics of modified Hb solutions and PFC solution 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 those of blood. This means that at a relatively low arterial O₂ pressure, substantial amounts of O₂ are being transported. In contrast, relatively high arterial O₂ partial pressures are necessary to maximize the O₂ transport of PFC emulsions (Figure 2.2 and Table 2.3).

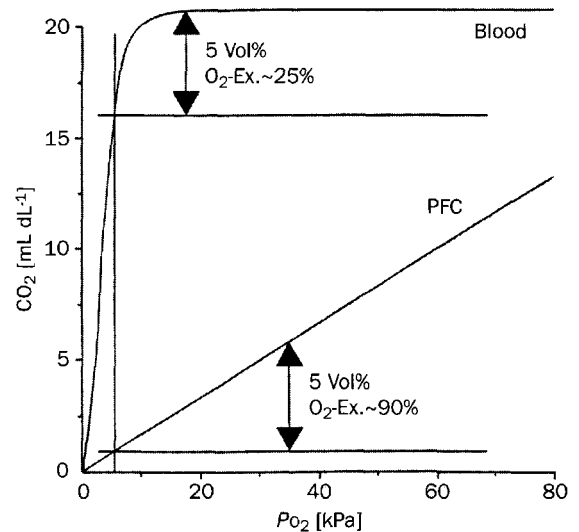


Figure 2.2 Oxygen transport characteristics of haemoglobin and perfluorocarbon (PFC). Note that 5% O₂ can be offloaded by both blood and PFC. PFC O₂ is more completely offloaded than blood-transported O₂.

Table 2.3 Advantages and disadvantages of haemoglobin (Hb) solutions and perfluorocarbon (PFC) emulsions

Hb-based solutions	PFC-based emulsions
Advantages	Advantages
Carries and unloads O ₂	Carries and unloads O ₂
Sigmoidal O ₂ -dissociation curve	Few and mild side effects
100% FiO ₂ not mandatory for maximum potency	No known organ toxicity
Easy to measure	Disadvantages
Disadvantages	100% FiO ₂ is mandatory for maximal efficacy
Side effects	Additional colloid often necessary, with potential side effects
Vasoconstriction	
Interference with laboratory methods (colourimetric)	

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2.4 RESUSCITATION ENDPOINTS

2.4.1 Metabolic considerations

2.4.1.1 HYPOTHERMIA

Hypothermia is almost always a complication of trauma and, thus, following 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, on balance, hypothermia 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.

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 °C 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 >32 °C can generally be rewarmed using external rewarming.

Methods of rewarming include:

- External:
 - remove wet or cold clothing and dry the patient,
 - infrared (radiant) heat,
 - electrical heating blankets,
 - warm air heating blankets.
- NB. 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 brainstem death.

2.4.1.2 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, as these are generally spun off, and whole blood has lost most of its platelets after 3 days of storage. Spontaneous bleeding rarely occurs if the platelet count is greater than $30\,000\text{ mm}^{-3}$. Levels as low as this are seen after the replacement of one to 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 10 units of blood, and replacement should be considered if the platelet level is $<50\,000\text{ mm}^{-3}$.

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, as several donors have contributed to a single pack of platelets.

Levels of clotting factors V and VIII decline quickly for 24 hours after collection. The rate of decline slows until clinically subnormal levels are reached at 7–14 days. 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.

2.4.1.3 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. The presence of acidosis and hypercapnia will tend to result in replacement of the potassium ions in the cell.

2.4.2 Physiology

Traditional endpoints of resuscitation have been limited to pulse rate, blood pressure, haemoglobin, urea, and electrolyte levels, but these were truly limited in their scope for 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 substrate subject as a result of reperfusion injury, the acute phase response, the risk of a second-hit phenomenon, coagulation deficiency, abdominal compartment syndrome (ACS), and immune compromise induced by trauma. Packing the abdomen and closing it inherently introduce an ACS 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 (Swan Ganz 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 ($>1\text{ mL kg}^{-1}\text{ h}^{-1}$) 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 intensive care unit (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 room is ultimately a surgical decision, taken when the goals of resuscitation are met, irrespective of the time of day or time since the original procedure.

What, then, are the indicators to admit an injured patient to the ICU and to ventilate mechanically?

There are no definite indicators for injured patients – only guidelines. In many units, admission to the ICU and elective ventilation are mandatory following damage control surgery, due to the physiological debility induced by the indications for and the effects of damage control. These include:

- respiratory failure with a PaO_2/FiO_2 ratio <2 , peak airway pressures >38 cmH₂O,
- hypothermia <34 °C due to acidosis, myocardial depression, coagulopathy etc., induced by global enzyme malfunction,
- metabolic acidosis with a base deficit >6 or a pH <7.10 ,
- massive blood transfusion (>10 units) that is associated with a mortality of >20 per cent,
- coagulation deficiency with an INR or PTT value >2 or platelet count $<50\,000/\text{mm}^3$ because of the risk of ongoing haemorrhage,
- requirement for inotropes to maintain cardiac output,
- evidence of ACS due to respiratory and renal failure induced by ACS, which are caused by the rise in intra-abdominal compartment pressure.

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

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

- inability to maintain an airway, e.g. severe facial fractures or Glasgow Coma Scale (GCS) <8 ;
- to achieve initial control of PO_2 and PCO_2 in head-injured patients, because avoidance of

hypoxia and maintenance of normocapnia (PCO_2 35–40 mmHg) are known to improve survival in head-injured patients; note that moderate hypocapnia (PCO_2 30–35 mmHg) has been shown to be of benefit only within the first 48 hours after injury;

- Injury Severity Score (ISS) >25 implies a mortality of >20 per cent; charts are available as guidelines;
- major burns – a rough indicator of survival is the equation:

$$\% \text{ burns} + \text{age over } 40 \left(\begin{array}{l} - \text{no. years under } 40, \\ + \text{no. years over } 40 \end{array} \right) = \% \text{ survival}$$

note that this equation was developed many years ago and is not always accepted in modern literature;

- any major co-morbid disease or extremes of age will upgrade any of the above indicators.

Elective admission to the 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 or TRISS (Trauma Score and Injury Severity Score combined), retrospectively measure outcome in populations and are particularly insensitive to determine 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 4/15) or complete quadriplegia levels C3–C4 or above. Guidelines should be developed beforehand by all healthcare disciplines involved.

Admission to the 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 the effects of ACS
- intravesical pressure monitoring to identify ACS,
- high-capacity (Alton Dean™/Level One™ apparatus) infusions of the freshest blood available to a Hb of 10–12 g%,
- platelet infusions to ensure a platelet count above $50\,000/\text{mm}^3$,
- infusion of cryoprecipitate as colloid to provide clotting factors, e.g. to INR/PTT <1.5 ,

- active rewarming using a warm 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 of 1–2 mL kg⁻¹ h⁻¹, and central venous pressures showing a rising trend.

The patient should be returned to theatre in case of deterioration or failure to improve – usually due to 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.

2.4.2.1 KEY REFERENCE

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2.4.3 When to ventilate?

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:

$$\text{oxygen delivery (DO}_2\text{)} = \text{cardiac index} \times \text{Hb} \times 1.39 \times \text{saturation}$$

Therefore, delivery is compromised when the cardiac index, Hb or Hb-carrying capacity is low, not only when the PaO_2 or saturation is low.

Hypoxic hypoxia implies that the PaO_2 is low and occurs with V/Q mismatch, alveolar hypoventilation, low inspired oxygen fraction, diffusion abnormality, and, in the presence of V/Q mismatch, a low cardiac output.

V/Q mismatch and diffusion defects occur with parenchymal disease such as pulmonary contusion or oedema, aspiration and adult respiratory distress syndrome (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 PaO_2 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 PaO_2 (mmHg)/ FiO_2 ratio (normal 400–500) or by the saturation; circulatory or anaemic hypoxia by the pH and the HCO_3^- . Adequacy of ventilation is assessed by the PaCO_2 . However, an elevated value is not necessarily an acute problem, provided that the pH is abnormal. For example:

- $\text{CO}_2 \uparrow$, $\text{HCO}_3^- \uparrow$, normal pH – chronic obstructive pulmonary disease (COPD),
- $\text{CO}_2 \uparrow$, $\text{HCO}_3^- \uparrow$, pH $\downarrow$ – acute on chronic respiratory failure,
- $\text{CO}_2 \uparrow$, HCO_3^- normal, pH $\downarrow$ – acute ventilatory insufficiency.

When to ventilate?

First, recognize that a problem might exist.

- Agitated, belligerent or obtunded patients.
- Respiratory distress or noisy breathing.
- Central nervous system depression or head injury.
- Severe maxillofacial injury.
- Severe chest injury, fractures or flail.
- 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),
- $\uparrow \text{CO}_2$ and respiratory acidosis,
- $\text{PaO}_2 < 60$ mmHg on re-breathing mask,
- haemodynamic instability or cardiac arrest,
- severe metabolic acidosis, pH <7.2.

2.4.4 Shock

2.4.4.1 PATHOPHYSIOLOGY

The traditional etiological 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

(Shoemaker, 1987).

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, for example haemorrhage, is remarkably similar, and consists of the acute phase response:

- hormonal: diabetogenic state,
- immune: immune suppression,
- coagulation: hypercoagulability,
- anticoagulation: clot lysis,
- temperature: pyrexia,
- leucocyte activation/aggregation: inflammatory response,
- arachnidonic acid derivatives (complement, prostaglandins).

Hypovolaemic shock results in vasoconstriction, anaerobic metabolism and clogging of the microcirculation. Anaerobic metabolism results in the production of 2 mol (47 kcal) of adenosine triphosphate (ATP) per mole of oxidized glucose as opposed to 36 mol (673 kcal) per mole of 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 leucocytes – 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,
- septic syndrome,
- multiple organ dysfunction syndrome.

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.

2.4.4.2 ENDPOINTS

The first endpoint is control of the cause!

For Shock Classes 1 and 2, the systemic insult is relatively minor, so that early control of the precipitating cause and simple volume resuscitation will prevent these events from being manifested. For more severe degrees of shock, such complex pathophysiological systems cannot be managed effectively by simply filling up the intravascular volume, and management cannot be delegated.

Volume resuscitation

There is no level-1 evidence of improved survival following 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 90 mmHg) until the bleeding is controlled may be of benefit, provided 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.

Blood pressure

Traditional manual measurements of blood pressure are notoriously inaccurate. We are interested in perfusion (flow), but measure a linear dimension thereof (pressure). Mean arterial pressure 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 blood pressure yields spuriously low values – use a blood pressure cuff and the Doppler probe or, preferably, intra-arterial catheters. Aim for a mean arterial pressure >70 mmHg.

Temperature

There is no enzyme system that functions under hypothermic conditions. A temperature >34 °C is associated with a mortality of 32 per cent. A temperature <32 °C in trauma patients is associated with mortality of >60 per cent. One degree of centigrade 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.

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 hours despite resuscitation is associated with ARDS (50 per cent), multiple system organ failure (36 per cent) and mortality (43 per cent). Failure to establish lactate levels of <2.5 mg/dL within 24 hours may be an indicator to use pulmonary capillary wedge pressure to guide inotrope and volume resuscitation. Hyperglycaemia, adrenaline infusion and infection may also elevate lactate levels. Base deficit is probably the poor man's alternative to lactate determinations.

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 antidiuretic hormone secretion and limit urine volume. A urine volume of 0.5–1.5 mL kg⁻¹ h⁻¹ without diuretics is normal.

Haemoglobin/haematocrit (see massive blood transfusion)

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. There is no difference in survival between patients (admitted to surgical ICU) transfused to Hb of 8–10 g/dL versus a Hb of 10–12 g/dL. However, the subgroup of trauma patients was too small to draw any conclusions.

Central venous pressure

Normal central venous pressure is 0–10 cmH₂O. Central venous pressure is an indicator, not a parameter, of cardiac filling. It (and pulmonary capillary wedge pressure) correlates poorly with volume of blood lost (vasoconstriction, decreased ventricular compliance). In general, use a bolus method, e.g. Ringer's 500 mL/h or colloid 250 mL/h, in previously healthy patients, until central venous pressure fails to increase for 30 minutes. For previ-

ously compromised patients, half the volume of bolus volume is suggested.

Oxygen extraction ratio

The oxygen extraction ratio (O₂ER) defines the efficiency with which oxygen is extracted compared to the amount delivered, and therefore gives an indication of tissue perfusion.

$$O_2ER = VO_2/DO_2 \times 100$$

(normal = 0.2–0.3)

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

$$O_2ER = SaO_2 - MvO_2 \propto SaO_2 - CvO_2$$

- hypovolaemia = 0.3–0.5,
- hypovolaemic shock = >0.5.

Gas exchange

SaO₂ measured by pulse oximetry differs by <3 per cent from SaO₂ measured by co-oximetry, and is accurate down to a Hb of 3 g/dL and blood pressure of 30 mmHg. End-tidal PCO₂ exhibits a linear relationship to cardiac output, with a correlation coefficient of 0.87 during resuscitation, and can be monitored non-invasively.

Intra-abdominal pressure

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 symphysis pubis. Pressure is normally <10 cmH₂O. Pressure >25 cmH₂O indicates incipient ACS and a possible need to relieve intra-abdominal pressure.

Coagulation

Stop the bleeding including the oozing!

Pulmonary capillary wedge pressure

This is equivalent to left atrial pressure (LAP) or left ventricular end-diastolic pressure (LVEDP).

Resuscitating to supra-normal values has been shown to differentiate between responders (corre-

sponding to survivors) and non-responders (equivalent to mortalities) and not to improve outcome. The current approach is to resuscitate to normal values.

The Swan Ganz 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.

Gastric mucosal pH

This measures local tissue (hypo)perfusion:

$pHi = 6.1 = \log^{10} \text{arterial } HCO_3^- / \text{saline } PCO_2$ or <0.03.

Gastric mucosal PCO_2 is largely derived from

gastric mucosal lactate, i.e. gastric mucosal pH measures lactate levels in gastric mucosa.

2.4.5 Recommended reading

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Part II

Decision making

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Surgical decision making 3

3.1 RESUSCITATION IN THE RESUSCITATION ROOM

3.1.1 Ideal practice

Patients with life-threatening injuries represent approximately 10–15 per cent of all patients hospitalized for injuries.¹ Some authors have defined extensive trauma in a patient as an Injury Severity Score (ISS) greater than 15.^{2–4} This is acceptable for triage purposes, but for the purpose of this discussion, extensive trauma can be defined as an ISS greater than 25. Most patients in this category have multisystem trauma. Deforming and destructive injuries can be obvious, but the surgeon or physician initially treating the patient must promptly conduct a systematic workup, so that all wounds, including occult mortal injuries, can be treated optimally.

The principles of management for patients with extremity 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.⁵ The majority of these patients either bleed to death or die from primary or secondary injuries to the central nervous system. In order 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 the 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. However, time is usually not available and the workup of the critically injured patient must often be rushed. In order 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 (ATLS®) Course⁶ is a model. We will use ATLS® as a paradigm for assessment, resuscitation and prioritization of the patient's injuries.

General guide times in the emergency department are:

- for the unstable patient, 15 minutes,
- for the stable patient, 30 minutes.

Within 15 minutes, the unstable patient should either be in the operating room or the intensive care unit (ICU).

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 neurologic status,
- measuring the core temperature.

Airway and breathing

Patients with extensive trauma who are unconscious or in shock benefit from immediate endotracheal intubation.^{7,8} To avoid spinal cord injury, the cervical spine must not be excessively flexed or extended during intubation. Oral endotracheal intubation is successful in the majority of 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 maxillofacial injuries 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, to establish high flow oxygen initially with a bag mask, and to proceed immediately to endotracheal intubation in the majority and surgical airway in a few.

Circulation

Simultaneous with airway management, a quick assessment of the patient will determine the degree of shock present. Shock is a clinical diagnosis and should be apparent. A quick first step is to feel an extremity. If shock is present, the extremities will be cool, pale, lack venous filling and have poor capillary refill. The pulse will be thready and consciousness will be 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 emergency department. A simple tube thoracostomy is the 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 emergency department alive. The diagnosis is often obvious. The patient has distended neck veins and poor peripheral perfusion, and a few will have pulsus paradoxus. In a very small number of patients with equivocal findings, ultrasonography may establish the diagnosis. Pericardiocentesis is an occasionally useful diagnostic or therapeutic aid. Proper treatment is immediate thoracotomy, preferably in the operating room, although emergency department thoracotomy can be life saving.⁹

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

Myocardial infarction from coronary occlusion is not uncommon in the elderly.

Air embolism^{10,11} is a syndrome that has only relatively recently been appreciated as important in injured patients. It represents air in the systemic circulation caused by a bronchopulmonary venous fistula. Air embolism occurs in 4 per cent of all major thoracic injuries. Thirty-five per cent of the time it is due to blunt trauma, usually a laceration of the pulmonary parenchyma by a fractured rib. In 65 per cent of patients it is due to gunshot wounds or stab wounds. The surgeon must be vigilant when pulmonary injury has occurred. Any patient who has no obvious head injury but has focal or lateralizing neurological signs may have air bubbles occluding the cerebral circulation. The observation of air in the retinal vessels on fundoscopic examination confirms cerebral air embolism. Any intubated patient on positive-pressure ventilation who has a sudden cardiovascular collapse is presumed to have either tension pneumothorax or air embolism to the coronary circulation. Doppler monitoring of an artery can be a useful aid in detecting air embolism. Definitive treatment requires immediate thoracotomy followed by clamping of the hilum of the injured lung to prevent further embolism, followed by expansion of the intravascular volume. Open cardiac massage, intravenous adrenaline and venting 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 four priorities are to:

- 1 gain access to the circulation,
- 2 obtain a blood sample from the patient,
- 3 determine where the volume loss is occurring,
- 4 give resuscitation fluids.

The fastest and most reliable way to gain access to the circulation is by a surgical cutdown on the saphenous vein at the ankle. This anatomically constant vein allows the physician to quickly achieve access with a large-diameter tube. Alternatively, the resuscitating physician can perform bilateral percutaneous femoral vein cannulation with a large-bore catheter or an 8 French introducer more commonly used for passing a pulmonary artery catheter. Some experienced physicians prefer to gain access through the subclavian or internal jugular vein, but this approach is associated with the complication of pneumothorax.

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

The third priority is to determine where the patient may have occult blood loss. Three sources for hidden blood loss are the pleural cavities (which can be eliminated by rapid chest X-ray), 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: 50 per cent of patients with significant haemoperitoneum have no clinical signs.^{12,13} Common sense dictates that if the chest X-ray is normal and the femur is 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 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.^{14,15}

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.

Crystalloid, synthetic colloids, such as gelatin, dextrans and hetastarch, 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 suggest 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 in whom 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.¹⁶ found that the survival in patients with 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 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 cross-matched blood. With extensive haemorrhage and massive transfusion (greater than 1 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⁻¹ h⁻¹ in the adult, 1.0 mL kg⁻¹ h⁻¹ in the child),
- maintain peripheral perfusion.

The only practical way to measure atrial filling pressure in the emergency department and imme-

diately in the operating room is by central venous pressure monitor. In elderly patients with extensive traumatic injuries, placing a pulmonary artery catheter may be prudent because it will be used to direct a sophisticated multifactorial resuscitation in the operating room or in the ICU. Resuscitation should be directed towards achieving adequate oxygen delivery and oxygen consumption. An important caveat is not to delay mandated therapeutic interventions to obtain the results of non-critical diagnostic tests.

Neurologic status

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

- determine the level of consciousness,
- observe the size and reactivity of the pupils,
- check eye movements and oculovestibular responses,
- document skeletal muscle motor responses,
- determine the pattern of breathing,
- 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 manifest by goal-directed or purposeful behaviour. The use of language is an indication of functioning cerebral hemispheres. If the patient attempts to protect himself from a painful insult, this also implies cortical function. Arousal is a crude function that is simple wakefulness. Eye opening, either spontaneous or in response to stimuli, is indicative of arousal and is a brainstem function. Coma is a pathologic 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 making sure the four primary reflexes (ankle, knee, biceps and triceps) are assessed and repeating this examination at frequent intervals, it is possible to both diagnose and monitor the neurologic status in the emergency department. An improving neurologic status

reassures the physician that resuscitation is improving cerebral blood flow. Neurologic deterioration is strong presumptive evidence of either a mass lesion or significant neurologic injury. Computed tomography (CT) scan 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 prior to infusion. Patients can be placed on warming mattresses and their environment kept warm using warm air blankets. Early measurement of the core temperature is important to prevent heat loss that will predispose to problems with coagulation. Hypothermia will shift the oxygen dissociation curve to the left, reduce oxygen delivery, reduce the liver's ability to metabolize citrate and lactic acid, and may produce arrhythmias.

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

- chest X-ray,
- lateral C-spine film, and
- plain film of the pelvis.

Selected patients – for example those with haematuria or significant deceleration injury – may require a single-film intravenous pyelogram (IVP) 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 X-ray facilities are juxtaposed to the emergency department; however, the basic X-rays 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.

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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 assure 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 a need for further intravenous access, an 8.5 French catheter can be introduced into the central venous system, either through the groin or through 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 in order to identify all life-threatening and other injuries.

An effective plan is to identify the patient's probability of survival. Once one determines 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 minutes
- 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 one-third of cases will occur within the next 30 minutes.

If the patient has arrested, cardiopulmonary resuscitation (CPR) should be initiated while other life-saving manoeuvres are being contemplated.

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

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, radiographic or other diagnostic aids, to determine precisely what is injured and to effect an operative plan for the management of 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 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 mass lesion with significant enough intracranial pressure to induce coning. This requires immediate decompression. Time can be saved by hyperventilation to induce hypocarbia and concomitant vasoconstriction.

Attention should be paid to monitoring the end-tidal CO_2 (ETCO_2), as a proxy for PaCO_2 . PaCO_2 should not be allowed to fall below 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 computed tomography (CT) scan to determine a specific site of the mass lesion and the type of haematoma. This will direct the surgeon specifically to the location of the craniotomy for removal of the haematoma.

Intravenous mannitol should be administered as a bolus injection in a dose of 0.5–1.0 g/kg. This should not delay any other diagnostic or therapeutic procedures.

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

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 a CT scan (see section on chest injuries). Once the diagnosis is 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.

NB. 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 in the neck veins. This is a clinical diagnosis and, once made, an immediate needle thoracostomy or tube thoracostomy should be performed to relieve the tension pneumothorax. The tube should then be placed to underwater seal.

The diagnosis of cardiac tamponade is often difficult to make clinically. It is usually associated with hypotension and elevated jugular venous pressure. There are usually muffled heart sounds, but these are difficult to hear in a noisy resuscitation suite. Placing a central line with resultant high venous pressures can confirm the diagnosis. If ultrasound is available, this is an extremely helpful diagnostic adjunct. Once the diagnosis is 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, together 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 – through either an anterolateral approach or a median sternotomy, with good light and assistance and the potential for autotransfusion and potential bypass – than it is to attempt heroic emergency surgery in the resuscitation suite. However, if the patient is in extremis with blood pressure in the 40 mmHg or lower range 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 distended to create tamponade. The end of the Foley should be clamped.

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

Massive haemorrhage from intercostal vessels secondary to multiple fractured ribs will often stop without operative 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 is frequently distended and dull to percussion. A definitive diagnosis can be made with a grossly positive diagnostic peritoneal lavage (DPL), ultrasound or CT scan.

DPL is easy to perform and gives a highly specific, but insensitive, answer immediately. Ten millilitres of grossly positive blood requires an immediate trip to the operating room.

Ultrasound is a useful tool, as it is specific for blood in the peritoneum, but it 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, it does require a trip to the CT scanner and a technician who can perform the scan immediately.

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 and which 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 intra-haematoma pressure that quickly exceeds venous and arterial pressure. Because approximately 70 per cent of pelvic bleeding is venous, compressing the haematoma usually stops the majority of bleeding from the pelvis. If the patient continues to be hypotensive, resuscitation should continue and an arteriogram should be performed. This will identify significant arterial bleeding in the pelvis, which then can 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.

3.2.5 Long bone fractures

Lone bone fractures, particularly of the femur, can bleed significantly. The immediate treatment for a patient who is hypotensive from haemorrhage from a femoral fracture is to put traction on the distal limb, pulling the femur into alignment. This not only realigns the bones, but also reconfigures the cylindrical nature of the thigh. This has an immediate tamponading effect on the bleeding in the muscles of the thigh. It is often 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 if there are any injuries to major vascular structures. A determination is then made as to the timing of arterial repair and bony fixation.

3.2.6 Peripheral vascular injuries

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

Every emergency department 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, it is not always possible, and the necessary equipment may not be available. If there is any doubt, consideration should be given to the use of the emergency department angiogram.

3.2.7 Summary

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

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

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

3.3 CURRENT CONTROVERSIES

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 ‘scoop and run’ is superior to any advanced life support 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 minutes. If minimal fluid resuscitation was carried out, the outcome was better, measured by either mortality or the development of multiple organ failure. 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, but 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 from Los Angeles County showed that patients transported

by private vehicle did better than patients transported by ambulance. Again, one must be circumspect in interpreting these data. There is no question that by transporting by 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 geographic settings and whether or not certain ALS techniques (airway) outweigh the disadvantages.

3.3.2 Systemic inflammatory response syndrome

A new concept (introduced in 1989) is systemic inflammatory response syndrome (SIRS). Since its introduction, 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 or 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 four primary symptoms associated with SIRS include:

- 1 temperature $<36^{\circ}\text{C}$ or $>38^{\circ}\text{C}$,
- 2 heart rate $>90/\text{min}$,
- 3 respiratory rate $>20/\text{min}$,
- 4 deranged arterial blood gases, i.e. $\text{PaCO}_2 <32$ mmHg, and white blood count $>12.0 \times 10^9/\text{L}$ or $<4.0 \times 10^9/\text{L}$ 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, which damage 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. One of the corollary concepts that has 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-operative management. Using a careful review of the surgical literature, a team of neurosurgeons using standards, guidelines and options (Eddy) has determined that some of the 'standard' treatment cannot be justified by the literature. An example is hyperventilation, which is almost routinely used 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 in order to increase cerebral perfusion pressure. Such practice guidelines are extremely useful to emergency physicians and surgeons.

3.3.4 Specific organ injury

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 echo, but it is probably still best to consider arteriogram the gold standard. Aortic disruption is further complicated by the best methodology to avoid paraplegia. It is most likely

that it cannot be avoided entirely because it probably represents an anatomic anomaly in about 5–8 per cent of the population and there is currently no way to predict this 5–8 per cent and then to optimally prevent it. Splenic injuries remain controversial, but have been further defined. It is now clear that anatomic injury severity scales do not correlate with physiologic 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 colostomy, it has not been defined by practice guidelines precisely which patients benefit from a colostomy.

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 in which 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 intensive care setting. Although the principles are sound, extreme care has to be exercised to avoid overutilization 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 systemic inflammatory response syndrome (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 has returned to an acceptable level proved to be life saving. The concept of staging applies to both routine and 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-operative control of other injuries, e.g. fractured pelvis,
- inability to approximate the abdominal incision,
- desire to reassess the intra-abdominal contents (directed re-look),
- evidence of decline of physiological reserve:
 - temperature $<34^{\circ}\text{C}$,
 - pH <7.2 ,
 - serum lactate $> \text{mmol/L}$,
 - prothrombin time (PT) $>16 \text{ s}$,
 - partial thromboplastin time (PTT) $>60 \text{ s}$,
 - >10 units blood,
 - systolic blood pressure $<90 \text{ mmHg}$ for more than 60 min,
 - operating time $>60 \text{ min}$.

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

3.4.2.1 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,

- intra-operative or post-operative embolization,
- intravascular shunting.

TECHNIQUE (SEE ALSO SECTION 6.1.5.4: 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 to temporarily control haemorrhage with a vascular clamp placed across the hilum.

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

Contamination control

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

- ligation or stapling of the bowel,
- resection of the damaged segment with clips, clamps or staples.

Temporary abdominal closure

Close the abdomen to limit heat and fluid loss and to protect the viscera. This depends on whether the abdomen can be approximated to achieve closure. If not:

- towel clips,
- Bogota bag (also known as temporary silo),
- Opsite® 'Sandwich' ('Vacpac'),
- mesh closure (e.g. with Vicryl® mesh).

The timing of the transfer of the patient from the operating theatre to the ICU is critical. Prompt transfer is cost-effective; premature transfer is counter-productive. 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 acido-

sis, hypothermia and hypoxia, and it may be possible to improve the coagulation status through these methods alone.

Adequate time should still be allowed for this, following which reassessment of the abdominal injuries should take place, as it is not uncommon to discover further injuries or ongoing bleeding.

3.4.3 Stage 3. Physiological restoration in the ICU

Priorities in the ICU are:

- The restoration of body temperature:
 - passive rewarming using warming blankets, warmed fluids etc.,
 - active rewarming with lavage of chest or abdomen.
- Correction of clotting profiles:
 - blood component repletion.
- The optimization of oxygen delivery:
 - volume loading,
 - haemoglobin optimization to 12 g/dL,
 - Swann Ganz pulmonary artery wedge pressure/pulmonary capillary wedge pressure (PAWP/PCWP) monitoring,
 - correction of acidosis to a pH >7.3,
 - measurement and correction of lactic acidosis to <2.5 mmol/L,
 - inotropic support as required.
- Avoidance of the abdominal compartment syndrome (ACS):
 - measurement of intra-abdominal pressure (IAP):

(a) Foley (bladder) catheter

(b) 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 with persistent bleeding despite correction of the other parameters merit early return to the theatre. Transfusion requirement of greater than 4 units in the normothermic patient is an indicator of

ongoing bleeding, and mandates re-operation.

Patients who develop major ACS must be re-looked early and any further underlying causes corrected.

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

The re-look operation should be carried out thoroughly, suspecting further, previously undiagnosed abdominal injury. If the patient's physiological parameters deteriorate again, 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

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3.5 ABDOMINAL COMPARTMENT SYNDROME (ACS)

3.5.1 Introduction

Raised intra-abdominal pressure (IAP) has far-reaching consequences for the physiology of the patient. The syndrome that results – ACS – has become a topic of interest to surgeons and intensivists in the last few years.

3.5.2 Definition

The concept of ACS was first reported by Fiestman in 1989, in four patients bleeding following aortic surgery.

To make the diagnosis requires at least three of the following:

- 1 appropriate clinical scenario (liver packing or large pelvic haematoma),
- 2 increased IAP (usually >20 mmHg – often >25 mmHg),
- 3 increase in $PaCO_2$ >45 mmHg,
- 4 decrease in tidal volume and rise in airway pressure.

3.5.3 Pathophysiology

The incidence of increased IAP (which we take as >18 mmHg) is 30 per cent of post-operative general surgery patients in intensive care units (ICUs). After emergency surgery, the incidence is even higher. The causes of acutely increased IAP are usually multifactorial. The first clinical post-operative reported cases of increased IAP were often after aortic surgery with post-operative haemorrhage from the graft suture line. In patients with peritonitis and intra-abdominal sepsis, tissue oedema with ileus is the predominant cause of increased IAP. Raised IAP in trauma patients is often due to a combination of both blood loss and tissue oedema. Patients suffering trauma and those requiring surgery for trauma constitute one of the commonest subsets of patients to develop intra-abdominal hypertension and ACS.

The common causes of increased IAP are shown below.

3.5.4 Causes of increased intra-abdominal pressure

- Tissue oedema secondary to insults such as ischaemia and sepsis, or over-transfusion.
- Paralytic ileus.
- Intraperitoneal or retroperitoneal haematoma.
- Ascites.
- Pneumoperitoneum.

3.5.5 Effect of raised intra-abdominal pressure on individual organ function

3.5.5.1 RENAL

In 1945, in a study of 17 volunteers, Bradley 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 be different, and the exact cause of renal dysfunction in the ICU is not clear, due to the complexity of critically ill patients. In a recent study, we found that out 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, as investigators have placed ureteric stents with no improvement in function. Other factors that may contribute to renal dysfunction include humeral factors and intra-parenchymal renal pressures.

The absolute value of IAP that is required to cause renal impairment is probably in the region of 20 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 central venous pressure (CVP), systemic vascular resistance, pulmonary artery pressure and pulmonary artery wedge pressure. 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 above 12 mmHg. In addition, recent studies of patients under-

going laparoscopic cholecystectomy show up to a four-fold increase in renin and aldosterone levels.

3.5.5.3 RESPIRATORY

In association with increased IAP, there is diaphragmatic stenting, exerting 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. Hypercarbia 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 ICU can sometimes be life threatening, requiring urgent abdominal decompression. Patients with true ACS undergoing abdominal decompression demonstrate a remarkable change in their intra-operative 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 a reduction in blood flow of between 11 per cent and 54 per cent was seen in the duodenum and stomach respectively at an IAP of 15 mmHg. Animals 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 cause severe rises in intracranial pressure (ICP).

3.5.6 Measurement of intra-abdominal pressure

The gold standard for IAP measurement involves using 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 mid-axillary line and the urinary tubing is clamped. Approximately 50 mL of isotonic saline is inserted into the bladder via a three-way stopcock. After zeroing, the pressure on the monitor is recorded.

3.5.6.1 TIPS FOR 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 size of the urinary catheter does not matter.
- The volume of saline instilled into the bladder is not critical.
- A CVP manometer system can be used, but it is more cumbersome than on-line monitoring.
- 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.

3.5.7 Treatment

3.5.7.1 GENERAL SUPPORT

The precise management of IAP remains somewhat clouded by many published anecdotal reports and uncontrolled series. Aggressive non-operative intensive care support is critical to prevent the complications of ACS. This involves careful monitoring of the cardiorespiratory system and aggressive intravascular fluid replacement.

3.5.7.2 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. In a prospective review of 88 post-laparotomy patients, we found that those with an IAP of 18 mmHg had an odds increased ratio for intra-abdominal sepsis of 3.9 (95% CI: 0.7–22.7). Abdominal evaluation for sepsis is a priority and this obviously should include a rectal examination as well as investigations such as ultrasound and CT scan. Surgery is the obvious mainstay of treatment in patients whose rise in IAP is due to post-operative bleeding.

3.5.8 Surgery for raised intra-abdominal pressure

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 over-statement and is not supported by level I evidence.

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

Wittman, in two separate studies in 1990 and 1994, prospectively evaluated outcomes in 117 and 95 patients respectively. A multi-institutional study of 95 patients concluded that a staged approach to abdominal repair with temporary abdominal closure (TAC) was superior to conventional techniques for dealing with intra-abdominal sepsis. Torrie and colleagues from Auckland reported their experience with 64 patients (median APACHE II score 21) undergoing TAC and found the mortality to be 49 per cent.

Indications for performing TAC include:

- abdominal decompression,
- to 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 fluid bags, Velcro, silicone and zips. Whatever technique is used, it is important that effective decompression be achieved with adequate incisions.

3.5.9 Tips for surgical decompression

- Early investigation and correction of the cause of raised IAP.
- On-going 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 and two suction drains placed laterally to facilitate fluid removal from the wound.
- If the abdomen is very tight, pre-closure with a silo should be considered.

Unfortunately, clinical infection is common in the open abdomen and is usually polymicrobial. Particular care needs to be taken in patients undergoing post-aortic surgery as 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, although this is often not possible due to persistent tissue oedema.

3.5.10 The future

The concept of IAP measurement and its significance are increasingly important in the ICU and it is 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.

3.5.11 Recommended reading

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3.6 CLOSURE OF THE ABDOMEN

3.6.1 Objectives

- The general principles of abdominal closure.
- The various techniques and indications for their use.
- Understanding the indications for re-operation or 're-laparotomy' ('second-look laparotomy').

3.6.2 Introduction: general principles of abdominal closure

In the majority of 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 adequately for the closure. This preparation includes:

- careful evaluation of the 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 Halstead's five principles of wound closure at this stage, 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 Eastridge (1997) 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 thus the need for speedy closure).
- 2 The amount of blood loss both prior to and during operation.
- 3 The volume of intravenous fluid administered.
- 4 The degree of intra-peritoneal 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), the subcutaneous tissue and the skin is obviously the desirable goal, and may be achieved when the conditions outlined above are optimal – that is, a stable patient with minimal blood loss and volume replacement, no or minimal contamination, no significant intercurrent problems and 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. The use of *retention sutures* should be avoided at all costs, and a wound that seems to require these is not suitable for primary closure.

Remember the 'one centimetre–one centimetre' rule as described by Leaper et al. (the so-called 'Guildford technique', Figure 3.1). This spacing seems to minimize tension in the tissues, and thus also minimizes the compromise of circulation in the area, and uses 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. The latter have the advantage of speed and, while being less haemostatic, nevertheless allow for a greater degree of drainage past the skin edges and less tissue reaction.

3.6.4.2 TEMPORARY CLOSURE

Delayed or temporary closure is elected when any combination of the factors listed above exists. In particular, multiply injured patients 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 contamination 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.

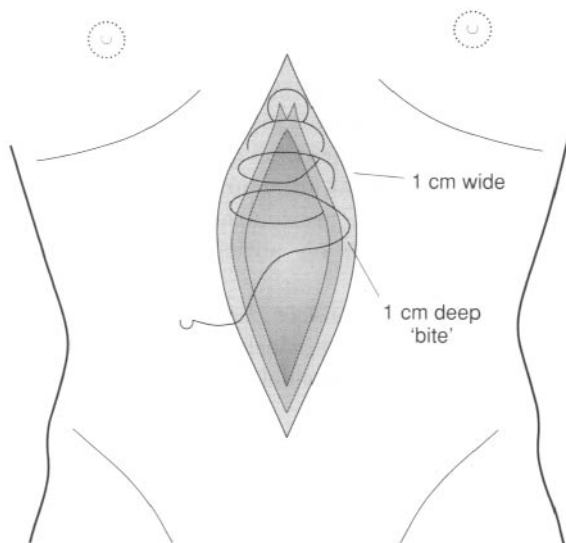


Figure 3.1 Skin closure – the 'one centimetre–one centimetre' rule.

3.6.4.3 LAPAROSTOMY

Laparostomy or open fascial closure has been developed for such circumstances. A variety of materials is used to provide an artificial component to the abdominal wall in this situation. These include:

- Non-absorbable meshes (Marlex and Mersilene): these 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 acid and membranes (PTFE or Gore-Tex®): these elicit minimal tissue reaction and in-growth and thus the risk of infection or fistula formation, but are considerably more costly.
- An acceptably cheap and readily available membrane-type material is the sterile polyethylene intravenous infusion bag cut open.

The membrane or mesh is utilized as an on-lay, with the margins 'tucked in' under the edges of the open sheath and the membrane fixed to these edges with monofilament interrupted or continuous suturing to maintain its position and prevent extrusion of the viscera around the edges.

The appreciable drainage of serosanguinous fluid that occurs is best dealt with by placing a pair of drainage tubes (e.g. sump-type nasogastric tubes or closed-system suction drains) through separate stab incisions onto the membrane and utilizing continuous low-vacuum suction.

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

Rotondo and Schwab have recently described the use of a folded sterile towel drape to reinforce the packing effect in those cases with marked oedema of the bowel in the 'quick-out' situation.

The use of laterally placed relaxing 'counter-incisions' should seldom be necessary to achieve an acceptable degree of approximation with this techniques 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.

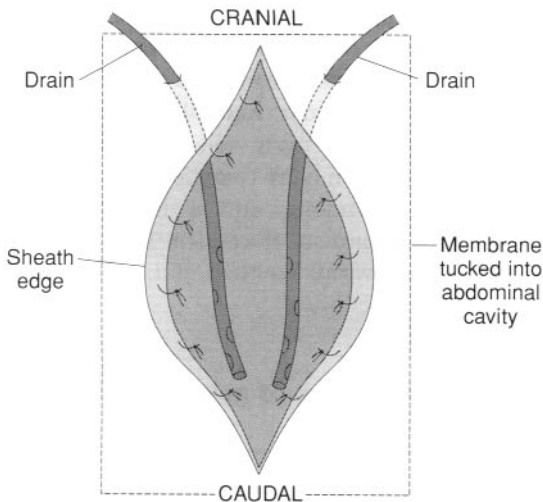


Figure 3.2 Dealing with drainage of serosanguinous fluid.

3.6.4.4 DELAYED CLOSURE

Delayed closure will be elected in the presence of risk for subcutaneous sepsis where primary sheath closure has nevertheless been made. This is usually undertaken by secondary suturing after an interval of 72 hours (or longer). Skin sutures may be placed but left untied at the primary operation, with an antiseptic dressing under an occlusive incise drape, or the skin edges may be approximated with sterile wound tapes of the Steristrip type.

3.6.5 Damage control and the 'quick out'

In the grossly unstable or coagulopathic case in which rapid and basic control of bleeding (usually by packing) and contamination has been undertaken, equally rapid temporary closure of the abdomen with subsequent re-operation for definitive procedures is a necessity.

The primary objectives of closure in such a case are the swift approximation of the wound edges to maintain the viscera within the abdominal cavity, the prevention of intra-abdominal contamination and the maintenance of intra-abdominal tamponade by the packing.

One example of such a 'quick out' technique is the use of Backhaus-type towel clips to oppose the skin and subcutaneous tissue, and preferably the superficial layer of the fascial sheath. The wound is then covered by an occlusive dressing sheet. Suction drain tubes as described above may also

be employed in this situation to minimize fluid leakage and maintain the integrity of the dressing.

3.6.6 Re-laparotomy

The circumstances under which re-laparotomy is undertaken have been touched on above. However, it is worthwhile to recollect that the 'second look' may be either:

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

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 more accurately to define 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) and ISS – are currently underway.

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3.7 MASSIVE LIMB TRAUMA: LIFE VERSUS LIMB

Injuries to the musculoskeletal system often appear 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. 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 topic 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.

During the past two decades, better understanding of the injury itself and technical advances in 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 cases, early or primary ablation might even be beneficial.

Therefore the management of the mangled limb remains a vexing problem and should thus be multidisciplinary and involve the combined skills of orthopaedic, vascular and plastic and reconstructive surgeons. Poorly co-ordinated management often results in more complications, increased duration of treatment, and a less favourable outcome for the patient. Successful limb salvage is defined by the overall function and satisfaction of the patient.

3.7.1 Complications of severe open fractures

If one compares primary to delayed ablation in terms of morbidity and cost, patients with limb salvage have more complications, more operations, and a longer hospital stay than patients with early below-the-knee amputations. There is slower recuperation and decreased motivation towards gainful employment.

In a review of 53 mangled lower extremities, Bondurant and associates¹ compared primary to 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), at greater cost (\$28 964 vs \$53 462). 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.

The complication rates for severe open fractures are significant (Table 3.1).

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 into consideration ATLS® 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, using a grading score to predict which injuries might eventually require amputation.

Table 3.1 Complications of severe open fractures

Skin and soft tissue	Skin and tissue loss, wound slough, coverage failure
Bone and fracture site	Compartment syndrome with necrosis of muscle/nerve injury Deep infection – acute/chronic Bone loss, delayed union, malunion/loss of alignment, non-union fixation problems – failure of hardware Bone re-fracture
Nerve	Direct injury or ischaemic damage Reflex sympathetic dystrophy
Vascular	Arterial occlusion, venous insufficiency Deep vein thrombosis, compartment syndrome
Joint motion	Associated joint surface fracture Contracture, late arthritis
Secondary	Ototoxicity, nephrotoxicity, myonecrosis from antibiotics Secondary spread of infection, sepsis/multiple organ failure/death
Psychosocial	Depression, loss of self-worth Economic hardship, questionable employment status, marital problems
Functional	Chronic pain Disability – muscle strength/endurance Decrease in activities of daily function Loss of ability to return to work, inability to participate in recreational activities
Cosmesis	Scars, bulky flaps

Table 3.2 Mangled Extremity Syndrome Index

Image Not Available

3.7.2 Mangled extremity syndrome (MES) (Table 3.2)

Gregory² retrospectively reviewed 17 patients with severe injuries (12 of the lower extremity) and proposed a mangled extremity syndrome. The injury was categorized according to the integument, nerve, vessel and bone injury. A point system quantitated injury severity, delay in revascularization, ischaemia, age of the patient, pre-existing disease and whether the patient was in shock.

If the total points are less than 20, a functional limb salvage can be expected. If the sum is greater than 20, limb salvage is improbable.

From Gregory et al.²

3.7.3 Predictive Salvage Index system (Table 3.3)

Howe et al.³ 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. Of their 21 patients, 43 per cent were amputated; the infrapopliteal injuries had the highest amputation rate (80 per cent).

It was suggested that if the score was 7 or less, the limb could be salvaged. If it was 8 or more, amputation was recommended.

Table 3.3 Predictive Salvage Index system

Table 3.4 Mangled Extremity Severity Score (MESS)

Image Not Available

Image Not Available

From Howe et al.³

3.7.4 Mangled Extremity Severity Score (MESS) (Table 3.4)

The MESS,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.

A MESS value greater than 7 predicted amputation.

3.7.5 NISSSA scoring system (Table 3.5)

Subsequently, McNamara and others⁵ 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,

From Johansen et al.⁴

ischaemia, soft-tissue injury/contamination, skeletal injury, shock/blood pressure, age) scoring system, which is thus more sensitive and more specific than the MESS.

A NISSSA score of greater than 11 predicted amputation.

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-threatening 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

Table 3.5 NISSSA scoring system

Image Not Available

From McNamara et al.⁵

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 as to 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 insensate 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.

3.7.6 References

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3.8 RESUSCITATION PRIORITIES: PAEDIATRICS

3.8.1 Introduction

The desired endpoints of paediatric resuscitation are identical to those sought for the adult trauma patient. 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 in the paediatric patient before they can be safely performed in the stress of a resuscitation situation. The requirement to use different equipment or smaller sizes of familiar equipment places additional stresses on the responsible practitioner and, indeed, the entire resuscitation team. The practitioner who undertakes to be responsible for the care of traumatized children must be a surgeon or, preferably, a paediatric surgeon, who is cognisant of their special needs and experienced in the practical delivery of the appropriate care. The need for referral should

be considered as soon as the patient will tolerate safe transfer to an appropriate facility.

3.8.2 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 due to dogged determination to achieve certain steps of resuscitation 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.

3.8.3 Resuscitation room

3.8.3.1 AIRWAY

The indications for airway control are identical to those of the adult patient. The clinical assessment of cervical spine injury is less reliable in the fearful, unco-operative 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. The greatest pitfalls are incorrect endotracheal tube placement, too small an endotracheal tube, and tube dislodgement due to 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.

3.8.3.2 VENTILATION

Hypoventilation is a prominent cause of hypoxia in the injured child. Because 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. The airway of the obligate nasal breather (the neonate or infant) must not be compromised with a nasogastric tube.

Once a controlled and monitored situation is obtained, one should avoid both barotrauma and volume trauma by providing about 6 mL/kg body weight tidal volume at the smallest pressure. Usually it is safer to permit mild to moderate hypercapnia (permissive hypercapnia) than to cause acute over-ventilation lung injury.

3.8.3.3 CIRCULATION

Most patients have hypovolaemic shock. Other forms of shock are more rare in children than in adult patients. The primary management of bleeding is surgical haemostasis. Most children respond rapidly to crystalloid resuscitation. *Do not delay the transfer of the unstable child to the operating room: establish good access and resuscitate in theatre while the surgeon stops the bleeding. Here, the anaesthetist is in charge!*

3.8.3.4 HAEMODYNAMICS

The small child has an effective compensation mechanism for hypovolaemic shock, dependent predominantly on an adequate heart rate, but a tendency to very rapid and calamitous decompensation. Tachycardia, peripheral vasoconstriction and signs of inadequate central nervous system perfusion predominate. The child may not necessarily be hypotensive. The urinary catheter is an invaluable aid to determine adequacy of resuscitation. The practitioner must recognize and treat shock aggressively. A heart rate of 60–80 beats per minute in an infant represents a life-threatening bradycardia and requires external cardiac massage.

3.8.3.5 VASCULAR ACCESS

Rapid vascular access is obtained, tailored to the severity of the child's shock and the practitioner's experience. Central lines are reserved for the larger child and more experienced operator.

3.8.3.6 FLUIDS AND BLOOD TRANSFUSION

Boluses are administered according to body weight and assessment of response. *Hypothermia* is assiduously avoided.

3.8.3.7 CARDIAC ARREST

In children, cardiac arrest usually is not 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.

3.8.3.8 RESUSCITATIVE THORACOTOMY

Resuscitative thoracotomy is usually futile and is not recommended for blunt trauma, but should be considered for children with witnessed cardiac arrest and penetrating thoracic injury.

3.8.3.9 NEUROLOGICAL ASSESSMENT

In general, children have a lower incidence of intracranial mass lesions requiring surgical drainage after blunt injury, compared to adults. The child with no localizing neurological signs, even with a severe head injury, who is haemodynamically unstable should not have surgery delayed in order to obtain a CT scan of the brain. Signs of transtentorial herniation – 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 CT scan and prompt neurosurgical management that supersedes all other priorities except the management of airway and treatment of shock. It may be necessary for the neurosurgeons and abdominal or orthopaedic surgeons to operate in two teams.

3.8.4 Recognition of injury patterns

Certain injury patterns of paediatric trauma are becoming apparent. It is important to take an accurate history of the mechanism of injury in order to detect associated injuries during the resuscitation stage.

The following complexes of injury mechanisms are associated with a particularly high incidence of injuries.

- 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.

3.8.5 Organ system injury: priorities

- Abdominal blunt trauma takes a lesser priority in a stable patient or in an 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 without 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.

3.8.6 Analgesia

Appropriate doses of morphine – 0.1 mg/kg 4-hourly or when required – greatly facilitate resuscitation and assessment and do not mask important clinical signs, but rather improve the patient's co-operation.

3.9 RESUSCITATION PRIORITIES: THE ELDERLY

3.9.1 Definition

Particularly in the more developed world, the population is ageing. In the United States in 1990, those aged over 65 accounted for 12.5 per cent of the population, and in 2040 it is expected that this will rise to >20 per cent of the population.

The definition of 'elderly' varies. Whereas conventionally the term is used to describe an age of 65 years or more, in trauma scoring systems the break-point for the elderly is 55 years of age. In the United States, the 12.5 per cent of the population over the age of 65 accounts for almost one-third of all deaths from injury.

3.9.2 Physiology

Age is associated with the following organ system characteristics.

3.9.2.1 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.
- Co-existing commonly prescribed medication can blunt normal physiological responses.

3.9.2.2 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.

3.9.2.3 NERVOUS SYSTEM

- Progressive atrophy of the brain.
- Deterioration of cerebral and cognitive functions:
 - cognition
 - hearing
 - eyesight
 - proprioception.

3.9.2.4 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).

3.9.2.5 MUSCULOSKELETAL

- Osteoporosis causing fracture in the presence of minimal energy transfer.
- Diminution of vertebral body height.
- Decrease in muscle mass.

3.9.3 Influence of co-morbid conditions

In addition to the typical changes listed above, the development of disease states commonly associated with the elderly can have a significant impact on

the response to injury. These disease states can include the following.

- Cardiac disease, including hypertension.
- Metabolic disease:
 - diabetes mellitus
 - obesity (Body Mass Index (BMI) >35).
- Liver disease.
- Malignancy.
- Pulmonary disease.
- Renal disease.
- Neurological or spinal disease.

3.9.4 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 any alteration in mental status is associated with brain injury, and only accept age-related deterioration after exclusion of injury.
- Be aware of the potential for poorer outcomes and sudden physiological deterioration.
- Be aware of the distinction between aggressive care and futile care.

3.9.5 Recommended reading

Kauder DR. Geriatric trauma. In Peitzman AB (ed.), *The Trauma Manual*, 2nd edition. Philadelphia, PA: Lippincott Williams and Wilkins, 2002, 469–76.

3.10 FUTILE CARE

In every environment there are circumstances in which the provision of adequate health care may not alter the outcome. Providing such health care may cause a significant drain on the resources available and denial to others of adequate care as a result. This 'rationing' of health care may be the result of operating theatres being in use, and consequently not available, inadequate numbers of 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 should be fully discussed with associated staff and family members.

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.

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Part III

Specific organ injury

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4.1 OVERVIEW

4.1.1 Introduction

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

Before World War II, non-operative management of penetrating neck trauma resulted in mortality rates of up to 15 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, because 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 require prompt exploration. However, initial assessment and management of these patients should be carried out according to Advanced Trauma Life Support® (ATLS®) Course principles.

A characteristic of neck injuries is rapid airway obstruction, and often difficult intubation. The key to management is early intubation. 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. 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.

NB. 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 due to the presence of blood, may result in catastrophe. Ideally, local anaesthetic spray should be used with sedation, and a cricothyroidotomy below the injury should be considered when necessary.

Control of haemorrhage should be done by direct pressure where possible. If the neck wound is not bleeding, do not probe or finger the wound, as a clot may be dislodged. If the wound is actively bleeding, the bleeding should be controlled by digital pressure or, as a last resort, a Foley catheter.

Patients with signs of significant neck injury, or who are unstable, should be explored urgently once rapid initial assessment is completed and the airway is 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.

4.1.2.2 INJURY LOCATION

Division of the neck into anatomic zones 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, the trachea, the oesophagus, the thoracic duct, the upper mediastinum and lung apices.

Zone II includes the area between the cricoid cartilage and the angle of the mandible. Enclosed within its region are the carotid and vertebral arteries, jugular veins, pharynx, larynx, oesophagus and trachea.

Zone III includes the area above the angle of the mandible to the base of the skull 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 zone I or zone III injuries can be difficult, and therefore the diagnostic workup may be more extensive than for zone II injuries (Figure 4.1).

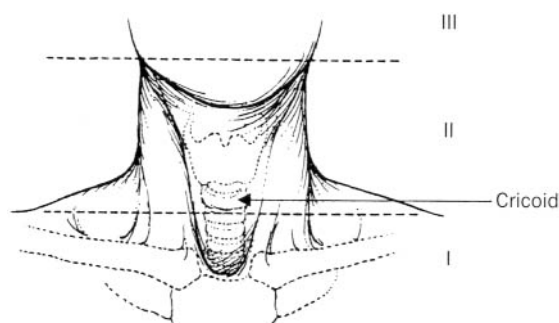


Figure 4.1 Zones of the neck.

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 due to cavitation.

4.1.2.4 FREQUENCY OF INJURY

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

4.1.3 Mandatory versus selective neck exploration

Recommendations for the management of patients with penetrating cervical trauma depend on the zone of injury and the patient's clinical status. If the

platysma is not penetrated, the patient may be observed. Mandatory exploration for penetrating neck injury in patients with signs of vascular or aerodigestive tract injury is still appropriate.

Mandatory exploration of all stable patients is controversial. Significant morbidity and mortality due to missed visceral injuries as well as the negligible morbidity caused by a 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-operative evaluation has been recommended. It is clear that missed injuries are associated with high morbidity and mortality.

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

4.1.4 Use of diagnostic studies

In the stable patient without 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 ARTERIOGRAPHY

Especially in zone I or zone III injuries, where surgical exposure can be difficult, angiography is invaluable to plan the conduct of operation. Arteriography should visualize both the carotid and vertebral arteries on both sides.

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

4.1.4.2 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 nearly 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 anatomic zones

4.1.5.1 VASCULAR INJURIES

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 tied in a knot, transected and left in place until the vessel has fully thrombosed. 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 patient 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. This is especially true 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 intra-operative decisions are influenced by the patient's pre-operative neurologic status. If the patient has no neurologic deficit pre-operatively, 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 embolization or haemorrhagic infarction.)
- Operative management of the patient with carotid injury and a pre-operative neurologic 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 pre-operative neurologic deficits and without evidence of retrograde flow at the time of operation. Thus, all carotid artery injuries should be repaired if technically feasible, except for those of patients in coma.

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 sternotomy and supraclavicular extension. The location of the vascular injury will dictate the definitive exposure. For right-sided great vessel injuries, median sternotomy with supraclavicular extension allows optimal access. On the left side, left anterolateral thoracotomy may provide initial proximal control. Further operation for definitive repair may require sternotomy, or extension into the right side of the chest, or up into the neck. Trap-door incisions, although recommended, are often difficult and are not commonly required. Never make the operation more difficult than necessary by inadequate exposure. Care must be taken to avoid injury to the phrenic and vagus nerves as they enter into the thorax. In the stable patient in whom the vascular injury has been confirmed by arteriography, the right subclavian artery or distal two-thirds of the left subclavian artery can be exposed through an incision immediately superior to the clavicle,

and resection of the medial half of the clavicle performed. However, in an unstable patient, this approach is inappropriate.

- Injuries to the internal jugular vein are 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 on arteriographic studies. These rarely require repair. Operative exposure may be difficult, and interventional radiology will usually be needed. If a vertebral artery injury is found at operation, pack the area. If this tamponades the bleeding, plan transfer from the operating room to the radiology suite for arteriography and embolization of the vertebral artery.

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 pre-operatively. 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 control of the airway.
- The next concern is to stop bleeding, either by digital pressure or by the use of a Foley catheter.
- The stability of the patient decides the appropriate diagnostic and treatment priorities.
- Adequate exposure of the area involved is critical.

4.2 ACCESS TO THE NECK

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

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 pre-operatively. 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, 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 (Figures 4.2 and 4.3). Fortunately, this is rarely required.

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, 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

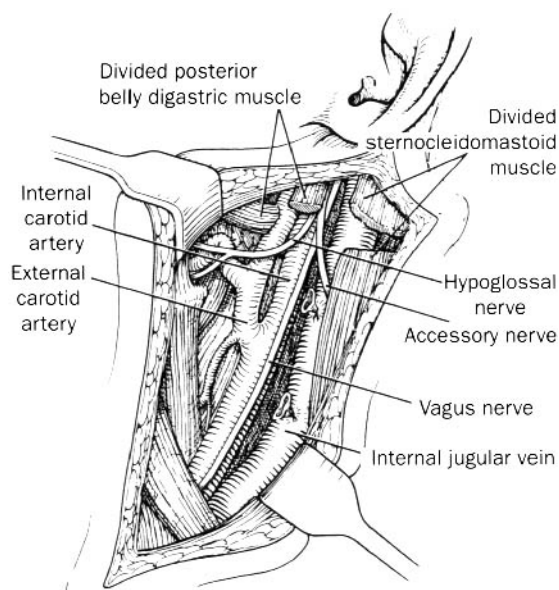


Figure 4.2 Approach to the left side of the neck with divided sternomastoid and digastric muscles.

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 stern-

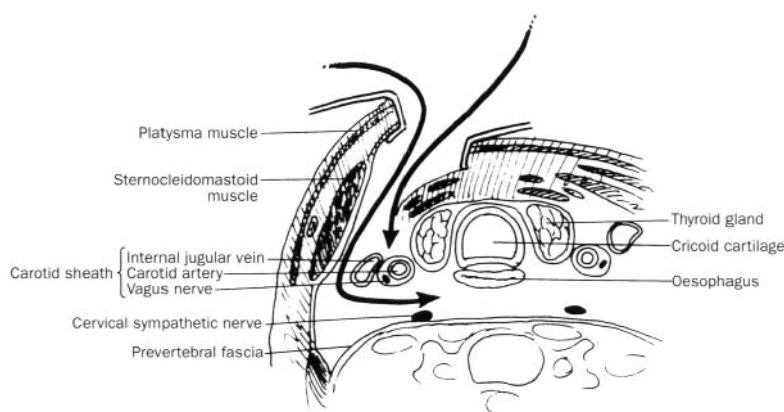


Figure 4.3 Approach to the neck showing retraction of platysma and sternomastoid muscles.

ocleidomastoid is divided and the supraclavicular fat pad is cleared by blunt dissection. This reveals the scalenius anterior muscle, with the phrenic nerve crossing it from the lateral side. Division of scalenius 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, as 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 are 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 cervicus 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 homeostasis. 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 United States during the early 1990s, there were approximately 180 000 deaths per annum from injury. Several investigators have shown that 50 per cent of fatalities are due to primary brain injury, 25 per cent to 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 following chest injury, usually the long-term consequences of hypoxic brain damage.

There are a number of important points in this regard.

- A definite proportion of these deaths occur virtually immediately (i.e. at the time of injury), for example rapid exsanguination following 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 mis-assessment 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) 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 require definitive surgery.
- Emergency room 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 as a consequence of 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 by 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.

- 1 *Immediately life-threatening injuries:*
 - airway obstruction due to any cause but specifically with laryngeal or tracheal disruption with obstruction or extensive facial bony and soft tissue injuries,
 - impaired ventilation due to tension pneumothorax, open pneumothorax or flail chest,
 - impaired circulation due to massive haemothorax or pericardial tamponade,
 - air embolism.
- 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 pathophysiologic 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 due to 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 mis-assessment 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 due to knives, missiles and impalement. Knife wounds and impalement usually involve low-velocity penetration, and mortality is directly related to the organ injured. Secondary effects such as infection are due to the nature of the weapon and the material (i.e. clothing and other foreign material) that the missile carries into the body tissue. Infection is also influenced by spillage of contents from an injury to a hollow viscous organ. In contrast, missile injuries can cause more extensive tissue destruction, related to the kinetic energy (KE), which is expressed as:

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

where M = mass and V = velocity.

Perhaps more appropriate is the concept of 'wounding energy' (WE), expressed as:

$$WE = \frac{1}{2} M (V_{EN} - V_{EX})^2$$

where M = mass, V_{EN} = velocity on entry and V_{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, upon 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, and 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 will require a fairly aggressive approach, particularly 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.²

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 with bleeding 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 of a transmediastinal injury, bilateral chest tubes may be indicated. X-ray is *not* required to insert a chest tube, but *is* useful after the chest tubes have been inserted to confirm proper placement.

For patients who are haemodynamically stable, X-ray remains the gold standard for the diagnosis of pneumothorax or haemothorax. In these patients it is preferable to have the X-ray completed before placement of a chest tube. The decrease in air entry may not be due to a pneumothorax but, especially following blunt injury, may be due to 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

This is the bony 'cage' constituted by the ribs, thoracic vertebral column and sternum, with the clavicles anteriorly and the scapula posteriorly (Figure 5.1). The associated muscle groups and vascular structures (specifically the intercostal vessels and the internal thoracic vessels) are the further components.

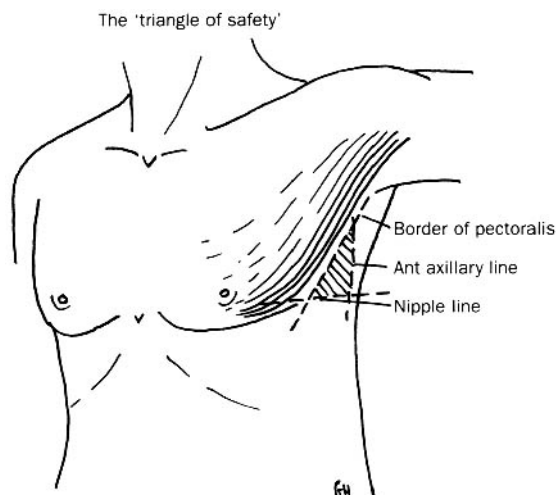


Figure 5.1 The chest wall. Remember the 'safe area' of the chest. This triangular area is the thinnest region of the chest wall in terms of musculature. This is the area of choice for tube thoracostomy insertion. In this area, there are no significant structures within the walls that may be damaged; however, take note of the need to avoid the intercostal vascular and nerve bundle on the undersurface 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. It 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 it can move up to 10 cm in deep breathing. During maximum expiration, the diaphragm may rise as high as the 5th intercostal space. Thus, any injury below the 5th intercostal space may involve the abdominal cavity.

5.1.5.3 THE CHEST CONTENTS

These are (Figure 5.2):

- 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 I of the neck.

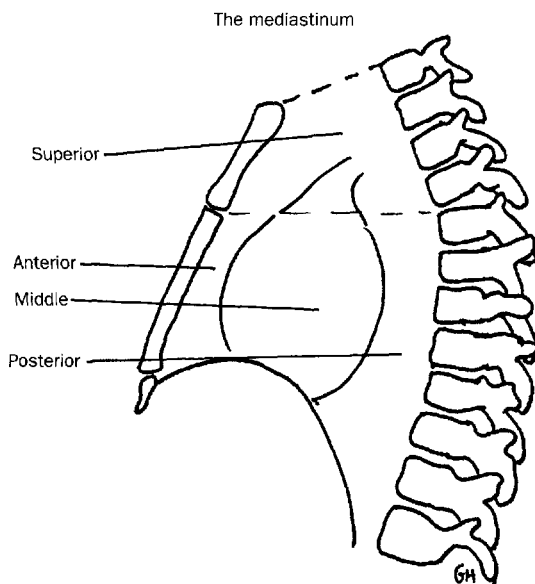


Figure 5.2 The chest contents.

Tracheobronchial tree

The trachea extends from the cricoid cartilage at the level of the 6th cervical vertebra to the carina at the level of the upper border of the 5th thoracic vertebra, where it bifurcates. The right main bronchus is shorter, straighter and at less of an angle compared to the left side. It lies just below the azygos–superior venacaval 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 superiorly 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. The visceral pleura is intimately applied to the surface of the lungs, and is

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 3rd costal cartilage to the xiphisternal junction. The majority of the anterior surface of the heart is represented by the right atrium and its auricular appendage superiorly, and by 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 less than 50 mL and up to more than 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, which is approximately 25 cm long, extends from the pharynx to the stomach. It starts at the level of the 6th cervical vertebra, and passes through the diaphragm about 2.5 cm to the left of the midline at the level of the 11th thoracic vertebra. The entire intra-thoracic 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 cisterna chyli overlying the 1st and 2nd lumbar vertebrae. It lies posteriorly and to the right of the aorta. It 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.

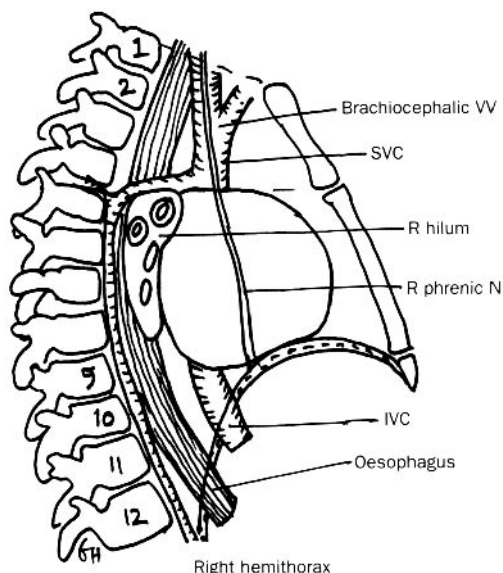


Figure 5.3 The right mediastinum.

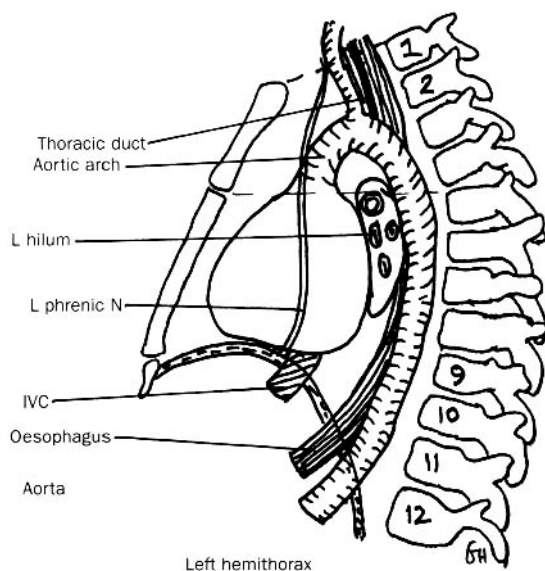


Figure 5.4 The left mediastinum.

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 co-operate 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. However, it is 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 X-ray is obtained early for 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 track. It is good practice to place metallic objects such as paper clips on the skin pointing to the various wounds on the chest wall to help determine the missile track. This 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.

Ultrasound has a role primarily in determining whether or not a patient has pericardial blood. Using the focused abdominal sonography for trauma (FAST) technique, pericardial blood can be detected. Similarly, transoesophageal echo is a useful adjunct in determining whether tamponade is present in the haemodynamically stable patient. Computed tomography is not routinely used in patients with penetrating chest injury. It might have some utility in determining the extent of pulmonary contusion caused by higher velocity injuries or shotgun blasts, but it 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 5th 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 wheal of 1 per cent lignocaine is placed in 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 about 2 cm onto 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 with 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 FG) 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, which may require operative 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 of these should be well under 1 per cent.

Routine antibiotics are not a substitute for good surgical technique.³

5.1.8.2 NON-OPERATIVE MANAGEMENT

As noted above, non-operative management can be used in the majority of 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-operative 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.⁴ Laparoscopy may have a small advantage in that, if the diaphragm has been penetrated, it also allows some assessment of intra-peritoneal 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 intra-peritoneal 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 their removal. Optimally, this should be done within 72 hours of injury, before the clot becomes too adherent to be removed safely 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 over-emphasized. 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 that the patient is not in a lateral decubitus position that would compromise exploration of the peritoneal cavity or pelvis. The surgeon must be comfortable about 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 can 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 take 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 5th intercostal space is preferred. This is usually 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 in whom 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 intra-thoracic viscera. Occasionally, a right anterolateral

thoracotomy is indicated in emergencies if air embolism is suspected (see section 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 5th or 4th intercostal space for a left anterolateral thoracotomy. Care should be taken in female patients 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-angle 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 (Marcain®) 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 post-operative period. It can then be supplemented with a thoracic epidural, if necessary, after the initial 12 hours of the post-operative period.

ERT is indicated in the agonal or dying patient with thoracic injuries.^{5,6} 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 pre-hospital setting and arrive with an electri-

cal 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 following civilian trauma is quite low, certainly less than 1 per cent of all major thoracic injuries.⁷ 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, and thin plastic sheets, 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, and removal of all foreign bodies, including clothing, wadding from shotgun shells or debris from the object that penetrated the chest. The majority 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 (ICU) and returned to the operating room in 24–48 hours for a free myocutaneous graft or alternative reconstruction. Complications include wound infection and respiratory insufficiency, the latter usually being due to 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 either dead or dying. The importance of making the diagnosis is that it is the most easily treatable life-threatening surgical emergency in the emergency department. '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 emergency department. 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 hilar, great vessel or cardiac wounds expire immediately after injury. Another 25 per cent live for periods of 5–6 minutes and, in urban centres, some of these patients may arrive alive in the emergency department after rapid transport. The remaining 25 per cent live for periods of up to 30 minutes and it is this group of patients that may arrive alive in the emergency department 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. Chest X-ray 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, the tube should be imme-

diately clamped and autotransfusion 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 X-ray.

The treatment of massive haemothorax is to restore blood volume. Essentially, all such patients will require thoracotomy. In approximately 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, tractotomy (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.⁸ If it is a distal bronchus, there may be persistent air leak for a few days, but it will usually close with chest tube drainage alone. If, however, there is 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's stain of the sputum and chest X-rays when necessary. If the Gram's stain shows the 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.⁹ It occurs in 4 per cent of all major thoracic trauma. Sixty-five per cent of the cases are due to penetrating injuries. 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, which 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: with focal or lateralizing neurological signs, sudden cardiovascular collapse, or 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 can occasionally 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 have air embolism. When a patient comes in to the emergency room 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 room. In the majority of patients, the left or right chest is opened, depending on the side of penetration. If a resuscitative thoracotomy has been carried out, it may be necessary to extend this across the sternum into the opposite chest if there is no parenchymal injury to the lung on the left. Definitive treatment is to over-sew the lacerations to the lung, in some instances 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. 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 ascending 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 secondary to penetrating trauma.

Cardiac injuries

In urban trauma centres, cardiac injuries are most common 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, with acute heart failure. Patients with tamponade due to penetrating injuries usually have a wound in proximity, decreased cardiac output, increased central venous pressure, 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 sustained interventricular septal defects, but represent less than 2 per cent of all patients with cardiac injuries. Pericardiocentesis is not a very useful diagnostic technique, but may be temporarily therapeutic. In cases in which the diagnosis of pericardial tamponade cannot be confirmed on clinical signs, echocardiogram is useful.

The treatment of all cardiac injuries is immediate thoracotomy, ideally in the operating room. In the patient who is in extremis, thoracotomy in the emergency department can be life saving. The great majority of wounds can be closed with simple sutures or horizontal mattress sutures of 3/0 or 4/0. Bolstering the suture with Teflon pledgets may occasionally be required, particularly if there is surrounding contusion. If the stab wound 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 magnification if necessary. The second option is to temporarily 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, chordae tendineae or septum. In most instances, these injuries are not immediately life threatening, but become evident over a few hours or days following the injury.

Complications from myocardial injuries include recurrent tamponade, mediastinitis and post-cardiotomy syndrome. Recurrent tamponade can be avoided by placing a mediastinal chest tube or leaving the pericardium partially opened following repair. Most cardiac injuries are treated through a left anterolateral thoracotomy, and only occasionally 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 herniation 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 surgical literature.^{12,13} The reason for this is that extensive injury to the great vessels results in immediate exsanguination into the chest, and most of these patients die at the scene of injury.

The diagnosis of penetrating great vessel injury is usually 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 proximity. These patients usually have a false aneurysm or arteriovenous 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 for long enough to permit the patient to reach the emergency department alive.^{14,15}

Complications of injuries to the great vessels include re-bleeding, false aneurysm formation and thrombosis. A devastating complication is paraplegia, which usually occurs following blunt injuries but, rarely, can develop after penetrating injuries, either because of associated injury to the spinal cord or because, at the time of surgery, important intercostal arteries are ligated. 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 occur somewhat more frequently 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 despite tube thoracostomy, and pleural effusion with extravasation of contrast on gastrograffin 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.¹⁶

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 they are less than 6 hours old and there is minimal inflammation and devitalized tissue present. A two-layer closure is all that is necessary. Post-operatively, the patient is kept on intravenous support and supplemental nutrition. Antibiotics may be indicated during the 24-hour peri-operative period. If the wound is more than 6 hours old and less than 12 hours 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 hours will not heal primarily when repaired. Open drainage, antibiotics, nutritional support and consideration of diversion are the optimal management. Complications following 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.¹⁵ 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 a conventional posterolateral thoracotomy, or via 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 5th 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 that of 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 intra-peritoneal 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 sutures. 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 it 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 following major injuries 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, following 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 emergency room thoracotomy (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. However, ERT's limited success in most circumstances prohibited the use of the technique for the next six to seven decades. 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 to differentiate early on between the definitions of thoracotomy:

- thoracotomy performed in the emergency department for patients in extremis, and
- resuscitative thoracotomy; i.e. in the operating theatre or ICU minutes to hours after injury in acutely deteriorating patients.

It is also important to differentiate between:

- patients with 'no signs of life', and
- 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 intra-thoracic 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 sub-diaphragmatic haemorrhage.

ERT has been shown to be most productive in life-threatening penetrating cardiac wounds, especially when cardiac tamponade is present. Even in established trauma centres, patients requiring ERT 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.

ERT 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. Human immunodeficiency (HIV) rates amongst the patient population at the Johannesburg Hospital Trauma Unit rose 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 these risks.

5.1.9.2 INDICATIONS AND CONTRAINDICATIONS

There are instances in which 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 intra-thoracic injury, especially penetrating cardiac injury ('salvageable' post-injury cardiac arrest),
- with severe post-injury hypotension (blood pressure <60 mmHg) due to cardiac tamponade, air embolism or thoracic haemorrhage.

Less clear benefit occurs in:

- those patients presenting with moderate post-injury hypotension (blood pressure <80 mmHg) potentially due to 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 emergency department to the operating theatre and the personnel available in applying the principles related to ERT. Whereas 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 minutes,
- there has been CPR for more than 10 minutes 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 only is present in the emergency department.

5.1.9.3 RESULTS

The results of ERT vary according to injury mechanism and location and the presence of vital and life signs.

ERT 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 to 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 outcome following ERT are:

- 1 signs of life at scene absent,
- 2 signs of life in emergency department 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 emergency department. If blunt injury is present, ERT is embarked on only if pulsatile electrical activity 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 other patients 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?

ERT 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 room for definitive repair or closure.

ERT should be terminated if:

- irreparable cardiac damage has occurred,
- the patient is identified as having massive head injuries,
- pulseless electrical activity is established,
- systolic blood pressure is <70 mmHg after 20 minutes,
- asystolic arrest has occurred.

5.1.9.5 CONCLUSION

Success in the management of thoracic injury in those cases requiring operation 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.

Table 5.1 Approach to the thorax – indications for surgery

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

The surgical approaches in current use include:

- anterolateral thoracotomy,
- median sternotomy,
- bilateral thoracotomy ('clamshell' incision),
- the 'trap-door' incision,
- posterolateral thoracotomy.

In the *unstable* patient, the choice of approach will usually be an anterolateral thoracotomy or median sternotomy, depending upon the suspected injury. In the case of the *stable* patient, the choice of approach must be planned after proper evaluation and workup have clearly identified the nature of the injury.

If time permits in the *more stable* patient, intubation (or re-intubation) 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 'trap-door' incision are complex and somewhat mutilating, with significant post-operative morbidity and difficulty in terms of access and closure.

5.1.11 References

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5.2 ACCESS TO THE THORAX

5.2.1 Anterolateral thoracotomy

This is the approach of choice for most unstable patients and is utilized for ERT (Figure 5.5).

- It allows rapid access to the injured hemithorax and its contents.
- It is made with the patient in the supine position, with no special positioning requirements or instruments.
- It has the advantages that it may be:
 - extended across the sternum into the contralateral hemithorax (the ‘clamshell’ incision or bilateral thoracotomy),
 - extended downwards to create a thoraco-abdominal incision,
 - converted into a ‘trap-door’-type 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 cases initially should be a midline laparotomy, the chest being entered only if no source of intra-abdominal bleeding is found.

5.2.1.1 TECHNIQUE

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

Incision is made through the 4th or 5th intercostal space from the costochondral junction anteriorly to the mid-axillary 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 periosteum 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.

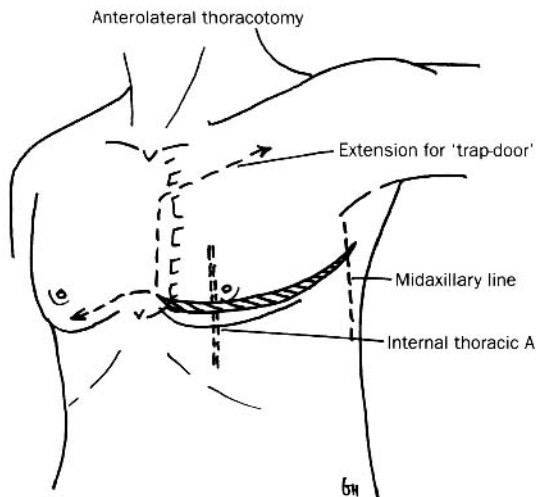


Figure 5.5 Anterolateral thoracotomy.

The periosteum 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 intra-thoracic inspection for identification of injuries is carried out after suctioning. In cases of ongoing bleeding, an autotransfusion suction device is advisable.

Note. It is important to identify the phrenic nerve in its course across the pericardium if this structure is to be opened – the pericardiectomy is made 1 cm anterior and vertical to the nerve trunk in order to avoid damage and subsequent morbidity.

5.2.1.2 CLOSURE

Following 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.

5.2.2 Median sternotomy

This incision is the approach of choice in patients with penetrating injury at the base of the neck (zone I) and the thoracic outlet (Figure 5.6).

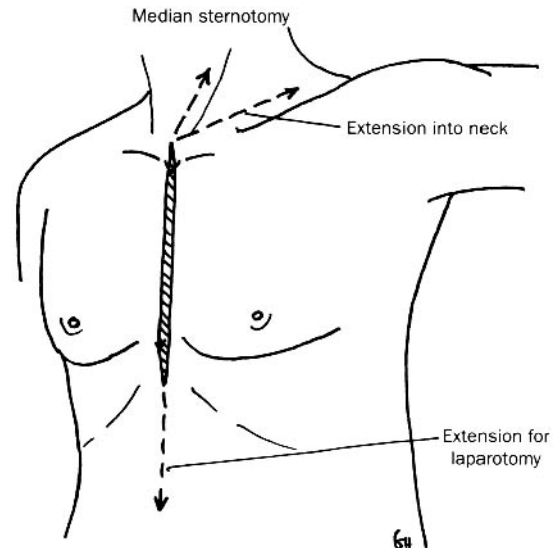


Figure 5.6 Median sternotomy.

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 a 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 post-operatively, especially in the emergency setting.

5.2.2.1 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 section (bisection) of the sternum is carried out with a saw (either oscillating or a braided wire Gigli saw) or a Lebschke knife, commencing from

above 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 the suprasternal space of Burns and must be controlled.

5.2.2.2 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.

5.2.3 Emergency department thoracotomy

5.2.3.1 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 number 20,21 blade,
- forceps,
- suitable retractor such as Finochietto's chest retractor or Balfour abdominal retractor,
- Lebschke's knife and mallet or Gigli saw for the sternum,
- large vascular clamps such as Satinski 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.

5.2.3.2 APPROACH

Two basic incisions are used in ERT. These are applied according to the best incision for the partic-

ular suspected injury (based on entrance and exit wounds, trajectory and, probably, 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 are 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 commonest site for urgent access. The incision is placed in the 5th intercostal space through muscle, periosteum and parietal pleura from the costochondral junction anteriorly to the mid-axillary line laterally, following upper border of the rib, and 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 the pleural cavities, pericardial cavity and even 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.

5.2.3.3 APPLICATIONS

Suspected cardiac injury and cardiac tamponade

Access is achieved via either lateral or midline sternotomy, the former allowing more rapid access, whereas the latter approach ensures better exposure. It is important to identify the phrenic nerve prior to 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 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 it is not beating, suturing precedes resuscitation.

Foley catheters may be used to temporarily control haemorrhage prior to definitive repair in the emergency department 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, as 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, as 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 are 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 over-sewing, segmental resection or pulmonary tractotomy.

Pulmonary tractotomy is a means of controlling tracts that pass through multiple lung segments when the extent of injury precludes pulmonary resection. It is a means of non-anatomical lung preservation in which linear staplers are passed along both sides of the tract formed, the lung is divided to allow

blood vessels and airways in the bases to be repaired, and the divided edges are then over-sewn.

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 the 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 infra-diaphragmatic haemorrhage in both blunt and penetrating trauma.

The thoracic aorta is cross-clamped inferior to the left pulmonary hilum and the area is exposed by elevating the left lung anteriorly and superiorly. The mediastinal pleura is dissected under direct vision and the aorta is separated by blunt dissection from the oesophagus anteriorly and from the pre-vertebral 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. the clamp is removed once effective cardiac function and systemic arterial pressure have been achieved, as the metabolic penalty rapidly becomes exponential beyond 30 minutes.

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

The incision extends, usually, as a 5th 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.

5.2.4 'Trap-door' thoracotomy

This is a combination of an anterolateral thoracotomy, a partial sternotomy and infraclavicular or supraclavicular incision with resection or dislocation of the clavicle (Figure 5.7).

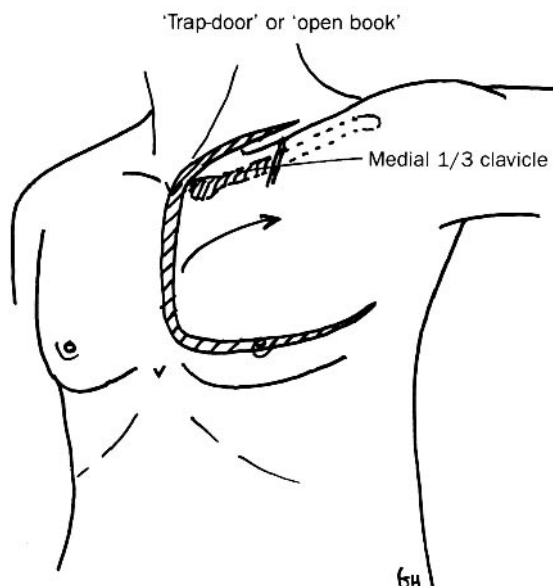


Figure 5.7 'Trap-door' incision.

It has the disadvantage of it being relatively more time consuming, and retraction of the bony 'trap door' created is often difficult. 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.

In general, this means of access is not recommended.

5.2.5 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.

5.2.6 Definitive procedures

5.2.6.1 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.

5.2.6.2 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. Whereas 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 temporising 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.

5.2.6.3 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

- This is usually performed to control massive intra-pulmonary 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 over-sewn.

Pulmonary tractotomy

- This is 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 over-sewn.
- 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.

5.2.7 Conclusion

Success in the management of thoracic injury in those cases requiring operation 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.

5.2.8 Recommended reading

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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 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 Advanced Trauma Life Support (ATLS®) 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 co-operative patient.

The diagnosis of injury following blunt trauma can be difficult and knowledge of the mechanism can be helpful. Shoulder harnesses can 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 intes-

tine and mesentery, especially when incorrectly placed above the iliac crest.

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:

- the patient with the normal abdomen,
- an equivocal group requiring further investigation,
- the patient with an obvious injury to the abdomen.

Virtually all penetrating injury to the abdomen should be explored promptly, especially in the presence of hypotension.

6.1.1.3 DIAGNOSIS

Diagnostic modalities depend on the nature of the injury:

- physical examination,
- diagnostic peritoneal lavage (DPL),
- ultrasound (focused abdominal sonography for trauma – FAST),
- computed tomography (CT) scan,
- 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:

- whether the patient has bled into the abdomen,
- whether the bleeding has stopped.

Thus, serial investigations of a quantitative nature will allow the best assessment of these patients. CT scanning 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 in which bleeding is taking place, e.g. 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. DPL remains one of the most common, most sensitive, cheapest and most readily available modalities to assess the presence of blood in the abdomen. Importantly, DPL can be performed without moving the patient from the resuscitation area. FAST is similarly useful, but is more operator dependent – which is important if the individual doing the scan is inexperienced in dealing with trauma patients. 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 intra-peritoneal in nature. This situation lends itself to FAST examination, as haemodynamic instability caused by intra-peritoneal haemorrhage is likely to be found at FAST examination, which may be somewhat quicker than DPL.

6.1.2 The abdominal contents

There are at least four complex abdominal injuries.

- 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. Unfortunately, experience comes from bad judgement.
- Pancreaticoduodenal injuries: these are challenging because of the difficulties of diagnosis, and because of associated retro-pancreatic vascular injuries, which are difficult to access. Missed injuries lead to a significant morbidity, and mortality.

- Aortic and caval injuries: these are difficult because access to these injuries and control of haemorrhage from them are especially problematic.
- Complex pelvic injuries with associated open pelvic injury: these are particularly difficult to treat and are associated with a high mortality.

Damage control approaches to these injuries may dramatically improve survival.

6.1.2.1 HEPATIC INJURIES

Repair and resection for the 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 anatomic resection has been well documented, but is usually not applicable to traumatic insults. Knowledge of the anatomy is important and also helps explain some of the patterns of injury following blunt trauma. In addition, there are differences in tissue elasticity that 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 tissues 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 retro-hepatic 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 anterior–posterior 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 pre-operatively 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. Virtually all penetrating injuries to the abdomen should be explored promptly, especially when they occur in conjunction with hypotension. The precise diagnosis in blunt injury can be difficult. Knowledge of the mechanism of injury, such as a history of rapid deceleration, may be helpful. Shoulder harnesses can cause blunt injuries to the liver and rib fractures that can cause direct laceration of the liver.

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 anatomy by manual compression and packing. If this is successful, and the bleeding is controlled, no further action may be required. Packing is performed using large abdominal packs, placed above, below and around the liver. The liver may need to be mobilized by division of the hepatic ligaments. Packs must *not* be forced into any splits or fractures, as this increases the damage and encourages haemorrhage. Most catastrophic bleeding from hepatic injury is venous in nature and therefore can be controlled by these liver packs. 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 there is concomitant injury to the bowel, with faecal soilage, it is appropriate to control this with a running suture, staples, an intestinal clamp, or by excluding the segment using tape ligature.

It is prudent at this time to enter the gastro-hepatic 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 hepatic (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 2-0 or 4-0 silk 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 scalpel. Then the normal liver tissue is gently compressed between thumb and forefinger, rubbing the normal parenchymal tissue away, leaving just the intact vessels for ligation or clipping. Forceful pinching or crushing of the liver tissue must be avoided, as 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, atrial caval shunt, non-shunt isolation (Heaney technique) and venovenous bypass. Liver packing can also 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 when expertise in more sophisticated techniques is not available, or when it is therapeutic in controlling the bleeding.

6.1.2.2 PANCREATIC TRAUMA

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 gastro-hepatic and omento-colic 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.

If there is obvious disruption to the pancreatic duct, it should be ligated with distal pancreatic resection (see section on pancreatic injury).

Diagnosis of blunt pancreatic trauma is much more problematic. Because 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; DPL and FAST are unhelpful; gastrograffin swallow has fair sensitivity. CT scan 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 can usually either be drained or, if there is strong suspicion of major ductal injury, 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.

6.1.2.3 AORTA AND INFERIOR VENA CAVA

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 the 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.

6.1.2.4 COMPLEX PELVIC FRACTURES

Complex pelvic fractures with open pelvic injury can be the most difficult 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 scan and occasionally 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 or a pelvic binder. The pneumatic anti-shock garment (PASG)/medical anti-shock trousers (MAST) can be used. These manoeuvres attempt to align the fractures anatomically, to prevent further movement, and to reduce further blood loss. More formal fixation can be performed in the operating room when time allows.

For patients with cardiovascular instability, urgent external stabilization of the pelvic bones or angiography may be required (see Chapter 7, section on pelvic injury).

Surgery

All open pelvic fractures should be taken to the operating room as soon as the necessary diagnostic studies have been carried out. In the case of the patient who is haemodynamically unstable, resuscitation is optimized in the operating room. The priorities facing the surgeon are to control the pelvic haemorrhage and to 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, after which the decision can be made about whether 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 of posterior pelvic stabilization. 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 soiling of the wound in the post-injury period. In general, all injuries involving the perineum and peri-anal area should have a diverting colostomy.

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 treated daily, either in the operating room or in the intensive care unit, with wound examination, debridement and gradual removal of

packs. 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 begun as soon as pelvic sepsis is controlled.

If adequate orthopaedic experience is unavailable, consideration should be given to early transfer of the patient to an institution with the necessary expertise as soon as his or her condition allows.

6.1.3 Retroperitoneum

Injuries to retroperitoneal structures are associated with a high mortality and 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. Because of the high incidence of intra-peritoneal and retroperitoneal injuries occurring simultaneously, the retroperitoneum is always approached via a transperitoneal incision. The patient is prepared and draped to widely expose the chest, abdomen, groins and thighs. A long midline laparotomy is performed and the peritoneal cavity is 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 trans-thoracic cross-clamping can be done prior to opening the abdomen (see Section 5.2, 'Access to the thorax').

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

The retroperitoneum can be divided into three areas:

- the central zone containing the aorta, vena cava, pancreas and duodenum,
- the lateral zones containing the kidneys, ureters and right or left colon,
- the pelvic zone containing the rectum, bladder, distal ureters and iliac vessels.

6.1.3.1 CENTRAL HAEMATOMAS

All central haematomas must be explored.

If the haematoma is not expanding, other abdominal injuries take priority. If the haematoma is expanding, it must be explored first. 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.

6.1.3.2 LATERAL HAEMATOMAS

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.

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.

6.1.4 Tissue adhesives in trauma

6.1.4.1 INTRODUCTION

Haemostyptic 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 afterbleeding.

Of the adhesives currently available, fibrin glue is the most suitable for treating injuries to the parenchymatous organs and retroperitoneum. 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 example is Tisseel®/Tissucol® (Baxter Hyland Immuno, Vienna). Fibrin is a biological two-component adhesive and has high concentrations of fibrinogen and factor XIII, which, together with thrombin, calcium and apro-

tinin, produce 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 injecting system.

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. It promotes the aggregation of thrombocytes when it comes 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.

6.1.4.2 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 nearly dry when the fibrin glue is sprayed onto it.

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.

Hepatic injuries

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 injuries

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 would a splenectomy.

After using one of the surgical techniques described above, 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 is 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 re-bleeding.

Pancreatic trauma

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 and without sutures, a worthwhile option in the treatment of such lacerations is fibrin sealing and collagen tamponade, for which adequate drainage is essential.

Injuries to the bowel

Serosal sutures and primary or secondary anastomoses can be secured with adhesives. Covering the anastomosis with collagen fleece can effectively 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 hours. Re-bleeding after the removal of the packs can necessitate re-packing. However, slight bleeding can be stopped effectively by spraying on adhesives.

6.1.5 Access to the abdomen

6.1.5.1 PRE-OPERATIVE MEASURES

During resuscitation, standard ATLS® guidelines should be followed. These should include:

- nasogastric tube or orogastric tube,
- urinary catheter.

6.1.5.2 ANTIBIOTICS¹

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

- no pathology found – no further antibiotics,
- blood only – no further antibiotics,
- small bowel or gastric contamination – continuation for 24 hours only,
- large bowel, minimal contamination – copious peritoneal washout, continuation for 24 hours only,
- large bowel, gross contamination – copious peritoneal washout, 24–72 hours of antibiotics.

The antibiotics commonly recommended include a second-generation cephalosporin or amoxicillin/clavulanate. There is some evidence that aminoglycosides should not be used in acute trauma, partly because of the shifts in fluids, which require substantially higher doses of aminoglycoside 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 twofold 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 intra-operatively, due to dilution of the pre-operative dose.

6.1.5.3 SPECIFIC PROCEDURES

- See under 'Retroperitoneum'.
- See under individual organ systems.
- See 'Abdominal vascular injury'.

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6.1.5.4 THE TRAUMA LAPAROTOMY

Temperature control is fundamental. Preparatory measures should be taken prior to commencement of the procedure. These include warming of the operating theatre, warming of all fluids infused, 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 the 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 sub-hepatic 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 as to 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 is assessed. It is prudent at this time to dissect the gastro-hepatic 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 '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.

6.1.5.5 SURGICAL APPROACH

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. Minimizing patient hypothermia by raising the operating room temperature to a higher than normal level, and warming of 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 (Figure 6.1). 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 MANOEUVRE

The hepatic flexure is retracted medially, dividing adhesions along its lateral border. The adhesions on the outer border of the duodenum are divided from below upwards, allowing the medial rotation of the duodenum. The posterior wall of the duodenum can be inspected, together with the right kidney, porta hepatis and inferior vena cava (Figure 6.2).

Initially, the Kocher manoeuvre is performed by dividing the lateral peritoneal attachment of the

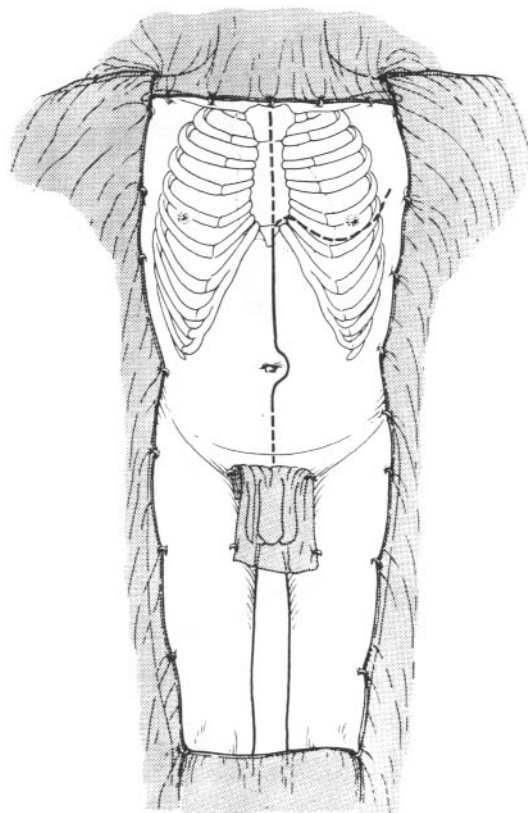


Figure 6.1 Exploration of the abdomen, showing the extent of tissue preparation and draping prior to surgery.

duodenum. The loose areolar tissue around the duodenum is bluntly dissected, and the entire second and third parts of the duodenum are identified. Medial traction is applied to the duodenum, which is then mobilized medially with a combination of sharp and blunt dissection. The dissection is carried all the way around the third part of the duodenum to expose the inferior vena cava and aorta.

By reflecting 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 can be achieved by mobilizing the ligament of Treitz and performing the Cattel and Braasch manoeuvre (Figure 6.3).

RIGHT MEDIAL VISCERAL ROTATION (CATTET AND BRAASCH MANOEUVRE²)

The right colon is mobilized along its entire lateral border, including the hepatic flexure, and reflected

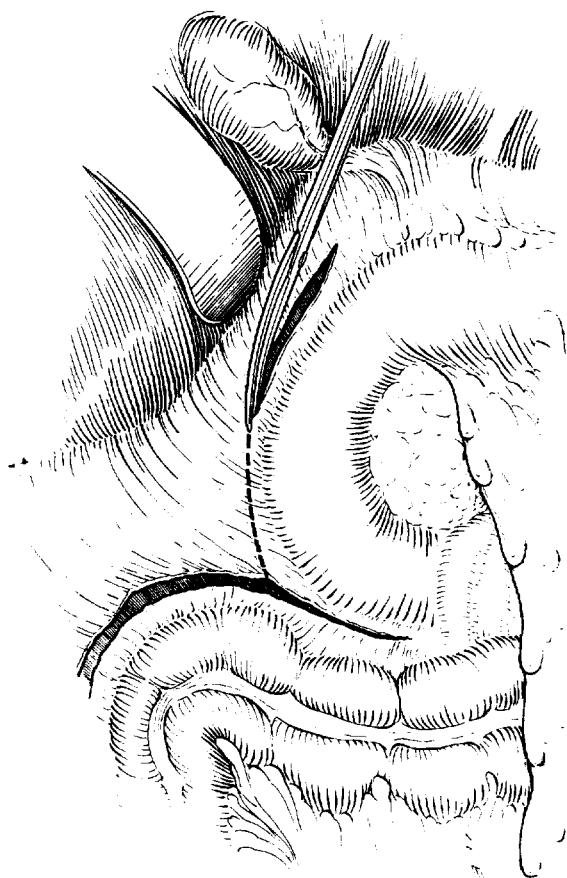


Figure 6.2 Kocher manoeuvre.

medially. The small bowel mesentery is mobilized. The right retroperitoneum is also exposed.

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 then be identified and any injuries inspected.

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. Access to the vena cava is also facilitated (Figures 6.4 and 6.5).

Exposure for repair of the aorta, distal body and

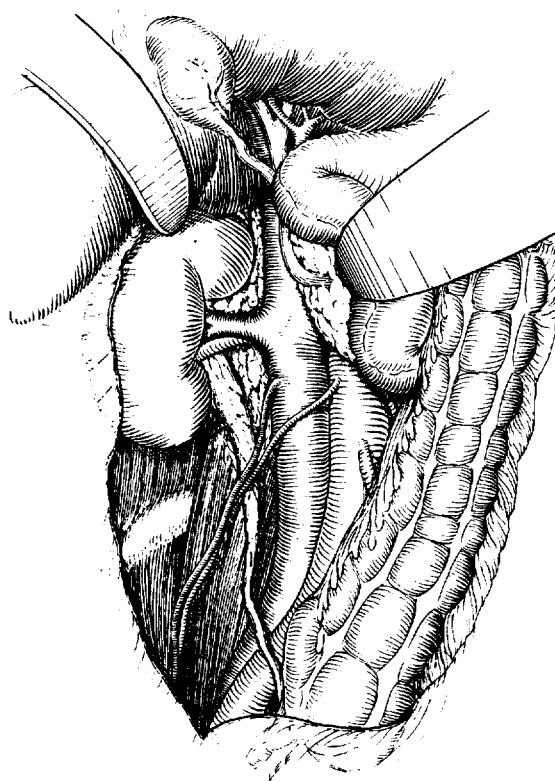


Figure 6.3 Reflection of the colon (Cattel and Braasch manoeuvre).

tail of pancreas is not ideal with the Cattel and Braasch manoeuvre, and better exposure can be obtained by performing a left medial visceral rotation (Mattox manoeuvre).

LEFT MEDIAL VISCERAL ROTATION (MATTOX MANOEUVRE³)

The left medial visceral rotation procedure entails mobilization of the spleno-renal 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 plexuses overlie the proximal aorta and need to be sharply dissected in order to identify the renal and superior mesenteric arteries (Figure 6.6).

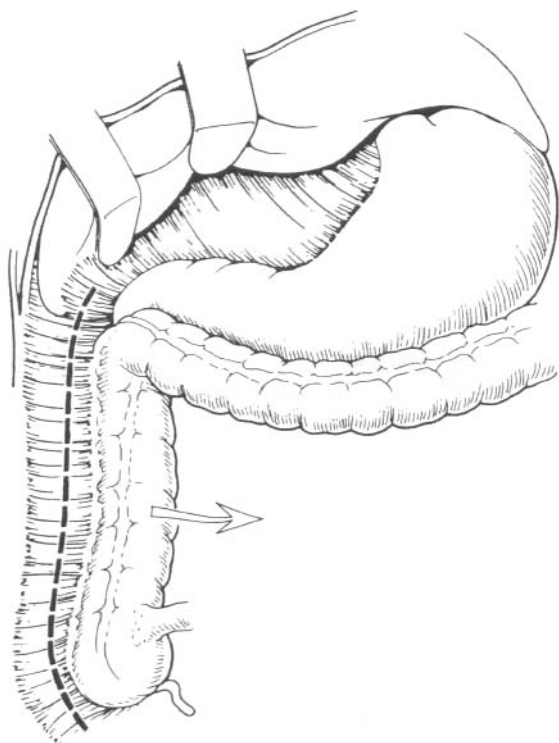


Figure 6.4 Rotation of the ascending colon.

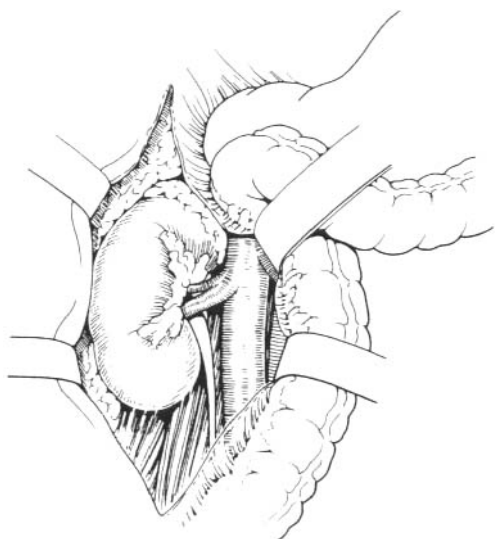


Figure 6.5 Rotation of the ascending colon.

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

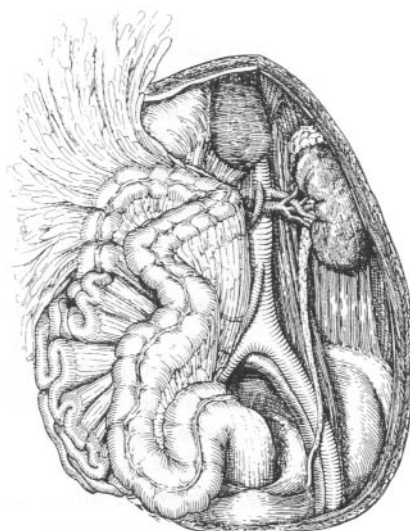


Figure 6.6 Left medial rotation.

well identified through this approach. The omentum is then grasped and drawn upward. A window is made in the omentum (via the gastro-colic ligament) and the operator'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 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, a Kocher manoeuvre is performed. Better exposure can be achieved using the Cattell and Braasch manoeuvre.

The Kocher 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. This manoeuvre requires mobilization of

the right colon (including the hepatic flexure) from right to left, so that 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 medial rotation of the spleen, descending colon and sigmoid colon to the right. This allows inspection of the left kidney, retroperitoneum and tail of the pancreas.

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 proven 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 proven reliable in ruling out full-thickness duodenal injury. Mobilization of the whole duodenum is mandatory for exclusion of duodenal injury.

6.1.5.6 REFERENCES

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6.2 THE LIVER

6.2.1 Overview

6.2.1.1 INTRODUCTION

Any surgeon who has been confronted with a patient exsanguinating from a liver injury will attest to the sense of helplessness experienced. In order to improve the outcome for these severely injured patients, the surgeon requires:

- 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.

6.2.1.2 RESUSCITATION

Haemodynamically *stable* patients without signs of peritonitis or other indication for operation 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 is mandated by haemodynamic instability should be transferred to the operating room 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 of fresh (if available) whole blood or equivalent.

6.2.1.3 MANAGEMENT

The patient's surgery should not be delayed by multiple emergency department procedures such as limb X-rays, unnecessary ultrasonography, and vascular access procedures. Brain scanning should be delayed until the patient is stable. The anaesthetist can continue resuscitation in the operating room.

The purpose of diagnostic investigation for stable patients is to help identify those patients who can be safely managed non-operatively, to assist decision making in non-operative management, and to act as a baseline for comparison in future imaging studies. Accurate, good quality computed tomography (CT) has enhanced our ability to make an accurate diagnosis of liver injuries.

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 or the intrahepatic cava or the portal structures are 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.

Penetrating wounds of the liver do not usually present a diagnostic problem, as 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 or by conservation or resection.

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 (FAST) 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 scan is an invaluable diagnostic aid and allows the surgeon to make decisions about operative management. Diagnostic peritoneal lavage is also quite useful, particularly when CT support services are inadequate.

6.2.1.4 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; see also Appendix B, 'Trauma scoring and scoring systems').

Hepatic injuries are graded on a scale of I to 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 to 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.

Most injuries (approximately 60–70 per cent) are managed simply by evacuating the free intra-peritoneal blood, and some will require drainage of the injury because of possible bile leak. Many of these patients can be managed non-operatively. Caution must be exercised because bile within the peritoneal cavity is not always benign.

Twenty-five per cent of liver injuries 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 temporarily controlled by direct compression, which requires a capable

Table 6.1 Liver Injury Scale (1994 Revision)

Grade ^a	Type of injury	Description of injury
I	Haematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
II	Haematoma	Subcapsular, 10–50% surface area: intraparenchymal <10 cm in diameter
	Laceration	Capsular tear 1–3 cm parenchymal depth, <10 cm in length
III	Haematoma	Subcapsular, >50% surface area of ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma >10 cm or expanding 3 cm parenchymal depth
	Laceration	Parenchymal disruption involving 25–75% hepatic lobe or 1–3 Couinaud's segments
IV	Laceration	Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud's segments within a single lobe
V	Vascular	Juxtahepatic venous injuries, i.e. retrohepatic vena cava/central major hepatic veins
VI	Vascular	Hepatic avulsion

^aAdvance one grade for multiple injuries up to grade III.

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 (less than 1 per cent of all liver injuries), haemostatic agents such as crystallized bovine collagen, fibrin glue and gel foam, use of the argon laser, and the hot air gun.

Approximately 5 per cent of all liver injuries require resection or debridement, 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, the portal triad, the intrahepatic

cava, are bi-lobar 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),
- veno-venous 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 due to using 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, prior to 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 anaesthesiologist and a surgeon capable of dealing with the problems rapidly.

Veno-venous 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 them 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 in controlling severe lacerations.

Injuries to the porta hepatis 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 HAEMATOMA

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 less than 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 grades III and IV haematomas are treated with exploration and selective ligation, with or without packing.

6.2.1.6 NON-OPERATIVE MANAGEMENT

Richardson and co-workers¹ managed approximately 1200 blunt hepatic injuries over a 25-year period. Non-operative management was used in up to 80 per cent of cases. During this period, deaths secondary to injury dropped from 8 per cent to 2 per cent.

Nearly 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 ultrasound, helical CT scan 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 (repeated daily, or as required) to assess change in size of the haematoma. Continued haemorrhage occurs in 1–4 per cent of patients. Hypotension may develop, usually within the first 24 hours after 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.

6.2.1.7 MORTALITY AND COMPLICATIONS

Overall mortality for patients with hepatic injuries is approximately 10 per cent. The most common cause of death is exsanguination, followed by multiple organ dysfunction syndrome (MODS) and intracranial injury. Three generalizations may be made regarding 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.

Post-operative 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 post-operative haemorrhage, the patient is best served by being returned to the

operating room. Arteriography with embolization may be considered in selected patients. If coagulation studies indicate that a coagulopathy is the likely cause of post-operative haemorrhage, correction of the coagulopathy must be a critical part of the strategy.

Perihepatic infections occur in less 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 12th 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 re-operation 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 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 intra-thoracic structures forms in patients with associated diaphragmatic injuries, resulting in a bronchobiliary or pleurobiliary fistula. Because of the pressure differential between the biliary tract and the thoracic cavity, most of these fistulae must be closed operatively.

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, into a bile duct or into 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.

6.2.1.8 INJURIES 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 the 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 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 common bile duct 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 utilized.

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.

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6.2.2 Access to the liver

6.2.2.1 INCISION AND EXPOSURE

The patient is placed in the supine position. Warming devices are placed around the upper body and lower limbs and 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 intra-pericardial inferior vena caval control is often easier than abdominal control adjacent to a severe injury. An Omnitract or Buckwalter-type automatic retractor greatly facilitates access.

6.2.2.2 INITIAL ACTIONS

Once the abdomen has been opened, intra-peritoneal 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. Auto-transfusion 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 subject 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 due to 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 a retrohepatic haematoma being evident, 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 anaesthesiologist time to restore the circulating volume before further blood loss occurs. Also, 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 most useful techniques for the control of hepatic haemorrhage are:

- 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 atriocaval shunt,
- Moore–Pilcher balloon,
- veno-venous 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 are 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.

- Packs should preferably be removed within 24–72 hours.
- 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 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 then 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 advantages of this approach are 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 only be performed for about 15 minutes 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 anatomic division between the left lobe and the right. 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 6.1.4, 'Tissue adhesives in trauma')

Fibrin glue has been used in treating both superficial and deep lacerations and appears to be the most effective topical agent. It can also be injected deep into bleeding gunshot and stab wound tracts to prevent extensive dissection and blood loss. Fibrin glue is 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 chromic catgut. 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 tightly may cut off the blood supply to viable liver parenchyma, resulting in necrosis.

Most sources of venous haemorrhage can be managed with intra-parenchymal 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 hepato-tomy 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. It can either be manufactured by the surgeon using Penrose rubber tubing or can be 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 falciform 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, anatomic resection produces good results, but in the uncontrolled circumstances of trauma, mortality 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 for whom 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 inferior vena cava above the right kidney (suprarenal) and above the liver (suprahepatic). 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 nearly 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 then passed caudally so that its end lies infrahepatically, below intrahepatic 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. At present, 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 either the use of 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 room within the next 48 hours.

6.2.2.6 FURTHER READING

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6.3 THE SPLEEN

6.3.1 Overview

The conventional management of splenic injury has been splenectomy. Over the last 20 years, the importance of splenic preservation as a means of prevent-

ing overwhelming post-splenectomy infection has been realized. The risk is said to be 0.5–1 per cent in adults, but in infants is up to 50 per cent.

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. It also gives rise to superior and inferior terminal branches that enter the splenic hilum. The splenic artery and the splenic vein are embedded in the superior border of the pancreas.

Three avascular splenic suspensory ligaments maintain the intimate association between the spleen and the diaphragm (spleno-phrenic ligament), left kidney (lienorenal/spleno-renal ligament), and the splenic flexure of the colon (spleno-colic ligament). The gastro-splenic 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

One-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 diagnosis of splenic injury is the computed tomography (CT) scan, as it will show whether blood is around the spleen and whether active bleeding is still taking place. If so, angiography with embolization can be considered.

Ultrasound

Focused abdominal sonography for trauma (FAST) ultrasound will show blood around the spleen and in the paracolic gutter. It will not show whether active bleeding is taking place. Serial ultrasound examinations may be necessary.

6.3.1.3 SPLENIC INJURY SCALE

Staging of splenic injury originally evolved from angiography studies of the spleen, which identified

specific injury patterns. This information was further divided 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 autopsy evaluation (Table 6.2; see also Appendix B, 'Trauma scores and scoring systems').

Table 6.2 Splenic Injury Scale

Grade ^a	Injury type	Description of Injury
I	Haematoma	Subcapsular <10% surface area
	Laceration	Capsular tear <1 cm parenchymal depth
II	Haematoma	Subcapsular 10–50% surface area; intraparenchymal <5 cm in diameter
	Laceration	Capsular tear 1–3 cm parenchymal depth that does not involve a trabecular vessel
III	Haematoma	Subcapsular >50% surface area or expanding; ruptured subcapsular or parenchymal haematoma; intraparenchymal haematoma ≥5 cm or expanding
	Laceration	>3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen)
V	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury with devascularized spleen

^aAdvance one grade for multiple injuries up to grade III.

6.3.1.4 MANAGEMENT

Non-operative management²

Management of hepatic and splenic injury has evolved with an increasing emphasis on non-operative management. Previously, diagnostic peritoneal lavage was an indication for laparotomy because of ongoing haemorrhage. However, stimulated by the success of non-operative

management of both hepatic and splenic injury in children, there has been a similar trend in adults.

The approach of non-operative management of blunt splenic injuries (or splenic salvage) in the paediatric population is well described, with a success rate of more than 90 per cent.

Non-operative management is contraindicated if there is a risk of other abdominal organ injury being present, which may require surgical intervention. Additionally, it is contraindicated where a significant brain injury is present and there is a risk of secondary brain injury from hypotension.

Indications for surgical intervention following a trial of non-operative management include:

- haemodynamic instability,
- evidence of continued splenic haemorrhage,
- associated intra-abdominal injury requiring surgery,
- replacement of more than 50 per cent of the patient's blood volume.

The advantages of non-operative management 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 workup, patients with grades I, II or III splenic injuries without haemodynamic instability, who have no associated intra-abdominal injuries requiring surgical intervention, and no co-morbidities to preclude close observation, may be candidates for non-operative management.

A large number of publications support non-operative management 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 re-bleeding of the spleen after non-operative management is acceptably low, reportedly in the range of 1–8 per cent. Re-bleed is considered more likely if a higher grade injury (grade IV) has been managed non-operatively.

6.3.1.5 REFERENCES

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Spleen and Liver (1994 Revision). *Journal of Trauma* 1995; **38**(3):323–4.

- 2 Alonso M, Brathwaite C, Garcia V et al. Practice management guidelines for the nonoperative management of blunt injury to the liver and spleen. In *Trauma practice management guidelines*. Eastern Association for the Surgery of Trauma, <http://www.east.org> 2002.

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 and spleno-phrenic, lieno-renal and spleno-colic ligaments are divided using scissors. Great care and gentleness must be exercised to avoid pulling on the spleen, avulsing the capsule and making a minor injury worse.

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

Spleen not actively bleeding

If not actively bleeding, the spleen can be left alone.

Splenic surface bleed only

These bleeds can usually be stopped by packing, diathermy or fibrin tissue glue.

Minor lacerations

These may be sutured using absorbable sutures. Some surgeons use Teflon® pledgets. Alternatively, omental patches may be placed.

Splenic tears

Ligation of segmental vessels at the hilum may be helpful in controlling the bleeding from a splenic tear. With deep tears, and in the absence of other life-threatening injuries, a partial splenectomy should be considered (see below).

Mesh wrap

If the spleen is viable, it can be wrapped in an absorbable mesh to tamponade the bleeding. A patch of absorbable material mesh (e.g. Vicryl®) slightly larger than the surface area of the spleen should be used. The slit is cut unto the mesh, into which the hilum of the spleen is placed. The mesh is then wrapped from the hilum and around the parenchyma. A running or purse-string suture is used to approximate the edges of the mesh.

Partial splenectomy

Diathermy and/or finger fracture techniques are employed. The procedure is usually reserved for a demarcated ischaemic pole, after ligating segmental vessels. Control of bleeding from the raw surface can be accomplished with mattress sutures, and a combination of packing, diathermy, or fibrin glue. The remnant may require mesh wrap.

Splenectomy

In the presence of other major injuries, or if the spleen has sustained damage at the hilum, a routine splenectomy should be carried out. The splenic vessels must be isolated and tied separately, as there is a small risk of subsequent fistula formation. 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 tail of the pancreas lies very close to the spleen and should be dissected free.

6.3.2.2 DRAINAGE

It is not routine to drain the splenic bed post-splenectomy. If the tail of the pancreas has been damaged, a closed suction drain should be placed in the area affected.

6.3.2.3 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.

6.4 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 they are substantially injured. Though the retroperitoneal location of the pancreas means that it is commonly injured, it also contributes to the difficulty in diagnosis, as the organ is concealed, and often results in delay with an attendant increase in morbidity. The increase, particularly, in penetrating injuries and in the incidence of wounding from gunshots has made pancreatic injuries more common. Because these organs are in the retroperitoneum, they do not usually 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 radiographic imaging to identify an injury to these organs early in their course.

In many cases the surgical management is relatively simple, but occasionally complex and technical surgical solutions are necessary. 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

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.

The pancreas 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, and 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 13 to 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 OF PANCREATIC INJURY

The central retroperitoneal location of the pancreas makes the investigation of pancreatic trauma a diagnostic challenge and, particularly if there are other 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 accurately assess 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 in conjunction with earlier work^{4,5} that showed an increase in late complications if ductal injuries were missed, the importance of evaluating the duct is evident.

Clinical evaluation and serum amylase activity

In a patient with an isolated pancreatic injury, even ductal transection may be initially asymptomatic or have only minor signs; but the possibility must be kept in mind. The level of 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 Jurkovich⁶ showed a positive predictive value of 10 per cent and a 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.⁷ At present, serum amylase has little value in the initial evaluation of pancreatic injury.

Diagnostic peritoneal lavage

The retroperitoneal location of the pancreas renders diagnostic peritoneal lavage 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 intra-operatively.

Ultrasound

The posterior position of the pancreas almost completely masks it from diagnostic ultrasound. In conjunction 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 (CT) scan has been advocated as the best investigation for the evaluation of the retroperitoneum. In a haemodynamically stable patient, CT scan with contrast enhancement has a sensitivity and specificity as high as 80 per cent.⁸ However, particularly in the initial phase, CT scan may miss or underestimate the severity of a pancreatic injury,⁹ 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, as 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 scan, will allow a reasoned decision to be made about the need for operative intervention.

Magnetic resonance cholangiopancreatography

New software has opened up investigation of the pancreas and biliary system to magnetic resonance imaging (MRI).¹⁰ However, to date there has been little work done in pancreatic injuries.

Intra-operative pancreatography

Intra-operative visualization of the pancreatic duct has been advocated in its investigation, 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,¹¹ 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 tail⁶ or needle cholecystocholangiogram.

6.4.1.5 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.

6.4.1.6 ORGAN INJURY SCALE

The Organ Injury Scale developed by the American Association for the Surgery of Trauma (AAST)¹² has been accepted by most institutions that regularly deal with pancreatic trauma (Table 6.3; see also Appendix B, 'Trauma scores and scoring systems').

Table 6.3 *Pancreas Injury Scale*

Grade ^a	Type of injury	Description of injury
I	Haematoma	Minor contusion without duct injury
	Laceration	Superficial laceration without duct injury
II	Haematoma	Major contusion without duct injury or tissue loss
	Laceration	Major laceration without duct injury or tissue loss
III	Laceration	Distal transection or parenchymal injury with duct injury
IV	Laceration	Proximal transection or parenchymal injury involving ampulla ^b
V	Laceration	Massive disruption of pancreatic head

^aAdvance one grade for multiple injuries up to grade III.

^bProximal pancreas is to the patient's right of the superior mesenteric vein.

6.4.1.7 OPERATIVE MANAGEMENT

Damage control

The origin of the concept of damage control was described by Halsted in the packing of liver injuries, as reported and repopularized by Stone in 1983,¹³ who advocated early packing and termination of the operation in patients who showed signs of intra-operative 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 are 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.¹⁴ showed that these require simple drainage and haemostasis, and this has become standard practice.¹⁵ 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 with a closed suction system.¹⁶

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 intra-operative pancreatogram 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 closed with a stapling device.¹⁷ An external drain should be placed at the site of transection, as there is a post-operative fistula rate of 14 per cent.¹⁸ 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 vessels usually leaves enough pancreatic tissue to result in an acceptably low rate of insulin-dependent diabetes.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.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 make the procedure contraindicated in an unstable patient with multiple associated injuries.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 overwhelming post-splenectomy infection post-operatively.

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.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 operative 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 are reserved for minor injuries in which the pancreatic duct is not involved and injuries to both organs are slight.²²

DUODENAL DIVERTICULIZATION

Duodenal diverticulization was developed to deal with the higher mortality of combined injuries to the duodenum and pancreas than of injuries to each organ in isolation.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^{22,23} 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 (see Figure 6.4). This is best achieved with access from the stomach through a gastrotomy and the use of a slowly absorbable suture. The stomach is decompressed with a gastrojejunostomy. Contrast studies have shown that the pylorus re-opens within 2–3 weeks in 90–95 per cent of patients, allowing flow through the anatomical channel. Feliciano et al.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 when 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, however, is primary closure, pyloric exclusion and gastro-enterostomy.

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,²⁴ is indicated only in the rare stable patient with this type of injury. The nature and severity of the injury and the co-existing 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.²⁵ The results of this operation vary and, when patients with major retroperitoneal vascular injuries are included, mortality can approach 50 per

cent.²⁰ Oreskovich and Carrico, however, reported a series of 10 Whipple's procedures for trauma with no deaths.²⁶

6.4.1.8 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. Buchler et al.²⁷ 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 Glasgow,²⁸ who found that somatostatin gave no benefit.

After pancreatic surgery, somatostatin can reduce the output from a pancreatic fistula.²⁸ However, retrospective work on the role of octreotide in pancreatic trauma differs. 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 at 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.³⁰ TPN is far more expensive, but may be used if enteral access distal to the duodenojejunal flexure is impossible.

6.4.1.9 PANCREATIC INJURIES 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 the management of solid organ injuries in children) is controversial. Shilyansky et al.³¹ reported that non-operative management of pancreatic injuries in children was safe for both contusion and pancreatic transection, and Keller et al.³² 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.³²

6.4.1.10 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. Pancreatic injuries have post-operative 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.^{5,33}

Most complications are treatable or self-limiting, however, and could be avoided by an accurate assessment of whether the pancreatic duct is damaged.³⁴ Pancreatic complications can be divided into those occurring early and late in the post-operative period.

Early complications

PANCREATITIS

Post-operative pancreatitis may develop in about 7 per cent of patients.³³ 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 post-operative pancreatic fistula is the most common complication, with an incidence of 11 per cent;³³ this increases when the duct is involved, and may be as high as 37 per cent in combined injuries.³⁵ Most fistulas are minor (less than 200 mL of 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.³⁶ 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 scan and operation 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,³⁷ but Kudsk et al.³⁸ reported pseudocysts in half their patients who were treated non-operatively 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 the duct 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 cyst-gastrostomy (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.

6.4.1.11 CONCLUSION

Pancreatic and combined pancreaticoduodenal injuries remain a dilemma for most surgeons and, despite advances and complex technical solutions, they still carry high morbidity and mortality. Pancreatic injury must be suspected in all patients with abdominal injuries, even those who initially have few signs. Accurate intra-operative 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.

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6.4.2 Access to the pancreas

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 gastro-colic ligament and open the lesser sac. A Kocher 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 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 identified through this approach. The omentum is then grasped and drawn upwards.

Anotomy is made in the omentum and the operator'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 KOCHER MANOEUVRE (SEE ALSO SECTION 6.1.5.5 'SURGICAL APPROACH')

If there is a possibility of an injury to the head of the pancreas, a Kocher manoeuvre is performed. 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. 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 CATTIL AND BRAASCH MANOEUVRE (SEE ALSO SECTION 6.1.5.5)

The inferior border of the proximal portion of the pancreas can be identified by the Cattil and Braasch manoeuvre. This is performed by taking down the descending colon and then mobilizing the caecum, the terminal ileum and the 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, 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.

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 of

Whipple's procedure continues to be significant in severely injured trauma patients. 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 to completely explore and identify any injuries.

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. Because these organs are in the retroperitoneum, they do not usually 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, the stomach, small bowel, transverse colon, liver, spleen or kidneys are frequently 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.

A pre-operative 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 pre-operative 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

Penetrating trauma is the leading cause of duodenal injuries in countries with a high incidence of civilian violence. Because of the retroperitoneal location of the duodenum, and its close proximity to a number of other viscera 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 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 the duodenum is crushed between spine and steering wheel or handlebar, or some other force is applied to it. They can be associated with flexion/distraction fractures of L₁–L₂ 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, the first and second parts). These injuries occur at the junction of free (intra-peritoneal) parts of the duodenum with fixed (retroperitoneal) parts. A high index of suspicion based on the mechanism of injury and physical examination findings may lead to further diagnostic studies.

6.5.1.2 CLINICAL PRESENTATION

The clinical changes in isolated duodenal injuries may be extremely subtle until severe, life-threatening peritonitis develops. In the vast majority of 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 cases in which the blunt injury causes intraperitoneal perforations.

6.5.1.3 DIAGNOSIS

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.^{3,4} 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

Although virtually all patients with blunt duodenal injury will eventually have increased white blood cell and amylase levels in diagnostic peritoneal lavage (DPL) fluid, DPL has a low sensitivity for duodenal perforations.⁸

Radiological studies may be helpful in the diagnosis. Plain X-rays 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 intra-peritoneal air and, although rarely seen, air in the biliary tree has been described.⁹ 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 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 gastrointestinal 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.¹⁰

Computed tomography (CT) scan has been added to the diagnostic tests used for investigation for 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.^{11,12} Its reliability in adults is more controversial. The presence of periduodenal wall thickening or haematoma without extravasation of contrast material should be investigated with a gastrointestinal study with gastrograffin. If normal, it should be followed by a barium study with contrast, if the patient's condition allows this.

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.¹³

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.

6.5.1.4 DUODENAL INJURY SCALE

Grading systems have been devised to characterize duodenal injuries (Table 6.4; see also Appendix B, 'Trauma scores and scoring systems').

Although useful for research purposes, the specifics of the grading systems are less important than several simple aspects of the duodenal injuries:

- the anatomic relation to the ampulla of Vater,
- the characteristics of the injury (simple laceration versus destruction of the 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 as mortality rises from 11 per cent to 40 per cent if the time interval between injury and operation is more than 24 hours.¹³

From a practical point of view, the duodenum can be divided into one 'upper' portion, which includes the first and second parts, and a 'lower' portion, which includes the third and fourth parts. The 'upper' portion has complex anatomic structures within it (the common bile duct and sphincter).

Table 6.4 Duodenum Injury Scale

Grade ^a	Type of Injury	Description of Injury
I	Haematoma	Involving single portion of duodenum
	Laceration	Partial thickness, no perforation
II	Haematoma	Involving more than one portion
	Laceration	Disruption <50% of circumference
III	Laceration	Disruption 50–75% of circumference of D2
		Disruption 50–100% of circumference of D1, D3, D4
IV	Laceration	Disruption >75% of circumference of D2
		Involving ampulla or distal common bile duct
V	Laceration	Massive disruption of duodenopancreatic complex
	Vascular	Devascularization of duodenum

^aAdvance 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.

ter) and the pylorus. It requires distinct manoeuvres to diagnose injury (cholangiogram, direct visual inspection) and complex techniques to repair it. The first and second parts of the duodenum are densely adherent, and dependent for their blood supply on the head of the pancreas; therefore, 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 parts of the duodenum, can generally be treated like the small bowel, and injury diagnosis and management are relatively simple, including debridement, closure, resection and reanastomosis.

6.5.1.5 SURGICAL MANAGEMENT OF DUODENAL INJURIES

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 hours to present. This is due to the gradual increase in the size of a haematoma as breakdown of the haemoglobin makes it hyperosmotic, with resultant fluid shifts into it. The diagnosis can be made by double-contrast CT scan or upper gastrointestinal 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 total parenteral nutrition, 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 to carefully explore the duodenum to exclude a perforation, leaving the intramural haematoma intact and planning nasogastric decompression post-operatively.

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, the 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 hours 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 oper-

ative procedures have been proposed. The methods of repair of the duodenal trauma as well as the 'supportive' procedures against dehiscence are described below.

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 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 onto 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 not technically 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 are appropriate.¹⁹ 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 also can 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. In order 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.²⁰ In trauma, the technique was introduced by Stone and Garoni as a 'triple ostomy'.²¹ This consists of a gastrostomy tube to decompress the stomach, a retrograde jejunostomy to decompress the duodenum, and an antegrade jejunostomy 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.²² The drawbacks of this technique are that several new perforations are made in the gastrointestinal tract, the inefficiency of the jejunostomy tube to properly decompress the duodenum, and the common scenario of finding that the drains fell out or were removed by the patient. The fashioning of a feeding jejunostomy at the initial laparotomy in patients with duodenal injury and extensive abdominal trauma (Abdominal Trauma Index >25) is highly recommended.

Duodenal diverticulization

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.²³ Truncal vagotomy and biliary drainage could be added. The disadvantage of duodenal diverticulization is that it is an extensive procedure that is 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, 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).

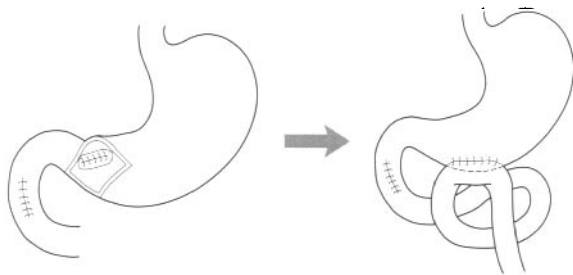


Figure 6.7 Pyloric exclusion and gastric bypass.

The closure of the pylorus breaks down after several weeks and the gastrointestinal continuity is re-established. This occurs regardless of whether the pylorus was closed with absorbable or non-absorbable sutures or staples.²⁴ 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.^{24,25} The long-term incidence of marginal ulceration in patients who have undergone pyloric exclusion is probably underestimated, as 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 gastrointestinal tract in 90 per cent of patients will be re-established within 3 weeks.^{26,27} 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.^{28,29}

The use of octreotide in the protection of the suture line in pancreaticojejunostomies after pancreaticoduodenectomies has been shown to be beneficial.^{30,31} 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 of bowel contamination and ligation of the common bile and pancreatic ducts should be the rule.³² Reconstruction should take place within the next 48 hours 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.^{1,32}

The role of pancreaticoduodenectomy in trauma is best summarized by Walt:³³

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 injuries³⁴ and suggested the following.

- Simple duodenal injuries with no ductal pancreatic injury (grades I and II) should be managed with primary repair and drainage (see Table 6.4).
- 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 necessitate a staged pancreaticoduodenectomy. Less extensive local injuries can be managed by intraluminal stenting, sphincteroplasty or re-implantation of the ampulla of Vater.^{35,36}

6.5.1.6 CONCLUSION

Upper gastrointestinal radiological studies and CT scan 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. The majority of 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. Detailed knowledge of the available operative choices and the situation in which each one of them is preferably applied is important for the patient's benefit.

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6.5.2 Access to the duodenum

See Section 6.4.2, 'Access to the pancreas'.

6.6 THE URO-GENITAL SYSTEM

Uro-genital 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, and in 1905 Evans and Fowler demonstrated that the mortality from penetrating intra-peritoneal bladder injuries could be reduced from 100 per cent to 28 per cent with laparotomy and bladder repair. Ambroise Pare observed death following a gunshot wound of 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 the presence of abdominal and pelvic injury.

6.6.1 Renal injuries

6.6.1.1 OVERVIEW

Diagnosis

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, whereas the majority of patients with significant abdominal trauma, but who are not shocked, will

have microscopic haematuria 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.

At laparotomy, it will become apparent whether or not the kidneys are the source of the shock; if they are, the options are to leave the kidneys alone at first and perform a single-shot on-table intravenous pyelogram (IVP), which has been found to give very good results, explore the injured kidney immediately, or pack the area around the kidney and get out, if in a damage control situation.

- *Penetrating trauma.* The default is to explore the kidney, unless it is obvious from observation or on-table IVP that the injury to the kidney is not the cause of the instability and there is no expanding haematoma, or damage control is required.
- *Blunt trauma.* On-table IVP 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.

STABLE PATIENT

The investigation of choice is the double-contrast or triple-contrast computed tomography (CT) scan; however, it can mis-grade the renal injury. More commonly, 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 sub-optimal studies and where older machines are used.

Contrast-enhanced ultrasound can also allow visualization of active intra-renal bleeds.

- *Penetrating trauma.* The individual trauma unit's accepted method of evaluation of penetrating torso trauma must be used, the renal visualization being provided by IVP/tomogram, followed by angiography if suspicious, or double-contrast or triple-contrast CT scan of the abdomen as a stand-alone investigation.

- *Blunt trauma.* The above investigations are reserved for children irrespective of urinalysis, and for adults with frank haematuria or blood pressure <90 mmHg.

Up to half of renal stab injuries, and up to one-third of gunshot injuries in one series, can be treated non-operatively, as long as excellent diagnostic methods can visualize them.

Post-operative care

Urinomas, infected urinomas, perinephric abscesses and delayed bleeding are the most common complications of renal conservation, and are often amenable to imaging and percutaneous, trans-ureteric 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.

6.6.1.2 ACCESS

Access should be by midline laparotomy, even if isolated renal injury is suspected, because the likelihood of other injuries is always present.

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 Mattox manoeuvre on the left or an extended Kocher's manoeuvre on the right can also afford good control of the aorta, inferior vena cava and renal vessels, if required (Figures 6.8 and 6.9).

The peritoneum over the aorta is opened, and the anterior wall of the aorta followed up to the left renal vein. After exposing the retroperitoneum from the right or the 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 6.10). The second method involves ligation of the distal renal vein at the inferior vena cava to improve the exposure of

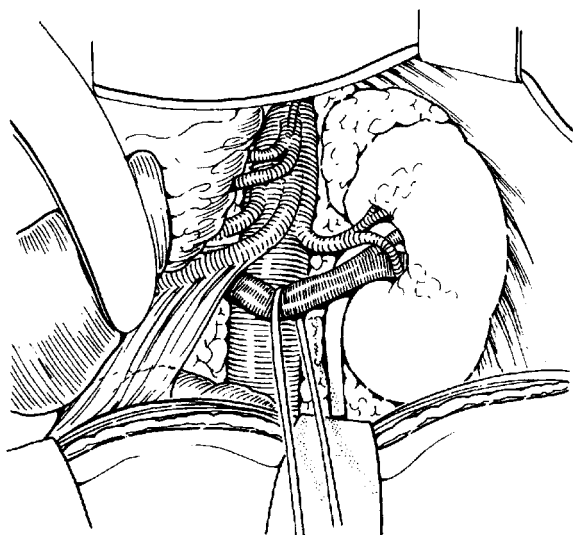


Figure 6.8 Access to the left kidney.

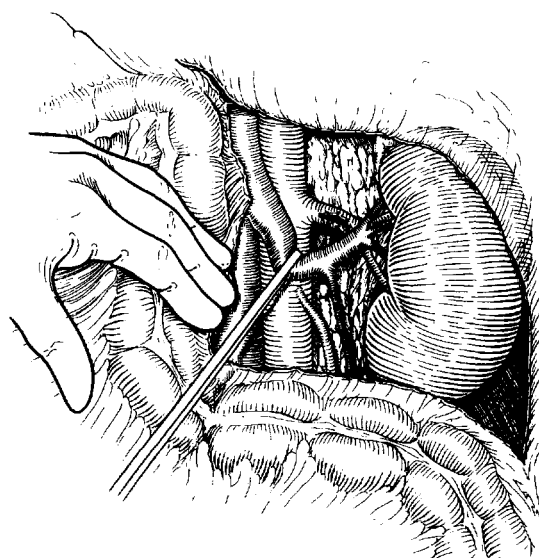


Figure 6.10 Mobilization of the left renal vessels.

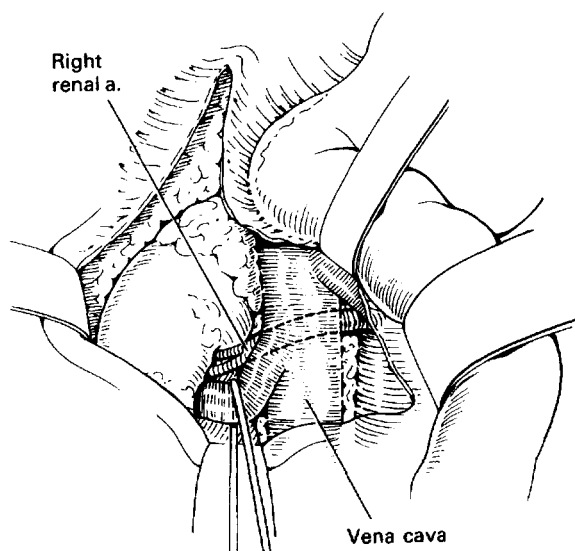


Figure 6.9 Access to the right kidney.

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 inferior vena cava. Dissection lateral to the inferior vena cava 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, as this may bleed profusely. The mobilized kidney now can be examined, debrided, trimmed and sutured with drainage (Figure 6.11).

The kidney tolerates a single ischaemic event much better than repeated short 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 minutes, Gerota's fascia is then opened, and the injured kidney debrided by sharp dissection and sutured, or partially amputated. The renal pelvis collecting system should be closed with a running absorbable suture to provide a watertight 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.

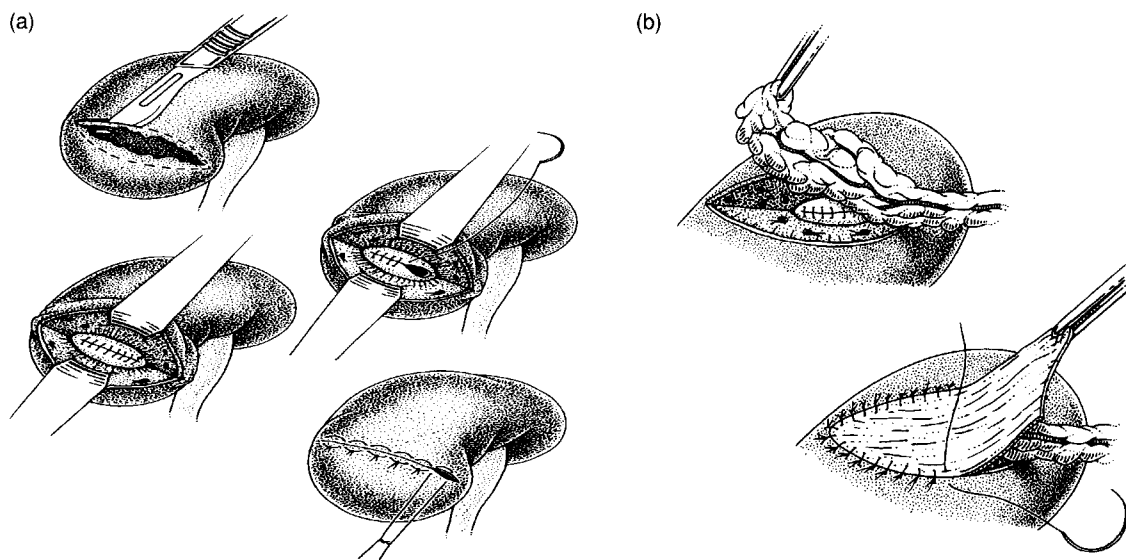


Figure 6.11 Repair of the kidney.

Nephrostomy tubes or ureteric stents can be used either immediately or at some later time in cases of major renal trauma with extravasation.

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6.6.2 Ureteric injuries

6.6.2.1 OVERVIEW

Diagnosis

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 occur in blunt trauma, especially in the paediatric population. Ureteric injuries may even be missed by high-dose IVP.

Treatment

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 IVP.

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 effected after delayed or missed presentation. Percutaneous nephrostomy can be used as a post-operative 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 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, for which 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 re-implantation.

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.

Complications

Complications comprise stricture with hydro-nephrosis, leakage from the anastomosis, and

infected urinomas, especially in late diagnosis, most of which are amenable to percutaneous management.

6.6.2.2 ACCESS

The procedures described for access to the retroperitoneal great vessels also allow perfect exposure of both ureters. Ureteric injuries are rare, and then often due to 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 peri-ureteric tissues should take place, except at the precise level of injury, so as to preserve the delicate blood supply (Figure 6.12). 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.

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6.6.3 Bladder injuries

6.6.3.1 OVERVIEW

Bladder injuries mainly are due to blunt trauma, and are found in about 8 per cent of pelvic fractures. Penetrating trauma is due to gunshot, stab, impalement or iatrogenic injuries, mostly in relation to orthopaedic pelvic fixation.

Diagnosis

Signs and symptoms vary from inability to void and frank haematuria, to vague abdominal or suprapubic tenderness and no haematuria in a small percentage of cases.

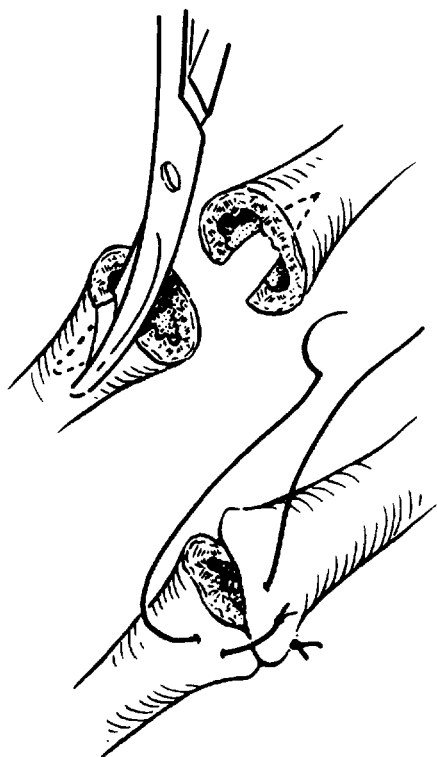


Figure 6.12 Repair of the ureter.

Intra-peritoneal injuries may be associated with a higher serum creatinine and urea, and low sodium, but this biochemical derangement takes some time to develop.

Sonar and CT can be of use only 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).

Retrograde cystography is the method of choice, as 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 also essential.

Contrast extravasation will delineate loops of bowel and the peritoneal contours in intra-peritoneal ruptures, and it will be tracking along the pelvic bones, scrotum, obturator areas etc. in extra-peritoneal ruptures.

Treatment

Urgent operative treatment is indicated in all intra-peritoneal, and some types of extra-peritoneal,

injuries, whereas others require delayed surgery upon failure of non-operative methods. The greatest majority of penetrating injuries require immediate surgery.

NON-OPERATIVE MANAGEMENT

Urethral or suprapubic catheter drainage, for up to 2 weeks, will allow most extra-peritoneal 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.

Extra-peritoneal 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 to be carried out through an intra-peritoneal 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 wound, *both* wounds to the bladder must be 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 extra-peritoneal ruptures, but for intra-peritoneal ruptures closure should be in separate layers.

A large-bore transurethral or suprapubic catheter, or both, can be used, the latter being fed extra-peritoneally into the bladder, and a drain left in Retzius space. A cystogram will be done in most cases after 10 days to 2 weeks, followed by removal of the suprapubic catheter.

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6.6.4 Urethral injuries

6.6.4.1 OVERVIEW

Urethral injuries can have the most disastrous consequences of all genito-urinary trauma, such as incontinence, long-lasting impotence and strictures.

Diagnosis

The 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. Fortunately, rupture of the female urethra is very uncommon.

Once rupture is suspected, two completely different approaches are practised and acceptable.

- 1 Retrograde urethrography is performed by placing a small Foley's catheter in the fossa navicularis, with the patient in the oblique position.
- 2 The preferable approach, however, is not to intervene with an emergency procedure at all. This is particularly important if a pelvic haematoma is present, due to 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) prior to its insertion. A cystogram and, if necessary, cystoscopy can then be done under controlled circumstances at a later stage.

Treatment

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, an open procedure during a laparotomy, or with a percutaneous method. The

isolated open method requires a lower midline laparotomy incision and an intra-peritoneal 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 ultrasound. 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.

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 are recommended for injuries to the anterior urethra, whereas for membranous urethra injuries, re-alignment and stenting over a Foley's catheter for 3 or 4 weeks may be sufficient. This can be achieved by an open lower midline laparotomy and passage of Foley's 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 time of injury.

Primary re-alignment may have better results than delayed repair.

6.6.5 Trauma to the scrotum

Ultrasound of the scrotum is indicated in the evaluation of blunt trauma to the testicle, and can differentiate between torsion, disruption and haematoma. The blood supply is so good that pene-

trating 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.

6.6.5.1 RECOMMENDED READING

Cline KJ, Mata JA, Venable DD, Eastham JA. Penetrating trauma to the male external genitalia. *Journal of Trauma* 1998; **44**(3):492–4.

Munter DW, Faleski EJ. Blunt scrotal trauma: emergency department evaluation and management. *American Journal of Emergency Medicine* 1989 **7**(2):227–34.

6.6.6 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.

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.

6.6.6.1 TREATMENT

Lacerations of the external genitalia and vagina can be sutured under local or general anaesthesia, and a vaginal pack left in for 24 hours to minimize swelling.

Intra-pelvic 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 for all patients suffering this type of injury.

There is increasing evidence that anti-human immunodeficiency virus (HIV) treatment is more effective if instituted within 3 hours of injury.

6.6.6.2 TRAUMA TO 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.

6.7 ABDOMINAL VASCULAR INJURY

6.7.1 Overview

The patient is prepared from 'sternal notch to knee'. It is critical to gain proximal and distal control, and this may necessitate a left lateral thoracotomy to gain access to, and control, the thoracic aorta, and groin incisions to control the iliac vessels.

Autotransfusion should be considered in all cases.

6.7.1.1 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 (Mattox manoeuvre) will reflect the spleen, colon, tail of pancreas and fundus of the stomach towards the midline. This provides access to the aorta, the celiac axis, the superior mesenteric artery, the splenic artery and vein, and the left renal artery and vein. In order 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 ischaemia or further injury.

6.7.1.2 LATERAL HAEMATOMAS

If these are not expanding or pulsatile, they are best left alone, as the damage is usually renal. Renal injuries can generally be managed non-operatively. The surgeon must be confident that there is no perforation of the posterior part of the colon in the paracolic gutters on either side.

6.7.1.3 PELVIC HAEMATOMA

Rapidly expanding or pulsating haematomas in this region may need exploration.

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 commenced if the patient is sufficiently stable.

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, as they are probably due to pelvic venous damage. These veins are notoriously fragile and unforgiving to any attempt at repair. In this situation, packing, and stabilization of the pelvis using external fixators or a sheet binder in the emergency situation, should provide control. Where angiographic facilities are available, the expanding or pulsatile haematoma may be packed firmly, and the patient sent directly to the angiographic suite from the operating theatre for embolization, without further exploration.

Contrast computed tomography (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.

6.7.2 Access

6.7.2.1 INCISION

The patient is prepared 'from sternal notch to knee'. It is critical to gain proximal and distal control, and this may necessitate a left lateral thoracotomy to gain access to the thoracic aorta, or groin incisions to gain control of the iliac vessels.

It is helpful to have all the apparatus available for massive transfusion, with adequate warming of all intravenous fluids.

6.7.2.2 AORTA

Control of the aorta can be achieved at several different levels depending on the site of injury (Figure 6.13) The supraceliac aorta can be exposed by incising the gastro-hepatic ligament and retract-

ing 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 blunt dissection. The oesophagus is then 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 for repair is not ideal with this approach, and better exposure can be obtained by performing a left medial visceral rotation procedure. This entails mobilization of the spleno-renal 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 plexuses, however, overlie the proximal aorta and need to be sharply dissected in order to identify the renal and superior mesenteric arteries.

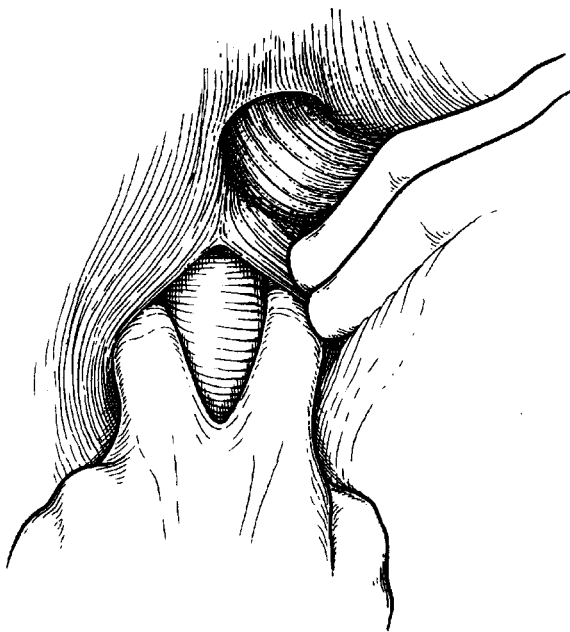


Figure 6.13 Control of the aorta by cross-clamping.

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 now 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.

6.7.2.3 COELIAC AXIS

The left colon is reflected to the right, together with the spleen and tail of the 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 prior to 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 injury should 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.

6.7.2.4 SUPERIOR MESENTERIC ARTERY

If the injury is behind the pancreas, the pancreas may need to be transected to gain access in those patients in whom the artery cannot be accessed by visceral rotation. More distally, the artery is accessed at the root of the small bowel mesentery. The superior mesenteric artery is a vital artery for the viability of the small bowel, and should always be repaired, using conventional techniques. If a period of ischaemia has elapsed, the artery should be shunted, using a temporary stent or 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 infra-renal aorta, away from the pancreas and areas of potential leak.¹

The graft must be tailored so that there is no tension, and the aortic suture line must be covered to prevent aorto-enteric 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.¹

6.7.2.5 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 re-operation to evaluate viability of the colon.

6.7.2.6 RENAL ARTERIES

Access is obtained by mobilizing the viscera medially. Access to the right renal artery proximally is more difficult, and it may be necessary to approach it through the mesocolon.

Repair is done using standard vascular techniques. However, the kidney tolerates warm ischaemia poorly, and its viability after 45 minutes 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.

6.7.2.7 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 veins are often adherent to the back wall of the common iliac artery, and attempts to mobilize them 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.

6.7.2.8 INFERIOR VENA CAVA²

Suprahepatic inferior vena cava

Access to the suprahepatic inferior vena cava can be obtained by incising the central tendon of the diaphragm or by performing a median sternotomy and opening the pericardium.

Infrahepatic inferior vena cava

The infrahepatic vena cava can be exposed by means of a right medial visceral rotation procedure (see Cattell and Braasch manoeuvre, Chapter 6.1.5.5, p. 103). 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. Kocherization 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 6.14–6.16).

Control is best achieved by direct pressure on the inferior vena cava above and below the injury, utilizing swabs.

Injuries to the posterior part of the inferior vena cava should always be expected with penetrating injury to the anterior part of the vessel. Not all bleeding posterior wounds need to be repaired.

It is very difficult to 'roll' the inferior vena cava to approach it posteriorly, due to multiple lumbar veins, so all injuries should be approached transcavally. Not all non-bleeding posterior wounds require repair. Provided it is infra-renal, ligation of the inferior vena cava is acceptable.

6.7.2.9 PORTAL VEIN³

The portal vein lies in the free edge of lesser omentum, together with the common bile duct and hepatic artery. It can generally be controlled with a Pringle's manoeuvre. If the injury is more proximal, it may be necessary to reflect the duodenum medially, or divide the pancreas (Figure 6.17).

The portal vein should be stented early to avoid venous hypertension of the bowel, which will make access to the area increasingly difficult. The stent

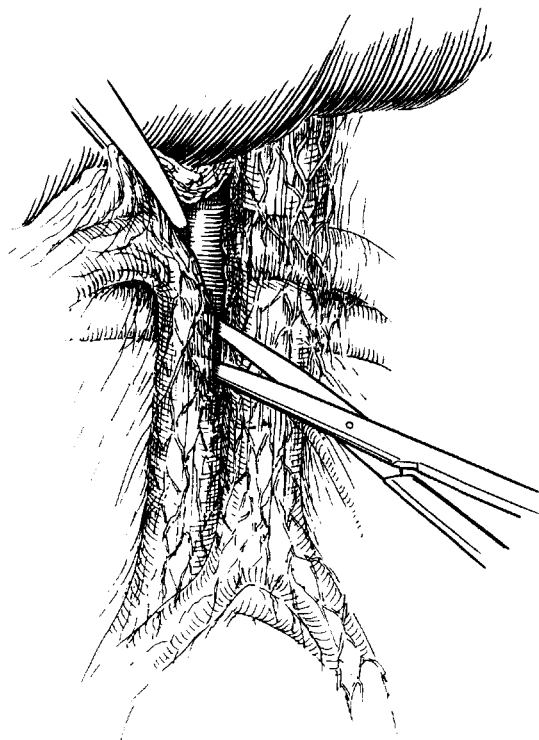


Figure 6.14 Control of the inferior vena cava.

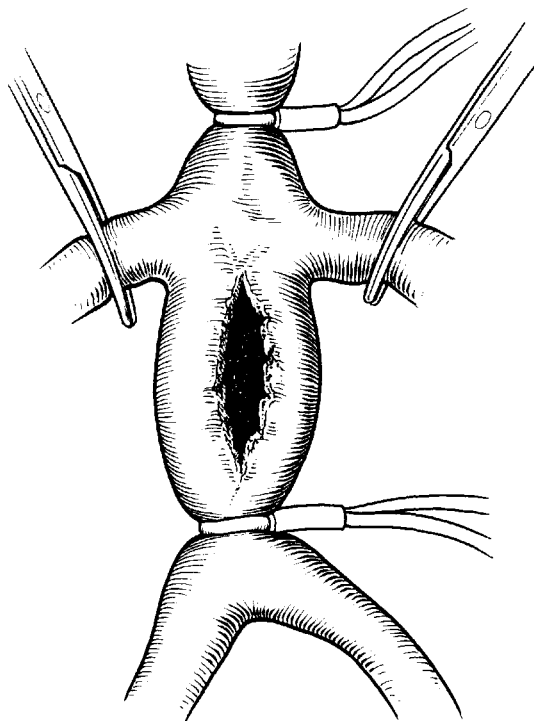


Figure 6.15 Control of the inferior vena cava.

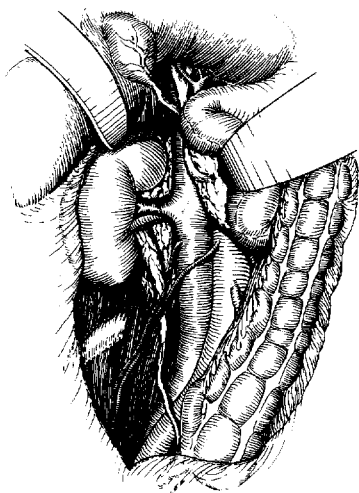


Figure 6.16 Access to the inferior vena cava.

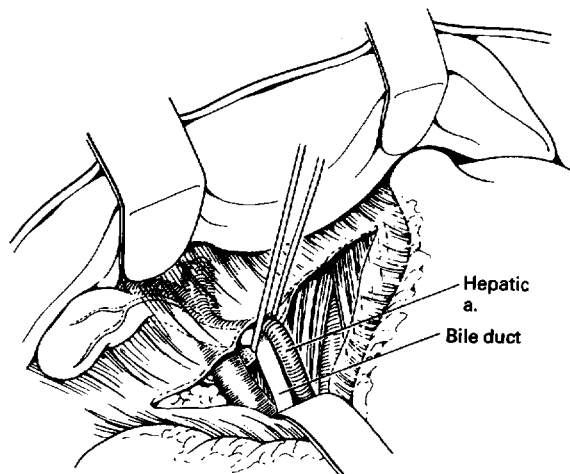


Figure 6.17 Access to the portal vein.

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, although it carries a high mortality.

6.7.3 References

- 1 Accola KD, Feliciano DV, Mattox KL et al. Management of injuries to the superior mesenteric artery. *Journal of Trauma* 1986; **26**:313–19.
- 2 Feliciano DV, Burch JM, Mattox KL et al. Injuries of the

inferior vena cava. *American Journal of Surgery* 1988; **156**(6):548–52.

- 3 Stone HH, Fabian TC, Turkleson ML. Wounds of the portal venous system. *World Journal of Surgery* 1982; **6**(3):335–41.

6.7.4 Recommended reading

Moore EE, Cogbill TH, Jurkovich GJ. Organ injury scaling III: chest wall, abdominal vascular, ureter, bladder and urethra. *Journal of Trauma* 1992; **33**(3):337–8.

7.1 INTRODUCTION

Fractured pelvis is a surgical problem because 65 per cent of patients with a fractured pelvis suffer associated injuries, and mortality is largely due to haemorrhage and infections in pelvic soft tissues, both of which can lead to multiple organ failure. Referral of trauma to surgeons should be based on a mechanism of injury indicating high energy transfer, abnormal vital signs – Glasgow Coma Score (GCS) <13, systolic blood pressure <90 mmHg, respiratory rate <8 or <30, and suspicion of multiple system trauma. 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 X-ray and cervical spine and pelvis X-rays for any blunt injury resulting in a patient being unable to walk). 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 upwards (retrograde urethrogram followed by 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.

7.2 ANATOMY

The surgical anatomy of the pelvis is a key to the pathogenesis of pelvic injuries.

- All iliac vessels, the sciatic nerve roots, including the lumbo-sacral nerve, and the

ureters cross the sacro-iliac joint and disruption of this joint may cause severe haemorrhage and sometimes causes nerve palsy. Fortunately, injuries to the ureters are rare.

- The sacro-iliac 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 quadriceps muscle paralysis.
- 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.
- The pelvic peritoneum that should tamponade pelvic haematomas can accommodate more than 3000 mL, as has been shown in post-mortem specimens.
- The pelvic inlet is circular and a structure that routinely gives way at more than one point, should sufficient pressure be applied to it.
- The pelvis also features the acetabulum, a major structure in weight transfer to the leg. Inappropriate treatment will lead to severe disability
- 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.

7.3 CLINICAL EXAMINATION

Clinical examination starts with gentle bimanual lateral and antero-posterior compression (not distraction!) of the pelvis. Any instability felt indi-

cates the presence of major pelvic instability, associated with life-threatening blood loss, requiring appropriate measures. An early pelvic X-ray 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, and may necessitate a retrograde urethrogram.

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 male patients, 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, femoral and sciatic nerves.

7.4 CLASSIFICATION

Pelvic ring fractures are classified into three types, according to their severity.

- 1 *Type I*: isolated fracture of iliac wing or pubic rami. These are stable fractures, to be treated conservatively.
- 2 *Type II*: 'open book' fractures, with horizontal (rotational) instability due to 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 sacro-iliac joint. These require internal or external stabilization.
- 3 *Type III*: horizontal and vertical instability, due to anterior and posterior fractures and/or disruptions (complete sacro-iliac disruption, displaced vertical sacral fracture). Type III is the result of major mechanical forces and is associated with major blood loss and related to injuries within the pelvis (bladder, urethra, rectum, vagina, sciatic and femoral nerve) and abdomen (bowel perforation) and laceration of the diaphragm.

7.5 RESUSCITATION

The priorities for resuscitating patients with pelvic fractures are no different from the standard priorities. 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 due to ongoing resuscitation) or haemodynamically unstable. Management is based on haemodynamic status.

- Haemodynamically normal: 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: because 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 of that segment, it can only do so in one dimension and will aggravate disruption outside of 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 external fixator with anterior compression if a Type II injury.
 - Apply a pelvic clamp in a Type III injury at the level of the sacro-iliac joint, with posterior compression.
 - If bleeding persists, undergo direct exploration of the pelvis with tamponade, exploration by area with suturing or ligature of lacerations of major blood vessels, repair of anatomical structures (bladder, rectum) where possible, cystostomy and/or colostomy with rectal wash out as required, and, if required and possible at this stage, internal fixation of the pelvic ring.
 - Tamponade pelvic area for any residual capillary bleeding. Leave appropriate drains.

- Effect temporary closure of the abdominal wall, leaving skin and subcutaneous tissue open.
- Haemodynamically unstable: these patients tend to exsanguinate rapidly and immediate measures are required to control bleeding. These measures include immediate application of a wet sheet or military anti-shock trousers (MAST) suit (often not available) immediately followed by definitive control.
 - If facilities allow and time permits, performing angiographic embolization provides better control of haemorrhage than external fixation.
 - Apply pelvic clamp in a Type III injury at the level of the sacro-iliac joint, with posterior compression.
 - Immediately control haemorrhage by aortic clamping. Undergo direct exploration of the pelvis, with tamponade, exploration area by area with suturing or ligature of lacerations of major blood vessels.
 - Consider damage control, with only packing of the pelvis and external fixator in Type II fractures. Repair anatomical structures (bladder, rectum) if possible, and undergo cystostomy and/or colostomy with rectal wash out as required.
 - Tamponade pelvic area for any residual capillary bleeding. Leave appropriate drains.
 - Temporarily close the abdominal wall, leaving skin and subcutaneous tissue open.
 - If bleeding persists, consider angiographic embolization.
- Head injuries are the most commonly associated major injuries. It is worthwhile to remember that 'C' precedes 'D' during resuscitation and management. Computed tomography (CT) scan and neurosurgical procedures have to wait for haemodynamic stability. Haemodynamic stability may be achieved only after damage control surgery.
- 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 scan is the diagnostic modality of choice in the stable patient. In all other patients, diagnostic ultrasound is preferred. If open DPL is performed, the entry point should be above the umbilicus to avoid entering extra-peritoneal haematomas tracking up the anterior abdominal wall. Perhaps the main benefit of DPL is to exclude intra-peritoneal haemorrhage. A low threshold should be maintained for laparotomy because of associated intra-peritoneal 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 washout of the rectum due to the risk of pelvic infection introduced by washing faeces into the pelvic cavity. Careful mechanical cleansing of the rectum, washout via a wide-bore tube after gentle anal dilatation, and adequate debridement performed in a stable patient make 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.

MAST/PASG (pneumatic anti-shock garment) control should only be considered a temporary measure in this condition, as it induces ischaemia in wide areas and compartment syndromes.)

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.

Associated injuries can only be managed once the patient is haemodynamically stable. Procedures for damage control may be the only available option.

In summary, a haemodynamically normal patient can be safely transferred for stabilization of unstable fractures within hours after injury and following control of the associated damage.

7.6 RECOMMENDED READING

- Scxalea TM, Burgess AR.. Pelvic fractures. In Mattox KL Feliciano DV, Moore EE, (eds), *Trauma*, 4th edition. New York: McGraw-Hill, 2000, 807–38.
- Fry RD. Anorectal trauma and foreign bodies. *Surgical Clinics of North America* 1994; **74**(6):1491–506.

Vascular injury must be assumed in any wound in the vicinity of major blood vessels. Unless they are markedly unstable, all trauma patients should have a complete vascular examination, including palpation of carotid, subclavian, brachial, radial, femoral, popliteal, dorsalis pedis and posterior tibial arteries. Neurologic function must also be assessed. Any injured part should be auscultated over the area of obvious or suspected injury.

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.

Recently, duplex scanning of blood vessels has been shown to be a useful adjunct in determining if an arteriogram is indicated. A positive duplex scan is valuable, but a negative one does not exclude vascular injury. A positive duplex scan or an ankle-brachial index <0.9 in a distal pulse is a mandatory indication for arteriogram and possible operation.

The gold standard for confirming a suspected vascular injury remains the arteriogram. However, arteriography should not be performed in the patient who is unstable and needs emergency laparotomy or thoracotomy: it should be delayed until after resuscitation and treatment of the life-threatening emergency.

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. The key, therefore, is to maintain a high index of suspicion based on the mechanism of injury and the proximity of vascular structures.

If doubt exists, an angiogram should be obtained!

Penetrating trauma also includes iatrogenic

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.

Blunt trauma may also cause peripheral vascular injuries, shear injuries being 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.

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.

8.1 SPECIFIC INJURIES

8.1.1 Injuries to the neck

All patients with zone 1 and 3 neck injuries should undergo arteriography as soon as vital signs are stable. Zone 1 injuries require arteriography because of the increased association of vascular injuries with penetrating trauma to the thoracic outlet. Arteriography helps the surgeon plan the surgical approach. Zone 3 injuries require arteriography because of the relationship of the blood vessels to the base of the skull. Often, these injuries

can best be managed by either non-operative techniques or manoeuvres remote from the injury site, such as balloon tamponade or embolization. Injuries in zone 2 may require arteriography if vertebral vessels are thought to be injured. However, arteriography will *not* rule out significant injury to the venous system, trachea or oesophagus.

8.1.2 Injuries to the chest

Arteriography is mandatory in any patient who has radiological evidence of a widened mediastinum following blunt chest trauma. Associated findings include obscuring of the aortic shadow, deviation of the trachea to the right, apical haematoma, and depression of the left main stem bronchus. Controversy surrounds some of the diagnostic techniques, and arteriogram is still the gold standard, although a good quality spiral computed tomography (CT) scan may be very helpful; however, CT scans still have a fairly high false-negative rate and are not a replacement for aortogram. Trans-oesophageal ultrasound has excellent resolution, but may miss injuries at the root of the aorta or at the aortic hiatus. Controversy also exists about operative management. In principle, the incidence of paraplegia is lower if cardiopulmonary bypass is used. Aorta-to-femoral-vein bypass using non-heparinized shunt is gaining popularity. Transmural pressure measurements and decompression may be a useful adjunct.

8.1.3 Injuries to the abdomen

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 room thoracotomy and aortic cross-clamp.

This procedure is not indicated in the severely shocked patient with blunt abdominal trauma, as 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 operative approach or interventional radiology is appropriate.

8.1.4 Extremity injury

Extremity vascular injuries are present in 25–35 per cent of all penetrating trauma to the extremities. A significant percentage of these patients have no physical findings suggesting vascular trauma; thus, routine use of arteriography has been advocated. Duplex scanning may now also play a useful screening role. Except for inconsequential intimal injuries and distal arteries, most extremity vascular injuries should be repaired.

Chemical vascular injuries may be treated with intra-arterial or intravenous administration of 10 000 units of heparin to prevent distal thrombosis. Reserpine (0.5 mg) also has 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 of 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 minutes or intravenously at the rate of 1000 units heparin per hour.

8.2 COMPARTMENT SYNDROME

Compartment syndrome occurs relatively commonly following trauma or ischaemia to an extremity. It is important to emphasize that reperfusion probably plays a major role. As such, the classical clinical findings may be absent prior to vascular repair. Once the diagnosis of compartment syndrome is made, urgent fasciotomy is indicated.

The measurement of intra-compartment pressure is invaluable when doubt exists about the diagnosis. It must be emphasized that a pulse may still be palpable, or recordable on the Doppler, even though a compartment syndrome exists.

Should there be doubt as to whether the compartment syndrome is significant, a fasciotomy should be performed.

8.3 FASCIOTOMY

Two techniques have been described:

- 1 fibulectomy
- 2 two-incision, four-compartment fasciotomy.

8.3.1 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.

8.3.2 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, 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 intra-compartmental pressures.

8.4 RECOMMENDED READING

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Part IV

Additional (optional) modules

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Critical care of the trauma patient 9

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 or 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 hours 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-threatening 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. Though 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 hospital 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, PaO_2 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 convection heat loss,
 - warmed ventilator circuits (airway warming),
 - warmed fluids,
 - external warming blankets,
 - radiator heaters,
 - pleural and peritoneal lavage,
 - arterio-venous warming circuits,
 - cardiopulmonary bypass.

9.3.2 Early life support phase (24–72 hours post-injury)

During this phase, treatment is focused on the management of post-traumatic respiratory failure and progressive intracranial hypertension in patients suffering from 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 during this phase 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, 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.
- Intracranial pressure monitoring and control.
- Fluid and electrolyte balance.
- Haematological parameters.
- Occult injuries.
- Delayed intracranial haematoma formation:
 - follow-up computed tomography (CT) scan of the head,
 - intra-abdominal injuries,
 - follow-up CT scan or ultrasound of the abdomen.
- Cervical spine injury:
 - completion of the radiographic 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 hours 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, whereas the more seriously injured enter a phase in which ongoing life support is necessary to prevent organ system failure. The 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 pressure area skin compromise. 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.
- Tracheostomy: early.

9.3.3.2 INFECTIOUS COMPLICATIONS⁴

- 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-INFECTIOUS CAUSES OF FEVER

- Drugs.
- Pulmonary embolism.
- Deep vein thrombosis.

9.3.3.4 PERCUTANEOUS TRACHEOSTOMY

Percutaneous tracheostomy has been shown to have fewer peri-operative and post-operative complica-

tions compared to conventional tracheostomy and is now the technique of choice in critically ill patients.⁵

Various techniques are described, with dilatation by forceps, 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 fiberoptic 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 MULTIPLE 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 systems 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 post-injury) is usually a consequence of shock or inadequate resuscitation, whereas a late onset is usually a result of severe infection.

MODS develops as a consequence of an 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 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 comorbid 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 commonest coagulation abnormality in trauma patients.
- 2 Consumption of clotting factors.
- 3 Hypothermia:
 - 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 the 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 occur. 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, 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 rewarming and replacement of blood products. Empirical transfusion of platelets, fresh frozen plasma and cryoprecipitate are 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, though, largely academic: the key step in the management of DIC is resolution of the condition predisposing to the coagulopathy. The condition will not resolve until the underlying cause is corrected; whilst this is achieved, component therapy is indicated.

9.5.2 Suggested transfusion guidelines

- 1 Start red blood cell transfusion based on pattern of injury, haemodynamic status,

response to resuscitation and arterial blood gas analysis (lactate/base deficit/haematocrit).

- 2 Start fresh frozen plasma (FFP), platelets, cryoprecipitate, based on presence of clinical coagulopathy and transfusion of 6–10 units of packed red blood cells (PRBCs) with anticipated transfusion of more PRBCs.
- 3 Aim for platelets $>50\,000\text{ mm}^3$.
- 4 Aim for prothrombin time (PT) $<16\text{ s}$.
- 5 Aim for fibrinogen $>100\text{ mg/dL}$.
- 6 Aim for haematocrit >20 and <30 per cent.

NB. Laboratory parameters may lag behind clinical events – do not wait for laboratory values.

- 1 Stop PRBC transfusion based upon haemodynamic stability, control of blood loss and haematocrit in the 20–30 per cent range.
- 2 Stop platelet transfusion when patient is not ‘oozing’ and platelet count $>50\,000\text{ mm}^3$.
- 3 Stop FFP transfusion when patient is not ‘oozing’ and PT $<16\text{ s}$.
- 4 Stop cryoprecipitate transfusion when patient is not ‘oozing’ and fibrinogen $>100\text{ mg/dL}$.

NB. Remember to use external warming blanket and warmed fluids for all patients.

9.5.3 Suggested protocol for massive transfusion

9.5.3.1 PATIENTS AT RISK

Hypovolaemic shock

- BP $<80\text{ mmHg}$ in adult.
- BP between 80 and 90 mmHg, unresponsive to 2-L crystalloid bolus.
- BP $<60\text{ mmHg}$ in child <12 years.

Major obvious blood loss

- Unstable pelvic fracture.
- Massive haemothorax.

9.5.3.2 IMMEDIATE TRANSFUSION REQUIRED

Give emergency blood

- O –ve blood or, if not available, O +ve.
- Only transfuse when need is *immediate*.
- Take care transfusing O +ve blood to women of child-bearing age.
- Emergency blood should be given via warming infuser.

Some delay acceptable

- 15-minute delay:
 - ask blood bank for *type-specific blood*.
- 60-minute delay:
 - ask blood bank for *cross-matched blood*.
- All protocols regarding identification of specimens and blood for transfusion must be followed despite urgency of the trauma setting.
- The commonest reason for a major incompatibility reaction is mis-identification by medical or nursing staff.
- Patients must all have two identity bands (wrist and ankle).

9.5.3.3 IN OPERATING ROOM AND ICU

- Alert blood bank that ‘massive transfusion’ is in progress.
- External warming blanket is mandatory.
- Cover patient’s head.
- All fluids to be warmed.
- Warm saline should be used to irrigate the abdomen, specifically to raise the core temperature.
- Patients undergoing massive transfusion (>6 units) should have 1 or more units of fresh frozen plasma per 6-unit PRBC transfusion.
- Platelets should be given if there is a massive transfusion requirement and there is ‘oozy’ bleeding.

9.6 RECOGNITION AND TREATMENT OF RAISED INTRACRANIAL PRESSURE

Early mortality in blunt trauma patients in the ICU is often caused by head injury. The primary goal in the 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 intracranial pressure. Consequently, monitoring and controlling intracranial pressure and cerebral perfusion pressure are a high priority in this phase of ICU care. Other conditions that worsen brain injury include:

- hypotension
- hypoxia
- hyperglycaemia
- hyperthermia
- hypercarbia.

9.7 RECOGNITION OF ACUTE RENAL FAILURE

Although the frequency of acute renal failure is relatively low, injured patients are at high risk of developing it 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 acute renal failure complicates the ICU management of a patient, increases the length of stay, and is associated with a mortality of approximately 60 per cent. Approximately one-third of acute post-traumatic renal failure cases are caused by inadequate resuscitation, and 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 the elderly 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 oxygen 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 radiographic lung volumes, or reluctance to cough and co-operate with chest physiotherapy. If the patient can co-operate, visual analogue pain scores may be helpful. Early pain control in the ICU is primarily achieved through the use of intravenous opioids. Other techniques are employed and tailored to the individual patient and injury:

- bolus opioids
- 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 the elderly, determination of living wills or other pre-determination documents is important.

9.11 ICU TERTIARY SURVEY⁶

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 the majority will be identified by a thorough tertiary survey.

9.11.1 Evaluation for occult injuries

Factors predisposing to missed injuries:

- mechanism of injury: reverify 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 Assess co-morbid conditions

- Medical history (including drugs and alcohol).
- Contact personal physicians.
- Check pharmacy records.

9.12 NUTRITIONAL SUPPORT⁷

Trauma patients are hypermetabolic and have increased nutritional needs due to the immunologic response to trauma and the requirement for accelerated protein synthesis for wound healing. Early enteral feeding has been shown to reduce post-operative 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 to total parenteral nutrition.

Head-injured patients appear to have similar outcomes whether fed enterally or parenterally. A recent Cochrane review⁸ confirmed that early (either parenteral or enteral) feeding is associated with a trend towards better outcomes in terms of survival and disability compared to later feeding. However, the superior results of enteral feeding over parenteral nutrition are not as clear for patients with brain injury. Parenteral and enteral nutritional support for brain-injured patients has

been compared in a randomised, prospective study.⁹ 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 due to lower total protein intake, cumulative caloric balance and negative nitrogen balance.

Enteral nutrition should be used when the gut is accessible and functioning.¹⁰ It 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.¹¹

Patients at risk include those with:

- major trauma
- burns.

It is critical to:

- determine energy and protein requirements,
- determine and establish a route of administration,
- set a time to begin nutrition 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. The majority 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, naso-enteric tubes are inconvenient, as they tend to dislodge and worsen aspiration and are uncomfortable.

9.12.1.2 MORE COMPLICATED

- Percutaneous endoscopic gastrostomy (PEG): PEG does not interfere with swallowing, is easy to nurse, and target feeding rates are more likely to be achieved compared to naso-enteric tubes; however, it is an invasive procedure with some risk.
- Jejunostomy: jejunostomy can be placed endoscopically or during laparotomy; rates of major complications should be less than 5 per cent.¹²

9.13 PREVENTIVE MEASURES IN THE ICU

9.13.1 Stress ulceration

Stress ulceration and associated upper gastrointestinal bleeding have been on the decline in most ICUs for the past decade. This is due, in great part, to the improved resuscitation efforts in the pre-hospital environment, emergency departments and operating room. Additionally, 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 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 blockade therapy (e.g. ranitidine) blocks the 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 blockade as the mainstay of therapy.

Perhaps the simplest and safest method of stress ulcer prevention is adequate resuscitation and early intra-gastric enteric nutrition. During the early resuscitative phase and while vasoactive drugs for the elevation of blood pressure are in use, it is not always prudent to provide nutrition enterally. It is in these circumstances that the use of acid blockade, cytoprotective agents or both is necessary.

9.13.2 Deep venous thrombosis and pulmonary embolus¹³

Pulmonary embolus (PE) from deep vein thrombosis (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 and long-bone fracture/pelvis fracture.

A high index of suspicion in these severely injured patients should result in preventative therapy and diagnostic screening measures being taken in the ICU. Unless hemorrhagic traumatic brain injury 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 ultrasound or duplex Doppler. Both of these are 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 being conducted now. This screening should be performed whenever clinical suspicion of DVT arises, within 48 hours 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 cava filter should be made. The lifetime risk of the filter appears to be quite low in several studies, with an obvious significant benefit in the prevention of death. An additional subgroup of patients to be considered for prophylactic inferior vena cava filter placement would be those with significant injuries who also have 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 inferior vena cava filters can result in a fatal PE rate of significantly less than 1 per cent of the trauma ICU population.

9.13.3 Infection

For patients with any open wounds from trauma, it is imperative that their tetanus immunization status is addressed. For those patients immunized within the previous 5 years, no additional treatment is generally needed; booster tetanus toxoid should be administered to those who have previously received the initial tetanus series but have not been re-immunized 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*, meningococcus and pneumococcus. Debate continues regarding 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 paediatric patients post-splenectomy. Due to the multiple strains of each organism, the immunizations are not foolproof in preventing overwhelming post-splenectomy infection. Therefore, patients must be carefully counselled to seek medical attention immediately for high fevers, and health care providers must be aggressive in the use of empiric antibiotics in patients who may have overwhelming post-splenectomy infection 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 more than 6 hours has elapsed since injury, moist dressings (to prevent tissue desiccation and further non-viable wound tissue) should be applied and changed twice per 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 wounds on the back and occiput (pressure may cause additional wound necrosis).

Thrombophlebitis and sepsis from intravenous cannulae are significant considerations, as these intravenous lines are frequently placed under less than optimal circumstances and technique in the field and in the resuscitation areas. Removal and replacement of all such lines as early as possible – but in every instance in less than 24 hours – 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 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 regarding the needs for routine antibiotics with tube thoracostomy.

For thoraco-abdominal injuries requiring operation, a single dose of broad-spectrum antibiotics is indicated. Prolonged courses of antibiotics, extending beyond 24 hours, 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 for whether the correct management should be any different from 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 Center for Disease Control, the diagnosis of pneumonia must meet the following criteria:

- rales 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,
- radiographic evidence of new or progressive infiltrate, consolidation, cavitation or effusion,
- isolation of virus or detection of viral antigen in respiratory secretions,
- diagnostic antibody titres for pathogen,
- histopathologic evidence of pneumonia.

Empiric antibiotic treatment for ventilator-associated pneumonia may not be adequate, and a recent international consensus supports a policy based on early initiation of high doses broad-spectrum antibiotics, with 'de-escalation of treatment' to narrow-spectrum antibiotics based on microbiological results.

Antibiotic guidelines should be formulated locally, and the empiric antibiotic regimen should be 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 RESPIRATORY

- '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 for ARDS.
- Prone ventilation may improve oxygenation in patients with ARDS or severe sepsis.
- Ventilator-associated pneumonia is the commonest hospital-acquired infection in ICUs.¹⁶

9.16 ORGAN DONATION

The identification of potential organ donors amongst 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*.

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Operating in austere or military environments 10

10.1 INTRODUCTION

Many military health care teams around the globe are now incorporating forward resuscitative surgery capabilities into doctrine and mission planning. Although these forward surgical teams provide only a limited trauma surgical capability, they aim to provide life-saving and limb-saving surgery to the select group of potentially salvageable patients who would otherwise suffer due to delays in evacuation from the fast-moving modern battlefield.

These teams should be capable of providing life-saving thoraco-abdominal 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 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 little 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.

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. The *raison d'être* of the team is delivery of the life-saving and limb-saving surgery as far forward as possible on the modern-day battlefield.

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 due to 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, neck, pelvis and groin.

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 health care 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 front lines, extended lines of logistics and delay in evacuation, the modern military doctor is likely to be called upon to treat civilians 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 TRIAGE

Effective triage is crucial in an efficient military health care system, which was 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 could return to the battle with minor treatment. Although we might now call this reverse triage, Larrey 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.

There are several factors that will influence decision making within the triage process at the forward surgery level. Staff safety and available time on the ground will influence which surgical interventions are possible and therefore which patients can go on the operating table. 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, as resupply 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 straightfor-

ward. 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.

The system for 'surgical triage' may be slightly different from 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 'Do they need to go on the table at all?'. Overtriage is a feature of all mass casualty situations; however, a rate of overtriage 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 (Hardaway, 1978). In a modern military setting, an accurate screening tool (perhaps focused abdominal sonography for trauma) would be very useful.

Triage is challenging; it requires difficult decisions to be made, but it remains crucial to the effective use and efficiency of the forward surgical teams.

10.4 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, however, may 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 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, for example 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 the 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, manpower and transport will be in short supply, so sound training and adhering to the principles of triage should ensure the 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, in which 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 the right patient arrives at the right time at the next level of medical care.

10.5 EVACUATION

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 minutes. 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. This concept of using cargo aircraft was originally validated in World War II 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.

10.6 RESUSCITATION

Hypovolaemia remains the commonest cause of death amongst those killed in action during military conflicts. Although the principles remain the same, the 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 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 of infused crystalloid for the acutely injured hypotensive patient is not feasible in the far-forward environment due to logistical constraints, but is also likely to be detrimental to the survival of the patient with uncontrolled haemorrhagic shock in whom surgical intervention is not immediately available. For patients with uncontrolled haemorrhagic shock, the goal of maintaining a systolic arterial pressure of 70–80 mmHg is now accepted by most practitioners treating the wounded forward of the first surgical capability.

Blood is the gold-standard fluid of choice in casualties with uncontrolled haemorrhagic shock, 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 health care 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 acquiring it. Intra-osseous access is an attractive option in these scenarios.

10.7 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-aid and buddy-aid levels; protocols to guide medical and paramedical staff in the provision of safe and effective analgesia are available.

10.8 BATTLEFIELD ANAESTHESIA

Surgery requires both adequate analgesia and anaesthesia. No single agent can provide an appropriate level of anaesthesia and analgesia, hence 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 the surgical exploration of body cavities, general anaesthesia is most frequently chosen, whereas a regional anaesthetic 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 pre-oxygenation of the patient's lungs is often not possible. There are several RSI cocktails used in the pre-hospital setting, most involving 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 inhalation 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, as 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 anaesthetic techniques should not be overlooked.

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 post-operative mechanical ventilation. Mass casualty situations are also a constant possibility in the military arena.

10.9 DAMAGE CONTROL SURGERY IN THE MILITARY SETTING

Damage control is a term used by the US Navy to describes the capacity of a ship to absorb damage, provide running repairs and still maintain mission integrity. The phrase has been adopted to describe the evolving concept of saving life after major trauma by deferring the treatment of anatomical disruptions and focusing on restoring the patient's physiology. In the civilian trauma centre setting, damage control is indicated for the severely injured patient who does not respond to the usual sequence of resuscitation and who appears to be dying. Rapid, abbreviated surgery is carried out to control bleeding and contamination and the patient is then subjected to extremely resource-intensive and personnel-intensive therapy. This process commits the patient (and the medical system) to ongoing resuscitation and further operative procedures.

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 hours, full

invasive monitoring, multiple operations, massive transfusion of blood and products and prolonged ICU stay, and to have a high risk of 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 US 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 for every casualty. Short, focused operative interventions can be used on peripheral vascular injuries, extensive bone and soft-tissue injuries and thoraco-abdominal penetrations in patients with favourable physiology, instead of *definitive* surgery for every injury. 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 physiologic 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 post-operative 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.

10.10 CRITICAL CARE

If TASC is going to be the norm for the far-forward surgeon, a critical care capability must be a part of the forward surgical team structure. The priorities will therefore be optimization of haemodynamic status, rewarming of the casualty, pain

relief and preparation for return to theatre, or evacuation, depending on the situation. The health care 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 rewarming 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.

10.11 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.

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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.

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, the examination is deemed incomplete and an alternative means of investigation is required, or FAST must be repeated at frequent intervals.

FAST is non-invasive and repeatable. Repeated scans have been shown to increase its sensitivity.

FAST is equally accurate when used by appropriately trained surgeons as by radiologists.

11.1.1 COMPARISON OF FAST WITH OTHER TECHNIQUES OF ABDOMINAL SCREENING

In blunt abdominal trauma, FAST has a sensitivity of approximately 86 per cent and a specificity of approximately 98 per cent for the detection of intra-abdominal injuries. The positive predictive value is approximately 87 per cent and the negative predictive value 98 per cent.

FAST examination relies on the detection of free intra-peritoneal fluid. In the hands of most operators, ultrasound will detect a minimum of 200 mL of fluid. Therefore false-negative examinations may occur (e.g. hollow viscus injury).

The retroperitoneum is not well visualized.

An International Consensus Meeting concluded that a negative FAST examination should be followed up by a period of observation of at least 6

hours and a follow-up FAST. The alternative is to use diagnostic peritoneal lavage (DPL) or computed tomography (CT) to confirm the ultrasound findings.

11.2 OTHER APPLICATIONS OF ULTRASOUND IN TRAUMA

11.2.1 Ultrasound in penetrating 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.

11.2.2 Ultrasound in thoracic trauma

The evaluation of fluid in the pericardium is a standard part of the FAST assessment after blunt trauma. A prospective, multi-centre evaluation of 261 patients with a penetrating precordial or transthoracic wound suspicious for cardiac injury demonstrated an accuracy of 97.3 per cent.

Ultrasound can be valuable in the detection of haemothorax; with sensitivity and specificity similar to those for the portable chest X-rays. However, the performance time for ultrasound is significantly shorter.

Ultrasound has been used to detect pneumothorax, with 95 per cent sensitivity.

11.3 CONCLUSION

Amongst 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, co-operative, 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 positive or 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.
- Ultrasound is useful in detecting thoracic injuries, particularly cardiac tamponade.

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Minimally invasive surgery in trauma **12**

Minimally invasive techniques have yet to be enthusiastically adopted by trauma surgeons, unlike their general surgical colleagues. However, indications for the use of these techniques are emerging. Physiological instability is a contraindication to minimally invasive techniques.

12.1 THORACIC INJURY

Persistent, non-exsanguinating haemorrhage can be investigated and occasionally treated by video-assisted thoracoscopic surgery (VATS).

New techniques in VATS can significantly improve the management of chest trauma.

- VATS can accomplish the 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

Suspected diaphragmatic injury due to both blunt and penetrating injury can be accurately evaluated by VATS. In the largest series of patients evaluated for suspected penetrating diaphragmatic injury, 171 stable patients with penetrating chest injury and without a separate indication for either thoracotomy or laparotomy were investigated with VATS. Sixty patients (35 per cent) had a diaphragmatic injury and the majority (93 per cent) of these were repaired using a laparotomy. Of the patients with diaphragmatic injury, 47/60 (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. Thoracoscopic pericardial windows performed in a series of patients with suspected tamponade showed no significant complications and were found to be accurate in 97 per cent of cases.

12.3 ABDOMINAL INJURY

12.3.1 Screening for intra-abdominal injury

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.

Laparoscopy has been used in attempts to minimize unnecessary laparotomies to assess whether or not an equivocal wound has breached the peritoneal cavity. However, sensitivity is poor for hollow viscus injury.

12.3.2 Splenic injury

Laparoscopic splenic preservation has been reported after trauma using fibrin glue, argon beam coagulator and splenic wrapping with mesh. Successful autotransfusion of haemoperitoneum aspirated from the peritoneum has also been reported.

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 peritonitis can be controlled via the laparoscope, which may be combined with endoscopic retrograde cholangiopancreatography (ERCP) to achieve control of biliary leaks.

Reports of successful laparoscopic treatment for intra-abdominal organs other than the liver or spleen are scarce.

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 to gain familiarity and expertise with their use.

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Skeletal trauma 13

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 dressings; 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 fractures¹

Sepsis is a constant threat to the healing of open fractures. The risk factors for infection include:

- severity of injury (especially the injury to the soft-tissue envelope of a limb),
- delay from injury to surgical care (>6 hours),
- failure to use prophylactic antibiotics,
- inappropriate wound closure.

13.2.2 Severity of Injury (Gustilo classification)

- *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 further stratified Grade III wounds.²

- *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 Antibiotics³

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, Trueta⁴ 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 Guidelines⁵) recommend antibiotics be discontinued 24 hours after wound closure for Grade I and II fractures. For Grade III wounds, the antibiotics should be continued for only 72 hours after the time of injury, or not more than 24 hours after soft-tissue coverage of the wound is 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 patients⁶

13.2.4.1 RESPIRATORY INSUFFICIENCY⁷

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 the risk of post-traumatic respiratory compromise after early, definitive fixation of fractures (i.e. within 48 hours), both for isolated injuries and for 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 INJURY⁸

In approximately 5 per cent of long bone fractures of the leg, the patient is physiologically unstable after initial resuscitation due to 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 the outcome.

13.3 AMPUTATE OR PRESERVE A SEVERELY DAMAGED LIMB?⁹

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 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 (see Section 3.7 on the management of the mangled limb).

13.4 COMPARTMENT SYNDROME^{10,11}

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 VENOUS THROMBO-EMBOLISM

Venous thrombo-embolism (VTE) is a significant problem following skeletal injury. VTE prophylaxis is an integral part of the management of all such patients.¹²

13.6 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

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In the Western world, the most common cause of death after trauma is severe brain injury, which contributes significantly to half of all deaths from trauma. The severely brain injured also have 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 the 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 scanning has been shown to reduce mortality for patients with acute extradural haematoma, as the time taken to diagnose and evacuate an intracerebral haematoma is critical in determining outcome. However, the majority of patients with brain injury do not have a lesion that is 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.

14.1.1 Associated injury

All patients sustaining a major mechanism of injury should be suspected of having a cervical spine injury. The avoidance of hypotension and hypoxia is 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 Score (GCS) of 5 or less after resuscitation, or a GCS of 8 or less 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 due to 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 (Cochrane Database) recently reviewed the available evidence and concluded that, whereas prophylactic anti-epileptics are effective in reducing early seizures, there is no evidence that treatment with prophylactic anti-epileptics 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 clear indications exist.

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Trauma systems

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 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, acute care and rehabilitation. The initial trauma systems did not consider the non-trauma centre hospitals, even though they cared for the majority of patients – those less severely injured. Rather, these systems were driven by the major or severely injured trauma patients 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, geographic location or population density. The trauma centre remains an essential component, but the system recognizes the necessity of having other health care 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 as follows.

- Administrative components:
 - leadership
 - system development
 - legislation
 - finances.
- Operational and clinical components:
 - injury prevention and control
 - human resources:
 - workforce resources
 - education
 - pre-hospital care:
 - emergency medical services
 - ambulance and non-transporting guidelines:
 - communications systems
 - emergency disaster preparedness plan
 - definitive care facilities:
 - trauma care facilities
 - inter-facility transfer
 - medical rehabilitation
 - information systems
 - evaluation
 - research.

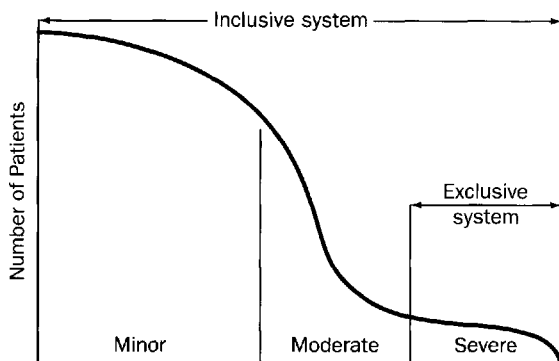


Figure A.1 Concept of an inclusive trauma system.

This can be graphically represented as shown in Figure A.2.

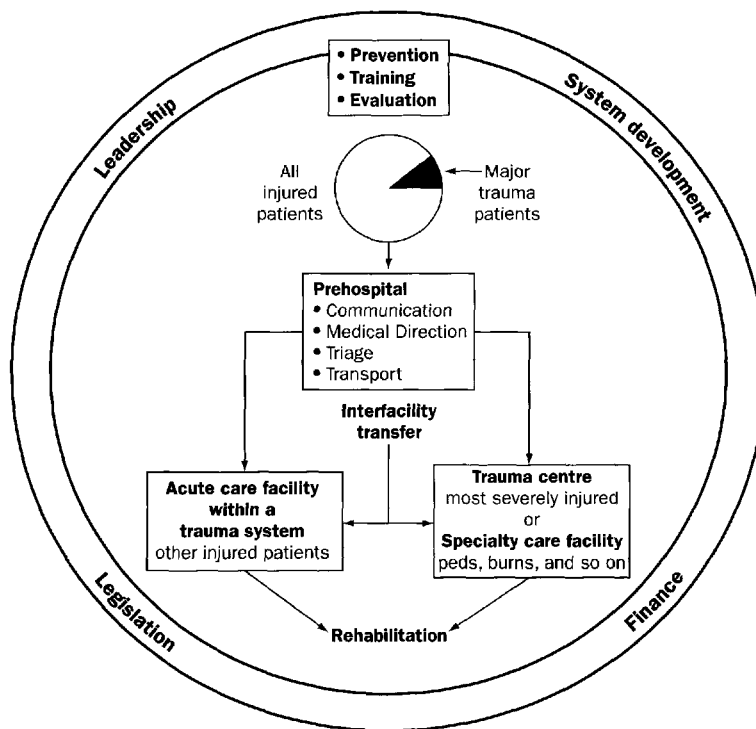


Figure A.2 The trauma 'wheel': components of a trauma system.

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 (the young, or the elderly), must be addressed by the system.

A.1.2 Prevention

Prevention reduces the actual incidence of injury, and is cost efficient for the system and for 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 only succeed 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 prior to trauma centre access. Triage guidelines must be accepted by all providers and used to determine which patients require access to trauma centre care. This co-ordination requires direct communication between 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 co-ordinates 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

- *Public support.* Public support is necessary for the enabling and necessary legislation to take place. The process takes place as follows.
 - 1 Identification of the need.
 - 2 Establishment of a patient database to assist with need and resource assessment.
 - 3 Analysis to determine resources available.
 - 4 Resource assessment formulated to identify the current capabilities of the system.
 - 5 Deficiencies highlighted and solutions formulated.
- *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 health care, 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.

- *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.
- *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 information from the hospitals managing the less severely injured, the data will be incomplete, and skewed towards major injury.

- *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. Its purpose was to evaluate the evidence regarding the efficacy of trauma systems. During the conference, the evidence was divided into three categories: that resulting from panel studies, registry comparisons, and population-based research.

- *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, autopsy results alone are inadequate, and panel studies vary regarding 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, MacKenzie¹ 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 a non-trauma centre is associated with fewer inappropriate deaths and possibly disabilities.
- *Registry study.* Jurkovich and Mock² 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 panel study. Their critique of trauma registries included the following six items: data are often missing, miscodings occur, there may be inter-rater reliability factors, the national norms are not population based, there is less detail about the causes of death, and 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.
- *Population-based studies.*³ Populated-based studies probably also fall into Class II evidence. They are not prospective, randomized trials, but, because of nature of the population-based evidence, they cover all aspects of trauma care, including pre-hospital, hospital and rehabilitation. 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 physiologic 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 three can be used pre-trauma and post-trauma system commencement. 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.

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Trauma scores and scoring systems

In principle, scoring systems can be divided into:

- physiological scoring systems
- anatomical scoring systems
- outcome analysis 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.

B.2 PHYSIOLOGICAL SCORING SYSTEMS

B.2.1 Glasgow Coma Scale (GCS)

The Glasgow Coma Scale, devised in 1974,¹ 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.

B.2.2 Revised Trauma Score (RTS)

Introduced by Champion et al., the Revised Trauma Score² evaluates blood pressure, GCS and respira-

Table B.1 *The Glasgow Coma Scale (GCS)*

Parameter	Response	Score
Eye opening	Nil	1
	To pain	2
	To speech	3
	Spontaneously	4
Motor response	Nil	1
	Extensor	2
	Flexor	3
	Withdrawal	4
	Localizing	5
	Obeys command	6
Verbal response	Nil	1
	Groans	2
	Words	3
	Confused	4
	Orientated	5

tory 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. A RTS =11 is suggested as the triage point for patients requiring at least Level 2 trauma centre status (surgical facilities, 24-hour X-ray etc.). An RTS of =10 carries a mortality of up to 30 per cent, and these patients should be moved to a Level 1 institution.

The difference between the RTS on arrival and the best RTS after resuscitation will give a reasonably clear picture of the prognosis. By convention, the RTS on admission is the one documented.

The RTS (non-triage) is designed for retrospective outcome analysis. Weighted co-efficients are used, which are derived from trauma patient populations and provide more accurate outcome prediction than the raw RTS (Table B.2). Because a severe

head injury carries a poorer prognosis than a severe respiratory injury, the weighting is heavier. The RTS thus varies from 0 (worst) to 7.8408 (best). It is the most widely used physiological scoring system in the trauma literature.

Table B.2 Revised Trauma Score (RTS)

Clinical parameter	Category	Score	x weight
Respiratory rate (breaths/min)	10–29	4	0.2908
	>29	3	
	6–9	2	
	1–5	1	
	0	0	
Systolic blood pressure mmHg	>89	4	0.7326
	76–89	3	
	50–75	2	
	1–49	1	
	0	0	
Glasgow Coma Scale	13–15	4	0.9368
	9–12	3	
	6–8	2	
	4–5	1	
	3	0	

The values for the three parameters are summed to give the Triage-RTS. Weighted values are summed for the RTS.

B.2.3 Paediatric Trauma Score (PTS)

The Paediatric Trauma Score³ has been designed to facilitate triage of children. It 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 (Table B.3). The PTS has been shown to accurately predict risk 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 (AIS)

The Abbreviated Injury Scale⁴ was developed in 1971. It grades each injury by severity, from 1 (least severe) to 5 (survival uncertain), within six body regions (head/neck, face, chest, abdominal/pelvic contents, extremities and skin/general). The AIS has

Table B.3 Paediatric Trauma Score (PTS)

Clinical parameter	Category	Score
Size (kg)	>20	+2
	10–20	+1
	<10	–1
Airway	Normal	+2
	Maintainable	+1
	Unmaintainable	–1
Systolic blood pressure (mmHg)	>90	+2
	50–90	+1
	<50	–1
Central nervous system	Awake	+2
	Obtunded/LOC	+1
	Coma/decerebrate	–1
Open wound	None	+2
	Minor	+1
	Major/penetrating	–1
Skeletal	None	+2
	Closed fracture	+1
	Open/multiple fractures	–1

The values for the six parameters are summed to give the overall PTS.
LOC loss of consciousness.

been periodically upgraded and AIS-90 is currently being revised.

In 1974, Baker et al. created the Injury Severity Score⁵ (ISS) to relate AIS scores to patient outcomes. 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 only considers 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 ISS but markedly different survival probabilities (an ISS of 25 may be obtained with isolated severe head injury or by a combination of lesser injuries across different regions). Also, the 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, the ISS was modi-

Table B.4 Injury Severity Score (ISS) body regions

Number	Region
1	Head and neck
2	Face
3	Thorax
4	Abdomen
5	Extremities
6	External

fied in 1997 to become the New Injury Severity Score⁶ (NISS) – the simple sum of the squares of the three highest AIS scores regardless of body region. The NISS is able to predict survival outcomes better than the ISS.

B.3.1.1 ANATOMIC PROFILE (AP)

The Anatomic Profile was introduced in 1990 to overcome some of the limitations of the ISS.⁷ In contrast to 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. 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 four-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 (MOTS).

A limitation of the use of AIS-derived scores is their cost. International Classification of Disease (ICD) taxonomy is a standard used by most hospi-

Table B.5 Component definitions of the modified Anatomic Profile (mAP)

Component	Body region	AIS severity
mA	Head/brain	3–6
	Spinal cord	3–6
mB	Thorax	3–6
mC	Front of neck	3–6
	All other	3–6

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.

tals and other health care 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.

Severity scoring systems have also been directly derived from ICD coded discharge diagnoses. Most recently, the ICD-9 Severity Score⁸ (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. The ICISS has been shown to be better than the ISS and to outperform the Trauma Score and Injury Severity Score analysis (TRISS) in identifying outcomes and resource utilization. However, mAP scores, AP and NISS appear to outperform ICISS in predicting hospital mortality.

There is some confusion as to which anatomic scoring system should be used; however, currently, the NISS should probably be the system of choice for AIS-based scoring.

B.3.2 Organ Injury Scalling (OIS) system

Organ Injury Scalling is a scale of anatomic injury within an organ system or body structure. The goal of OIS is to provide a common language amongst 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,^{1,9} and at the end of this appendix.

B.3.3 Penetrating Abdominal Trauma Index¹⁰ (PATI)

Moore and colleagues facilitated the identification of the patient at high risk of post-operative complications when they developed the Penetrating Abdominal Trauma Index scoring system for patients whose only source of injury was penetrating abdominal trauma.² 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 post-operative morbidity associated with the particular injury.

The severity of injury was estimated by a simple modification to the AIS, where 1 = minimal injury and 5 = maximal injury. The sum of the individual organ score times risk factor comprised the final PATI. If the PATI is 25 or less, the risk of complications is reduced (and where it is 10 or less, there are no complications); if it is greater than 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 post-operative complications (46 per cent of patients with a PATI score >25 developed serious post-operative complications, compared to 7 per cent of patients with a PATI score <25). Further studies have validated the PATI scoring system.

B.4 OUTCOME ANALYSIS

B.4.1 Glasgow Outcome Scale¹¹

For head-injured patients, the level of coma on admission or within 24 hours expressed by the GCS 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 B.6 Outcome related to level of consciousness

Death (D)
Persistent vegetative state (PVS)
Severe disability (SD)
Moderate disability (MD)
Good recovery (GR)

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 B.7 Outcome related to signs in the first 24 hours of coma after injury

	Dead or vegetative (%)	Moderate disability or good recovery (%)
Pupils:		
reacting	39	50
non-reacting	91	4
Eye movements:		
intact	33	56
absent/bad	90	5
Motor response:		
normal	36	54
abnormal	74	16

Outcome scale as described by Glasgow group.

B.4.2 Major Trauma Outcome Study (MTOS)

In 1982, the American College of Surgeons Committee on Trauma began the ongoing Major Trauma Outcome Study, a retrospective, multi-centre study of trauma epidemiology and outcomes.

The MTOS uses TRISS analysis¹² methodology to estimate the probability of survival, or $P(s)$, for a given trauma patient. $P(s)$ is derived according to the formula:

$$P(s) = 1/(1 + e^b)$$

where e is a constant (approximately 2.718282) and $b = b_0 + b_1(RTS) + b_2(ISS) + b_3(\text{age factor})$. 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

Table B.8 Coefficients from Major Trauma Outcome Study Database

Blunt	Penetrating
$b_0 = -1.2470$	-0.6029
$b_1 = 0.9544$	1.1430
$b_2 = -0.0768$	-0.1516
$b_3 = -1.9052$	-2.6676

expectation of survival. Each patient's values can be plotted on a graph with ISS and RTS axes (Figure B.1). The sloping line represents patients with a probability of survival of 50 per cent; these PRE charts (from PREliminary) are provided for those with blunt versus penetrating injury and for those above versus below 55 years of age. Survivors whose co-ordinates are above the $P(s)50$ isobar and non-survivors below the $P(s)50$ isobar are considered atypical (statistically unexpected) and such cases are suitable for focused audit.

In addition to analysing individual patient outcomes, TRISS allows comparison of a study population with the huge MTOS database. The 'Z-statistic' identifies if study group outcomes are significantly different from expected outcomes as predicted from MTOS. Z is the ratio $(A - E)/S$, where A = actual number of survivors, E = expected number of survivors, and S = 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, the more biased Z will be. This bias can be misleading, for example 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.

B.4.3 A Severity Characterization of Trauma (ASCOT)

A Severity Characterization of Trauma, introduced by Champion et al. in 1990,^{13,14} is a scoring system that uses the AP to characterize injury in place of the ISS. Different coefficients are used for blunt and penetrating injury and the ASCOT score is derived from the formula $P(s) = 1/(1 + e^{-k})$. The ASCOT model coefficients are shown in Table B.9. ASCOT has been shown to outperform TRISS, particularly for penetrating injury.

B.5 SUMMARY

Trauma scoring systems and allied methods of analysing outcomes after trauma are steadily

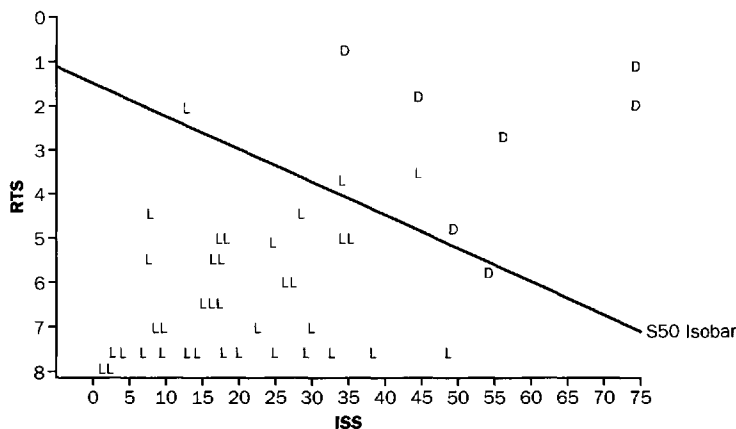


Figure B.1 PRE chart. Survivors (L) and Nonsurvivors (D) are plotted on a graph, using the weighted Revised Trauma Score (RTS) and Injury Severity Score (ISS) of each. The S50 isobar denotes a probability of survival of 0.50.

Table B.9 Coefficients derived from MTOS data for the ASCOT probability of survival, P(s)

k-coefficients	Type of Injury	
	Blunt	Penetrating
k ₁	-1.157	-1.135
k ₂ (RTS GCS value)	0.7705	1.0626
k ₃ (RTS SBP value)	0.6583	0.3638
k ₄ (RTS RR value)	0.281	0.3332
k ₅ (AP head region value)	-0.3002	-0.3702
k ₆ (AP thoracic region value)	-0.1961	-0.2053
k ₇ (AP other serious injury value)	-0.2086	-0.3188
k ₈ (age factor)	-0.6355	-0.8365

MTOS, Major Trauma Outcome Study; ASCOT, A Severity Characterization of Trauma; RTS, Revised Trauma Score; GCS, Glasgow Coma Scale; SBP, Systolic Blood Pressure; RR, Respiratory Rate; AP, Anatomic Profile.

evolving and have become increasingly sophisticated over recent years. They 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 for 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 only occur 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.5.1 References

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B.6 SCALING SYSTEM FOR ORGAN-SPECIFIC INJURIES

Table 1	Cervical Vascular Organ Injury Scale	Table 16	Rectum Injury Scale
Table 2	Chest Wall Injury Scale	Table 17	Abdominal Vascular Injury Scale
Table 3	Heart Injury Scale	Table 18	Adrenal Organ Injury Scale
Table 4	Lung Injury Scale	Table 19	Kidney Injury Scale
Table 5	Thoracic Vascular Injury Scale	Table 20	Ureter Injury Scale
Table 6	Diaphragm Injury Scale	Table 21	Bladder Injury Scale
Table 7	Spleen Injury Scale	Table 22	Urethra Injury Scale
Table 8	Liver Injury Scale	Table 23	Uterus (Non-pregnant) Injury Scale
Table 9	Extrahepatic Biliary Tree Injury Scale	Table 24	Uterus (Pregnant) Injury Scale
Table 10	Pancreas Injury Scale	Table 25	Fallopian Tube Injury Scale
Table 11	Oesophagus Injury Scale	Table 26	Ovary Injury Scale
Table 12	Stomach Injury Scale	Table 27	Vagina Injury Scale
Table 13	Duodenum Injury Scale	Table 28	Vulva Injury Scale
Table 14	Small Bowel Injury Scale	Table 29	Testis Injury Scale
Table 15	Colon Injury Scale	Table 30	Scrotum Injury Scale
		Table 31	Penis Injury Scale
		Table 32	Peripheral Vascular Organ Injury Scale

Table 1 *Cervical Vascular Organ Injury Scale*

Image Not Available

Table 2 Chest Wall Injury Scale

Image Not Available

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*

Image Not Available

Advance one grade for multiple wounds to a single chamber or multiple-chamber involvement.

From Moore et al.³ with permission.

Table 4 Lung Injury Scale

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
Haemothorax is scored under Thoracic Vascular Injury Scale.
From Moore et al.³ with permission.

Table 5 Thoracic Vascular Injury Scale

Image Not Available

Increase one grade for multiple Grade III or IV injuries if >50% circumference. Decrease one grade for Grade IV injuries if <25% circumference.
From Moore et al.³ with permission.

Table 6 *Diaphragm Injury Scale*

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
From Moore et al.³ with permission.

Table 7 *Spleen Injury Scale (1994 revision)*

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁴ with permission.

Table 8 *Liver Injury Scale (1994 revision)*

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁴ with permission.

Table 9 *Extrahepatic Biliary Tree Injury Scale*

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 10 *Pancreas Injury Scale*

Image Not Available

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.

Table 11 *Oesophagus Injury Scale*

Image Not Available

Advance one grade for multiple lesions up to Grade III.

From Moore et al.⁵ with permission.

Table 12 *Stomach Injury Scale*

Image Not Available

Advance one grade for multiple lesions up to Grade III.

From Moore et al.⁵ with permission.

Table 13 Duodenum Injury Scale

Image Not Available

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.

Table 14 Small Bowel Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁶ with permission.

Table 15 Colon Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.
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.
From Moore et al.⁶ with permission.

Table 16 Rectum Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.

From Moore et al.⁶ with permission.

Table 17 Abdominal Vascular Injury Scale

Image Not Available

From Moore et al.² with permission.

Table 18 *Adrenal Organ Injury Scale*

Image Not Available

Advance one grade for bilateral lesions up to Grade V.
From Moore et al.¹ with permission.

Table 19 *Kidney Injury Scale*

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
From Moore et al.⁷ with permission.

Table 20 *Ureter Injury Scale*

Image Not Available

Advance one grade for bilateral up to Grade III.
From Moore et al.² with permission.

Table 21 *Bladder Injury Scale*

Image Not Available

Advance one grade for multiple lesions up to Grade III.
From Moore et al.² with permission.

Table 22 *Urethra Injury Scale*

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
From Moore et al.² with permission.

Table 23 *Uterus (Non-pregnant) Injury Scale*

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 24 Uterus (Pregnant) Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 25 Fallopian Tube Injury Scale

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 26 Ovary Injury Scale

Image Not Available

Advance one grade for bilateral injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 27 Vagina Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 28 Vulva Injury Scale

Image Not Available

Advance one grade for multiple injuries up to Grade III.
From Moore et al.⁵ with permission.

Table 29 Testis Injury Scale

Image Not Available

Advance one grade for bilateral lesions up to Grade V.
From Moore et al.¹ with permission.

Table 30 Scrotum Injury Scale

Image Not Available

From Moore et al.¹ with permission.

Table 31 *Penis Injury Scale*

Image Not Available

Advance one *grade* for multiple injuries up to Grade III.
From Moore et al.¹ with permission.

Table 32 *Peripheral Vascular Organ Injury Scale*

Image Not Available

From Moore et al.¹ with permission.

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Appendix C

The Definitive Surgical Trauma Care (DSTC™) Course: requirements and syllabus



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C.1 BACKGROUND

Injury (trauma) remains a major health care 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, and
- traditional surgery training, which has been organ specific.

Consequently, surgeons can finish training with suboptimal skills in this field, in which 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 between Howard Champion (USA), David Mulder (Canada), Donald Trunkey (USA), Stephen Deane (Australia) and Abe Fingerhut (France) in October 1993.

This postgraduate surgical course for 16 participants assumes competence with assessment and resuscitative measures, which have become standardized through the ATLS® 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 final standardized course will have special relevance for surgeons in countries where major trauma rates are high and for 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 2 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 Uniform 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 currently working on bringing the course to Chile.
- There was a Swedish Trauma Surgery Course, which 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 1 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. There were some issues identified by the faculty and registrants and these have been addressed and incorporated. In particular, the didactic points in relation to organ support and pathophysiology have been placed in the manual.
- Since 1996, successful courses have been held world wide.

C.3 COURSE DETAILS

C.3.1 Definition

The DSTC™ Course is a Registered Trademark (TM) of the International Association for the

Surgery of Trauma and Surgical Intensive Care (IATSIC). IATSIC is an Integrated Society of the International Society of Surgery (ISS) based in Pratteln, Switzerland.

Only courses recognized by IATSIC may be called DSTC™ Courses.

C.3.2 Recognition

Application can be made to IATSIC for recognition of any course. Provided the minimum requirements have been met, as laid down below, IATSIC will recognize the course, which will then be entitled to be called a DSTC™ Course and to carry the IATSIC logo.

C.3.3 Mission statement

The DSTC™ Course is designed to train 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 human material, either cadaver or prosected, and/or animal tissue.

C.3.4 Eligibility to present

The DSTC™ Course can be presented by any tertiary academic institution or recognized surgical organization.

C.3.5 Course overview

The course is a mixture of manual-based and lecture-based learning, case discussion and surgical skills demonstration and practice.

- Manual addressing 'Physiology and Organ Support'.
- Theory session addressing technical surgical strategies.
- Practical session, using cadavers, addressing approaches to injured areas.
- Practical session, using animal laboratories, addressing organ injuries.
- Session addressing strategic thinking in trauma surgery.
- One session on troubleshooting (what if what I do doesn't work?).

C.3.6 Course materials

The course is over 2 days, with the following course materials:

- a manual to supplement the course content,
- practical and technical skill stations,
- patient scenarios in acute trauma surgery,
- practical work laboratory.

C.3.7 Course Director

The Course Director must be a full member of IATSIC, in addition to the requirements below.

C.3.8 Course Faculty

The Course Instructor Faculty must have completed an ATLS® Instructor Course, a Royal College of Surgeons 'Train the Trainer' Course, or an equivalent Educator Course. All Faculty members must have fully attended a previous DSTC™ Course. A faculty of seven surgeons for a course of 16 surgeons in four groups of four is required.

C.3.9 Course participants

All course participants must be Licensed Medical Practitioners, and should be *actively involved in the definitive surgical care* of the trauma patient. Attendance at the entire course is mandatory.

C.3.10 Practical skill stations

Different material may be needed for the acquisition of practical skills, depending on local constraints. However, full local ethical committee and any other necessary legal approvals must be obtained and submitted to IATSIC before a course can be held.

C.3.11 Course syllabus

In order for IATSIC to recognize a course as a valid DSTC™ Course, it 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', which may be added as required, at the discretion of the local organizing committee and as required for local needs.

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 may be called DSTC™ Courses.

Appendix D

Definitive Surgical Trauma Care (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

D.2 THE CHEST

- 1 Incisions
 - 1.1 Antero-lateral 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 Lung wounds
 - 5.1 Oversewing
 - 5.2 Stapling
 - 5.3 Partial lung resection
 - 5.4 Tractectomy
 - 5.5 Lobectomy
- 6 Access to and repair of the thoracic oesophagus
- 7 Access to and repair of the diaphragm
- 8 Compression of the left subclavian vessels from below
- 9 Left anterior thoracotomy
 - 9.1 Visualization of supra-aortic vessels
- 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 rotation – Mattox manoeuvre
- 3 Right visceral rotation
 - 3.1 Kocher's manoeuvre
 - 3.2 Cattel and Braasch manoeuvre

- 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 infra-hepatic inferior vena cava**
 - 3.2 Control of supra-hepatic inferior 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**
 - 1.2 Cattell and Braasch manoeuvre**
 - 1.3 Division of ligament of Treitz**
- 2 Repair of the duodenum**

D.8 THE GENITO-URINARY 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 intra-peritoneal rupture**
 - 3.2 Repair of extra-peritoneal 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**
 - 1.2.1 Supra-hepatic inferior vena cava**

- 1.2.2** Infra-hepatic inferior vena cava
- 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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