Emergency Management of Severe Burns

AUSTRALIAN & NEW ZEALAND
COURSE MANUAL

ANZBA
Australian & New Zealand Burn Association
Care • Prevention • Research • Education

Presented by the Australian & New Zealand Burn Association
19th Edition – 2024 revision

ASSESS | RECOGNISE | STABILISE | TRANSFER

The EMSB Committee

of

The Australian and
New Zealand Burn Association Limited
ACN 054 089 520

Emergency Management of Severe Burns

(EMSB)

COURSE MANUAL

19th edition
2024 revision

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CONTENTS

PROLOGUE

The Australian and New Zealand Burn Association Limited

The Australian and New Zealand Burn Association (ANZBA) was formed in 1976 by a group of medical and nursing staff who were drawn together by their common interest in improving the quality of care their burn patients received.

Since then this group has expanded to now include a truly multidisciplinary group of burn care professionals who are interested in teaching, care, research, and prevention of burn related problems.

The multidisciplinary nature of the Association is an extension of everyday burn care philosophy, as practiced in Burn Services throughout Australia and New Zealand.

The Association has an important role in the promotion of the Minimum Standards of Burn Care in Australia and New Zealand and it is in this context that this manual and the related EMSB course have been developed. It is hoped this initiative will improve standards of burn care for the patient with severe burn injuries.

Contributors to the EMSB Course

The Emergency Management of Severe Burns (EMSB) course was first developed in 1996 by the following people who generously gave of their time and expertise:

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A special thanks is owed to Mani M. Mani, M.D., Kansas USA

The Course has been reviewed and updated in:

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Contribution to the chapter on Frostbite & Hypothermia by **Andrew Lindford**, Finland, is also noted.

A special acknowledgment is made of the comprehensive revision and updating of the manual undertaken in 2018 by **David Milliss**.

A special acknowledgement is also made to **Cath Frances** for her illustrations used throughout the manual and course.

The course has become 'international'. It is now taught by multiple partner societies in countries including the United Kingdom, The Netherlands, South Africa, Indonesia, Finland, Norway, Hong Kong and Germany. The course has also been taught in multiple other countries including – Bangladesh, Fiji, India, Malaysia, Nepal, Papua New Guinea, Kenya and Sri Lanka. Regardless of where it is taught, the same standard for participants in order to pass remains the same.

The EMSB Manuals and Courses have been adapted to address country specific circumstances, whilst retaining standardised examination processes and pass marks throughout the world. In addition, ANZBA has organised several courses in differing countries with a multinational faculty and ANZBA will endeavour to continue this approach. An EMSB certificate is therefore valid worldwide and is an expression that the recipient is knowledgeable in the initial treatment of burn injuries.

For suggestions and comments please contact the Chair of the EMSB Committee via the Secretariat of the Australian & New Zealand Burns Association (info@anzba.org.au)

EMSB OVERVIEW TIMETABLE – VENUE – DATE

Key Faculty : Course Director *Faculty*

Key Coordinator *Faculty*

07:00–07:30	Faculty Meeting	*All Faculty*
07:30–07:55	Registration	*Faculty / Faculty*

Part One – Lectures

08:00–08:15	Welcome & Burn Pathophysiology	*(15 min)*	*Faculty*
08:15–08:30	Burn Size & Depth	*(15 min)*	*Faculty*
08:30–08:55	Emergency Examination & Treatment	*(25 min)*	*Faculty*

08:55–09:10	**Morning Tea**	***(15 min)***

Part Two – Small Group Sessions (25 min + 2 min turnover) **Timekeeper:** *Faculty*

09:10–09:37	ROTATE ROOMS	Burn Area & Fluid Management	*(27 min)*	*Faculty / Faculty*
09:39–10:04		Referral & Transfer	*(25 min)*	*Faculty / Faculty*
10:06–10:31		Escharotomy	*(25 min)*	*Faculty / Faculty*
10:33–10:58		Burn Depth & Wound Management	*(25 min)*	*Faculty / Faculty*
11:00–11:25		Airway Management & Inhalation Injury	*(25 min)*	*Faculty / Faculty*

11:25–12:00	**Lunch**	***(30 min)***

Part Three – Small Group Sessions (25 min + 2 min turnover) **Timekeeper:** *Faculty*

12:00–12:25	ROTATE ROOMS	Paediatric	*(25 min)*	*Faculty*
12:27–12:52		Chemical	*(25 min)*	*Faculty*
12:54–13:19		Electrical	*(25 min)*	*Faculty*
13:21–13:46		E.M.S.B. Approach	*(25 min)*	*Faculty*

13:50–14:10	**Demonstration of Case Simulation**	***(20 min)***	*Faculty / Faculty (all Faculty present)*

14:10–14:20	**Break**	***(10 min)***

Part Four – Multiple Choice Exam & Simulations **Timekeeper:** *Faculty*

14:20–15:40	Group A MCQ Exam				*Faculty*
	Group B Case Simulations				
14:20–14:38	– simulation practice 1	(1st Patient as a Group)	*(18 min)*		
14:40–14:58	– simulation practice 2	(2nd Patient as a Group)	*(18 min)*	Pt A	*Faculty / Faculty*
15:00–15:12	– simulation test 1	(3rd Patient Individually)	*(12 min)*	Pt B	*Faculty / Faculty*
15:14–15:26	– simulation test 2	(3rd Patient Individually)	*(12 min)*	Pt C	*Faculty / Faculty*
15:28–15:40	– simulation test 3	(3rd Patient Individually)	*(12 min)*	Pt D	*Faculty / Faculty*

15:40–15:50	**Break & Turnover**	***(10 min)***

15:50–17:10	Group B MCQ Exam		
	Group A Case Simulations		
15:50–16:08	– simulation practice 1	(1st Patient as a Group)	*(18 min)*
16:10–16:28	– simulation practice 2	(2nd Patient as a Group)	*(18 min)*
16:30–16:42	– simulation test 1	(3rd Patient Individually)	*(12 min)*
16:44–16:56	– simulation test 2	(3rd Patient Individually)	*(12 min)*
16:58–17:10	– simulation test 3	(3rd Patient Individually)	*(12 min)*

~17:12–17:24	***Re-sits of Simulations if required (with 4th Patient individually as required)***

~17:26-17:40	**Summation and Presentations**

Please note that the number of candidates electing to re-sit failed simulations at the end of the day will affect the actual finishing time of the course. ***All participants are expected to stay until the end of the course and should arrange travel plans accordingly.***

CHAPTER 1

Introduction – Epidemiology & Aetiology

Introduction

The patient with a severe burn injury presents a difficult challenge. In addition to the pain and distress experienced by the patient, the implications of this life-threatening injury extend beyond mere survival. Potentially permanent changes to appearance, function and independence impact not only on employment and income, but also on the general wellbeing and long-term quality of life for both the patient and their families.

It is widely accepted that the trauma patient who is seen, assessed and treated early by skilled personnel will heal more quickly than the patient whose treatment is delayed. This is also true for the burn patient (1,2). It is important the right treatment is instigated quickly not only to save a person's life but to maximise their functional outcome.

The Emergency Management of Severe Burns (EMSB) course is based on the principle that timely emergency assessment, recognition, management, stabilisation and transfer provide the best chance of recovery.

The aim of this course is to provide candidates with sufficient information regarding the presentation, diagnosis and initial management of the patient with severe burn injuries, to enable them to deal competently with this urgent and often life-threatening problem.

This course was written by members of the ANZBA EMSB Committee and EMSB Faculty, combining current research findings and recommendations as well as their considerable personal experience across multiple disciplines involved in all aspects of burn care. While regional variations may exist in practice there is a consensus on the basic principles of management.

The EMSB course follows the trauma management approach as taught by the Royal Australasian College of Surgeons (RACS) in their Early Management of Severe Trauma course (EMST). The EMST course is the Australian version of the Advanced Trauma Life Support Course (ATLS) that is taught worldwide. The EMST course is the accepted trauma management teaching system for medical practitioners in Australia and New Zealand.

The EMSB course provides trauma management guidelines and protocols specific to burns that are in addition to content to the EMST course. One significant difference is the approach to fluid resuscitation. In the late 1920's Underhill suggested burn patient mortality was due to loss of fluid similar to plasma (3). This was the beginning of the development of current fluid and electrolyte replacement regimens unique to the resuscitation of patients with severe burn injuries.

The EMSB course is designed to be a 'Stand Alone Course' and provides sufficient information to define the Minimum Standards of Emergency Burn Care (as determined by the Australian and New Zealand Burn Association).

The EMSB course covers the principles of the emergency management of severe burns during the first 24 hours from time of injury. The course is appropriate for all health care professionals who may find themselves involved with the initial assessment, management and stabilisation of a patient with severe burn injuries – from those working in isolated areas to members of the burn team working in a receiving Burn Service.

The EMSB course also seeks to emphasise the benefit to patients whereby all emergency burn care providers have knowledge of the same guidelines for emergency burn care. This facilitates initial care, appropriate referral, communication and transfer of care. The EMSB course is delivered in five separate but complimentary sections.

1. Course Manual

This manual contains the complete syllabus of the course. **Candidates are expected to read the manual and be thoroughly familiar with its contents before attending the course**. The "Structure of the EMSB" image (page 23) is a vital summary tool outlining the most important aspects of the course.

2. Formal Lectures

These occur at the beginning of the course, giving a brief outline of the course and highlights some important concepts. They are **NOT** a substitute for reading the manual.

3. Small Group Sessions

These teach important practical aspects and concepts in the assessment and management of a patient with severe burn injuries. They maximise opportunities for candidates to discuss these topics, and to use their own clinical experience, at their own level, to explore these topics with experienced faculty.

4. Simulated Burn Cases

Volunteers are 'made-up' (moulaged) to simulate burn injuries. This gives candidates an opportunity to apply their knowledge and skills in a clinically relevant manner.

5. Examination

At the end of the course, there is a multiple-choice exam and a simulation test using a moulaged burn case.

Successful candidates will receive an official certificate from the Australian and New Zealand Burn Association. If unsuccessful, remediation in the form of re-testing or even repeating the entire course at another time may be necessary to achieve the required standard.

Please note that the finishing time of the course is variable as it is dependent on the number of candidates who need to repeat a simulated burn case.

The Team Concept of Burn Care

Since the Second World War, advances in burn care have resulted in a marked decrease in the mortality and morbidity from severe burns (4). Early fluid resuscitation, improved nutrition, the introduction of topical antimicrobials (particularly those containing silver) along with other infection control measures and the introduction of protocols for surgery that promote early excision and closure of the burn wound have all contributed to the remarkable improvements in survival (4).

Burn Team

It is now well established that the team approach to complicated medical issues leads to improved patient care (5-7). Truman Blocker was possibly the first clinician to demonstrate the value of the multidisciplinary team approach to burn care (4). Blocker was involved in the response to a fertiliser explosion in Texas in 1947 that killed 560 people and injured more than 3,000. As the advances in burn care developed, the concept of a Burn Team approach that brings significant patient benefits also developed. The Burn Team approach is now considered to be part of standard burn care (6). The Burn Team consists of a multidisciplinary group whose individual skills are complementary to each other. Team members recognise the benefits of interdisciplinary collaboration and the use of shared guidelines in providing the best quality care to the patient with burns.

Burn Service

The specially trained staff of a Burn Team are able to operate more effectively within a purpose built acute care facility of a Burn Service (burn unit or burn centre). The concentration of specialist team members within one facility has the added advantages of cost efficiency, knowledge-sharing and the development of high levels of expertise by individual team members. The Burn Service approach ensures patients receive the best care possible.

Pre-hospital Clinicians

Ambulance and other first responders provide essential pre-hospital care for burn patients by initiating first aid, stabilising the patient and providing rapid transfer of the burn patient to an appropriate facility. This early management assists the burn patient's chance of survival and optimal outcome.

Emergency Department

Most burn patients will be assessed and receive their initial treatment in an Emergency Department. A highly effective working relationship between the Burn Service and Emergency Department is essential to ensure high quality care.

Intensive Care

Many patients with severe burn injuries are admitted at some stage of their hospitalisation to an Intensive Care Unit (ICU). A close working relationship between the Burn Service and Intensive Care Unit is also essential to provide optimal care and outcome for the patient.

Surgeons

Burn Surgery is a sub-specialty of Plastic Surgery, General Surgery and Paediatric Surgery. Burn Surgeons have a particular interest in the management of these seriously injured patients, in wound healing, rehabilitation, and related research.

Anaesthetists

Burn Anaesthetists provide specialised anaesthetic skills and techniques in treating the patient with severe burn injuries, managing severe blood loss and maximising the area of burn wound surgery that can be treated at any one time as well as providing pain management and sedation for burn wound care.

Nurses

The Burn Nurse is a pivotal member of the team, providing day-to-day continuity of care. Burn nurses have specialist expertise in wound care, skin graft and wound coverage care and discharge planning of the patient with severe burn injuries. They liaise closely with all members of the burn team throughout the patient journey, focusing not only on the physical but also the psychosocial recovery and rehabilitation of patients.

Physiotherapist & Occupational Therapist

Therapists play an indispensable role in the care and rehabilitation of the burn patient. Rehabilitation begins from the time of admission to the Burn Service and continues well after discharge in the outpatient setting. Burn therapy is a specialised sub-discipline and is critical for optimal functional outcome.

Speech Pathologist / Speech Language Therapist

The Speech Pathologist provides comprehensive clinical assessment and management of severe burn patients. Problems they assess and manage may include swallowing, voice and communication disorders as a result of the burn injury or secondary complications.

Dieticians

Optimal nutrition is necessary to counteract the extreme catabolic response that occurs with a severe burn injury. Specialised Dieticians attached to Burn Services provide appropriate calculated feeding regimens utilising various routes of administration.

Psychosocial Clinicians

Social workers, psychiatrists, psychologists, hospital play specialists/child life therapists and other support workers are members of the Burn Team, providing necessary support and treatment for the wide variety of psychosocial problems that burn patients commonly encounter. The patient's ability to function in society in the long term is as dependent on this psychosocial adjustment as it is on the physical outcome.

Rehabilitation

Post-burn rehabilitation begins at the time of admission and in the minor burn can often be managed as part of outpatient care. Patients with severe burn injuries require intensive rehabilitation to enable the attainment of maximum function, employment and independence, this is best facilitated by experienced rehabilitation personnel.

The Epidemiology of SEVERE Burns

Burns can cause devastating injuries and are a worldwide concern, especially in developing countries. Burn injuries are common but related death is uncommon (9). Globally, burns are the fourth most common type of trauma after traffic accidents, falls and attacks by another person (10). The incidence increases in areas of armed conflict. In Australia, burns are the fifth most common type of trauma with the highest incidence in the 15–24 year age group (11). Most burn injuries are unintentional (93%), either accidental or through carelessness and inattention (12). The Burn Registry of Australia and New Zealand (BRANZ) reports that, for adult patients, drug and/or alcohol is involved in 15% of cases (12).

Approximately 1% of the population of Australia and New Zealand each year[1] suffer a burn requiring medical treatment. Of these, 3–4% are hospitalised. Of those hospitalised, 16% are categorised as having a high threat to life and 5% require time in the Intensive Care Unit (ICU) (13,14).

Statistics in this manual define severe burns as >20% TBSA for adults or >10% for children or any burn requiring admission to an adult or paediatric ICU.

Severe burns are expensive to treat. It has been reported that treatment of an adult with a 70% total body surface area (TBSA) burn may cost approximately $700 000 for acute hospital treatment alone (15). To this figure must also be added the potentially lifelong cost of rehabilitation, time off work, and loss of earning which represent a substantial cost to both the patient and their community (15).

A 'child' is defined as a person less than 16 years of age. This is consistent with the definition adopted by the majority of Australian and New Zealand Burn Services and the Burn Registry of Australia and New Zealand (BRANZ). The data presented in the following discussion and tables are sourced from the BRANZ database of admissions of severe burns **admitted** between January 2015 and December 2019 (16) to one of the 17 Burn Services throughout Australia and New Zealand.

In both adults and children, the commonest location where burn injuries are sustained is in the home. In children, 80% of accidents occur in the home, with the most dangerous rooms being the kitchen and the bathroom. Most scalds in children and the elderly occur in these two rooms. Other high-risk places are those containing dangerous (and often easily accessible) chemicals or flammable liquids such as the laundry, garage and garden shed.

[1] Population of Australia and New Zealand as of November 2020 was 30,322,117

Home	79%
Other residential	7%
Place of recreation	4%
Trade & service area	0%
Roadway	3%
Other	5%

TABLE 8.1 –
Children's Place of Severe Burn Injury (%)

Home	56%
Other residential	6%
Place of recreation	7%
Trade & service area	6%
Roadway	9%
Other	16%

TABLE 1.1 –
Adult's Place of Severe Burn Injury (%)

Scald	61%
Flame / Explosion	23%
Contact	8%
Chemical	1%
Electrical	4%
Friction	4%
Other	1%

TABLE 8.2 –
Causes of Children's Severe Burn Injury (%)

Flame / Explosion	73%
Scald	11%
Contact	7%
Chemical	3%
Electrical	3%
Friction	0%
Other	0%

TABLE 1.2 –
Causes of Adult's Severe Burn Injury (%)

All burns occurring from suspected non-accidental injury necessitate referral to a Burn Service and warrant further investigation.

Military burns

Combat and non-combat settings result in quite different patterns of burn injury. The incidence and injury pattern will differ between the acute phases of deployment compared with more stable or on-going deployment. Burn injuries outside of the conflict zone occur in the same manner as in civilian life.

Historically, burn injuries constitute 5–20% of all injuries in the conflict setting (17–19). Figures for burn injuries sustained in combat compared with those in the non-combat situation vary depending upon the nature of the conflict. In the Vietnam War, when looking at burn injuries, combat associated injuries accounted for 46% and non-combat associated injuries for 54% (20). More recently, in Middle East operations combat related burn injuries accounted for 62–74% of all burns with the remainder being non-combat related (17–19). Overall mortality from burn injuries in the military remains around 4% (17,19).

Combat related burn injuries are commonly associated with other injuries (inhalation injury, shrapnel injury, multiple trauma) (19-21). The majority of non-combat related burn injuries are a result of waste burning, fuel mishaps and munitions mishaps (18,19). Waste burning and fuel mishaps (flame/fire) account for 56% of non-combat related thermal injuries.

Summary

- *A multidisciplinary Burn Team is essential for appropriate and long-term management of severe burn injuries.*
- *Burn injuries requiring medical attention affect approximately 1% of the population per year.*
- *Burn injuries are frequently caused by carelessness and inattention. Burns occurring from suspected non-accidental injury require both recognition and necessitate further investigation.*
- *The majority of burn injuries in all age groups occur in the home.*

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Chapter 2

Local and General Response to the Acute Burn Injury

Introduction

Burn injuries can affect any part of the body but primarily involve injury to the skin. The skin is the largest organ of the body and has several major functions:

- control of fluid loss
- regulation of body temperature
- physical protection against mechanical, chemical, osmotic, thermal and UV radiation sources
- physical barrier against microbial invasion
- involvement in immune activity
- metabolic – synthesis of vitamin D, cytokines and growth factors
- sensory interface / organ
- psycho-social interface.

Local Response

Extended exposure to temperatures greater than 40°C leads to tissue damage such as protein denaturation and loss of cell plasma membrane integrity. Depending on the age of the patient and the skin thickness, cell necrosis can occur after one hour of exposure to 45°C and can occur within one second at 60–70°C (1–3). Similar tissue damage can be seen with electrical, friction, chemical and ultraviolet insults.

Jackson Burn Wound Model

In 1953, Jackson, a surgeon from Birmingham, England, proposed a burn wound model that is still used today (1–6). Jackson separated the local tissue changes in a burn wound into three zones (Figure 2.1).

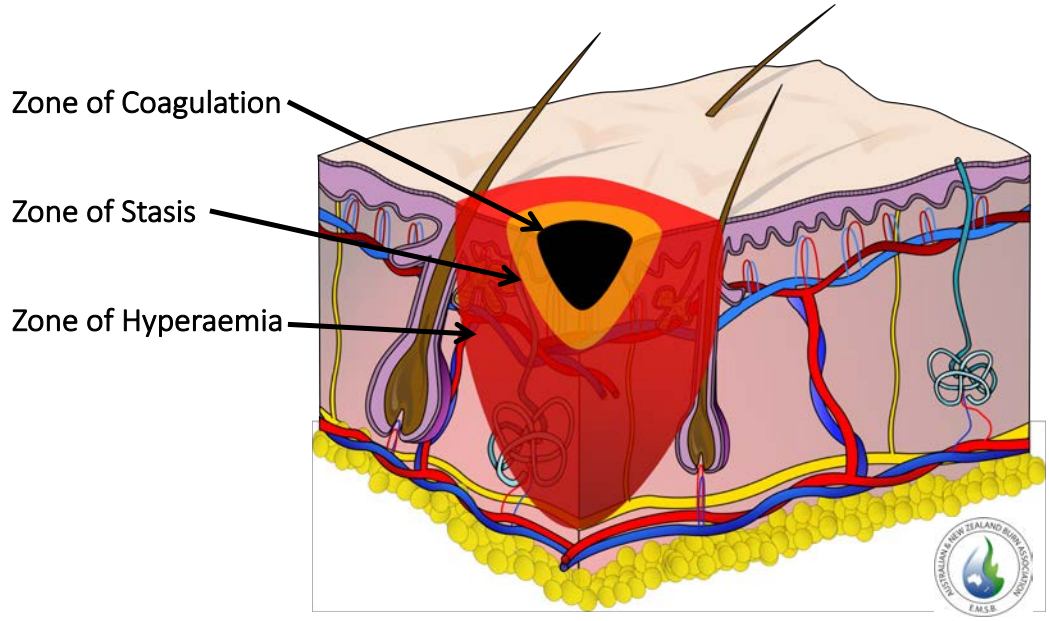

FIGURE 2.1 – Jackson's Burn Wound Model

1. The central **Zone of Coagulation –** this area sustains the greatest direct damage from the burn insult; it is characterised by irreversible necrosis with complete destruction of tissue.
2. The middle **Zone of Stasis** – the tissue here is damaged and there is impaired vasculature and perfusion; the tissue viability is under threat but is potentially recoverable.
3. The outer **Zone of Hyperaemia** – this zone appears superficially as red and it is hyperaemic; this tissue is not under threat and recovers.

In the necrotic and damaged tissues of the Zone of Coagulation and the Zone of Stasis there is rapid activation of inflammatory processes (1,5,6,8–10) along with a rapid increase in the quantity of inflammatory mediators.

These inflammatory mediators:

- come from mast cells in the damaged tissue;
- are released by macrophages and neutrophils that infiltrate the damaged tissue;
- are produced locally by platelets and;
- are produced locally by the endothelial cells lining the blood vessels.

Local inflammatory mediators released include histamine, serotonin, bradykinin, nitric oxide, oxygen-free radicals, prostaglandins, thromboxane, tumor necrosis factor (TNF) and interleukins. Histamine and bradykinin are thought to increase vascular permeability while other mediators can cause further tissue damage and necrosis (so-called pro-apoptotic factors). The combination of these effects leads to local oedema (increased vascular permeability and reduced interstitial hydrostatic pressure), tissue ischaemia (vascular damage and occlusion) and continued cellular damage and necrosis in the Zone of Stasis.

Initially, Jackson thought that the Zone of Stasis always progressed, through worsening perfusion, to complete cessation of blood flow leading to necrosis and extension of the Zone of Coagulation (4,5). Clinically this is seen as progression of the depth and extent of the burn injury. That is, on presentation, areas of the burn wound appear viable but, subsequently, over 3–5 days, the tissue becomes necrotic. However, by 1969, Jackson realised that the Zone of Stasis was not always avascular and did not inevitably progress to necrosis. With intervention the tissue could be protected from further injury and remain viable (5,7).

In the acute stage of the management of the patient with severe burn injuries, ways to protect the Zone of Stasis include:

- first aid (prompt and appropriate)
- rapid assessment and management using the principles of primary survey ('ABCDE' to ensuring adequate oxygenation and perfusion)
- timely and adequate fluid resuscitation
- avoiding vasoconstriction (e.g. first aid with ice cold water, hypothermia) and excess tissue oedema.

General Response

As the size and depth of a burn increases so does the inflammatory response. As the burn injury approaches 20% TBSA in adults and 10% TBSA in children the amount of inflammatory mediators released from the site of the burn injury causes clinically significant systemic effects (1,7,9,10). The inflammatory response is no longer confined to the local injury but becomes systemic with impacts in organs and tissues distant from the burn wound.

Systemic effects of the acute severe burn injury include:

Vascular

In unburned soft tissue there is widespread increased vascular permeability. This leads to generalised loss of fluid and plasma proteins from the intravascular compartment resulting in oedema formation. There is significant oedema formation at the burn site. Hypovolaemia is the most important immediate result from this combination. Correction of hypovolaemia can be lifesaving in the first hours after a major burn injury. Fluid resuscitation aims to restore and preserve tissue perfusion at the same time acknowledging that oedema formation may worsen. The increased vascular permeability begins to resolve after 12–24 hours.

Normal Capillary Exchange

Substances pass through the capillary wall in one of three ways:

1. *Diffusion* is the mechanism of transfer of very small particles such as oxygen, carbon dioxide or sodium. It implies these particles cross the capillary wall (membrane) easily and the direction is determined by the concentration gradient, moving 'downhill' from more concentrated to less.
2. *Filtration* is the mechanism of transfer of water and certain other substances. The amount of water filtered through the capillary depends on the balance of forces pushing water in and out across the capillary wall, as well as factors in the capillary wall. The

forces causing movement across the capillary wall are summarised by Starling's Hypothesis (5,10). Starling's Hypothesis states net fluid movement is the difference between the forces moving fluid out (hydrostatic pressure in the capillary pushing fluid out plus the colloid osmotic pressure in the interstitial fluid pulling fluid out) and the forces moving fluid in (hydrostatic pressure in the interstitial space pushing fluid back in and plasma colloid osmotic pressure pulling fluid in).

3. *Large molecule transport* is less well understood. Large molecules probably cross the capillary wall mostly by passing through spaces between the endothelial cells. Most capillaries are 'semi-permeable': easily permeable to water and small particles, such as sodium and chloride, but relatively impermeable to large molecules such as albumin.

Normal variations in filtration occur because of local factors in the capillary wall (e.g. kidney capillaries let out much more water than muscle capillaries) as well as the factors mentioned in Starling's Hypothesis. The capillary hydrostatic pressure depends on the pressure of the blood flowing in and the resistance to blood flowing out (controlled by pre- and post-capillary sphincters respectively). Normally most capillaries undergo cycles of active blood flow, interspersed by long periods of low flow and hence low pressure. The colloid osmotic pressure of the plasma is almost totally dependent on the serum albumin concentration. The colloid osmotic pressure of the interstitial fluid is due to the small amount of albumin and the ground substance between cells.

Abnormal Capillary Exchange in the acute burn setting

Vasodilatation is one of the major vascular responses to inflammation and causes:

- increase in capillary hydrostatic pressure
- recruitment of all capillaries (as opposed to only a few under normal circumstances)
- distension of the capillary wall that increases the surface area of the capillary membrane and opens the spaces between endothelial cells
- pooling of blood in venules.

There is a marked increase in the permeability of the capillary membrane. This causes increased transport of substances by all three mechanisms, diffusion, filtration and large molecule transport. However, large molecule transport is most affected, and there is a dramatic increase in the movement of albumin across the capillary membrane. This causes mass movement of albumin out of the circulation and into the interstitial space leading to an increase in interstitial colloid osmotic pressure.

Tissue damage by burning may produce breakdown of the intercellular matrix. This can also contribute to a rapid increase in the colloid osmotic pressure of the interstitial space. Another effect of burn injury on the intercellular matrix is uncoiling of long molecules, which is thought to cause expansion of the space and thereby lower its hydrostatic pressure.

Heart

Cardiac output is reduced after burn injury as a result of myocardial depressant mediators, decreased blood volume, reduced venous return and increased pulmonary and systemic vascular resistance due to increased levels of catecholamines. In the first 24 hours reduced cardiac output persists even after restoration of blood volume. Between 24–48 hours post-burn, a hyperdynamic state develops with reduced peripheral resistance, increased oxygen consumption and an increased cardiac output.

Lungs

In large burns there is an increase in pulmonary vascular resistance. However, pulmonary oedema is not common but may occur late in the resuscitation phase or after. Reduced cardiac output and hypoproteinemia may be contributing factors for post-burn pulmonary oedema. While acute lung injury can occur in patients with severe burn injuries it is usually associated with an inhalation injury, excess fluid resuscitation or sepsis.

Kidneys

Acute kidney injury (AKI) may occur early, especially if there has been inadequate fluid resuscitation or secondary to compartment syndromes or later in association with sepsis.

Gastrointestinal tract (GIT)

Effects seen in the GIT include oedema, ischemia, mucosal atrophy and bacterial translocation. These effects can develop within hours of the injury.

Metabolic Response

After approximately 72 hours post-burn a hypermetabolic and catabolic response develops that may persist for months.

Other intermediate to long term effects

- Immunosuppression.
- Growth inhibition in children.
- Muscle weakness and wasting.

Summary

- *The local effect of burn injury on the skin and subcutaneous tissues causes three zones of injury. Lack of timely and effective management results in progression of the intermediate zone of stasis to the irretrievable zone of coagulation.*
- *Normal capillary exchange is disturbed leading to loss of albumin from the circulation and oedema formation.*
- *A severe burn injury becomes a systemic threat to life due to the large amounts of inflammatory mediators released. Generalised effects on the circulation, metabolism, temperature control, immune competence, gut integrity and acute lung injury. Growth inhibition, particularly in children, is a common long-term complication.*

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CHAPTER 3

Emergency Examination and Treatment of Severe Burns

Introduction (1–3)

Rapid assessment and treatment can be life-saving for a patient with a burn injury.

A patient with a burn injury falls into one of three categories:

1. an isolated burn injury;
2. a burn with an obvious other injury and;
3. a burn with a concealed other injury.

Life-threatening, non-burn injuries may be missed because the obvious and serious burn injury can be very confronting and distracting. The history of the mechanism of injury and the circumstances around the event should alert burn management personnel to the possibility of co-existing other injuries.

Suspicion for other injuries should be considered in:

- road traffic accidents, particularly with ejection or at high speed
- blasts or explosions
- electrical injuries, especially high voltage
- jumping or falling from significant heights.

Non-communicative patients, whether unconscious, intubated, psychotic, or under the influence of drugs and alcohol, should be regarded as potentially having other injuries and treated accordingly.

Staff should don personal protective equipment (PPE) such as eye protection, mask, gown and gloves prior to attending any patient.

Pre-hospital Care

Emergency treatment at the scene

- Ensure personal safety, the safety of others and of the patient.
- The focus is on airway, control of external bleeding and shock, immobilisation and immediate transfer.
- Obtain relevant information regarding the patient and the event and circumstances surrounding the injury.

First aid at the scene

- First aid consists of:
    - stopping the burning process, followed by;
    - cooling the burn wound (effective in first 3 hours – see Chapter 7)
- Immediate burn care at the scene is important, can greatly alter outcomes and can significantly limit burn progression in terms of depth and size.

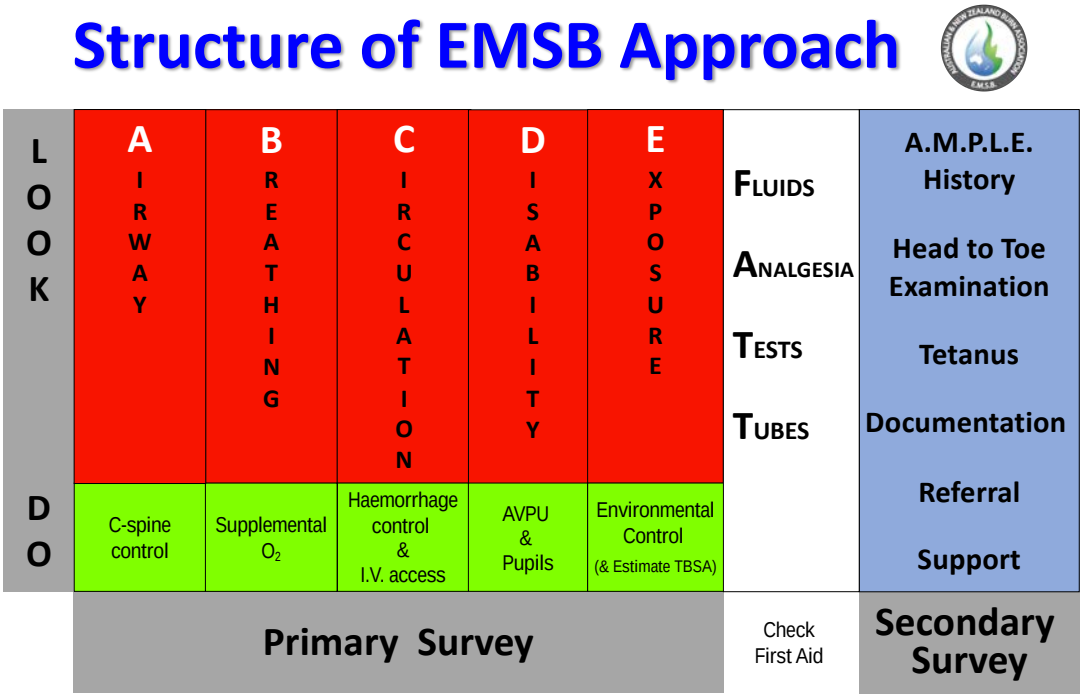

FIGURE 3.1 – Structure of EMSB

Primary Survey

Depending upon location, trauma systems may differ slightly in their approach to the Primary Survey. The goal of the Primary Survey is to recognise and manage life-threatening injuries. This course recommends an approach that targets injuries commonly associated with severe burn injury. Do **not** get distracted by the obvious burn injury.

The primary survey consists of:

A. Airway maintenance with cervical spine control

B. Breathing and ventilation with supplemental oxygen

C. Circulation with haemorrhage control and establish IV access

D. Disability - neurological status (AVPU) and check pupils

E. Exposure with environmental control and estimation of %TBSA burn

A. Airway Maintenance with Cervical Spine Control

- Check for a patent airway:
  - speak to the patient and listen for a response
  - if the airway is not patent, clear the airway of foreign material and open the airway with simple manoeuvres such as chin lift/jaw thrust; keep movement of the cervical spine to a minimum and never hyperflex or hyperextend the head and neck
  - consider adjuncts such as an oro-pharyngeal or naso-pharyngeal airway. If these adjuncts fail consider early intubation with an endotracheal tube. Loss of airway is often progressive in a burn patient so securing a patent airway will become more challenging with time.

- Maintain cervical spine control in all patients until cervical spine injury is excluded.

B. Breathing and Ventilation with Supplemental Oxygen

- Always provide supplemental **oxygen –** 100% high flow (15 L/min) via a non-rebreather mask; attach pulse oximeter.
- Expose the chest.
- Inspect, palpate the chest and auscultate ('look, feel, listen').
- Assess respiratory rate, depth, symmetry and work of breathing.
- Exclude pneumothorax, haemothorax and significant rib injury.
- If required ventilate via a bag and mask or intubate the patient if necessary.
- Have heightened concern in the following situations:
  - a respiratory rate (RR) <10 or >30 per minute in adults or an elevated RR in children that is age related

- circumferential chest burns in adults or chest and abdominal burns in children; an escharotomy may be required to improve ventilation.

C. Circulation with Haemorrhage Control

- Specifically look for evidence of haemorrhage:
  - external… manage with direct pressure or tourniquet
  - chest… consider chest drain
  - abdomen… consider need for FAST (Focused Assessment with Sonography for Trauma) scan and/or urgent laparotomy
  - pelvis… pelvic binder may be life saving
  - long bones… reduce and splint
- Check both centrally and peripherally:
  - pulses
  - capillary refill… normal return is ≤2 seconds
    >2 seconds suggests significant hypovolaemia (or the need for escharotomy (or fasciotomy) on a limb)
  - skin temperature
  - consider whether abnormal peripheral pulse/capillary refill/temperature is a result of a systemic (and therefore life-threatening) insult or from a local injury
- Check blood pressure and heart rate
- Insert 2 large bore IV lines, preferably through unburned tissue.
- Take blood for FBC, blood biochemistry including glucose and liver function tests, blood gas with lactate, crossmatch, coagulation studies, carboxyhaemoglobin level (if carbon monoxide poisoning suspected), alcohol and drug screen, β-hCG.

Clinical shock requires immediate fluid resuscitation. If it is a result of uncontrolled haemorrhage blood products should be commenced.

The **EARLY** appearance of clinical signs of shock in the burn patient is **not** likely to be explained by an extensive burn. Other haemorrhagic and non-haemorrhagic causes of shock must be excluded.

***Note*:** Whilst checking the peripheral pulses is technically not a 'life saving' assessment it can be done extremely quickly and adds important information about the status of the patient, their limbs and their burn.

D. Disability: Neurological Status

- Establish level of consciousness using the AVPU scale:

  A - **A**lert

  V - Response to **V**ocal stimuli

  P - Responds to **P**ainful stimuli

  U - **U**nresponsive

- **Pupils** - are there localising signs? A markedly dilated pupil may indicate intracranial pathology or ophthalmic trauma.
  - response to light should be brisk and equal

Note: There are several causes of restlessness and decreased level of consciousness such as hypoxaemia, carbon monoxide/cyanide poisoning, trauma and drugs and alcohol. The Glasgow Coma Scale (GCS) is used in the Secondary Survey.

E. Exposure with Environmental Control

- Warm the room if possible.
- **Remove** all clothing (including nappies), watch, jewellery and piercings.
- **Log roll** the patient to visualise posterior surfaces.
- **Keep the patient warm** and cover the burn wound and the patient.
- **Estimate the burned area** using the 'Rule of Nines' and/or Palmar Surface method (see Chapter 5).

Fluids, Analgesia, Tests and Tubes (FATT)

(The 'FATT' between the primary and secondary survey)

Fluid Resuscitation (See Chapter 6)

- Fluids are given initially as per the modified Parkland formula:
  - Adult (>20% TBSA): 3 mL x weight (kg) x % TBSA burn
  - Child (>10% TBSA): 3 mL x weight (kg) x % TBSA burn **+ maintenance**
- Balanced crystalloids (e.g. Hartmann's™ solution or Plasma-Lyte™ 148) are the recommended fluid (consider blood products early if haemorrhagic shock evident).
- Intravenous fluids should be warmed whenever possible.
- Half of the calculated fluid volume is delivered over the first eight hours **from the time of the burn** and the remainder of the calculated volume over the subsequent 16 hours.
- The adequacy of resuscitation needs careful monitoring with an indwelling urinary catheter:

- **hourly urine output is the recommended clinical measure of adequate fluid resuscitation and organ perfusion** (see Chapter 6);
- if urethral trauma is suspected, another means of urinary output measurement is required;
- other parameters may be of assistance: pulse rate and fullness, blood pressure, respiratory rate, pulse oximetry and arterial blood gas analysis as appropriate.

- Adjust resuscitation fluid infusion rate to maintain desired hourly urinary output (see Chapter 6).

Analgesia

- Burns are very painful. Give intravenous analgesia (e.g. morphine 0.05–0.1 mg/kg). Intravenous administration is preferred for rapid onset and reliable absorption. Avoid intramuscular administration due to poor absorption.
- The choice of analgesic will depend on availability, preference and experience of the clinician.
- Titrate to effect (avoid over-sedation in conscious patients; small frequent doses are safer than single large doses).

Tests

- Radiology (XR or CT)
  - cervical spine
  - chest
  - abdomen / pelvis
  - head
- Sonography
  - FAST scan to assess abdomen and cardiac windows
- Ensure blood tests have been taken.

Tubes

- An indwelling urinary catheter is imperative to allow monitoring of urine output to allow accurate titration of fluid resuscitation.
- **Nasogastric tube**
  - insert a nasogastric tube for major burns (>20% in adults, >10% in children), if there are associated injuries, or to decompress stomach for air transfer. Gastroparesis is common.
- Consider endotracheal intubation where indicated (see Chapter 4).

Check Adequacy of First Aid

- If first aid (20 minutes of cool running water) has not yet been administered (or inadequately administered), it can be completed at this stage if it is still within three hours from the time of burn.
- Care must be taken to ensure the patient does not become hypothermic.

Secondary Survey

The Secondary Survey commences after life-threatening conditions have been assessed and treated.

It consists of two components:

1. An 'AMPLE' history
2. A head-to-toe examination (to identify all other injuries)

1. History

A – **A**llergies

M – **M**edications

P – **P**ast illnesses

L – **L**ast meal

E – **E**vents & circumstances related to injury

Events and circumstances related to the injury

- **Burn**
  - what was the duration of exposure?
  - was the patient in an enclosed space?
  - what type of clothing and did it ignite?
  - for a scald, what was the temperature and composition of the fluid?
  - for a chemical burn, what was the composition and concentration?
  - for an electrical burn, what was the voltage and current (AC or DC)?

2. Head-to-toe examination

- **Head**
  - Eyes… penetrating injuries are often missed
    check for corneal injuries
    check visual acuity, check for foreign bodies
  - Scalp… lacerations, boggy masses

- **Face**
  - Facial skeleton… orbital rim and zygomatic integrity
    nasal bone fracture; exclude septal haematomas
    stability of the mid-face
    mandibular integrity
  - Check for missing teeth / malocclusion
  - CSF leak from the nose, ears or mouth
  - Soot, blisters, oedema of the tongue or pharynx

- **Neck**
  - Inspect and palpate. Consider imaging. Always suspect cervical fracture
  - Lacerations deep to platysma require exploration in operating theatre and/or angiography
  - Check the pulses (palpate and auscultate)

- **Chest/Back**
  - Cough, productive of soot
  - Altered voice or brassy cough
  - Examine whole chest – anterior, flanks, axillae and back
  - Assess the bony cage of the thorax; ribs, clavicles and sternum
  - Check breath sounds and heart sounds
  - Circumferential burns may need escharotomy if restricting ventilation

- **Abdomen**
  - Requires frequent re-evaluation especially for increasing tenderness and distension. Evidence of intra-abdominal free fluid (often detected by FAST scanning) in the context of an unstable patient mandates urgent surgical review.
  - If there is a seat belt bruise over the abdomen, consider intra-abdominal pathology such as ruptured viscus.
  - If assessment of the abdomen is unreliable, equivocal or impractical (e.g., presence of an extensive abdominal burn, head injury or intoxication) further investigation with a CT scan is strongly indicated in patients with multiple injuries.
  - Pregnancy should always be considered in women of childbearing age; ask directly and/or test urine or blood (β-hCG).

- **Perineum**
  - Bruising, meatal blood; if present do not proceed with urinary catheter insertion until reviewed by a trauma surgeon, or urethrogram has been performed

- **Rectum**
  - Blood, lacerations, sphincter tone, high-riding prostate

- **Vagina**
  - Foreign bodies, lacerations

- **Limbs**
  - Contusion, deformity, tenderness, crepitus
  - Identification and splinting of fractures. Femoral fractures may be associated with significant haemorrhage.
  - Assess all extremity pulses regularly.
  - Inelastic circumferential (or near circumferential) extremity burns in conjunction with developing oedema will result in progressive limb ischaemia. Initially venous return is obstructed; with increasing pressure arterial perfusion compromised resulting in tissue ischaemia. This may produce the classic signs of decreasing limb

perfusion; **pain** (often disproportionately severe considering the associated burn)**, paraesthesia** (or numbness)**, pulselessness** and **paralysis**. In these circumstances an **escharotomy** is indicated to preserve or restore adequate circulation. (see Chapter 7).

- **Pelvis**
  - Rapid access to radiology in most emergency departments/trauma units precludes the need to test pelvic stability by springing the pelvis.
  - Identification of a fractured pelvis and the application of a pelvic binder should be completed (if not already placed by pre-hospital personnel).

- **Neurological**
  - Glasgow Coma Scale to be done if AVPU any lower than "A" (see Appendix 1).
  - Motor and sensory assessment of all limbs.
  - Paralysis or paresis indicates a major injury and immobilisation with spinal boards and semi-rigid collars is indicated.
  - Decreased level of consciousness may be due to:
    - hypoxaemia/hypercapnia; which may be due to airway obstruction or a breathing issue
    - cyanide or carbon monoxide poisoning
    - hypovolaemia from undiagnosed bleeding or under-resuscitated burn shock
    - head trauma
    - hypoglycaemia
    - intoxication from alcohol or other drugs.

Documentation

- Take notes and document all observations, procedures and interventions
- Seek consent for photography and procedures

Tetanus prophylaxis

- Give tetanus prophylaxis if required:
  - Australia: https://immunisationhandbook.health.gov.au
  - New Zealand: https://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/new-zealand-immunisation-schedule

Re-evaluate

- Re-evaluate the **Primary Survey**, particularly:
  - airway patency
  - respiratory compromise
  - circulation status
  - neurological deterioration
  - adequate fluid resuscitation.
- Laboratory tests:
  - blood biochemistry, including glucose, liver function tests
  - blood gas with lactate
  - carboxyhaemoglobin level
  - full blood count
  - crossmatch, coagulation studies
  - alcohol level, other drug screening tests
  - β-hCG
  - urinalysis
- Review imaging
  - need for additional imaging as directed by findings (e.g. imaging of limbs, angiography)
- Electrocardiogram

Support and Reassure Patient, their Relatives and Staff

Burn injuries can be associated with significant emotional overlay in the patient, their relatives and friends which need to be addressed. Physical, psychological and social effects on the patients, families and staff are substantial. Early and intensive intervention from psychiatry and adjunct psychosocial professions optimises long-term outcomes.

Challenging patients include intoxicated, highly distressed or mentally unwell patients. Involvement from other hospital services for management of these patients is recommended.

When a reasonable explanation of the mechanism of the burn is lacking, the possibility of non-accidental injury must be considered in **all** age groups, but particularly in children. Accurate documentation is important and reporting to the appropriate authority will enable proper investigation.

The decision to palliate patients in the first 24 hours should be made on the basis of minimal chance of recovery and following consultation with senior clinicians, patient and family (where possible) using multiple factors including quality of life, potential complications and patient's wishes.

Definitive Care

Definitive burn care is described elsewhere in this manual. Transfer to a Burn Service where other specialised services are available is indicated in accordance with ANZBA referral criteria (See Chapter 12).

Summary

- *A burn injury may be associated with obvious or concealed other injury and is a potential distractor for other more immediately life-threatening problems.*
- *A systematic approach to trauma will prioritise the identification and treatment of life-threatening injuries (the primary survey). This is followed by a systematic evaluation of the burn injury (extent %TBSA, depth and distribution).*
- *Following the primary survey, treatment of the burn injury begins in the 'FATT' with fluid resuscitation, monitoring and analgesia.*
- *A secondary survey to identify all other injuries (an 'AMPLE' history and head-to-toe examination) begins once all life-threatening conditions are addressed.*
- *Definitive care in the severely burned or complicated patient, as defined by the ANZBA referral criteria, often requires transfer to a Burn Service. Transfer requires careful preparation and documentation.*
- *The following points should be the focus of regular re-evaluation:*
  - *airway or respiratory compromise from inhalation injury or oedema under constricting eschar*
  - *concealed (intra-cavity) bleeding*
  - *neurological deterioration*
  - *adequacy of fluid resuscitation, titrating primarily to urine appearance and output*
  - *peripheral circulatory insufficiency from oedema and inelastic eschar or dressings*

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CHAPTER 4

Inhalation Injury

Introduction

Inhalation injury is defined as the toxic and deleterious effect of heat and the chemical products of combustion on the airways and lungs (1). Inhalation injury is seen in a third of patients with severe burns (2–6). Inhalation injury can result from breathing in hot gases/steam and/or smoke and irritant chemicals. Inhalation injury may be seen more frequently in explosions or entrapment in enclosed spaces.

Historically, inhalation injury has consistently been associated with increased mortality (3-9). In a 2015 nationwide analysis of American burn inpatients, inhalation injury was associated with a four-fold increase in mortality compared to burns without associated inhalation injury ($p < 0.001$) (9).

In another review published in 2014 the mortality rate of patients with inhalation injury was 17.9% compared to 0.7% in patients without inhalation injury (10). In the Burns Evaluation and Mortality Study (BEAMS) their prediction model using multivariant analysis did not show an association between inhalation injury and mortality (11). However, by comparing survivors and non-survivors in that study, non-survivors were statistically more likely to have an inhalation injury (48% versus 39.8%, $p = 0.001$) (11).

Data from BRANZ for admissions between January 2015 and December 2019 showed adult patients with severe burns are twice as likely to die if they had an associated inhalation injury (8,7% versus 18.6%) (2). Overall, the evidence would suggest inhalation injury in a burn patient is associated with increased mortality.

Classification of Inhalation Injury

Inhalation injury refers to damage to the respiratory tract or lungs from heat, smoke or chemical irritants and to systemic toxicity from certain chemicals. An inhalation injury can be broadly classified according to the site of injury (5) see Table 4.3:

1. At and above the larynx – upper airway injury (oropharynx)
2. Below the larynx –
    a. lower airway injury (tracheobronchial tree)
    b. lung parenchymal injury
3. Systemic toxicity

An inhalation injury can involve any of these sites, systemic toxicity or a combination. Inhalation injury can occur in isolation or in association with cutaneous burns. The severity of the inhalation injury depends on the composition of the inhaled components, the size of

the particulates and the magnitude of exposure (temperature, intensity and duration). Resultant respiratory effects may not be directly proportional to the degree of smoke exposure. These differences are likely due to the varied composition of the inhaled material and differences in host response.

Pathophysiology (5,7,8,10,12–15)

1. At and above the Larynx – Upper airway (oropharyngeal) injury

Thermal airway injury from the inhalation of hot gases is generally limited to the supraglottic airway. The upper airway acts as a very efficient heat exchanger and readily dissipates heat and in conjunction with reflex laryngeal closure protects the subglottic airway from thermal injury.

The major pathophysiological changes seen in the oropharynx are similar to the changes seen with cutaneous thermal burns (see chapter 2). The oedema resulting from microvascular dysfunction and fluid administration is readily apparent in the soft tissues of the face and oropharynx. The formation of oedema can result in increasing obstruction and deformation of the upper airway which progress **rapidly over the first hours** of the burn injury.

Commencement of fluid resuscitation can **accelerate** the rate of oedema formation and obstruction. This is more likely to occur in children who have relatively narrower airways, shorter necks and soft tissues that are more easily distorted by oedema. Burns involving more than 20% TBSA in adults and more than 10% in children result in a widespread inflammatory response which may lead to the airway becoming oedematous even if there is **no** direct injury to the oropharynx. It should also be noted that inhalation of steam and blast injuries can cause thermal injuries beyond the glottic opening (i.e. below the larynx).

2. Below the Larynx

With the exception of steam inhalation and blast injuries the injury to the lower airways is usually from toxic chemicals in smoke and carbon particles (soot) coated with irritant chemicals. If the airways are heavily soiled with soot there may be early mechanical clogging of the airways.

a. Tracheobronchial Injury

There are many and varied chemicals in smoke depending on the material that is burning. In general, the inflammatory reaction to these chemicals in the airways is similar to the reaction in skin. The inhalation of toxic gases and liquids such as chlorine, ammonia and acids can also result in a similar inflammatory response.

The chemicals dissolve in the water in the secretions from the mucosa resulting in acidic and alkaline damage to the mucosa. The inflammatory reaction includes:

- hyperaemia of the airway mucosa
- increased microvascular permeability and vasodilatation
- proteinaceous exudate formation with inflammatory cell infiltrate
- direct bronchial mucosal injury and mucosal oedema
- shedding of the bronchial lining

- airway cast formation
    - bronchorrhoea
    - bronchoconstriction
    - loss of hypoxic pulmonary vasoconstriction.

As stated above, in addition to the typical chemical injury, if the airways are heavily soiled with soot there may be early mechanical obstruction.

Clinically, these changes can be seen as: airway obstruction, bronchospasm, alveolar collapse (atelectasis), impaired gas exchange from ventilation-perfusion mismatch and pneumonia.

b. Lung parenchymal injury

Lung parenchymal injury includes cellular dysfunction, pulmonary oedema and pulmonary microvascular clots. These changes are due to increased bronchial circulation delivering inflammatory mediators and toxic material or cells into the pulmonary microcirculation and then to the lung tissue, amplifying the inflammatory cascade. Exudate and cast formation in the airways may result from leaked protein and exudate into the lung interstitium.

These lung parenchymal changes are delayed and dependent on the severity of the airway injury. A shorter delay is associated with more severe injury. The parenchymal injury from inhalation injury can ultimately result in Acute Respiratory Distress Syndrome (ARDS). ARDS is characterised by:

- bilateral pulmonary infiltrates, not explained by cardiogenic pulmonary oedema
- hypoxaemia
- decreased pulmonary compliance and increased work of breathing.

3. Systemic Toxicity

Many materials produced today are synthetics and petrochemical-based materials. These materials burn hotter and faster than natural materials such as wood. When they burn they also produce gases and smoke that are more toxic than natural materials. The most common gases associated with morbidity and mortality in burn patients are carbon monoxide and cyanide.

Other toxic gases and chemicals, among many, that are generated during a fire include hydrogen chloride, phosgene, ammonia, sulphur dioxide, hydrogen sulphide and acrolein. These products of combustion can cause chemical injuries to the airways and lung parenchyma. It should be remembered that as the oxygen in the atmosphere is consumed by the fire the immediate environment can become hypoxic leading to asphyxiation in the victim.

Diagnosis of inhalation injury

Airway and lung parenchymal injury (5–8,12–15)

An inhalation injury should be suspected from the history. The clinical signs and symptoms of an inhalation injury can present early or evolve over a period of time. As with all trauma patients, the patient with suspected or diagnosed inhalation injury must be repeatedly re-evaluated. This is a potentially fatal injury.

An inhalation injury has significant clinical implications and early recognition is important. An inhalation injury is associated with increased morbidity (e.g. airway obstruction, acute lung injury, pneumonia), mortality and an increase in fluid requirements. Clinical diagnosis is based on **history** (e.g. circumstances of the event, chemicals or materials involved, enclosed space, explosion, etc.) and **physical examination** (e.g. singed facial hair, soot on the face and in the sputum, head and neck burns, hoarseness and stridor, etc.). Currently there is limited consensus on the diagnosis and severity grading for inhalation injury, with limited evidence supporting bronchoscopy and helical chest CT (1).

A chest x-ray (CXR) should be obtained in all patients, but a normal CXR does not rule out an inhalation injury. The presence of pulmonary changes on the initial CXR has been associated with increased mortality. If there has been an inhalation injury the CXR will often show changes after 2 to 3 days.

While fibreoptic bronchoscopy is often considered necessary to confirm the diagnosis, the procedure carries significant risk and may not always provide an accurate picture. Bronchoscopy performed soon after injury may not show any airway injury at all. Airway and lung injury may be the result of a systemic response from significant cutaneous burns. Furthermore, bronchoscopy is limited to views of relatively proximal airways and cannot identify any changes or the degree of changes more distally. Bronchoscopy is not uniformly performed in Australia and New Zealand unless there is gross soiling of the airways. Scoring systems to quantify the severity of an inhalation injury have been proposed but are not widely used in Australia and New Zealand.

Circumstances of the event
- heat source: flame, scald, steam
- degree of smoke present
- chemicals involved
- explosion
- enclosed space
- duration of exposure
- level of consciousness

TABLE 4.1 – History relevant to inhalation injury

Observe for...	Listen for...

Burns to mouth, nose and pharynx
Other head and neck burns
Singed facial hair
Soot on the face and sooty sputum
Breathing difficulty:
- increased rate
- increased effort…
  - flaring of nostrils/tracheal tug
  - use of accessory respiratory muscles
	Productive cough
	Hoarse, brassy cough
	Croup-like breathing
	Change of voice
	Inspiratory stridor

TABLE 4.2 – Clinical signs suggestive of an inhalation injury

Type of Inhalation	Mechanism	Timing	Signs / Symptoms
At and above the larynx	Oedema (internal / external) Injury Physical obstruction	Developing over 0–24 hours	Hoarseness or weak voice Drooling Brassy cough Restlessness Respiratory difficulty Increasing stridor
Below the larynx	Tracheobronchial airway Lung parenchymal injury	Gradual onset 12 hrs to 5 days	Increasing hypoxaemia Pulmonary oedema Respiratory failure/ARDS
Systemic toxicity	Absorption into bloodstream	Potentially fatal at scene	Reduced LOC Coma Confusion Drowsiness/Headache Visual disturbances

TABLE 4.3 – Change in Clinical Presentation of inhalation injury over time

Systemic toxicity (5–8,12,14–18)

Patients with systemic toxicity can present early with respiratory distress and/or obtundation at the scene. Early death may occur. Life-saving prehospital rescue and resuscitation may be required. The respiratory distress at the scene of the fire may be due to hypoxia, as the oxygen is consumed by the fire itself.

Carbon monoxide (CO)

Carbon monoxide (CO) is an odourless, colourless gas produced by the incomplete combustion of many fuels such as wood, paper and cotton. CO produces its effect through two mechanisms.

First, CO binds readily to haemoglobin (Hb), displacing oxygen (O_2), to form carboxyhaemoglobin (COHb). CO has an affinity for Hb 240 times more than O_2. This leads to reduced O_2 delivery to the tissues (especially the heart and brain).

Second, CO inhibits the intracellular cytochrome system in the mitochondria, preventing the cells from using O_2. Patients who have CO poisoning exhibit a range of symptoms and signs. They are often confused and disorientated and have a reduced level of consciousness (LOC) similar to patients with hypoxaemia, head trauma and alcohol and drug intoxication. It is important to consider CO poisoning in these circumstances. Apart from the acute effects of CO poisoning there may be delayed effects, predominantly affecting the central nervous system (CNS).

Diagnosis of CO poisoning is dependent on direct assessment of the COHb level in the blood. This is obtained using co-oximetry measurements from a blood gas analyser. Patients who have CO poisoning are often confused and disorientated, exhibiting symptoms similar to those of hypoxia, head trauma and acute drug and alcohol intoxication. It is important to consider CO poisoning in this clinical setting.

Pulse oximetry in this setting is unreliable as the probe is unable to distinguish between O_2Hb and COHb. This may give a false higher reading of the O_2 saturation of the victim. Patients may not look cyanosed; and the classical ‘cherry red’ skin appearance described in pathology texts is rarely seen in survivors. It should be noted that at the scene and in transit victims are usually given high flow oxygen which will displace the CO from haemoglobin. Therefore, the delayed measuring of COHb levels at the hospital may not give an accurate picture as to the true levels of COHb at the scene.

CO-Hb, %	Signs and Symptoms
0–10	None (non-smoker < 3%, smoker <10%) Tightness across forehead, slight headache, dilation of the cutaneous blood vessels
20–30	Headache and throbbing in the temples
30–40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting
40–50	Same as above, greater possibility of collapse; syncope, increased pulse and respiratory rates
50–60	Syncope, increase respiratory and pulse rates, coma, intermittent convulsions, Cheyne-Stokes respiration
60–70	Coma, intermittent convulsion, depressed heart action and respiratory rate, possible death
70–80	Weak pulse, slow respiration leading to death within hours
80–90	Death in less than 1 hour
>90	Death within minutes

TABLE 4.4 – Symptoms at various COHb levels
Reproduced from: Einhorn IN. Physiological and toxicological aspects of smoke produced during the combustion of polymeric materials. Environ Health Perspect. 1975; 11:163.

Cyanide (CN)

Cyanide (CN) is a colourless gas with an odour of bitter almonds. It is produced from the combustion of nitrogen and carbon containing compounds such as wood, silk, cotton, paper, plastics and glues. It is rapidly absorbed through the lungs.

Cyanide acts by reversibly binding to the intracellular cytochrome system resulting in tissue hypoxia. It is converted to thiocyanate in the liver and then excreted through the kidneys. CN poisoning can lead to loss of consciousness, convulsions, coma and death. Most patients with inhalation systemic poisoning will have mixed CO and CN poisoning. Elevated CN levels are directly related to mortality. From reviews of CO and CN levels in fire victims it is thought CN is more likely to be the cause of death than CO.

Diagnosis of cyanide poisoning at the scene of injury and later may be difficult. CN poisoning results in CNS, respiratory and cardiovascular dysfunction. Blood CN levels are not readily available in the clinical setting. Arterial oxygen partial pressures (PaO_2) are not reduced by cyanide. Clinical signs of hypoxia or metabolic acidosis despite an adequate PaO_2 and oxygen delivery may suggest cyanide toxicity. However, a metabolic acidosis can be seen in other settings such as under-resuscitation, trauma, CO poisoning and ambient oxygen-deficient surroundings. If CN poisoning is strongly suspected the relevant poisons information resource centres should be contacted.

Management of inhalation injury (6–8,12–16, 20)

Management should begin at the scene of injury. The first priority is to remove the victim from the source of fire/injury to reduce exposure time. In line with the EMSB approach management of inhalation injury is focused on:

1. Airway patency (including cervical spine control)
2. Breathing (ventilation) adequacy; add supplemental oxygen (high flow if possible)
3. Frequent evaluation (for impending airway obstruction and deteriorating oxygenation)

A brief general approach to managing the airway and ventilation in inhalation injury includes the following procedures:

1. Airway patency (involving cervical spine control)

- Basic manoeuvres
  - clear secretions, blood, foreign bodies
  - jaw thrust/chin support (avoid backward head lift)
- Adjuncts
  - suction
  - insertion of an oropharyngeal airway (e.g. Guedel's airway) or nasopharyngeal airway
  - laryngeal mask as a rescue technique to oxygenate until a definitive airway is secured
- Protected airway
  - intubation via ETT
  - surgical airway

2. Breathing (ventilation) adequacy (with supplemental O_2)

- Mask and bag ventilation
- ETT and lung ventilation/mechanical ventilation

Airway management can become difficult over time as oedema from local injury develops, as well as, from the impact of fluid resuscitation. A normal initial airway assessment is not a good predictor of later obstruction. Frequent reassessment for airway patency is required.

Isolated superficial or mid-dermal facial burns sustained from a flash-over in the outdoors, even in the presence of singed facial hair, are unlikely to require intubation.

Need to maintain or protect a patent airway	- impending or risk of airway obstruction - reduced level of consciousness - haemodynamic instability - clearing of secretions (soot, blood etc.) - facial trauma - deep dermal or full thickness facial burn - to facilitate safe transportation / investigation
Need for mechanical ventilation	- deteriorating oxygenation
If in doubt about the airway and for patient safety, **INTUBATE EARLY**	

TABLE 4.5 – Possible indications for intubation

Specific Treatment of Systemic Toxicity / Poisoning

Carbon monoxide (CO)

CO poisoning is treated by giving supplemental oxygen (via non-rebreathing mask; resuscitation bag and mask; intubation). Treatment should not be delayed. The additional oxygen will displace CO from the haemoglobin in the blood and facilitate the washout of CO from the body.

The half-life of COHb in room air is around 250 minutes, and around 50 minutes when breathing in 100% oxygen. The use of hyperbaric oxygen in the burn injury setting is not established. The half-life of COHb in 100% O_2 at 2–3 atmosphere is around 25 minutes. However, hyperbaric facilities are not really accessible, there is no firm evidence of benefit, there is the risk associated with transfer to the facility and burn care and resuscitation may be hindered or delayed.

Cyanide (CN)

Treatment with hydroxocobalamin has been proposed as a relatively safe approach with few side effects. Hydroxocobalamin (Vitamin B_{12}a) binds intracellular cyanide to form non-toxic cyanocobalamin which is then directly excreted by the kidney. Dosages of 5g in adults over 15 minutes (can be repeated once) and 70 mg/kg in children (can be repeated once) have been suggested. Treatment of cyanide poisoning is based on a high index of clinical suspicion in cases with cardiorespiratory collapse at the scene, significant altered mental status, or severe metabolic acidosis associated with high lactate concentrations (19,20).

Nitrates have been used previously but nitrates increase levels of methaemoglobin in the blood that then bind with cyanide to form cyanmethemoglobin which is then metabolised in the liver. Raised methaemoglobin levels may impair oxygen transport. Other antidotes have been suggested but have significant side effects that limit or preclude their clinical use.

Military Aspects of Inhalation Injury

Up to 17% of military burn casualties have an associated inhalation injury and the civilian population has a similar incidence of up to 20% (21–23).

Guidelines from Australian, British and American military sources recommend that a definitive airway (commonly intubation) should be established early for those personnel with evidence or suspicion of an airway and/or inhalation injury and for evacuation (22–29). While it is acknowledged that establishing a definitive airway may be difficult, if not impossible, in forward positions, it should be performed as soon as possible.

Military personnel may be exposed to a variety of chemicals in the form of gases, vapours or aerosols that have an impact both on the lungs and systemically. These include hydrogen fluoride from automatic fire suppression systems, ammonia, phosgene, chlorine and perfluoroisobutylene (PFIB) from burning Teflon that was originally designed as corrosive, heat and cold resistant, water repellent surface linings of military vehicles and aircraft (29).

Summary

- *Inhalation injury and the related systemic toxicity/poisoning are potentially fatal.*
- *Diagnosis of inhalation injury and/or systemic poisoning depends upon the clinical suspicion and recognition from the history and examination.*
- *Emergency treatment consists of providing respiratory support with oxygen and protection of a patent airway, undertaking endotracheal intubation where necessary. Cervical spine control must be maintained.*
- *Patients with an inhalation injury or suspected inhalation injury should be referred to a Burn Service for ongoing care after initial emergency stabilisation.*

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CHAPTER 5

Burn Wound Assessment

Introduction

Irrespective of the mechanism of a burn injury the amount of tissue damage is dependent on the temperature or the strength of the injuring agent and the duration of exposure to that agent (1–3). To determine the extent of the injury the first step is to assess the depth of the burn wound. The size of the relevant burn wound (in terms of % TBSA) can then be estimated. Note that only dermal or full thickness burn wounds are included in the calculation of % TBSA for resuscitation. Epidermal burns are **not** included in this calculation.

Towards the end of the 19th century it was first noted that there was a relationship between burn size and mortality. In 1884 Schjerning found that if two thirds or more of the body was burned death always occurred, if half of the body was burned death could be expected and if no more than one third of the body was burned, death usually didn't occur (4). Mortality remains related to burn size and has improved since that time but the seriousness of the burn wound remains dependent on both the depth and the size of the burn wound (5–7).

The impact of the burn wound on the patient is influenced by the seriousness of the burn wound and the physiological reserve of the patient (e.g. extremes of age, pre-existing illness, comorbidities such as trauma, inhalation injury).

Structure and Function of the Skin (8–10)

The skin is a self-renewing barrier between the body and the environment. The skin is the largest organ of the body and accounts for around 16% of the total body weight. The skin has several major functions:

- control of fluid loss
- regulation of body temperature
- physical protection against mechanical, chemical, osmotic, thermal and UV radiation sources
- physical barrier against microbial invasion
- involved in immune activity
- metabolic – synthesis of vitamin D, cytokines and growth factors
- sensory interface
- psycho-social interface.

There are two main skin types: thin skin with hair follicles that covers most of the body and thicker hairless glabrous skin that can be found on the palms, soles and flexor surfaces of digits.

The skin consists of two sections call the epidermis and dermis (see Figure 5.1).

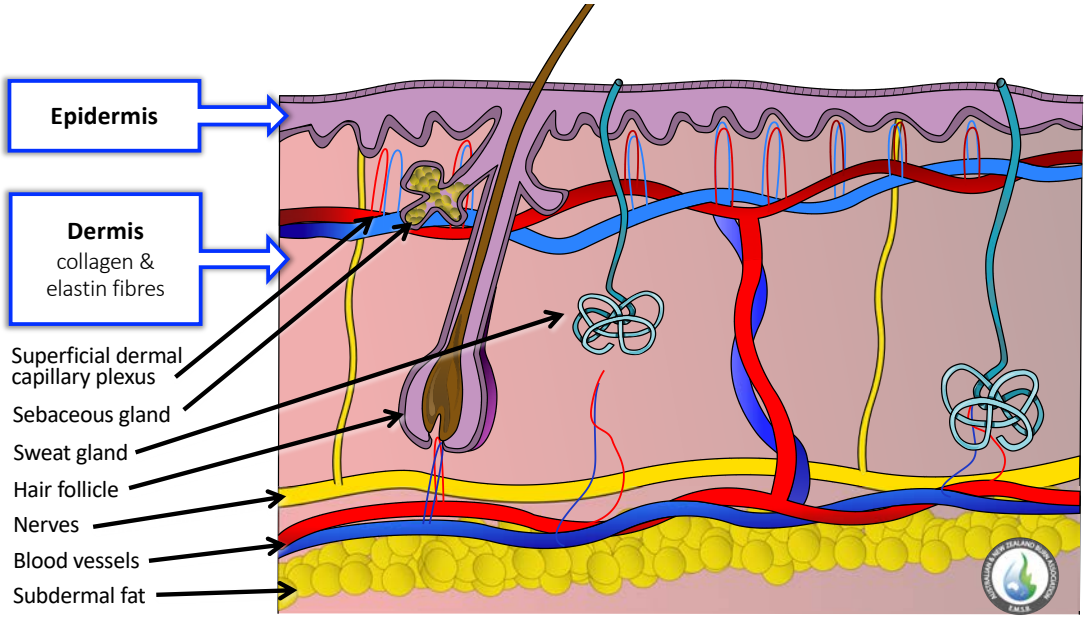

FIGURE 5.1 – Layers of the skin

The epidermis is mostly made up of keratinocytes that undergo continuous renewal and arise from the basal layer of the epidermis. There are other cell types in this layer that serve various functions. Beneath the epidermis is the dermis. The dermis is a thicker, irregular connective tissue layer comprised mostly of collagen and elastin fibres. The fibroblast is the main cell of the dermis. The fibroblast is responsible for the production and turnover of fibrous and elastic dermal proteins. It is the dermis that gives the skin strength and durability.

The dermis contains nerves, blood vessels, lymphatics and epidermal appendages such as hair follicles, sebaceous glands and sweat glands. The lining of these structures can act as a reservoir from which epithelial regrowth can occur under control of growth factors to heal superficial and some mid-dermal wounds. This process is called epithelialisation. The dermis itself cannot regenerate but heals by fibrosis and scarring. The superficial dermal capillary plexus is particularly important in maintaining the viability of the superficial dermis. Beneath the dermis lies the subcutaneous tissues (hypodermis) that is comprised of a layer of loose connective tissue, fat tissue and, in the head and neck region, muscle tissue (e.g. platysma).

As the depth of the burn progresses below the epidermal layers, more and more structures are damaged or destroyed. The loss of these reserve epithelial cells and damage to blood vessels reduces the ability for spontaneous wound healing from the wound bed. Re-epithelialisation can only occur from the edges and is usually associated with significant contracture and scarring.

The structure of skin of the nose and external ears is unique in that the skin is very closely applied to the underlying cartilage with little or no subcutaneous fat. The nerves and blood vessels run between the skin and the cartilage. The skin and cartilage in these areas is readily injured causing significant damage and deformity.

Estimation of the Depth of a Burn Injury (9–12)

The current approach to describe burns is using anatomical depth of the burn wound rather than degrees (as in 1st degree, 2nd degree etc.). Depth of the burn wound can be described as **epidermal**, **dermal** or **full thickness**. Dermal burns can be described as **superficial, mid or deep dermal**. Full-thickness burns extend through the entire thickness of the epidermis and dermis and may penetrate more deeply into underlying structures.

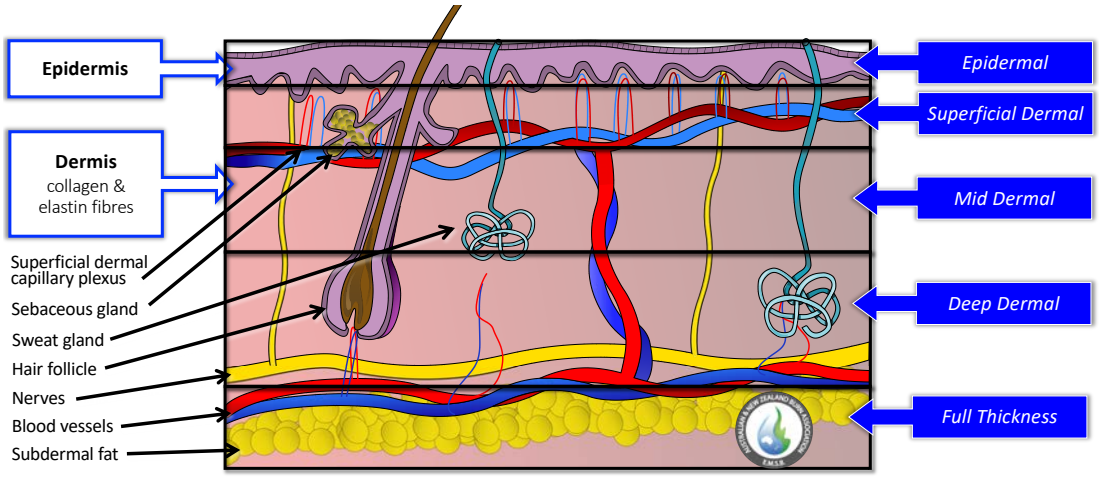

FIGURE 5.2 – Depth of burn injury

Most burns are a mixture of areas of different depths (heterogeneous). Burn depth is dynamic and can progress over time, particularly in the first 48–72 hours. For this reason, the estimation of burn depth may not be completely accurate at the initial assessment.

Clinically, the best time to assess the depth of the burn accurately is 3–5 days post burn. This allows time for the Zone of Stasis to declare whether it will worsen to become part of the Zone of Coagulation or recover. Sometimes it is challenging to differentiate between the different depths of dermal burns. There are a number of technologies, including non-contact Laser Doppler Imaging (L.D.I.), available to assist in determining the depth of the burn wound. However, these remain as adjuncts and repeated serial clinical assessment by an experienced burn clinician is the standard method used to determine burn depth.

Epidermal Burns

Epidermal burns involve the outer layers of the epidermis. Common causes of this type of burn include sunburn and minor flash injuries. These burns are red and painful but do not develop blisters. Digital pressure to 'shear' the epidermis should be 'negative' (negative Nikolsky sign). If the epidermis shears, then it suggests a dermal burn injury.

Healing, without scar formation, occurs within 3–7 days through regeneration from the epidermal basal layer.

Epidermal burns are **NOT** included in estimations of burn size for resuscitation fluid calculations. Burn depth progression over the next 48 hours may result in the loss of the epidermis and necessitate reassessing previous fluid requirement calculations.

Dermal Burns

Dermal burns involve the epidermis and layers of the dermis. They can be divided into superficial, mid and deep dermal burns.

Superficial Dermal Burns

Superficial dermal burns involve the epidermis and the superficial layer of the dermis (the papillary dermis). These burns characteristically form blisters. Blistering may not occur immediately. The epidermal layer covering the blister fluid is dead. The blister fluid is formed from the outpouring of inflammatory oedema from the viable base. The exposed wound bed underlying the blister is pink, moist, has brisk capillary refill, is hypersensitive to touch and often extremely painful. Superficial dermal burns heal spontaneously, through epithelialisation, within 10–14 days and permanent scaring is rare.

Mid-Dermal Burns

A mid-dermal burn lies between a superficial dermal burn, that will heal relatively quickly and a deep dermal burn that will not. At the mid-dermal level there are fewer surviving epithelial cells in the epidermal appendages and epithelialisation is slower. Capillary refill may be sluggish and blisters may still be present. The burn area is usually a darker pink than that of a superficial dermal burn but not as dark as a deep dermal burn. Sensation to light touch may be decreased but pain usually persists. Healing usually occurs within three weeks.

Deep Dermal Burns

The deep dermal burn involves damage or destruction of virtually all essential structures required for spontaneous healing. These burns have a dark red blotchy appearance due to the presence of haemoglobin from red blood cells that have leaked from the damaged blood vessels. There is significant reduction or absence of capillary refill and sensation. Healing does not usually occur spontaneously.

Full-Thickness Burns

In full thickness burns both epidermal and dermal layers of the skin are destroyed. These burns may appear white, waxy, dry, leathery or charred and black. There is no capillary refill and sensation is lost. Full thickness burns do not heal spontaneously. The burn injury may involve deeper structures such as fat, tendons, muscle and bone.

Depth	Blisters	Colour	Capillary Refill	Sensation	Healing
Epidermal	None Negative Nikolsky	Red *epidermis*	Present	Present	Yes
Superficial Dermal	Present Positive Nikolsky	Pale Pink *dermis*	Present	Painful	Yes
Mid Dermal	Present	Dark Pink *dermis*	Sluggish	+/-	Usually
Deep Dermal	+ / -	Blotchy Red *dermis*	Significantly reduced or absent	Absent	No
Full Thickness	None	White *dermis*	Absent	Absent	No

TABLE 5.1 – Assessment of Burn Depth

Estimation of the Area of the Burn (4,11,13–19)

Early fluid replacement strategies based on body weight did not take into account different body sizes and shapes. In 1944 Lund and Browder developed diagrams where clinicians could easily identify burn areas and then quantify a percent of the body surface area that was injured. In 1947, Pulaski and Tennison put forward the 'Rule of Nines' method of estimating burn surface areas and in association with Wallace published the method in 1951. These estimation rules have been used to create apps for smart phones to assist in calculating burn size, and link to fluid resuscitation at the same time. The NSW Trauma app created by ITIM (New South Wales Institute of Trauma and Injury Management) is a good example of such an application available on the Google and Apple app stores.

The 'Rule of Nines' provides a relatively quick and easy estimation of the size of the burn as a percentage of total body surface area (TBSA) (see Figure 5.3). The 'Rule of Nines' divides the body surface into areas of nine percent or multiples of nine, with the exception of the perineum that contributes one percent in adults. Another tool used as an adjunct to the 'Rule of Nines' or in isolation for smaller or scattered burns is the 'Palmar Surface method' (see Figure 5.4). The palmar surface of the hand is defined as the areas from the distal wrist crease to the closed tips of all five digits of the patient's hand, which approximates to 1% of the TBSA. The use of digital tools may assist in calculating TBSA and fluid resuscitation but this should be clarified with the receiving Burn Service.

The 'Rule of Nines' is relatively accurate in adults but does not take into account the different body proportions of a smaller child without modification. Children have proportionally smaller legs and larger heads than adults. For these reasons the Paediatric 'Rule of Nines' should be used (see Figure 5.5). This can be modified for different ages to enable accurate surface area calculations, specifically to the head and lower limbs (see Chapter 8). For every year of life (expressed as a whole number), 1% should be deducted from the head and distributed equally (i.e. halved) to the lower limbs.

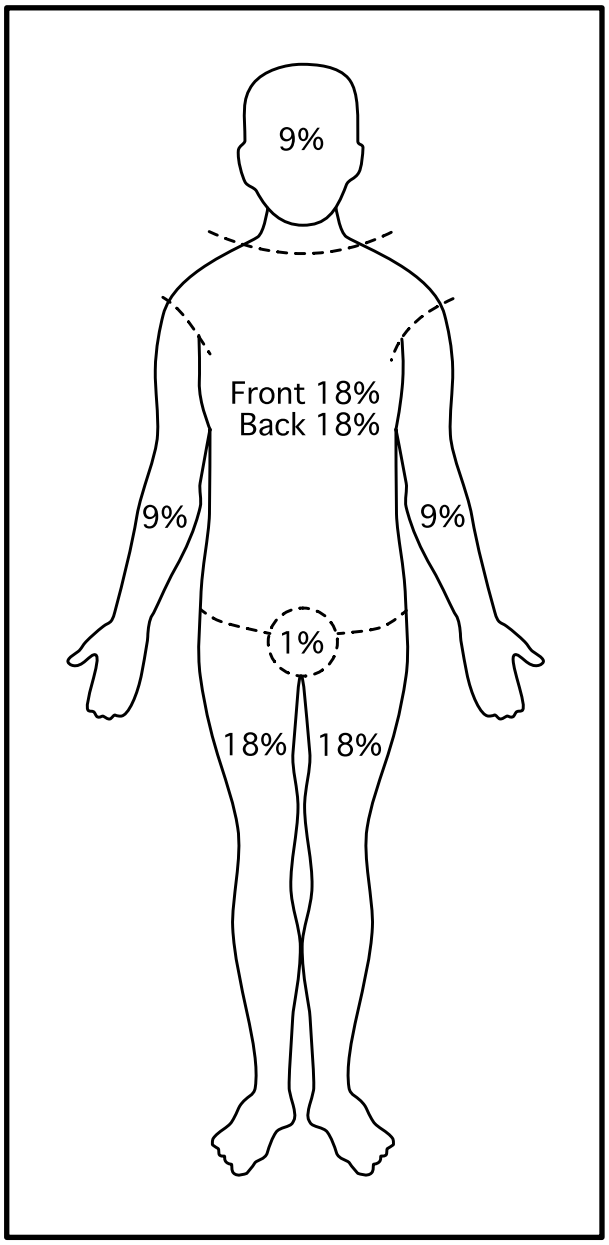

FIGURE 5.3 – Adult Rule of Nines

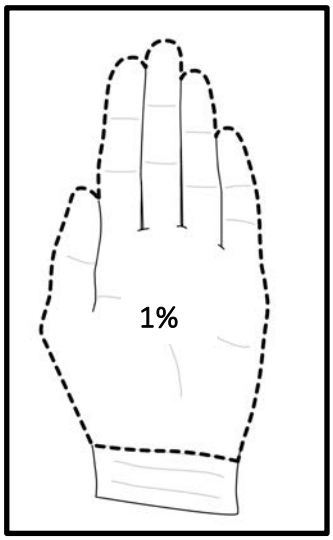

FIGURE 5.4
– Palmar Surface = 1%TBSA

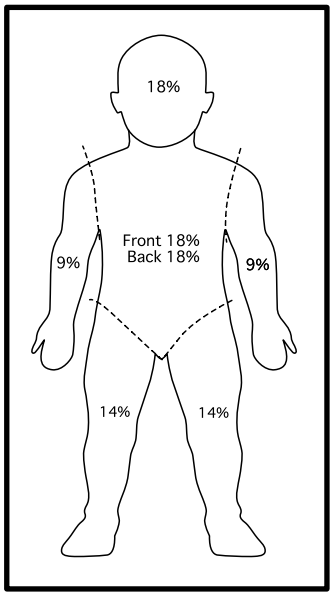

FIGURE 5.5
– Paediatric Rule of Nines

For example:

- a 1 year old child
  - has a 17% (18 - 1) TBSA head
  - and 14.5 (14 + 1/2) TBSA for each lower limb (see Figure 5.4)
- a 4 year old child
  - has a 14% (18 - 4) TBSA head
  - and 16% (14 + 4/2) TBSA for each lower limb (see Figure 5.4).
- From 9 years of age the adult calculations apply, including 1% TBSA being allocated to the perineum.

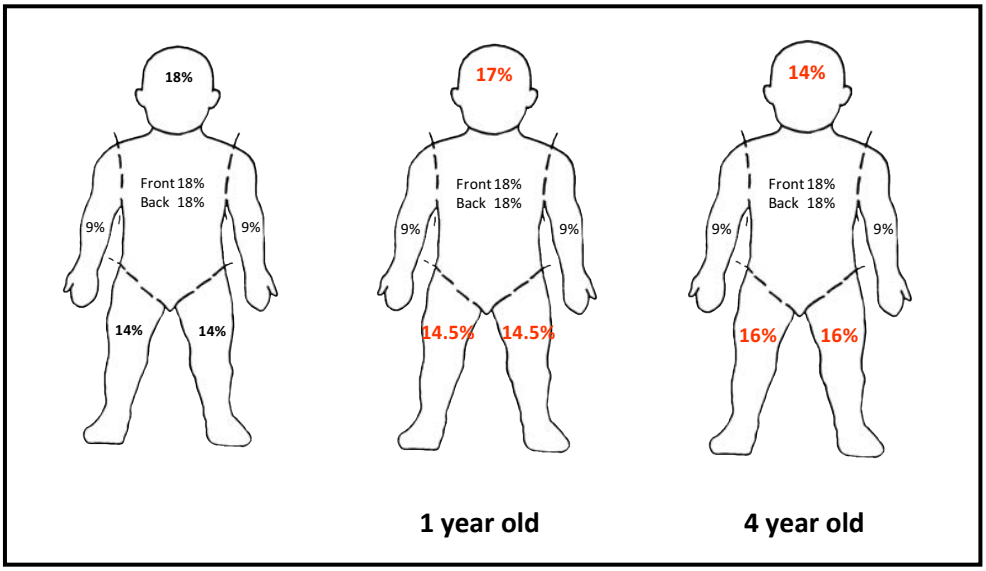

FIGURE 5.6 – Paediatric adjustments to 'Rule of Nines' for a 1 and 4 year old

Body Surface Areas in Overweight Patients (20)

As the body weight and Body Mass Index (BMI) increase beyond the norm the proportional contribution of various major body segments to the total body surface area changes.

There is no universally agreed adjustment for overweight patients. Close clinical assessment guided by your Burn Service is required in these exceptional circumstances.

Summary

- *The severity of a burn is determined by the depth and surface area burn.*
- *The mortality from a burn is related to the severity of the burn injury and the physiological reserve of the patient.*
- *It is possible to diagnose the depth of the burn by clinical examination of the burn wound.*
- *The 'Rule of Nines' and the 'Palmar Surface method' enable assessment of burn extent to be calculated for both adults and children of varying ages.*

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CHAPTER 6

Burn Shock and Fluid Resuscitation

Introduction (1–6)

A severe burn injury can result in shock associated with reduced blood volume, cardiac output and end-organ perfusion. Fluid resuscitation refers to the administration of intravenous fluids acutely after burn injury to correct intravascular volume depletion and the resulting tissue and organ malperfusion (burn shock). Fluid resuscitation is essential in the early management of major burns in order to counteract this burn shock. Fluid resuscitation is needed if the burn wound is estimated at >20% TBSA in adults or >10% TBSA in children.

The basis for current fluid therapy in burn patients began in 1921 following a fire in the Rialto Theatre in Connecticut, USA. Frank Underhill studied the composition of blister fluid of victims and noted its similarity to plasma. He replicated it in a solution of electrolytes and protein, and suggested mortality in burns was due to fluid loss and not toxins.

Following a review of victims of the Cocoanut Grove Nightclub fire in Boston in 1942, Moore and Cope quantified the amount of fluid required per area burn that would provide adequate resuscitation. This was followed by the development of several formulae for calculating the amount of fluid required based on the percentage of TBSA burn **and** the body weight.

By the late 1960's Baxter and Shires who were working at the Parkland Hospital in Dallas, Texas, had developed a fluid resuscitation formula, without colloid, for the first 24 hours, the use of which became widespread across the world and is known as the Parkland formula. A modified version of this is used today and detailed below.

Pathophysiology of Burn Shock (2,7–10, see Chapter 2)

A burn injury leads to loss of plasma (fluid and proteins) into the burn wound itself. A severe burn (>20% TBSA in an adult and >10% TBSA in a child) leads to plasma loss not just into the burn wound but also into unburned tissue. This is due to the widespread increased vascular permeability that occurs in large TBSA burns resulting in:

- hypovolaemia with reduced plasma volume
- reduced cardiac output
- decreased urine output
- increased systemic vascular resistance with reduced peripheral blood flow.

Fluid Resuscitation in Severe Burns

The primary goal of fluid resuscitation is to quickly restore intravascular volume with the minimum amount of fluid required to maintain adequate tissue perfusion and avoid tissue ischaemia. Under-resuscitation may result in burn shock.

Fluid resuscitation is needed when the burned surface area is >20% TBSA in adults or >10% TBSA in children. Fluid should be administered through two large bore cannulae (at least 16G in adults), preferably inserted through unburned skin. Intraosseous access or central venous access may be required.

A trauma patient with burns showing early signs of shock must have haemorrhagic and other non-haemorrhagic pathology excluded. For example, a patient with 60% TBSA burns who has jumped from a burning building and showing early signs of shock may have a fractured pelvis, long bone injury or internal injuries.

Assessment of the burn (see Chapter 5)

The depth and size of the burn determines the extent of fluid lost from the intravascular space and thus needs to be estimated. Epidermal burns involving only the epidermis are **not** included in estimates of the TBSA burn for calculation of fluid resuscitation. All deeper burn depths are included.

The size of the burn is estimated using the 'Rule of Nines' and the 'Palmar Surface method'. Take account of the differences between adults and children when using the 'Rule of Nines' tool. The use of digital tools, including smartphone apps may assist in calculating TBSA and fluid resuscitation but this should be clarified with the receiving Burn Service.

Formula used for fluid resuscitation (1,3,5,6,12–15)

Several studies have shown a tendency for over-resuscitation when using the original Parkland formula which recommended 4 mL x kg body weight x %TBSA burn. Significant complications associated with over-resuscitation has prompted EMSB to recommend using a **modification** of the **Parkland formula** for both adults and children:

Modified Parkland Formula

3 mL balanced crystalloid x kg body weight x %TBSA burn

This formula gives an **estimated** 24 hour fluid resuscitation requirement from the time of the burn injury, **not** from the time of presentation.

Half the calculated volume is given in the first 8 hours and the remaining half given over the subsequent 16 hours.

The rationale is to match fluid resuscitation with the gradual resolution of the widespread increased vascular permeability that begins around 8–12 hours post-burn. This abrupt change at 8 hours implied by the formula fails to reflect accurately the gradual resolution of increased permeability and highlights that the formula is **not** 'set and forget'.

Fluid resuscitation in a severe burn requires constant monitoring of the urine output and (at least) hourly adjustment of the rate of resuscitation fluid administration to match. The modified Parkland formula provides an estimation of the fluid requirement over the first 24 hours and allows calculation of a 'starting' rate for resuscitation.

The aim in providing fluid resuscitation is to give the least amount of fluid needed to maintain tissue perfusion. Formulae are only guides and the resuscitation fluid infusion rate needs to be adjusted according to the generally accepted end-point of hourly urine output.

Monitoring Adequacy of Fluid Resuscitation (1,3,5,6,13,14)

The most commonly used method for determining the adequacy of resuscitation is monitoring the hourly urine output (UO). It is essential that a urinary catheter is inserted if the burn size is >20% TBSA in adults and >10% TBSA in children. Urine output in this context is used as a general reflection of tissue perfusion, renal blood flow and cardiac output. The goal of fluid resuscitation is to achieve the following output:

Adult… 0.5 mL / kg / hr ≈ 30–50mL / hr
Child… 1.0 mL / kg / hr

It is assumed adequate tissue/organ perfusion is being maintained if UO is kept at or near these levels. A low UO indicates poor tissue perfusion and likely cellular injury. A high UO indicates excessive fluid resuscitation that will lead to excess oedema formation.

Adjust fluid resuscitation rate according to the urine output:

If the UO is ***low*** increase the fluid resuscitation rate:
- increase the next hour's fluid by 10–20% (or more if the UO is significantly low) of the planned volume and continue to review hourly.

Avoid giving fluid boluses unless the patient is hypotensive. Low urine output in normotensive patients is addressed by increasing the rate of fluid administration.

If the UO is ***high*** it is important to reduce the fluid resuscitation rate:
- reduce the next hour's fluid by 10–20% of the planned volume and continue to review hourly.

It is vitally important to accurately document fluid balance during resuscitation as this information is helpful in guiding on-going fluid management at the receiving Burn Service.

Serum lactate levels and base deficit can be used as indirect indicators of global tissue perfusion. Elevated lactate levels and acidaemia may suggest the need for increased fluid resuscitation. Other parameters that need to be monitored include heart rate and blood pressure (to ensure adequate vital signs), haematocrit and electrolytes. In select cases transthoracic echocardiography can also provide information on cardiac function and volume status.

Goal directed fluid resuscitation based on Central Venous Pressure (CVP) or Pulmonary Artery Occlusion Pressure (PAOP) is an unreliable measure of fluid responsiveness particularly in burn patients.

Choice of Resuscitation Fluid (1,3,5,6,12–18)

There is some variation in practice in the exact choice of resuscitation fluid between Burn Services.

Crystalloid Solutions

Crystalloids are aqueous solutions with varying amounts of mineral salts that are freely permeable through membranes. The main ions determining the tonicity (a measure of the effective osmotic pressure gradient) of the solution are sodium and chloride.

'Balanced crystalloid' or 'physiological' solutions such as Hartmann's™ solution (lactated Ringer's solution) or Plasma-Lyte™ 148 have electrolytes such as lactate, acetate or gluconate added. These solutions may be associated with hypotonicity and metabolic alkalosis however these effects are not common and generally not clinically significant. Increased lactate levels following infusion of Hartmann's™ solution are usually only seen in patients with significantly impaired liver function. Lactic acidosis is usually a reflection of inadequate resuscitation and impaired tissue perfusion or similar clinical circumstances. Although there is rapid redistribution of these solutions into the extravascular compartment their use remains the main practical approach to initial fluid resuscitation.

'Normal Saline' (NaCl 0.9%, Na: 154 mEq/L) was used in the past for burn resuscitation but, due to effects on the kidneys (reduced renal blood flow and reduced glomerular filtration rate) and because the use of large volumes may cause a hyperchloremic metabolic acidosis, it is **not** recommended as the first choice.

Colloid Solutions

Colloid fluids contain large molecules in a carrier solution which is usually a crystalloid. Colloid solutions can be natural based or semi-synthetic. The most commonly used colloid solution is albumin.

With intact capillaries large molecules are less likely to leak into the extravascular compartment. In the burn wound or in a severe burn with widespread increased capillary permeability there is leakage of large molecules into the extravascular compartment where they may exert an osmotic effect drawing fluid into the extravascular compartment and increasing oedema formation.

Semi-synthetic colloid solutions such as starches, gelatins and dextrans have been associated with a range of adverse effects such as anaphylaxis, impaired coagulation and renal impairment. Therefore, their use is not recommended.

Albumin is commonly presented as 4% or 20% albumin in a saline solution. The role of albumin in the first 24 hours post-burn remains unclear. Half of burn clinicians surveyed in an international study added albumin or Fresh Frozen Plasma (FFP) to the resuscitation regimen within the first 24 hours. Indications for starting colloids are either rescue therapy or large fluid requirements, or routine by some clinicians, but it is currently not supported by high-quality evidence.

Colloids do not affect fluid leakage from the burn wound, but albumin given 8–12 hours post burn can reduce fluid leakage and oedema from unburned tissue.

Colloids, particularly albumin, may also reduce resuscitation volumes and restore cardiac output better than using crystalloids alone. While the use of albumin solutions within the first 24 hours post-burn may be beneficial, the dose, timing, indications and method of administration remain unclear.

Maintenance Fluid in Children (19–21, see Chapter 8)

Children are at risk of hypoglycaemia due to limited glycogen storage, particularly if fasting. To prevent hypoglycemia, children (<16 years) receiving fluid resuscitation should also receive maintenance fluid containing glucose at a calculated hourly rate from the time the infusion starts.

Maintenance fluids for children are calculated as mL / kg / hr using:

- For the first 10 kg of body weight : 4 mL / kg / hour
  + (plus)
- For each kg over 10kg and up to 20 kg body weight: 2 mL / kg / hour
  + (plus)
- For each kg over 20kg of body weight: 1 mL / kg / hour.

The recommended maintenance fluid choice is 0.9% (normal) saline with 5% dextrose. This choice aims to reduce the risk of iatrogenic hyponatremia due to administrations of hypotonic solutions with excessive free water. This represents a change from the previous recommendation and is in response to evolving evidence to support the use of 0.9% (normal) saline, or similar tonicity crystalloids for maintenance fluids in unwell children.

This calculation of maintenance fluids for children is discussed in greater detail in Chapter 8.

Haemochromogenuria

Deep tissue injury, particularly muscle injury, can cause release of myoglobin and haemoglobin (haemochromogens). This is commonly seen in high voltage electrical injury, blunt trauma or tissue ischaemia. These haemochromogens colour the urine a dirty red (haemochromogenuria).

Acute renal failure will occur as a result of both deposition of these haemochromogens in the proximal tubules and a direct toxic effect on the kidneys. Increase the resuscitation infusion rates in order to increase the urine output:

- **Adult**… 1–2 mL / kg / hr
- **Child**… 2 mL / kg / hr

In addition to protecting the kidney, the source of the haemochromogens should be identified and any treatment instigated (e.g. fasciotomies for muscle compartment syndrome).

Concerns with fluid resuscitation (1,3,5,14,16–18)

Acute fluid overload

Infants, the elderly and those with cardiac disease should be monitored particularly closely during fluid resuscitation. In burn patients pulmonary oedema is uncommon due to a disproportionately greater increase in pulmonary vascular resistance than systemic vascular resistance. Pulmonary oedema can occur in patients with pre-existing impaired myocardial function and may require invasive monitoring, inotropic support, ventilatory support and closer monitoring of fluid management.

Fluid creep

The term 'fluid creep' describes over-resuscitation of burn patients above the predicted amounts resulting in a range of oedema-related effects. These effects include airway oedema, intra-abdominal hypertension (IAH), abdominal compartment syndrome (ACS), limb compartment syndromes, progression of burn wound depth, increased ventilation requirements, orbital and cerebral oedema and increased risk of death.

Causes of fluid creep include inattention to monitoring and failure to titrate down fluid administration by clinicians, having excessive UO targets, overestimation of the %TBSA burn and excessive initial infusion rates.

Best recommended practice includes closely monitoring hourly UO, reducing infusion rates if the UO is high and positioning of the patient to reduce the effects of oedema (limb elevation, head up position, etc.).

Need for extra fluid

Low urine output generally indicates inadequate fluid resuscitation. The appropriate first response is to increase the rate of infusion. However, there are a number of situations where it is expected that extra fluid resuscitation is required. These situations include:

- inhalation injury
- electrical injury
- delayed resuscitation
- pre-existing dehydration (e.g. firefighters and intoxicated patients)
- associated trauma.

Military Aspects of Fluid Resuscitation

Burn injuries constitute up to 20% of all injuries in the conflict setting (22–24). Twenty percent of these burn injuries are severe burns requiring fluid resuscitation (25). Other

injuries that may impact on the management of the patient may also impact fluid resuscitation and should be considered.

A study from conflicts in the Middle East showed that of the patients who presented to a Combat Support Hospital with >20% TBSA burn injury 60% did not receive any fluid resuscitation and of those that did the volume administered was in excess of guidelines (26). While it is acknowledged that treatment is difficult during combat evacuation, fluid replacement should start as soon as possible (27–30). Both under-resuscitation and over-resuscitation may result in increased morbidity and mortality (26,30). Improvements in morbidity and mortality have occurred when treating personnel use a modified Brooke formula of 2 mL x kg body weight x %TBSA burn for the volume suggested for the first 24 hours and use formal fluid charting in the field (29,31).

The current approach used in combat **pre-hospital fluid resuscitation** for burns more than 20% TBSA involves the 'Rule of 10s' in which, for adults who weigh between 40–80 kg, the initial hourly infusion rate = %TBSA x 10. For every 10 kg above 80 kg the hourly rate is increased by 100 mL / hr (29,32).

Summary

- *Fluid resuscitation is life-saving*
- *Fluid resuscitation is needed for… adult : >20% TBSA burn*
  child : >10% TBSA burn
- *Calculate first 24 hour resuscitation fluid requirements (from the time of the burn injury) using:*
  - *the modified Parkland formula*
    - *3 mL x kg body weight x %TBSA burn (deeper than epidermal)*
  - *use Hartmann's™ solution or Plasma-Lyte™ 148*
  - *half in first 8 hours / half in next 16 hours*
- *Maintenance fluids are required for children:*
  - *calculate using '4:2:1' rule*
  - *use 0.9% (normal) saline with 5% dextrose*
  - *maintain infusion rate steady – do not adjust up or down.*
- *Monitor hourly urine output with a urinary catheter…*
  - *adult : 0.5 mL / kg / hr*
  - *child : 1.0 mL / kg / hr*

  If haemochromogens are seen, aim for twice normal UO.
- *Adjust resuscitation fluid rate up or down to achieve the target hourly urine output.*

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CHAPTER 7

Early Management of the Burn Wound

Introduction

Early care of the burn wound is important and has the potential to significantly limit burn wound progression and depth. The aim of early management is to stop the burning process and minimise or prevent further complications. Remember the burn wound is both dynamic and heterogeneous. Do not assume all areas of the burn injury are equally deep or will remain as initially assessed.

First Aid

First aid consists of:

1. stopping the burning process
2. cooling the burn wound

First aid reduces tissue damage and the production of inflammatory mediators. Appropriate first aid can maintain the viability of the Zone of Stasis and minimise progression of the burn.

1. Stop the burning process (1–3)

If the patient is still on fire, the flames should be extinguished using the Stop, Drop, Cover (face) and Roll technique. A non-flammable blanket may also assist in smothering the flames. Clothing should be removed. Remember to ensure the safety of the rescuers so they are not injured.

In a scald burn, the clothing that is soaked with the hot liquid acts as a reservoir of heat. The clothing should be removed as quickly as possible to stop the burning process. This includes nappies in children.

All jewellery should be removed because it can both retain heat and produce a tourniquet effect especially on the limbs and digits; even in unburned areas.

2. Cooling the burn wound (1–12)

Cooling the burn wound reduces the local inflammatory reaction and may prevent conversion of the Zone of Stasis to the Zone of Coagulation. The recommended technique is to use cool running water for 20 minutes, commencing as soon as possible. The recommended temperature of the water is 15°C. Cooling is effective within the first three hours after the injury but the effect is greater the earlier the cooling is started.

Ice or iced water must not be used for cooling. The extreme cold causes vasoconstriction and may worsen the tissue injury. Where cool running water is not available other methods such as immersion in containers, spraying or sponging water on to the wound may be used with limited effect. If these methods are used they must be refreshed frequently as heat from the body makes them ineffective.

Hydrogels are not effective for first aid treatment. In burns >20% TBSA in adults or >10% TBSA in children they should not be used due to the risk of hypothermia (see ANZBA Consensus Statement at www.anzba.org.au).

It is important to simultaneously cool the burn wound and to keep the patient warm. Children are at even greater risk of hypothermia during the application of first aid due to their relatively higher body surface to volume ratio. Cooling the surface of the burn wound may also have an analgesic effect.

Hypothermia in the Burn Patient

Systemic hypothermia frequently accompanies severe burns unless pre-emptive preventative measures, constant monitoring and proactive steps are taken. This is especially the case in young children, who have a higher surface area to body mass ratio, thinner skin and less effective thermoregulation (13). Hypothermia increases complications such as coagulopathy, cardiac arrhythmias and metabolic instability, which ultimately increase morbidity and mortality (14,15).

In the pre-hospital phase, cooling of the burn wound for first aid should be undertaken judiciously. Where possible, areas of the body not requiring cooling should remain covered. If possible, the temperature of the room in which the patient is undergoing first aid should be kept warm. Once cooling has been completed, the affected areas should be covered (16,17).

After exposure for the primary survey, resuscitation of the patient with severe burn injuries should ideally be undertaken in a suitably warmed room. Keeping the patient covered with warm blankets and external warming devices should be employed. Resuscitation fluids should be also warmed.

Management of the Burn Wound

Dressing (1,2,10–12,18,19)

Once first aid has been completed the wound should be washed with an appropriate solution such as saline, soap and clean water or aqueous chlorhexidine gluconate 0.1% solution. In preparation for transport the patient will need a dressing on the burn wound.

If the patient will arrive at the receiving Burn Service within eight hours from the time of the burn, the wound may be dressed with **cling film**. Cling film is available in large sizes, is non-adherent, pliable and transparent. It can limit evaporation and heat loss.

Soft white paraffin is generally recommended for faces. Cling film should not be used on the face and it should not be applied circumferentially. If the patient is unable to be transported to the receiving service within eight hours, topical antimicrobial dressings may be used after discussion with the receiving Burn Service.

Dressings should be applied to take into account potential oedema formation. Primary dressings should not be constrictive and should not be applied circumferentially. Further information on dressings can be found in Appendix 4 Selecting an Appropriate Dressing.

Elevation

Oedema increases the distance between capillaries and cells. Elevation can limit oedema formation and theoretically may improve tissue oxygenation, nutrition and subsequent survival. Elevation of the burn wound area (head and/or limbs) is recommended during initial treatment and transport.

Elevation of an affected limb may not only limit oedema formation but may improve the limb circulation. This is especially important in circumferential or near-circumferential burn injuries to the limbs.

Escharotomy (2,4,12,20–27)

When a burn injury affects the whole of the dermis, as in deep dermal or full thickness burns, the skin loses its elasticity and is unable to expand if there is underlying oedema. When the distribution of this inelastic tissue is circumferential or near-circumferential this can be problematic especially with the need for fluid resuscitation for a severe burn. The inevitable swelling will produce different harmful effects depending on the area involved.

To address the effects of this inelasticity and oedema formation it may be necessary to decompress the burn wound eschar surgically by incising the burned skin down to healthy subcutaneous tissue, extending beyond the burn wound at both ends into either unburned skin or until the area involved is no longer circumferential or near-circumferential (i.e. where it is no longer needed). This procedure is called an escharotomy. There are recognised incision lines for escharotomies (see figure 7.1).

Neck

If there is a circumferential or near-circumferential burn to the neck and the airway is threatened an anterior midline incision may be needed.

Torso

Rigidity of the chest wall decreases compliance and this may reduce ventilation especially when compounded by subcutaneous oedema. In adults this is seen with circumferential or near-circumferential burns of the chest with or without involvement of the abdomen.

In younger children breathing is principally diaphragmatic. Due to the inelasticity of burned skin, ventilation problems may be seen when the front of the chest and abdomen are burned *even without the injury extending to the posterior aspect*.

Limbs

In deep dermal or full-thickness circumferential or near-circumferential limb burns, oedema causes increased pressure under the rigid burn skin which may interfere with circulation, especially in the context of fluid resuscitation. The resulting ischaemia may be rapid or slow and progressive and can be missed if neurovascular observations and patient assessment are not performed. This may be detected by the appearance of one or more of the following signs or symptoms:

- increased tension/pressure in the limbs or stiffness
- decreased perfusion… coolness and pallor of the skin
  reduced capillary refill
  weak or absent palpable pulses
  Doppler Ultrasound changes
- increasing pain at rest, worse with passive movement
- loss of distal circulation.

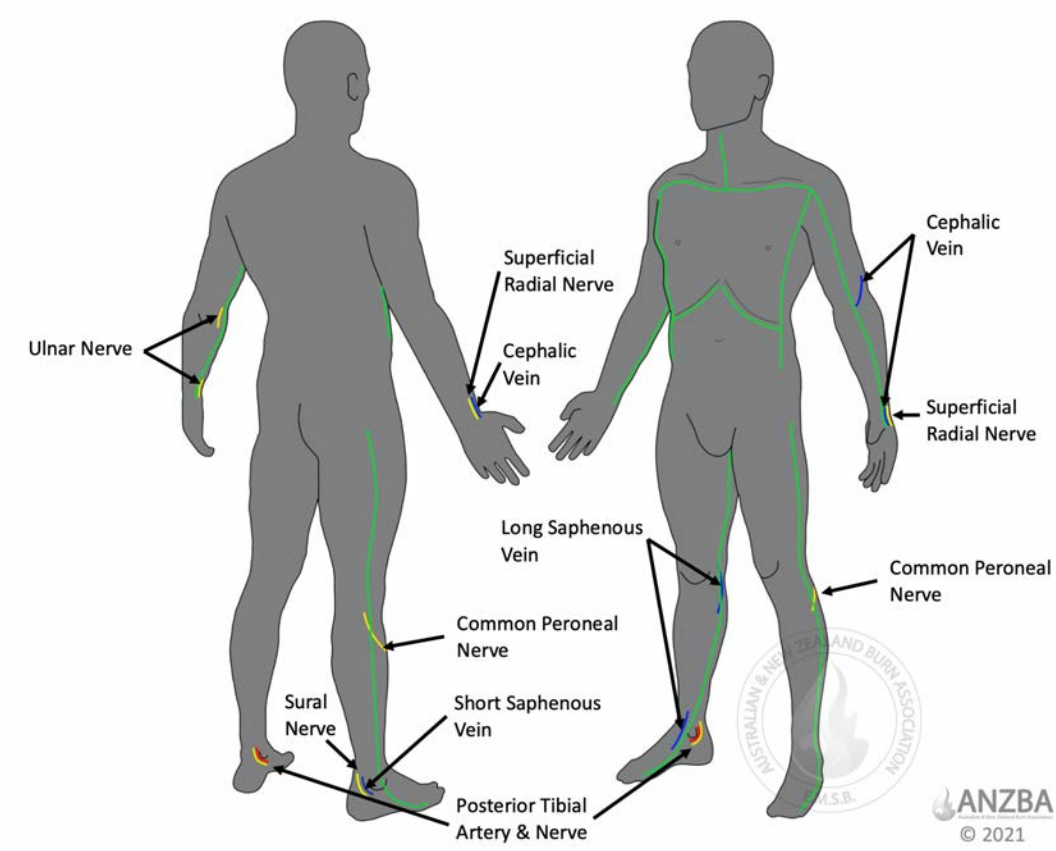

FIGURE 7.1 – Incision lines for escharotomy (in green)

Procedure

Informed consent for the procedure should be obtained from the patient whenever possible. In the conscious patient local anaesthetic is necessary in the unburned skin at the upper and lower limits of the intended escharotomy incision. A general anaesthetic is recommended for children.

The affected limb/chest/neck should be prepped and draped appropriately. The limb needs to be positioned in its anatomical position. The resting forearm naturally lies in pronation, especially when oedematous so it must be supinated before marking and incising. Intended lines of incision should be marked with a marker pen.

For the torso, the incisions run longitudinally along the anterior axillary lines (from unburned skin) to the costal margin or to the upper abdomen (to unburned skin). In severe cases it may be beneficial to connect these incisions across the midline in the upper chest just below the clavicles and across the upper part of the abdomen.

Escharotomy incisions in the limbs run along the mid-axial lines. In the upper limb the incision should go in front of the medial epicondyle to avoid damage to the ulnar nerve.

In the lower limb the medial incision should avoid the posterior tibial neurovascular bundle that lies behind the medial malleolus and also avoid exposing the medial malleolus itself. Laterally, care should be taken to avoid the common peroneal nerve where it crosses the neck of the fibula.

Further detail is in Appendix Three.

The equipment should include a scalpel or cutting diathermy, and some means of achieving haemostasis. Artery forceps and ties, diathermy or topical haemostatics such as calcium alginate are useful. Following the escharotomy the wounds should have an appropriate dressing applied such as calcium alginate or multi-layer impregnated gauze. Limbs should be elevated post-procedure with hourly neurovascular observations.

Fasciotomy (21–27)

A fasciotomy is a different surgical procedure to an escharotomy. It is indicated in compartment syndrome which is caused by the swelling of muscle tissue within one of the fascial compartments of the limb. Compartment syndrome can develop following high-voltage electrical, crush or deep burn injuries involving muscle.

The surgical procedure follows different incision lines and involves opening the deep fascia of the relevant compartment to allow the swollen muscle to freely expand. This will relieve the increased compartment pressure and restore muscle perfusion. Fasciotomy should only be performed under general anaesthetic in an operating theatre and by an experienced surgeon.

Escharotomy	Fasciotomy
• Divides eschar	• Divides deep fascia
• Can be performed at bedside	• Performed under GA in theatre
• Prophylactic or therapeutic	• Prophylactic or therapeutic (haemochromogenuria)

TABLE 7.1 – Differences between Escharotomy and Fasciotomy

Blisters (4,10,11,12,28-31)

Blisters develop when fluid accumulation leads to separation of the epidermal and dermal layers. Blisters indicate that the burn injury is deeper than the epidermis and has extended into the dermis. Blisters are mostly seen in superficial dermal burns. Remnants of blisters may be seen in deeper burns. Blisters may remain intact or rupture early.

Management of blisters remains controversial. There is no definitive evidence for any of the options including leaving the blister intact, de-roofing completely or evacuation of blister fluid whilst leaving the skin as a biological dressing. Leaving small blisters intact may reduce infection rates. De-roofing the blister can be painful and adequate analgesia should be used. While it is unclear if blister fluid helps or inhibits healing, advocates of de-roofing suggest that removal of blisters allows for accurate assessment of the wound bed, as blisters may hide a deep dermal burn. De-roofing of the blister also allows direct treatment of the burn wound bed. One suggested approach involves leaving small blisters intact and de-roofing blisters that are larger (>5mm), tense, associated with pain, restrict movement or are over joints.

Summary

- *First aid consists of stopping the burning process and then cooling the burn wound whilst keeping the patient warm.*
- *Immediate treatment of the wound should be kept simple but aim to prevent complications such as infection and constriction, whilst assisting with early wound closure.*
- *Escharotomy should be considered in any circumferential (or near circumferential) deep dermal or full thickness burn to the chest (to allow ventilation) or limbs (to prevent distal circulatory obstruction) to release the rigid eschar.*

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Chapter 8

Paediatric Burns

Introduction (1,2)

In this manual the definition of a 'child' is a person less than 16 years of age. This definition is consistent with that adopted by the majority of Australian and New Zealand Burn Services and the Burn Registry of Australia and New Zealand (BRANZ).

As with adults the long-term outcomes of a child with burns is related to the initial assessment and management. Early and appropriate management will reduce complications, scarring and later surgical intervention.

A higher proportion of the paediatric population suffer burn injuries compared to adults. Scald burns are the most common cause of burn injury in children. The pattern of the mechanism of a burn injury in children varies with age, for example, the incidence of flame burns increases with age. Eighty percent of incidents occur in the home, most commonly in the kitchen and bathroom.

The numbers below reflect severe burns defined as >10% for children or any burn requiring admission to an ICU.

Home	79%
Other residential	7%
Place of recreation	4%
Trade & service area	0%
Roadway	3%
Other	5%

TABLE 8.1 –
Children's Place of Severe Burn Injury (%)

Scald	61%
Flame / explosion	23%
Contact	8%
Chemical	1%
Electrical	4%
Friction	4%
Other	1%

TABLE 8.2 –
Causes of Children's Severe Burn Injury (%)

The basic concepts of emergency burn care in adults apply equally in children. Conduct a Primary Survey to detect and correct any immediate life-threatening conditions. Institute treatment (FATT), followed by a Secondary Survey.

Differences Between Adults and Children (1–3)

Body size and proportions

Children have a greater body surface area to weight ratio than adults. This means children will have higher fluid requirements compared to an adult for any burn size. Consequently, fluid resuscitation in a child is commenced at a burn of >10% TBSA compared to >20% TBSA in an adult.

In children, the head and neck are comparatively larger than in an adult and the legs are comparatively smaller. In a child up to one year of age the head and neck constitute 18% TBSA (9% in an adult) and each leg constitutes 14% TBSA (18% in an adult).

Skin

Children have higher evaporative water losses and higher heat losses making them very susceptible to hypothermia.

Children have thinner skin than adults. They will have a deeper burn than an adult for any given temperature. They also have fewer epidermal appendages which may have an impact on healing. Water at 60°C will cause:

- in infants: a full thickness burn injury in less than one second
- in an older child: a full thickness burn injury in 5 seconds
- in an adult: a full thickness burn injury after 20 seconds.

Children's avoidance reactions are slower. A child may not withdraw as quickly, or even fail to withdraw altogether, thus increasing the duration of exposure to the insult leading to a deeper burn injury.

Airway

There are several differences between the paediatric and adult airways. Issues that can impact on the airway itself or on securing the airway include:

- larger head in proportion to the rest of the body
- large tongue
- large tonsils and adenoids
- long floppy epiglottis
- small mouth
- no teeth or loose teeth
- soft palate extends more anteriorly (recedes to become hard palate as the child grows)
- trachea… smaller in absolute diameter
  narrower at the cricoid cartilage level
  softer more pliable
  shorter in length

These issues put a child with burns at a higher risk of airway obstruction and can make securing the airway, including endotracheal intubation, difficult.

Occult or hidden airway obstruction is common in children and related to the narrow airways, a large tongue, large tonsils and adenoids, and frequent upper respiratory tract infections.

Breathing/ventilation

Children can effectively compensate for respiratory failure by increasing their respiratory rate and work of breathing. However, when decompensation occurs it is often sudden and potentially fatal.

The indicators of compromise include:

- change in mental state (listlessness or irritability)
- subcostal recession, grunting
- hypoxaemia
- respiratory acidosis.

Circulation

Compensatory mechanisms in a child mean circulation may be well maintained even in large fluid deficits. Little overt warning of circulatory collapse may be given until late in the progression of the pathophysiology of shock. Useful signs of shock and hypoxia in the adult, such as agitation and tachycardia, are more difficult to interpret in children because they can occur for other reasons such as pain or anxiety.

Hypotension is a late sign of hypovolaemia in children and indicates decompensation of homeostasis. By the time it occurs the child is rapidly and dangerously deteriorating towards irreversible shock. Therefore greater reliance must be placed on the alternative signs of decreased circulation.

The indicators of compromise include:

- tachycardia (age appropriate)
- capillary refill >2 seconds (sternum)
- mottled or pale cool peripheries
- organ dysfunction: tachypnoea, altered mental status / lethargy
- metabolic acidosis (lactic acidosis / base deficit).

Initial Assessment

The initial assessment of children with burns is the same as with an adult and follows the EMSB approach to the assessment and management of a patient with severe burn injuries (ABCDE, FATT, Secondary Survey).

An accurate history is essential and should include the mode of injury, the time of the accident, what first aid was given, what the child was wearing and, in the case of scalds, how hot was the liquid at the time of the incident. The medical history should be ascertained including immunisation history. A social history is important as is evaluation of the history of the injury and physical findings where there may be inconsistencies indicating a non-accidental injury.

Management (1–4)

First Aid

The principles of first aid for a burn injury are the same in children as in adults (see Chapter 7). However, children are at a much greater risk of hypothermia than adults. This risk is greatest during the application of first aid, exposing the patient to assess the wound, and during procedures and transport. If first aid has not been given to the child prior to presentation it should be given **AFTER** the Primary Survey.

Care should be taken to limit the application of cool water only to the burn wound and to keep the rest of the child warm. (e.g. warm blankets, overhead heating). Active warming of a child after burn first aid should be undertaken.

Airway

The anatomical differences seen in children place them at greater risk of airway obstruction. The narrower airways in a child mean less oedema is needed to cause airway obstruction.

Children requiring fluid resuscitation, even without burns to head and neck, may develop generalised oedema including oedema of the airway.

Total Body Surface Area

In children the head and neck are comparatively larger than in an adult and the legs are comparatively smaller (see Chapter 5). In a child up to one year of age the head and neck constitute 18% TBSA and each leg is approximately 14% TBSA.

For every year of life (expressed as a whole number), the surface area of the head decreases in size by 1% and each leg gains 0.5% TBSA (see Figure 8.1). Adult proportions are attained at nine years of age.

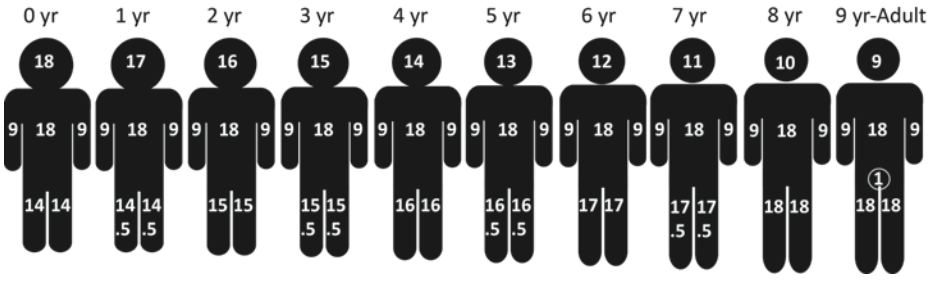

FIGURE 8.1 – Changing body proportions with age
Modified with permission from Mike Fuery, Ambulance, Victoria

Fluid Management (1–6)

Resuscitation Fluid (see Chapter 6)

Children have greater fluid losses than adults with an equivalent burn injury. Fluid resuscitation in children is commenced at >10% TBSA compared to >20% TBSA in an adult. Children require maintenance fluid in addition to the resuscitation fluid (see below).

Resuscitation fluid is calculated using the same formula as for adults:

Modified Parkland Formula

3 mL balanced crystalloid x kg body weight x %TBSA burn

Epidermal burns are NOT included in the %TBSA calculation for fluid resuscitation.

The recommended resuscitation fluid is Hartmann's™ solution or Plasma-Lyte™ 148. Two intravenous cannulae should be inserted into peripheral veins (percutaneously or via cut-down) through unburned skin if possible. Intraosseous access or central venous access may be required.

This formula gives an **estimated** fluid resuscitation requirement for the first 24 hours from the time of the burn injury, **not** from the time of presentation.

Half the calculated volume is given in the first 8 hours from the time of the burn injury and the remaining half given over the subsequent 16 hours.

The rationale is to match fluid resuscitation with the gradual resolution of the widespread increased vascular permeability that begins around 8–12 hours post-burn. This abrupt change at 8 hours implied by the formula fails to reflect accurately the gradual resolution of increased permeability and highlights that the formula is **not** 'set and forget'.

Fluid resuscitation in a severe burn requires constant monitoring of the urine output and (at least) hourly adjustment of the rate of resuscitation fluid administration to match. The modified Parkland formula provides an estimation of the fluid requirement over the first 24 hours and allows calculation of a 'starting' rate for resuscitation.
The aim in providing fluid resuscitation is to give the least amount of fluid needed to maintain tissue perfusion. Formulae are only guides; the resuscitation fluid infusion rate needs to be adjusted according to the generally accepted end-point of hourly urine output.

Maintenance fluid administration rate (see below) remains constant.

Adequacy of resuscitation is assessed using hourly urine output. For children the recommended urine output is 1 mL / kg / hr.

Adjust fluid resuscitation rate hourly according to the urine output:

If the UO is ***low*** increase the fluid resuscitation rate:
- increase the next hour's fluid by 10–20% (or more if the UO is significantly low) of the planned volume and continue to review hourly.

Avoid giving fluid boluses unless the patient is hypotensive. Low urine output in normotensive patients is addressed by increasing the rate of fluid administration.

If the UO is ***high*** it is important to reduce the fluid resuscitation rate:
- reduce the next hour's fluid by 10–20% of the planned volume and continue to review hourly.

It is just as important to reduce the resuscitation fluid administration rate if the urine output is above 1 mL / kg / hr. Over-resuscitation may lead to heart failure, pulmonary oedema, cerebral oedema, abdominal and limb compartment syndromes and compromise of the Zone of Stasis.

Accurate fluid balance documentation is vitally important during resuscitation as this information is helpful in guiding on-going fluid management at the receiving Burn Service.

Maintenance Fluid

Limited hepatic glycogen stores expose all children to the risk of hypoglycaemia, particularly in association with hypothermia. In prolonged fasting, even adolescents may benefit from the provision of glucose. For this reason, the maintenance fluid should contain glucose, e.g. 5% dextrose as part of an isotonic crystalloid solution.

The current recommended maintenance fluid of choice is 0.9% (normal) saline with 5% dextrose. Regular blood glucose measurements should be performed during initial stabilisation and transport.

Hourly maintenance fluid requirements can be calculated using the following '4:2:1' formula:

- For the first 10 kg of body weight : 4 mL / kg / hour
  + (plus)
- For each kg over 10kg and up to 20 kg body weight: 2 mL / kg / hour
  + (plus)
- For each kg over 20kg of body weight: 1 mL / kg / hour.

The total maintenance fluid is the sum of these three calculations.

The maintenance fluid is given at a constant hourly rate. The rate is **NOT** adjusted based on the urine output. It is commenced as soon as possible after presentation without adjustment to 'catch up' for delayed presentation.

Figure 8.2 illustrates examples of the application of the '4:2:1' formula in calculating the hourly maintenance fluid requirements for a 7 kg, 17 kg and a 27 kg child.

kg		Rate Rule		mL / hr
7	x	4	=	28
		2		
		1		
		TOTAL		**28 mL / hr**

7 kg

kg		Rate Rule		mL / hr
10	x	4	=	40
PLUS				
7	x	2	=	14
		1		
		TOTAL		**54 mL / hr**

17 kg

kg		Rate Rule		mL / hr
10	x	4	=	40
PLUS				
10	x	2	=	20
PLUS				
7	x	1	=	7
		TOTAL		**67 mL / hr**

27 kg

FIGURE 8.2 – Maintenance fluid for 7 kg, 17 kg and 27 kg children

Other Differences

Escharotomies

Indications for limb escharotomies are the same in children as in adults.

Trunk escharotomies, however, may be necessary in children with less extensive burns than in adults. This is because younger children breathe by diaphragmatic movement and abdominal wall rigidity is more likely to restrict tidal volume. For this reason interference with ventilation may occur from trunk burns that are not fully circumferential. If a full-thickness burn involves the anterior and lateral aspects of the chest and the upper half of the abdomen, trunk escharotomies should be considered.

Gut

Children are more prone to gastric dilatation than adults and tend to swallow air when crying. An adequately sized nasogastric tube on free drainage is therefore often recommended in the initial assessment phase and during transport particularly if aerial evacuation is needed.

Children's high metabolic rate and their nutritional needs for growth mean that they have less tolerance of nutritional deprivation. Enteral feeding should be established as soon as possible after arrival at the definitive treatment centre, as it prevents loss of gut function and maintains nutrition.

Emotional Aspects

When a child sustains a burn injury the consequences for the child and their family's psychological, emotional, social and financial well-being can be profound.

The impact will be determined by a wide range of factors, not necessarily including the size of the burn. Interventions are aimed at promoting the psychosocial well-being of family members so the primary caregivers are in an optimal position to provide effective support for their child.

Psychosocial support is required for both the patient and their family.

Non-accidental Injury

Children's burns vary from momentary lack of supervision, through neglect, to the other extreme of deliberate abuse or self-harm.

Health professionals have legal obligations to report non-accidental injuries. Staff should follow local processes and abide by local regulations. These statutory requirements differ from state to state in Australia, and in New Zealand.

Suspicion of non-accidental injury meets the referral criteria to a Burn Service.

Suspicion of non-accidental injury may be raised by:

- delay in presentation
- vague or inconsistent history from different observers
- history not compatible with pattern of injury
- history not compatible with the physical capabilities of the child
- presence of other signs of trauma
- certain patterns of injury (e.g. cigarette marks or bilateral 'shoes and socks' scalds).

Summary

- *The **principles** of burn care are the same for children as for adults.*
- *Factors that modify the care of children are:*
  - *different body proportions*
  - *different fluid dynamics*
  - *thinner skin*
  - *different psychosocial needs*
- *Major physical differences in care are:*
  - *tendency to hypothermia*
  - *increased depth of injury for a given mechanism or agent*
  - *increased fluid and glucose requirements*
- *The psychosocial background and emotional needs of the child with a burn injury and their family may be very different to those of adult burn patients.*

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CHAPTER 9

Electrical Injuries

Introduction

Electrical injuries account for 2.5% of adult burn admissions and 1% of paediatric burn admissions across Australia and New Zealand (1). Admissions due to electrical injuries can be as high as 17% of burn admissions in developing countries (3).

Electrical injuries can take a number of forms. In addition to thermal burns at the sites of contact with the electrical source and ground, there may be deep tissue damage from the electrical current passing through the body, and/ or cutaneous burns from 'flash over' or the ignition of clothing. The normal electrical functions of nerves and muscles in the path of the current may also be disrupted, resulting in sensory changes, muscle spasm/tetany or cardiac arrest. Additional injuries may be sustained if the victim is thrown or falls as a result of the electrical discharge.

Principles of Electricity (4,5,7–9,11,12)

In order for electricity to flow, a complete pathway, or circuit, must be formed by conductive materials, although in the case of very high voltages, a spark can pass through the air – lightning being the most extreme example.

The severity of an electrical injury depends on the type and amount of current flow, voltage and resistance (both of which affect current flow), the duration of contact and the pathway of current flow in the body.

Current (I)

Current is the flow of electrons from atom to atom down an electrical gradient. Current is measured in amperes (A).

There are two types of current: alternating current and direct current. Current flow that changes direction periodically in a circuit (to and fro) is called alternating current (AC). The frequency of this current direction change is measured in Hertz (Hz).

Current that flows in one direction in a circuit is called direct current (DC).

Voltage (V)

Voltage is the electrical difference (potential) between two points. It can be thought of as the electric 'pressure' that causes current to flow. Voltage is measured in volts (V).

In electrical injuries in humans, except for lightning, voltage is the only physical property of the circuit that is actually known. Voltage levels of circuits can be divided into low voltage (<1000 volts) and high voltage (>1000 volts).

Resistance (R)

Resistance is the degree to which a material or substance hinders or impedes the flow of current. Resistance is measured in ohms (Ω). Resistance of the different tissues in the body plays an important part in determining injury patterns and severity.

Ohm's Law

The relationship between voltage, resistance and current within a circuit is described by Ohm's law. This states that voltage equals the current multiplied by the resistance. Rearranged, it states that the amount of current flowing is directly proportional to the amount of the voltage of the source of electricity and inversely proportional to the resistance of the material/substance through which the current flows.

$$V = I \times R \quad \textit{or} \quad I = V / R$$

Practically this means the higher the voltage the greater the current for a given resistance, and the higher the resistance the lesser the current for a given voltage.

Thermal Energy (Q / E)

The heat generated (measured in joules) from the passage of current through a substance (in this case the human body) can be described by Joule's Law:

$$Q = I^2 \times R \times t$$

(heat generated) = (current squared) x (resistance) x (duration of exposure)

That is, as the amount of current flow increases, or the resistance increases so does the amount of heat that is generated.

Types of Electrical Circuits

	AC	**DC**
Low Voltage (<1,000V)	Domestic Power Supply	Car Batteries, Electroplating
High Voltage (>1,000V)	High Tension Power Lines Substation	Electric railway systems

TABLE 9.1 – Types of Electrical Circuits

In this classification the amount of current is not considered separately to voltage as, in general, both increase together. Through a succession of transformers the higher voltages are gradually reduced to lower voltages used in households and industry.

Low Voltage (<1,000 V)

AC circuits: In Australia and New Zealand the standard single-phase household electrical supply is provided as alternating current at 50 cycles per second at 240 volts. Some industrial supplies are 3-phase AC at 415 volts.

The three wires that connect to power outlets include two 'live' wires, called active and neutral, and an 'earth' wire that runs to a common conductor driven into the ground on the outside of a building. In order for a person to sustain an injury from this type of power supply, they must contact a live wire and complete the circuit by simultaneously contacting the ground or the earth wire.

Circuit breakers or fuses are placed to interrupt current flow in a circuit if it exceeds a certain limit. Current overload of wiring can lead to overheating and electrical fires. A fuse operates once and then must be replaced whereas a circuit breaker can be reset manually or automatically. In the absence of an RCD (see below) a fuse will 'blow' in the event of a current leak to ground, but this is much slower and less protective.

Safety switches or residual current detectors (RCDs) are designed to protect people by detecting leaking current from switches, wiring or appliances and interrupting the electricity supply. Generally RCDs turn off the electricity supply within 0.03 seconds. They can be reset manually and also have a test button.

Electrosurgery (diathermy) involves use of high frequency AC current, and the voltage is usually in the <1000V range. In monopolar diathermy, the active handpiece creates an intentional/controlled burning of tissue by concentrating the current at a narrow point. The circuit is completed by the dispersive pad which must contact a wide, well-conducting part of the patient. If the contact is narrow, then a burn injury may also result at this point. Another route by which a burn injury can inadvertently occur is by the creation of alternative circuits to ground, for example through a surgical assistant with a holed glove.

DC circuits: Low voltage direct current is used in car batteries, the electroplating industry, electrolyte purification and electrocautery (current passes through a wire to heat it and it is the wire that contacts the tissues). Rooftop solar panels generate direct current and are becoming increasingly commonplace, also potentially posing a risk.

High Voltage (>1,000 V)

AC circuits: While high voltage is defined as greater than 1,000 volts, voltages in the usual settings are around 11,000–33,000 volts. These levels are commonly seen in high tension electricity transmission cables; voltage levels may be even higher in electricity distribution power stations and substations. Voltage levels of 25,000 volts AC are now seen in many new railway systems.

DC circuits: Many existing urban railway electrification systems throughout the world use DC voltages ranging from 1,500–3,000 volts. High voltage direct current (HVDC) systems are also used for electricity transmission throughout the world (including Europe, Asia, South America, Australia, New Zealand). Voltage levels range from 100,000 to 800,000 volts. Such systems are used for transmission of electricity over very long distances (100–1,000s of kilometres) and not for general urban use. High voltage direct current is becoming increasingly common with electric cars, especially at high-voltage DC 'super-charging' stations.

Lightning is an ultra-short (around 10–30 microseconds) DC electrical discharge that involves extremely high voltages (tens to hundreds of millions of volts) and large amounts of current flow (>200 000 amperes). Temperatures in the surrounding air rise rapidly to extremely high levels (c. 30,000°C). Lightning also produces shock waves that may cause trauma to the victim.

Pathophysiology (4–16)

The damage to the body from electrical injury is caused by the effect of electrical current on different body tissue. The most common and obvious effect comes from the conversion of electrical energy to thermal energy producing burn injuries. The current can also have direct effects on certain tissues including cardiac arrhythmias, asystole, central apnoea and peripheral tetany (limbs, respiratory muscles). Disruption of cell membranes from the passage of current without heat generation is called electroporation. Electroporation can lead to cell death and tissue necrosis. How much this effect contributes to the overall injury is currently not known. Exposure to electricity can also involve associated trauma. Overall, the degree of injury from exposure to electricity depends on:

1. Type of current
2. Amount and duration of current flow
3. Voltage (determines current flow)
4. Resistance (determines current flow)
5. Duration of contact
6. Pathway of current flow

1. Type of Current

Exposure to DC usually causes a strong muscular contraction that may throw the patient back from the source of electricity; therefore limiting the duration of exposure and minimising electrical injuries.

By contrast AC exposure typically causes muscle spasm or tetany and prevents the patient from letting go from the source of electrical discharge. This will prolong the duration of exposure and increase the risk of electrical injuries.

2. Amount and Duration of Current Flow

In general, the extent of an electrical injury depends on the intensity of the electric current (i.e. the amount of current flow). As seen above from Joule's Law, current flow is a major determinant of heat generation in tissues, and the longer the duration, the greater the damage.

3. Voltage

Low voltage circuits (e.g. 240 volts) in the home can carry 10–15 amperes of current. Higher voltage circuits (e.g. 415 volts) can carry 40+ amperes. Current flow in other high voltage circuits can reach 1,000 A (high tension lines) or >12,000 A (lightning). High voltage electricity can also discharge through the air; 1,000 volts will only jump a few

millimetres, 5,000 volts will bridge 1 cm, and 40,000 volts, 13 cm. Injury can therefore occur without direct contact with the source.

4. Resistance

As previously stated, as the resistance of a substance/tissue increases so does the heat generated from the passage of current through the substance/tissue. In the human body resistance varies in the different tissues. This is partly due to their natural properties (e.g. nerves) and partly due to their fluid and electrolyte content.

The average resistance of the interior of the body is around 500-1,000 Ω. In order of **increasing** resistance, the resistance of various tissues is as follows:

- blood, body fluids (high water content) – least resistance
- nerves (natural conductors)
- muscle
- dry skin
- tendons
- bone – highest resistance

Skin presents an external barrier/resistance to the passage of current which depends on:

- **the thickness of the skin**: dry calloused skin in adults has a resistance of around 100,000 Ω; normal skin may have a resistance of around 40,000 Ω; the thin skin of a small child will have considerably less resistance
- **the moistness of the skin**: wet skin (sweat, saliva, water) may reduce the resistance to 1000 Ω or less; skin immersed in water (e.g. bath) or a moist mucous membrane offers almost no resistance at all

As the affected skin blisters and deteriorates the resistance to the passage of current is greatly reduced and more extensive damage occurs.

5. Duration of Contact

The longer the duration of exposure to the passage of current at all voltage levels the greater the degree of tissue damage.

6. Pathway

The pathway of the current also determines the type and severity of injury. Contact point wounds usually show evidence of significant skin damage with full thickness burns often seen. The most common contact points are the hands, wrists and heels. However, children often present with oral burns from contact with electric cords or power points.

Vertical pathways through the body can involve all vital organs including the nervous system, heart and lungs. Passage of current horizontally from arm to arm may affect the heart, lungs and spinal cord. Passage of current through lower parts of the current (e.g. straddle pathway) may cause significant local damage.

Electrical Burn Injury Patterns (2,4–7,9–19)

Low Voltage DC

Significant current can flow from car batteries, particularly where metal objects are in contact with the skin. Temperatures of 1,000°C or more can develop in metal objects such as rings, watches and tools. Local contact injuries range from superficial to full-thickness burns.

Low Voltage AC

If the current passes through the thorax, cardiac arrhythmias or cardiac arrest may occur without tissue damage (superficial or deep). There may also be tetany of the respiratory muscles causing apnoea. If there is tetany that prevents the patient from letting go of the current source there may significant local contact point wounds. These wounds are due to the concentration of current flow over a small area for a prolonged period of time.

High Voltage (DC+AC)

Exposure to high voltage current can cause injury in several ways:

- passage of current through contact with the electrical source or where a high-voltage arc reaches the patient
- flash burns from a high-voltage arc travelling through the air without directly contacting the patient
- clothes can be ignited causing typical flame burns
- associated trauma (e.g. the patient is thrown from the source with DC)

High voltage current transmission can result in both cutaneous and deep tissue injury. Contact points are always full thickness. Arcing can also occur when the current jumps from skin surface to skin surface (typically across flexed areas of the body).

Any deep tissues in the path of the current may be injured. This can involve internal organs but commonly affects the limbs and other muscular areas. The direct damage to deep tissues such as muscle and bone can lead to significant hidden injuries out of proportion to the cutaneous injury seen in the contact point wounds. There may be damage to the blood vessels leading to vascular compromise and further secondary tissue ischaemia and necrosis (in particular muscle). Deep tissue injury is commonly manifested in signs such as haemochromogenuria and compartment syndrome. Management requires both additional fluid resuscitation to preserve renal function and fasciotomy to minimise pressure necrosis of swelling muscle beneath unyielding fascia.

When electricity is conducted through high resistance tissues such as tendon and bone a substantial amount of heat may be generated. The increase in bone temperature continues even after the current flow has ceased causing secondary thermal damage and, due to the depth of the bone, heat escape is slow. There may be osteonecrosis and tissues close to the heated bone (e.g. periosteum, muscle, blood vessels and nerves) may sustain considerable damage.

Other injuries include a range of neurological complications including spinal cord injury and delayed brain atrophy.

High voltage injuries are associated with an increased length of stay, more complications and more surgical procedures (fasciotomy, amputation).

Lightning

Lightning is a form of DC discharge characterised by extremely high voltage and current delivered over a short duration. Variable patterns of injury may occur.

The mortality rate for lightning injuries is around 5–10% which is significantly higher than that of other electrical injuries (4). Survivors may have permanent disabilities (5).

The mechanism of cardiopulmonary arrest from lightning can be primary asystole and/or central apnoea causing secondary cardiac arrest. These situations may be responsive to prolonged cardiopulmonary resuscitation.

Survivors of lightning strikes typically do not have deep cutaneous burn wounds, deep tissue injuries (muscle) or haemochromogenuria. Cutaneous burns are seen in 90% of lightning injuries but only around 5% are deep burns. Similar to other high voltage injuries, if clothes are ignited there may be additional flame burns.

Acute and prolonged neurological complications are common. These range from altered mental state, loss of consciousness and seizures to paralysis, peripheral neuropathies and autonomic dysfunction.

Lightning may also be responsible for an unusual pattern on the skin which has an arborescent or 'splashed on' appearance like a fractal pattern. Known as Lichtenberg figures, they are considered pathognomonic of a lightning strike but are uncommonly seen and typically resolve over 24 hours.

Transient vasospasm can produce a pulseless, cold, blue limb similar to that seen with other high voltage electrical injuries or compartment syndrome that resolves within a few hours.

Associated blunt trauma can result from the patient being thrown. Barotrauma can result in ruptured ear drums and contused internal organs e.g. lungs, GIT. Cataracts from corneal damage and other ocular injuries can also occur.

Voltage	Skin	Deep tissue	Cardiac arrhythmias
Low voltage (<1000 V)	Contact point wounds	Mostly at site of contact point wounds	Immediate cardiac arrest or transient ECG changes possible
High voltage (>1000 V)	Full-thickness contact point wounds Flashover burn	Extensive muscle damage Rhabdomyolysis Compartment syndrome	Transthoracic current may cause myocardial damage and delayed arrhythmias
Lightning	Contact point wounds Superficial or dermal flashover burns ('Lichtenberg figures')	Eardrum perforation and corneal damage	Respiratory arrest – needs prolonged CPR

TABLE 9.2 – Overview of electrical injuries

Management (4,5,8,10–12,17–23)

General approach

Safety is paramount. No attempt should be made to provide medical care until the source of the current has been turned off or the patient has been safely moved away from the source. Once clear of the power source the primary survey begins as with any burn injury. Follow the EMSB approach to care using the primary survey. Any patient with a significant electrical injury has a mechanism for significant trauma. Maintain a high index of suspicion for secondary or concealed traumatic injuries associated with the electrical injury such as blast/barotrauma, falls or cervical spine injury.

If the patient is in cardiopulmonary arrest, follow local protocols. Prolonged resuscitation is recommended due to documented survival in a number of these patients. Asystole has different implications in these settings and outcomes are not as bad as expected as for other causes of asystole. Follow the initial EMSB approach with a thorough secondary survey including the history of the event.

Specific situations

Low voltage AC injury

- Management of the resultant wound will depend on the site and depth rather than the mechanism of injury.
- ECG monitoring is required only if there has been a history of cardiac arrest, loss of consciousness, ECG abnormalities on admission, or pre-existing heart disease; otherwise the patient can be discharged (assuming the wound is not an issue).
- There may be long-term physical and psychological sequelae.

High voltage AC/DC injury

- Cutaneous burn wounds should be treated as for any other cutaneous burn. There may be unseen deep tissue injuries particularly in the limbs involving bone, muscle and other tissue. There may be non-viable tissue beneath viable-looking muscle necessitating thorough exploration at any surgical procedure; fasciotomy is commonly required.
- Examine and re-evaluate the patient as per the secondary survey for dysfunction in any of the organ systems; there may be delayed onset of complications, especially neurological.
- ECG monitoring is required.
- Intravenous fluid is mandated in a high voltage electrical injury irrespective of cutaneous burn size. A urinary catheter is essential to monitor adequacy of fluid resuscitation and to identify the presence of haemochromogens which is associated with rhabdomyolysis. The aim is to maintain urine output at 1-2 mL / kg / hr in adults, and > 2.0 mL / kg / hr in children (see Chapter 6 for further discussion).
- There may be long-term physical and psychological complications.

Lightning

- Cutaneous burn wounds are treated as any other cutaneous burn; deep burns and muscle damage are uncommon.
- Current evidence suggests the need for ECG monitoring in this situation.
- Patients can present with fixed dilated pupils in the absence of irreversible brain injury; this should be kept in mind when making decisions on providing resuscitation. There may be a transient paralysis present that is usually associated with widespread vasospasm; both usually resolve. There may be delayed onset of neurological complications. Neurological injury causes the greatest number of long-term problems for survivors.

	Low voltage	High voltage	Lightning
Wound	Depends on site and depth Dressing as appropriate	Monitor capillary refill Dressing as appropriate	Depends on site and depth Dressing as appropriate
ECG monitoring	If history of cardiac arrest, abnormal ECG on arrival or cardiac PMHx	Required	Required
I.V. Fluid	Should not be required	I.V. fluid required Monitor for haemochromogenuria and adjust to maintain urine output	Should not be required
Long term	May be physical and psychological effects	May be physical and psychological effects	May be physical and psychological effects

TABLE 9.3 – Overview of management of electrical injuries

Summary

- *Avoid injury to those rendering assistance.*
- *Treat cardiac and respiratory arrest.*
- *Assess and manage associated trauma.*
- *ECG monitoring (24 hours) is required for all high voltage electrical injuries. It is also required if there has been loss of consciousness or an abnormal ECG on arrival in low voltage injuries.*
- *Patterns of injury are specific to low voltage, high voltage and lightning strike.*
- *Standard burn resuscitation formula may be inadequate.*
- *Haemochromogenuria is common in high voltage injury and requires insertion of a urinary catheter and maintenance of a urine output of 1–2 mL / kg / hr in adults, or >2 mL / kg / hr in children, until urine clears.*
- *High voltage injury involving limbs may require fasciotomy.*
- *All electrical burns should be discussed with a Burn Service regarding referral and transfer.*

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CHAPTER 10

Cutaneous Chemical Burn Injuries

Introduction

There is a widespread potential for exposure to dangerous chemicals. There are over 40,000 chemicals listed on the public Australian Inventory of Chemical Substances (1).

Chemicals may cause skin injuries, ocular burns and systemic effects. Chemical burns are commonly smaller in size compared to thermal injuries, but they are more likely to involve deeper injuries.

The challenge for burn clinicians in treating chemical injuries include:

- the wide variety of chemical agents
- inexperience in treating chemical injuries due to the low frequency of clinician exposure to patients with chemical injuries
- and the potential for ongoing tissue destruction (2).

One main difference between thermal and chemical burns is the continuation of the injury process with chemical burns. Chemical burns need referral (and possible transfer) to a Burn Service.

Epidemiology

Chemical burns are relatively uncommon, accounting for 6% of all burn injuries in adults in Australia and New Zealand and 2% of all burn injuries in children (3). The areas more commonly affected tend to include the face, eyes and extremities (2,4,5).

Classification of Chemicals

Chemicals can be classified according to the chemical reaction that occurs (2,6). The following is one approach to classifying chemicals that cause cutaneous injuries:

Acids

Acids are proton (H^+) donors; examples include hydrofluoric acid (HFA), hydrochloric acid, sulphuric acid, chloric acid, formic acid and phenols.

Bases

Bases are proton (H^+) acceptors; and alkali are bases that dissolve in water. Examples include sodium hydroxide, potassium hydroxide, calcium oxide and ammonia.

Although there are strict differences between bases and alkalis the terms are often used interchangeably. For the purposes of this course we will use the term alkali only.

Organic Hydrocarbons

Organic hydrocarbons are compounds containing only hydrogen and carbon atoms; examples include petrol and bitumen.

Elemental Compounds

These include elemental sodium, potassium and lithium. Accidental exposure to these compounds is usually seen in the laboratory however release of lithium may come from damaged or exploding lithium-ion batteries.

Miscellaneous

Vesicants (blistering agents), desiccants (tissue dehydrating agents) e.g. sulphuric and concentrated hydrochloric acid-muriatic acid.

Table 11.1 lists some common chemical compounds and where they may be found in products in industry and the household.

ACIDS	
Hydrofluoric acid (HFA)	Rust removers, cleaning agent for stone, marble, alloy wheels, glass etching Military automatic fire suppression systems Li-ion batteries damage/explosion
Hydrochloric acid	Toilet bowl cleaners Dye manufacturing Swimming pool cleaners
Sulphuric acid	Toilet bowl cleaners Drain and metal cleaners Car batteries Munitions and fertiliser manufacturing
Phenols	Deodorants Sanitisers Disinfectants Production of resin adhesives
Cresols	Degreasing agents

ALKALIS	
Sodium hydroxide Potassium hydroxide	Drain cleaners Oven cleaners
Sodium hypochlorite Calcium hypochlorite	Disinfectants Household bleach Pool chlorinating solutions Deodorants
Calcium oxide	Cement

TABLE 10.1 – some common chemical compounds

Pathophysiology (2,4,6–8)

The extent of damage from chemicals is dependent on several factors:

- type of chemical (e.g. acid, alkali)
- concentration
- strength. The strength of an acid or alkali refers to how readily the chemical donates protons (H^+) making it an acid, or how avidly it accepts protons (OH^-) making it a base. The pH scale is a measure of the strength of an acid or a base in aqueous solution from 1 (a strong acid) to 14 (a strong base) and is related to how the chemical dissociates in water. A pH of 7 is considered 'neutral'. In general, the greater the deviation from a neutral pH of 7, the greater the strength of the acid or alkali and the greater the potential for tissue damage but this is not always true. HFA is a weak acid but highly corrosive.

  The *term* 'strong' or 'weak' refers only to the degree of ionisation (pH). *It does not refer to the concentration of the acid or alkali.* It is possible to have a concentrated solution of a weak acid, and a dilute solution of a strong acid.
- volume involved
- duration of contact
- location: ocular, thin skin, inhalation, ingestion
- mechanism of action.

In addition to tissue damage some chemicals can also cause injury through exothermic (heat producing) reactions and through systemic toxicity.

Mechanism of Action

Acids

Typically cause coagulation necrosis of superficial tissues (pH <2).

Alkalis

Typically cause liquefactive necrosis (pH >11.5). Liquefactive necrosis allows deeper penetration of the chemical leading to ongoing injury even after removal of the initial insult. Alkalis also cause dehydration of cells, dissolution of cell membranes and the chemical reactions produce heat (exothermic) further worsening the injury.

Organic Compounds

Act by dissolving lipid membranes of cells and through denaturation of proteins.

Elemental Compounds

React with water (sweat, mucous membranes etc.) to form strong alkali which then cause severe alkali burns.

General Management of Cutaneous Chemical Burns

As with all trauma patients, the initial assessment and management should follow the EMSB approach with particular reference to airway, breathing and circulation (2,5,6). However, some points relevant to patients with chemical burns include (6,7):

- remove the patient from danger, protecting self at the same time
- ensure appropriate personal protection equipment (PPE) is used by clinicians, other relevant staff and relatives if appropriate (from gloves, gowns, goggles to full encapsulation with self-contained breathing apparatus (SCBA))
- consider the potential for ocular injuries, inhalation injuries and systemic toxicity
- removal of residual chemicals is a first aid priority (equivalent to 'stopping the burning process'). As long as chemicals remain in contact with the tissues there is the potential for ongoing tissue damage.

As stated above, the duration of the chemical contact with the tissues is a major determinant of injury severity.

First aid treatment begins with removal of the patient from the situation of exposure, removal of all clothing and jewellery and brushing off any dry chemicals from the patient avoiding spread to unaffected areas.

The clothing, jewellery and any chemicals should be treated as hazardous material and handled appropriately.

Water Irrigation

The mainstay of the treatment of chemical burns, including ocular burns, is copious water irrigation for at least 30 minutes to 2 hours (or longer for alkali burns). Water irrigation acts to both wash away and dilute the chemical (5,6,8). Irrigation should begin as soon as possible after exposure (2,4,6). Assessment of the effectiveness may be measured using litmus paper to maintain the pH balance between 5–11. Run-off from the irrigating water and chemicals should not be allowed to flow across unaffected skin or structures (e.g. eyes). Similarly patients should not be placed in a bath as the chemicals may be spread to unaffected areas. Care should be taken to avoid hypothermia, it is reasonable to use slightly warm water for irrigation, the cooling effect is unwanted (4).

There are circumstances where irrigation with water is contraindicated. These situations include exposure to elemental sodium, elemental potassium, elemental lithium, phenols and dry lime. Areas affected by elemental compounds should be covered with mineral oil and any fragments removed with forceps and placed in mineral oil (7). Sulphuric acid and concentrated hydrochloric acid (muriatic acid) may theoretically liberate heat when irrigated with water but current literature suggests that the best approach is early decontamination with copious water irrigation (7).

Neutralisation, where an acid is neutralised by a weak alkali or an alkali is neutralised by a weak acid, is theoretically possible. However several issues prevent neutralisation from being a widespread treatment option (6). These include:

- determining which neutralising agent to use

- delay in decontamination while determining and locating the neutralising agent
- the neutralising agent itself may cause injury
- difficulty matching pattern of injury and dispensing neutralising agent
- excessive use of the neutralising agent may push the patient past neutral or near neutral pH measurements
- the chemical reaction of the neutralisation may be exothermic (heat producing).

A chelating agent, Diphoterine®, has been used for both acid and alkali burns and other chemicals as it has binding sites for both hydrogen ions, hydroxide ions and other compounds (4,6).

Specialist advice for common and rare mechanisms should be sought from relevant organisations. Ocular and inhalation injuries require further referral for specialist management.

Specific Agents

Hydrofluoric Acid (2,6,7,11–23)

Hydrofluoric acid (HF) is a highly corrosive inorganic acid of elemental fluorine. HF is widely used in glass etching, the electronics industry and cleaning solutions for metals, stone and marble. HF is available in solutions from <1% up to 70%. Concentrations above 40% may fume when containers are opened. Even dilute solutions can rapidly penetrate the skin and exposure of even small areas of the body to HF can be fatal (e.g. 2% TBSA).

Initial superficial skin damage removes the normal protective barrier and allows deeper penetration of the chemical where toxic and reactive fluoride ions are released (as well as hydrogen ions). These fluoride ions cause extensive *liquefactive* necrosis and osteolysis, which is different from the usual pattern of necrosis seen in acid injuries. The fluoride ions also binds with calcium ions and magnesium ions in the tissues which can cause significant hypocalcaemia and hypomagnesaemia. Hyperkalaemia may also be seen. These electrolyte disturbances along with a direct cardiotoxic effect of fluoride ions can lead to cardiac arrhythmia that are difficult to treat.

High concentrations of HF (>15–50%) cause immediate pain (out of proportion to physical findings) and tissue damage. Lesser concentrations may cause erythema, swelling and discomfort. Exposure to weak solutions may take hours to produce symptoms but can still cause severe injury. The majority of HF injuries involve the hands and low concentrations of 1–3%. Inhalation and ocular injuries can also occur.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.
- Remove all clothing and jewellery (handle appropriately).

- Immediate copious water irrigation for at least 30 minutes to 2 hours. This will not only wash away the chemical but dilute the concentration to less dangerous levels. Inactivation of the fluoride ion (treatment continued until the patient is pain free)
    - topical application of 2.5% calcium gluconate gel; rubbed in by the clinician wearing double gloves. An injured hand can be placed in a glove containing the gel. Reapply initially every 30 minutes until resolution of pain, then every 4 hours
    - subcutaneous injection of 10% calcium gluconate (0.1–0.2 mL via 25g needle); seek expert advice
    - intravascular administration of calcium gluconate; intraarterial/intravenous (Bier's block) using calcium gluconate; seek expert advice
    - do not use calcium chloride as it can be an irritant
    - local anaesthetic in either a ring block or regional block will provide pain relief, but does not treat the injury itself and should be avoided.
- Resolution of pain with the application of calcium indicates that the fluoride ion has been neutralised. If pain returns then more calcium is indicated.
- Excision of contaminated areas is sometimes necessary (and may be lifesaving in severe cases). This may include removal of fingernails to allow application of the calcium gluconate gel (HF rapidly penetrates fingernails) with short-acting local anaesthesia.
- The systemic effects need to be monitored by repeat electrolyte measurements and ECG monitoring. Intravenous administration of calcium and/or magnesium may be necessary as well as treatment of hyperkalaemia.

Cement Burns (2,4–7,24–27)

Cement is a dry powder containing various calcium oxides (calcium oxide is also known as lime). When dry cement combines with water or sweat it forms wet cement and releases calcium hydroxide which is a very corrosive chemical. The reaction is also exothermic.

Wet cement contains around 65% calcium oxide by weight. It has a pH of 10–12 which rapidly rises to >12 as the chemical reaction proceeds. Dry cement is very hygroscopic (highly water absorbent) and may cause injury through desiccation of tissues.

The chemical injuries from cement burns are typically found in the lower limbs. The surface injuries produced include erythema, oedema and blistering. Cement is also abrasive and coupled with rubbing from clothing and footwear causes minor skin damage that allows deeper penetration of the chemical. Early symptoms include mild irritation and there may be little or no pain. Presentation is often delayed (sometimes days later) resulting in significant morbidity from deep/full thickness burns requiring excision and grafting. Inhalation injuries from breathing in dry cement dust can occur. Ocular injuries can arise from exposure to both dry and wet cement. Contact dermatitis may be seen due to exposure to the chromate and cobalt in the cement.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.

- Remove all clothing and jewellery (handle appropriately).
- Brush off any residual dry chemical.
- Copious water irrigation should be initiated as soon as possible and continue for minimum of 30 minutes to 2 hours, depending on patient's pain level.

Caustic Soda (sodium hydroxide) (7,28,29)

Caustic soda is another example of a strong alkali. It is commonly found in household cleaners and drain cleaners. In household exposure the injury is usually small but in the industrial setting the injury may be life threatening due to the large %TBSA involvement. In household exposure first aid is often inadequate and presentation delayed.

Systemic effects are not seen but ocular involvement can be devastating due to the rapid corneal penetration. Caustic soda exposure can involve ingestion and inhalation of the chemical. Inhalation of sodium hydroxide dust, mist, or vapour may cause irritation of the mucous membranes of the nose, throat, and respiratory tract. The severity of damage depends on the concentration of the chemical and the volume involved.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.
- Remove all clothing and jewellery (handle appropriately).
- Brush off any residual dry chemical.
- Copious water irrigation should be initiated as soon as possible and continue for minimum of 30 minutes to 2 hours, depending on patient's pain level.

Petrol (2,7,8,10,30)

Petrol is a mixture of hydrocarbons. Following prolonged contact with skin the hydrocarbons dissolve the lipids in cell membranes resulting in skin necrosis and allowing absorption of more hydrocarbons.

Most injuries are dermal thickness in nature but if the contact is longer than 2 hours there may be deep/full-thickness burns. Apart from petrol flame burns (more easily identified) burns caused by dousing in petrol or contact with liquid petrol, commonly seen in motor vehicles, may be overlooked.

Systemic toxicity can be seen 6–24 hours after exposure. Systemic toxicity (more often seen after ingestion or inhalation) can affect the lungs, kidneys, liver, nervous system, heart and the immune system. Seek specialist care for system toxicity.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.

- Remove all clothing and jewellery (handle appropriately).
- Copious water irrigation for minimum of 30 minutes to 2 hours, depending on patient's pain level.

Bitumen (2,7,31–35)

Bitumen is a petroleum product obtained during the refining of crude oil. Bitumen is a complex mixture of hydrocarbons and elements such as nickel, lead, chromium and mercury. It is a dark-brown/ black viscous liquid. It is used to bind sand and gravel aggregates to form asphalt which is commonly used as road paving material.

To remain in liquid state it needs to be kept above 100–150°C. It is often kept at higher temperatures to facilitate spraying of the compound. Therefore, a bitumen burn causes both chemical and thermal injuries with the thermal component often being the most significant.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.
- Remove all clothing and jewellery (handle appropriately).
- Copious water irrigation to address the thermal component (until the bitumen has cooled).
- Removal of the bitumen:
  - use a solvent product to 'dissolve' the bitumen;
  - products that can be used include… petroleum jelly
    vegetable oil, paraffin oil, baby oil
    De-Solv It™
  - do not use liquid solvents like alcohol, acetone, kerosene or petrol as they may have a toxic effect on the skin and are not as effective
  - cover the involved areas with the product, cover with a dressing and change appropriately (usually every 4 hours). Sometimes removal of the bitumen may be achieved within 1–2 hours.

Note: circumferential bitumen contact may cause impairment of circulation and longitudinal splitting of the 'cast' may be necessary.

Cutaneous Chemical Burns in the Military Setting

White Phosphorous (7,36–41)

White phosphorous is a waxy translucent compound and is commonly used in the manufacture of smoke, tracer, illuminatory and incendiary munitions (in particular, hand grenades, mortars and other artillery rounds). It is also used in the manufacture of fertilisers and fireworks.

White phosphorous burns fiercely in the presence of air and is quickly oxidised to phosphorous pentoxide. The auto-ignition temperature (temperature at which combustion can occur in the absence of an ignition source) is 30^{o}C. In this very exothermic reaction the white phosphorous bursts into a yellow flame and produces a dense white smoke containing phosphorous pentoxide. Phosphorous pentoxide when it combines with water in the tissues produces phosphoric acid which is also corrosive to the tissue.

Particles of white phosphorus in contact with skin continue to burn until the source of oxygen is removed, the particles burn out or they are removed. White smoke issuing from a wound should alert the clinician to the possible presence of white phosphorous particles.

Clothing may ignite from the burning white phosphorus. The majority of injuries from white phosphorous munitions explosions are due to ignition of clothing and are managed as per flame burns. White phosphorous thus causes both chemical and thermal cutaneous injuries.

Phosphorous may also be absorbed through the skin and injured tissues and produce systemic toxicity. This may be fatal even when only small areas are involved (<10% TBSA). Systemic effects that can be seen include:

- hypocalcaemia and hyperphosphataemia which may cause ECG abnormalities including ventricular arrhythmias
- hepatotoxicity
- nephrotoxicity

The dense white smoke is irritant to mucosal surfaces where in combination with water it becomes phosphoric acid. An inhalation injury can occur in white phosphorous burns especially if the explosion occurs in an enclosed space.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE (note the potential hazard from white phosphorous smoke inhalation).
- Primary Survey.
- Remove all clothing and jewellery (handle appropriately).
- Brush off any particles. Be aware that the particles are still potentially hazardous and should be placed under water.
- Copious water irrigation; keep particles wet (wet pads etc.) especially during transport. Be mindful of the potential for hypothermia. This stops combustion by removal of the O_2 source, cools the burn wound and dilutes the phosphoric acid.
- Remove particles as soon as possible; it is possible to use a Wood's lamp to fluoresce the particles; do not use copper sulphate as it has not been proven to be better than water irrigation and it may be harmful due to the systemic absorption of copper.
- Debridement of injured tissue (including white phosphorous particles) may be required.

Vesicants (7,33,42–45)

Vesicants, or blistering agents, can cause injury to the skin, the eyes and the airways. Vesicant compounds include mustard (sulphur mustard), Lewisite and phosgene oxide. Lewisite and phosgene oxide have probably not been used on the battlefield.

Mustard remains the main concern in the military setting because it is both incapacitating and lethal, it is easy to manufacture and there are known large stockpiles. Mustard may act as an alkylating agent that denatures DNA or interact with glutathione affecting cell membranes leading to cell death and liquefactive necrosis. It is a liquid at room temperature but becomes a vapour as the temperatures rises (>37.7°C).

There can be a delay from the time of exposure to the onset of symptoms from 2–12 hours depending on the dose. However, within minutes the mustard becomes fixed to the tissues (10%) or is absorbed into the circulation (90%). Because of this fixation, fluid in the blisters that develop do not contain mustard. Thus, decontamination should start immediately after exposure.

Defence force personnel may also be exposed to mustard in sea operations from both explosions and contamination in the water as the mustard will float on top of the water. The skin injuries range from erythema to blistering. Skin injuries are more prevalent in warm, humid areas of the body. Eye injuries range from conjunctivitis to corneal damage leading to temporary or permanent loss of vision. Inhalation injuries affecting the airways can be significant and severe exposure usually results in damage to the lower airways. Inhalation injuries account for most of the deaths from mustard exposure.

Systemic toxicity, which is usually seen with very high dose exposure, can result in bone marrow suppression, nausea, vomiting and diarrhoea, and CNS effects such as apathy, depression, abnormal muscular movements, agitation and coma.

Management

- Remove the patient from danger, protecting self at the same time.
- Staff should don appropriate PPE.
- Primary Survey.
- Remove clothing and jewellery (handle appropriately).
- Water irrigation – immediately after exposure.
- Inhalation and ocular injuries need referral to specialist care, steroid antibiotic ointment may be used for ocular injuries.

Summary

- *Agents capable of causing chemical burns are commonplace.*
- *Most chemical burns require copious irrigation with water.*
- *Hydrofluoric acid burns require treatment with calcium gluconate.*
- *Systemic toxicity is possible after exposure to hydrofluoric acid or petrol.*
- *Alkali burns require irrigation with water for an even longer period than other chemical burns.*
- *Chemical injuries to the eye also require copious irrigation, and then referral.*

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Chapter 11

Frostbite and Hypothermia

Introduction

Cold injuries may be divided into two groups: (1) peripheral cold injury and (2) systemic hypothermia. In Australia and New Zealand, localised peripheral cold injuries are relatively rare. When they do occur these patients are typically managed by a Burn Service, particularly if there are multiple extremities involved. Systemic hypothermia, however, is a common and often missed accompaniment to trauma, especially in severe burn injuries. The presence of hypothermia in these situations can lead to serious complications, morbidity and increase mortality (1,2).

1.Peripheral Cold Injury

There are three types of peripheral cold injury: (1) frostnip, (2) tissue-freezing injury (commonly known as frostbite) and (3) non tissue-freezing injury, see Table 11.1. (3–9)

Frostnip	Mild, completely reversible, cold injury characterised by skin pallor and numbness
Tissue-freezing injury ('frostbite')	Below 0.5°C; tissue freezes resulting in formation of intracellular ice crystals and microvascular occlusion
Non tissue-freezing injury	Results from chronic exposure to high humidity and low temperatures, without tissue freezing (also known as chilblains or perniosis). Trench foot is caused by chronic exposure to damp environment at temperatures 1–10°C

TABLE 11.1 – The three types of peripheral cold injury

Frostbite

Frostbite injuries have been well documented over the past 200 years mostly afflicting military personnel. In recent times an increasing number of civilian cases have been described especially in populations exposed to cold winters and also amongst mountaineers. Frostbite often occurs in otherwise healthy adults and presents a significant clinical challenge.

Historically frostbite injuries have traditionally been classified by **degree**, with a first degree injury affecting the epidermis, second and third degree injuries associated with blistering (containing clear and haemorrhagic fluid, respectively) and a fourth degree injury penetrating the deeper structures including muscles, tendons, and bone, with resultant tissue loss (10). A more useful classification for predicting prognosis and risk of amputation assesses severity of frostbite according to clinical signs of distal ischaemia (pallor/cyanosis,

altered sensation, cold digits and delayed capillary refill) (11).

The distal extremities are most commonly affected (in 90% of cases), but the shins, cheeks, nose, ears, and corneas may also be injured (6,9). The severity is related to the environmental temperature and more importantly to the duration of exposure.

Frostbite is rarely a result of extreme weather conditions alone. **Risk factors** include environmental and behavioural factors (homelessness, sporting or military activities); substance misuse, especially alcohol; psychiatric illness; peripheral vascular disease; drugs (prescribed and illicit); and trauma. It is most commonly seen in adult men (4–9,12,13).

Liquefied Petroleum Gas (LPG) has a low boiling point and is stored in a pressurised, cooled liquid form. Exposure to the skin can result in severe cold burns similar to frostbite due to the rapid drop in temperature.

Initial Management of Frostbite

All potentially severe cases of frostbite should be discussed with a Burn Service. Any other life-threatening or concomitant traumatic injuries should be evaluated and prioritised according to the **primary survey**.

Hypothermia, if present, should be appropriately managed. The standard acute treatment for all degrees of frostbite consists of rapid rewarming in water heated to 37–40°C with a mild antibacterial agent (povidone-iodine or chlorhexidine) added, for at least 30 minutes until complete thawing (14). This should only be performed once there is no further risk of refreezing (6–10).

Intravenous opioid analgesia should be administered as required for pain. Massage of the affected area is contraindicated as it may cause mechanical trauma (6). Fluid resuscitation is not needed unless the patient is clinically dehydrated.

Post-rewarming Management (3,6–10, 25)

- Debridement of clear blisters to prevent thromboxane-mediated tissue injury. High concentrations of prostaglandin F2α and thromboxane B2—which may cause platelet aggregation, thrombosis, and ischaemia—have been found in fluid from frostbite blisters.
- Haemorrhagic blisters imply a deeper injury and although some still advocate these should be left intact, the current expert consensus is to debride all frostbite blisters.
- Elevation of affected extremity to reduce oedema.
- Prophylactic oral penicillin is recommended to prevent wound infection because of the inactivation of the normal skin streptococcicidal properties by local tissue oedema.
- Advise patients to cease smoking to avoid further reduction in distal perfusion.
- Application of topical aloe vera (thromboxane inhibitor) to debrided blisters every six hours is recommended by many centres.
- Systemic antiprostaglandins (regular ibuprofen). Aspirin is a less selective antiprostaglandin and is not generally recommended. However, in severe cases of frostbite with confirmed distal thrombosis, aspirin is preferable due to its more potent platelet inhibitory effects.

- Prophylactic tetanus toxoid when indicated according to standard immunisation schedules:
  - Tetanus cover
    - Australia: https://immunisationhandbook.health.gov.au
    - New Zealand: https://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/new-zealand-immunisation-schedule
- Early mobilisation of frostbitten digits (fingers, thumbs and toes) is important to preserve function.

Adjuvant therapy

Various adjuvant therapies have been proposed in recent years. Many of these are limited to case reports or animal studies and convincing evidence for their use is lacking. These include: hyperbaric oxygen therapy (14,15), surgical or chemical sympathectomy (8,9) and Pentoxifylline, a phophodiesterase inhibitor (6,7,9).

Another vasodilatory drug, **iloprost** (synthetic prostacyclin analogue) has been used with some success (5,16–18), but is not widely available. It is administered via an intravenous infusion for 6 hours duration and repeated over 2–3 days. It dilates systemic and pulmonary arterial vessels and also affects platelet function.

Thrombolysis

Only in the last 10 years has a major therapeutic advance been made in the early use of thrombolytic agents to reduce the incidence and morbidity associated with distal extremity tissue loss (3,4,5,11,12,18,19).

Recent reports have described the successful use of tissue plasminogen activator (tPA) in acute (<24 hours) cases of severe frostbite. An approximate combined 75% digital salvage rate has been reported (5,9,16,18,19). Immediate digital subtraction angiography should be carried out in cases of severe frostbite presenting within 24 hours of injury.

Severe frostbite refers to clinical signs of ischaemia involving one or more digits at (or proximal to) the proximal interphalangeal joint level (or interphalangeal joint level in the thumb). The use of a hand-held Doppler can aid diagnosis. If thrombosis is confirmed on angiography, tPA should be administered if there are no contraindications. It can be delivered intra-arterially or intravenously and requires monitoring on a high-dependency unit. This is likely to become the standard treatment in severe cases of frostbite.

Complications of frostbite

Short-term complications after rewarming include compartment syndrome, infection, and ischaemia of the distal extremities, with potential tissue necrosis and subsequent tissue loss. Long term sequelae include cold sensitivity, sensory loss, hyperhidrosis, chronic pain and loss of function associated with distal amputation (6,8,9).

The role of surgery in frostbite

Surgery is not usually indicated in the acute phase and should be delayed until the frostbitten area is thoroughly demarcated. However, fasciotomy may be indicated in cases of

compartment syndrome and early amputation in cases of sepsis.

2. Systemic Hypothermia

In some cases, systemic hypothermia may be more pronounced and present as a major issue in the management of a burn patient. This may result from environmental factors, overzealous cooling of the burn wound or from the inability to keep the patient warm during transportation.

Significant hypothermia can be classified into mild, moderate and severe (see Table 11.2). In all cases, rewarming (passive and active) should be instituted. Methods for rewarming are detailed in Table 11.2 (21–24).

If a patient suffers a cardiorespiratory arrest, the patient will need to be rewarmed to normothermia before resuscitation is declared futile (24). In practice, a successful outcome in patients who have moderate to severe hypothermia is very rare. The conditions necessary for successful outcome (rapid and profound cooling, usually in association with immersion in frigid waters) are typically absent in most presentations in Australia and New Zealand. Instead, inability to successfully warm a patient may of itself be an indicator of futility in resuscitation (23). Expert emergency or intensive care opinion should be sought in all such extreme cases.

Severity of Hypothermia	Temp range (°C)	Symptoms and signs	Treatment
Mild	32 – 35°C	• Tachycardia • Tachypnoea • Altered behaviour: impaired judgement • Amnesia, slurred speech • Uncontrollable shivering • Cold diuresis	• Prevent further heat loss (remove wet clothing) • Measure core temperature to determine severity* • External rewarming by blankets, warm air devices, radiant heat, warm room
Moderate	28 – 32°C	• Hypopnoea • Bradycardia, arrhythmias (< 30°C) • ECG J waves • Reduced cardiac output • CNS depression • Pupillary dilation and loss of light reflex • Hyporeflexia • Shivering ceases	• Warm air devices, radiant heat • Be prepared for vasodilatation and shock from surface rewarming • Heated IV fluids • Consider measures used in severe hypothermia if external rewarming inadequate
Severe	<28°C	• Apnoea, pulmonary oedema • Hypotension, VF, pulselessness • Marked myocardial depression • Coma, fixed pupils, areflexia • EEG activity decreased/absent	• Intubate and ventilate with warmed humidified oxygen • DC shock for VT arrest, consider amiodarone • Lavage (gastric, bladder, peritoneal, pleural) • Warm water bath immersion

TABLE 11.2 – Hypothermia classifications and treatment
*Rectal, oesophageal or bladder. Do NOT use tympanic thermometry. Beware false readings if rectal probe inserted into stool

Hypothermia in the Burn Patient

Systemic hypothermia frequently accompanies severe burns unless pre-emptive preventative measures, constant monitoring and proactive steps are taken. This is especially the case in young children, who have a higher surface area to body mass ratio, thinner skin and less effective thermoregulation (20). Hypothermia increases complications such as coagulopathy, cardiac arrhythmias and metabolic instability, which ultimately increase morbidity and mortality (1,2).

In the pre-hospital phase, cooling of the burn wound for first aid should be undertaken judiciously. Where possible, areas of the body not requiring cooling should remain covered. If possible, the temperature of the room in which the patient is undergoing first aid should

be kept warm. Once cooling has been completed, the affected areas should be covered (21,22).

After exposure for the primary survey, resuscitation of the patient with severe burn injuries should ideally be undertaken in a suitably warmed room. Keeping the patient covered with warm blankets and external warming devices should be employed. Resuscitation fluids should be also warmed.

Summary

- *Cold injuries can be divided into peripheral cold injuries and systemic hypothermia.*
- *Peripheral cold injuries can further be divided into three – frostnip, tissue-freezing injury (frostbite) and non tissue-freezing injury.*
- *Management of frostbite includes rapid rewarming with adequate analgesia. Thrombolysis represents a recent development in the management of frostbite. Surgical management of frostbite is delayed to allow for full demarcation and to allow maximal recovery of tissues.*
- *Systemic hypothermia can be extremely problematic with the burn patient. Rewarming, both active and passive, needs to be undertaken.*

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CHAPTER 12

Referral and Transfer

Introduction (1,2)

The patient with severe burn injuries requires immediate assessment and stabilisation, either at the scene of accident or at the nearest hospital. Once stabilised the patient will need transfer to a Burn Service.

Initial treating personnel should complete a primary and secondary survey and evaluate the patient for potential referral and transfer. In Australia and New Zealand expert multidisciplinary care is readily available at Burn Services and early contact is advised. Burn injuries may occur in conjunction with other traumatic injuries and the burn patient must be evaluated, assessed and managed with this in mind.

Documentation needs to accompany the patient to provide the receiving Burn Service with a record that includes detailed information about the burn injury (mechanism, size, depth, distribution) and past medical history (co-morbidities), observations including hourly fluid balance, medications, procedures and therapies (including completion of first aid).

Referral Criteria

Burn patients may present with burns of varying depth, size and mechanisms of injury. They may also present with other factors that have an impact beyond the obvious burn injury.

The Australian and New Zealand Burn Association has identified a number of burn injury characteristics and co-morbidities that require referral to a Burn Service (see Table 12.1).

All patients with these injuries and co-morbidities should have early consultation with a Burn Service. If there are local resources that are appropriate, some patients may not need transfer, but generally, patients fulfilling any of the criteria listed in Table 12.1 will need transfer. If the patient has a pre-existing condition that could make management more difficult or the risk of injury greater, a specialised team is needed to maximise the likelihood of a good outcome.

Those patients with concurrent trauma should be admitted to a Burn Service or a trauma service depending upon the severity of the associated trauma and the seriousness of the burn. The decision is based on the clinical findings at the time of emergency assessment and discussion between the local trauma team and the Burn Service personnel. If the associated trauma poses the greater immediate risk the patient may initially be treated in a trauma unit until stable, prior to transfer to the Burn Service. Burn care must be provided concurrently and transfer arranged after the patient has recovered from the immediate effects of multiple trauma. Should the burn injury present the dominant threat to morbidity or mortality then primary transfer to the Burn Service is indicated. The priorities are a matter for medical

judgement and should be discussed by the referring doctor, the burn specialist and the trauma or intensive care specialist.

S – Size	>10% TBSA (adult)
	> 5% TBSA (child)
	> 5% TBSA full thickness (in any age)
P – Person	Pre-existing illness
	Pregnancy
	Extremes of age
A – Area	Face / Hands / Feet / Perineum / Major joints
	Circumferential (limb or chest)
	Lungs (inhalation)
M – Mechanism	Chemical / Electrical
	Major trauma
	Non-accidental (including suspected)

TABLE 12.1 – ANZBA referral criteria

Patients at the extremes of age (both young and old) pose particular challenges. Their pathophysiological responses are less predictable and benefit from the experience and expertise of a specialised team.

The burn team approach, of bringing together doctors, nurses, physiotherapists, occupational therapists, psychiatrists, psychologists, social workers, speech pathologists and dietitians in a management team has a significant and beneficial effect on the outcome of major burn injuries.

Communication (3–7)

To assist the transfer process a standardised communication framework is recommended. There are multiple variations of ISBAR (Identify, Situation, Background, Assessment, Recommendation) in use, but all share a common goal of formalising the transfer of important clinical information in a time efficient manner. In addition the ISBAR framework can be used for virtually any form of clinical handover. An example as it applies to a burn referral is shown in Table 12.2.

I - Identify	**Referring clinician** (you) – designation, location (± contact) **Receiver** – confirm person receiving information (designation, location) **Patient** information (name, hospital identification & DOB (age) *Not just patient, so if contact is lost, referring or receiving service can re-establish contact.*
S - Situation	State the immediate clinical situation and level of concern Summary of injuries / problems
B - Background	***Burn injury*** – 'E' in AMPLE - how / what / where & when did the injury occur ***Patient*** – 'AMPL' in AMPLE ALSO social issues/care and protection concerns
A - Assessment	Assessment / summary of **Primary & Secondary Survey** Assessment / summary of **Burn** – size & depth '**Vitals**' – HR, BP, RR, SaO_2, temperature & urine output
R - Recommendation	Decide best place for care – typically transfer to Burn Service Discuss transfer procedure - mode, timing, who is responsible (referrer or receiver) - escort, family members & other supports Treatment advice - resuscitation, wound care, escharotomy (? prior to transfer) Preparation for transfer - Circulatory… IV access & fluids - Urinary catheter - Pain… IV narcotics - G-I system… gastric drainage - Wound… wash & cover / tetanus *PREVENT HYPOTHERMIA*

TABLE 12.2 – ISBAR Framework

Influence of Geographic Situation (8)

Urban Areas

For burn injuries that occur in cities that have an established Burn Service, patients requiring hospital admission should be transported to that service without unnecessary delay after a primary survey has been conducted, so that resuscitation and definitive care can begin as soon as possible. The only exception to this rule is in those cases that require immediate life-saving intervention, such as endotracheal intubation.

When transport to a Burn Service can occur within an hour, unnecessary delays to begin intravenous fluid resuscitation may not be in the patient's best interests. Nevertheless, significant delays can occur in 'short transports', necessitating fluid resuscitation to be established prior to transport to avoid the development of burn shock. This is of greater significance in children and the elderly, as long delays in beginning fluid resuscitation can compromise care and outcome. Advice from retrieval experts may assist in this decision.

Rural and Remote Areas

In rural and remote areas, because of distance and sparse facilities, and also because of logistical challenges it may not be possible to transfer the patient immediately. Patients may be transferred by road, aeroplane or helicopter or any combination of these modes. The use of telemedicine facilities may also assist in decision making by allowing the expertise of the Burn Service to be virtually present.

It may occasionally be necessary to treat patients in these areas for extended periods of time. In these circumstances it is the responsibility of the local treating staff to liaise with the staff at the Burn Service regarding appropriate management to ensure the patient is in the best possible condition when transfer is possible.

Only in the most exceptional circumstances are the patient's best interests served by continuing treatment at a local or district hospital. Sacrificing good physical care for the perceived advantages of 'keeping the family together' is detrimental, particularly as the emotional care of the burn injured patient is as specialised and as important to their long-term outcome as the physical care. All Burn Services recognise this need and have facilities for relatives to stay as well as personnel trained to help both patient and family achieve an optimal emotional outcome.

Preparation for Transfer (2,9)

Patients who are physiologically stable are capable of safe transfer over long distances, even after massive injury. It is therefore essential for the patient to be stabilised prior to starting their journey. Preparation of the patient for transfer should follow the EMSB structure (see Chapter 2):

- Primary survey – ABCDE
- FATT
- Secondary survey
- Wound management (including completion of first aid)
- PREVENT HYPOTHERMIA
- Documentation
- Tetanus cover
    - Australia https://immunisationhandbook.health.gov.au
    - New Zealand: https://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/new-zealand-immunisation-schedule

Transfer Processes

Early contact with a Burn Service should be initiated when any patient may need to be transferred. Once the decision to transfer has been made, the receiving Burn Service will be responsible for arranging a bed and the referral team will be responsible for arranging transport. Transfer procedures should be followed in accordance with local protocols.

The method of retrieval is determined by discussion between the referring centre, the retrieval team and the receiving Burn Service.

The responsibility of the referring team is to stabilise the patient and to document the findings of the primary and secondary survey, the burn injury and the care given. Times of events, tests, hourly fluid balance and treatment, including doses and timing of medications, are all important. Ensure copies of all documentation accompany the patient on transfer.

Military aspects of Documentation and Transfer (10,11)

All exercises and operations have Health Support Plans (HSP) drawn up. Remembering that prior preparation prevents all sorts of poor performance, the health practitioner should ensure they have access to a copy and understand the relevant HSP in advance in order to prepare effectively. Effective relationships (and clarity of expectations) with other elements in the health/evacuation chain should be developed as soon as possible, preferably in advance of deployment, and evacuation routes should be tested prior to or immediately on deployment.

The tactical environment can obviously influence emergency management of burns in a multitude of ways. Evacuation of casualties may be delayed by threat, weather or by mission priorities. Evacuation by air, particularly by opportune Aero-Medical Evacuation (AME) flying with doors open, carries a very high risk of hypothermia for burns patients that must be protected against. Cognitively impaired casualties should be disarmed but anyone else on evacuation from a forward environment should be left equipped to fight.

In the austere environment the value of good quality information increases exponentially. It may be impossible for the Destination Medical Facility (DMF) to reach forward to troops, ships or units in contact or on patrol. It is vital therefore to ensure good clinical information moves with the casualty and this may take many forms, from field treatment cards to pages torn from a notebook. As always, a picture paints a thousand words and the IT/comms systems in many deployed environments can facilitate transmission of images of injuries or diagrams of surface burns.

Tactically, casualties may be identified only by call-signs until they are formally identified at the DMF. Immediate casualty management at the DMF can be tailored to the casualty if good information is received prior to the casualty's arrival. Evacuation is activated on receipt of a 9-liner but this is essentially only logistic information. MIST (Mechanism, Injury, Signs/Symptoms, Treatment) is a pro-forma for rudimentary but efficient transmission of clinical information. Personnel at point of injury should transmit (push) a MIST as soon as possible after a 9-liner, and if health practitioners at the DMF do not receive a MIST shortly after a 9-liner they should endeavour to reach forward for (pull) that information.

Summary

- *Patients with any burn injury that meets the **ANZBA Criteria for Burn Service Referral** (Table 12.1) should be assessed and stabilised while referral is initiated.*
- *Early contact with the receiving Burn Service is essential for adequate preparation and preparedness.*
- *Retrieval and placement are the responsibility of the referral unit. The retrieval team will provide help in the stabilisation of the patient.*
- *Thorough documentation is essential for the successful transfer of care from the referring hospital to the Burn Service.*

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CHAPTER 13

Burn disasters

Introduction

A disaster is defined as a situation in which the clinical need of a patient outweighs the resources available resulting in an adverse outcome. This can occur with any number of simultaneous admissions or when additional admissions overwhelm a health service already running at capacity.

The EMSB course allows clinicians to systematically assess, triage, stabilise and transfer a burn patient. It facilitates communication between clinicians with standardised communication, classification and framework for burn care in the acute setting which is essential for disaster preparedness.

Burn Disaster planning / Preparedness

Burn societies such as ANZBA and the International Society of Burn Injuries recommend that individual countries have their own disaster planning system to address its own particular needs.

To effectively write a burn centre disaster plan, it is critical to understand the framework in which it will operate. Plans need to be made in consultation with other services within individual hospitals as well as at a local, regional, and national level to ensure they complement each other. Plans should reflect the manner in which government agencies are able to work together to respond to events. Plans should be robust and flexible in nature.

Table-top exercises are a useful way to test the feasibility and detail of disaster plans. The Emergo Train System (ETS), for example, is a useful tool. Designed in Sweden it can be used for education, training and simulation of disasters as well as having the ability to evaluate systems and hospital preparedness. Including key stakeholders in any exercise is essential.

Training in disasters for staff is also useful. The Major Incident Medical Management and Support (MIMMS) course provides participants with a systematic approach to medical and healthcare management during a disaster. It provides background knowledge and practical skills for working in a mass casualty situation. People successfully completing the MIMMS course can be placed on a deployment roster to be sent to the field in a disaster situation if required.

Identification of a Burn Disaster

It is essential that there is an indication within the plan which takes into account at what point the plan should be activated. A major disaster resulting in mass casualties is obvious. What is not so obvious is the impact of continued individual admissions ('slow creep') and the effect this has on not only the ability of the Burn Service to provide care, but also the ongoing impact on delivery of other health services due to resource capacity constraints.

Due to the nature of burn injuries and the specialised resources to care for them Burn Services can rapidly become overwhelmed.

A number of points need to be considered when designing a mass casualty plan. Factors such as when to implement a plan, a coordinated approach to triage, the ability to predict resource requirement (including the acute and rehabilitation phase) as well as taking into account the existing workload are all factors to be considered during the development process.

Transport and Allocation of Patients

Following a major burn disaster one of the priorities is to transport patients in a timely fashion and ensure the appropriate distribution of patients across Burn Services to fit the available resources. Depending on the number of patients and the availability of local resources patients may be distributed across multiple jurisdictions. Patients not able to be accommodated in a Burn Service hospital may be supported by burn clinicians as appropriate. Patients with inhalation injury only and no cutaneous injury may be managed in an Intensive Care Unit and not require Burn Service input.

Ongoing Implications

The management of an incident involving multiple complex burn patients will have an impact on facilities long after the initial event. Patients are likely to require ventilation for prolonged periods of time, multiple trips to the operating theatre over weeks to months, rehabilitation services over months to years as well as prolonged and intensive use of resources. Forward planning needs to take these implications into account.

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Neurological Assessments

AVPU

Alert	Patient aware of examiner, respond to environment around them spontaneously, follows command, opens eyes spontaneously and track people and objects
Verbally responsive	Patient's eyes do not open spontaneously, opens in response to a verbal stimulus towards them. Patient is able to react to stimulus directly and in a meaningful way
Painfully responsive	Patient's eyes do not open spontaneously, opens only in response to a painful stimulus. Patient may move, moan or cry out in response to pain.
Unresponsive	Patient does not respond spontaneously, neither to verbal or painful stimuli

Adult Glasgow Coma Scoring

	RESPONSE	**SCORE**
Eye Opening	Spontaneous	4
	To name	3
	To pain	2
	None	1
Best Verbal Response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Best Motor Response	Obeying	6
	Localizing	5
	Withdrawal	4
	Abnormal Flexion	3
	Extension	2
	None	1
		15

Severity of Head Injury

Severe	GCS < 9
Moderate	GCS 9 – 12
Minor	GCS 13 – 15

Tetanus Protocol

Guide to tetanus prophylaxis in wound management [139] (p414)

History of tetanus vaccination	Time since last dose	Type of wound	DTPa, DTPa-combinations, dT, dTpa, as appropriate.	Tetanus immunoglobulin* (TIG)
≥3 doses	<5 years	All wounds	NO	NO
≥3 doses	5–10 years	Clean minor wounds	NO	NO
≥3 doses	5–10 years	All other wounds	YES	NO
≥3 doses	>10 years	All wounds	YES	NO
<3 doses or uncertain †		Clean minor wounds	YES	NO
<3 doses or uncertain †		All other wounds	YES	YES

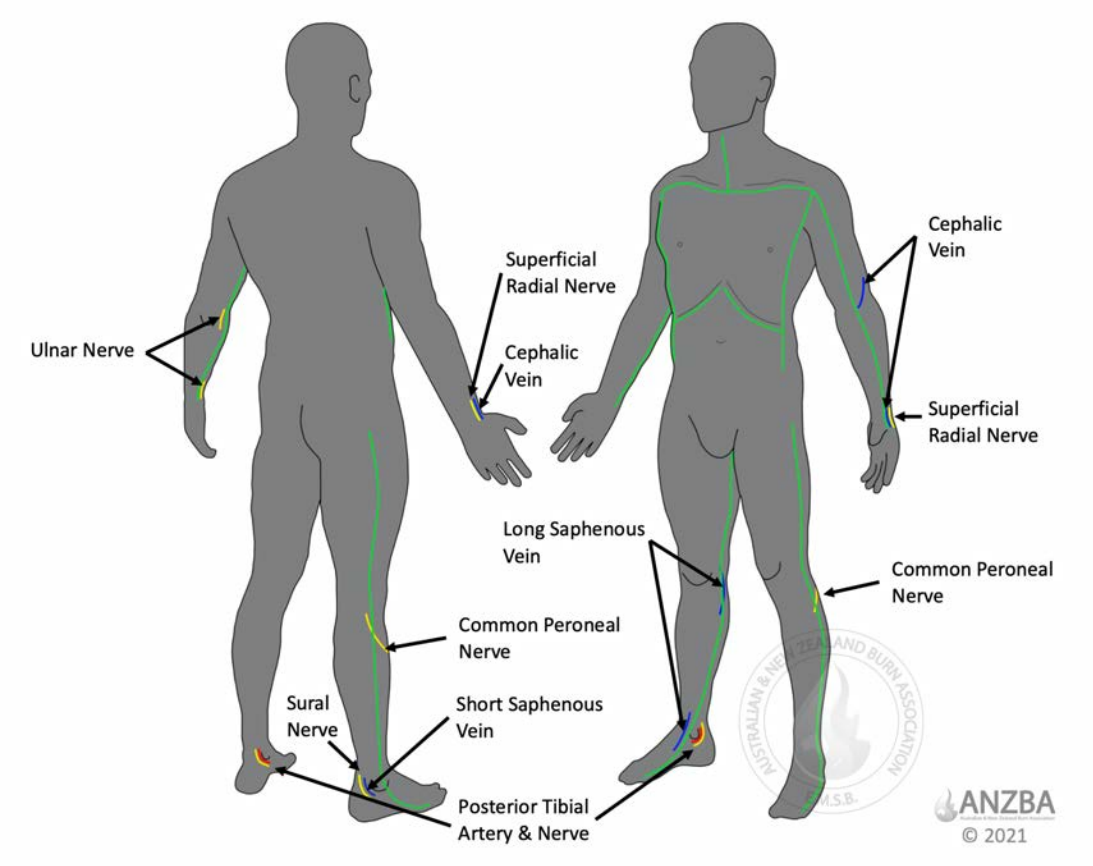
The recommended dose for TIG is 250 IU, given by IM injection using a 21 gauge needle, as soon as practicable after the injury. If more than 24 hours has elapsed, 500 IU should be given.

† Individuals who have no documented history of a primary vaccination course (3 doses) with a tetanus toxoid-containing vaccine should receive all missing doses. See Section 1.3.5, Catch-up.

Immunisation schedules:

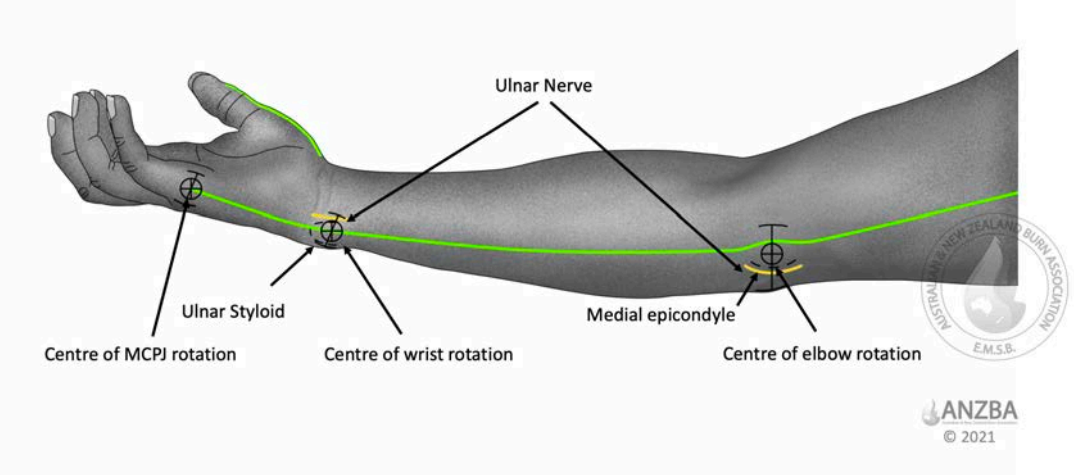
- Australia https://immunisationhandbook.health.gov.au
- New Zealand: https://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/new-zealand-immunisation-schedule

Recommended Escharotomy Incisions



Recommended escharotomy incision lines highlighted in green.

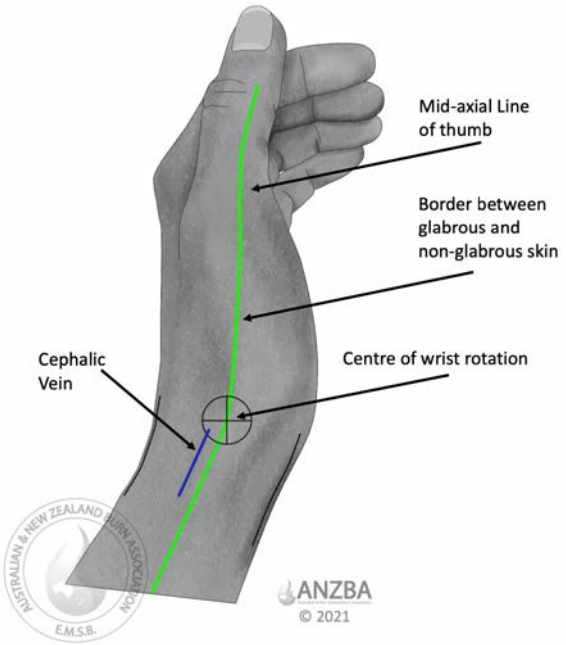
Escharotomy incisions on the chest should run along the anterior-axillary line and are joined superiorly at the level of the clavicle, and below at the subcostal margin. Note that these can be moved to the mid-axillary line to avoid the breast.



Medial Upper Limb

Escharotomy incision follows the mid-axial line which passes through the centre of rotation of the elbow, wrist and metacarpal-phalangeal joint (MCPJ) **EXCEPT** at the elbow where it should deviate more anteriorly to avoid the ulnar nerve.

Note that the escharotomy does **NOT** extend onto the ulnar border of the little finger. The escharotomy incisions for the thumb can also be seen in this view (details below)

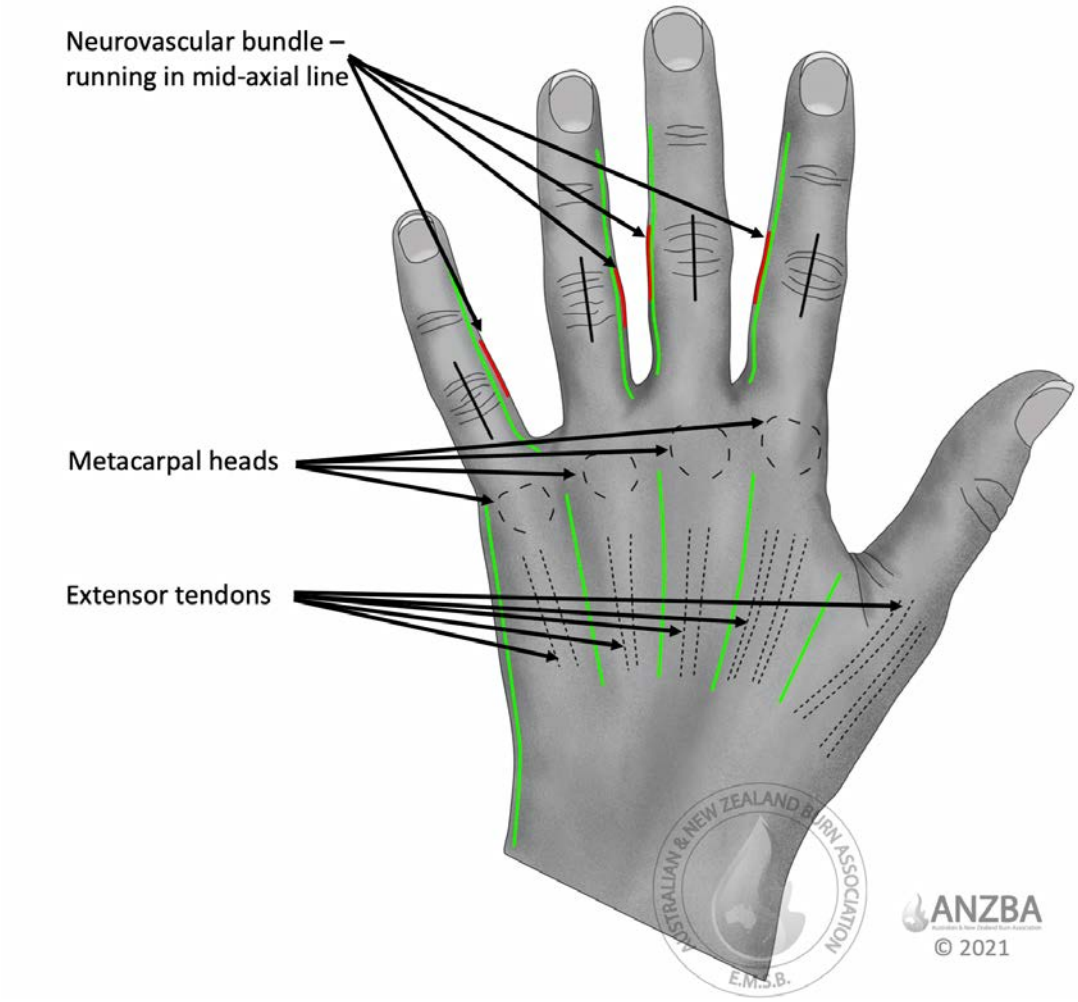


Radial Border of Hand / Thumb and Distal Forearm

Escharotomy incisions on the radial border of the forearm extends from the mid-axial line (passing through the centre of wrist rotation) and then along the border between the glabrous and non-glabrous skin of the thenar eminence and then along the mid-axial line of the thumb itself.

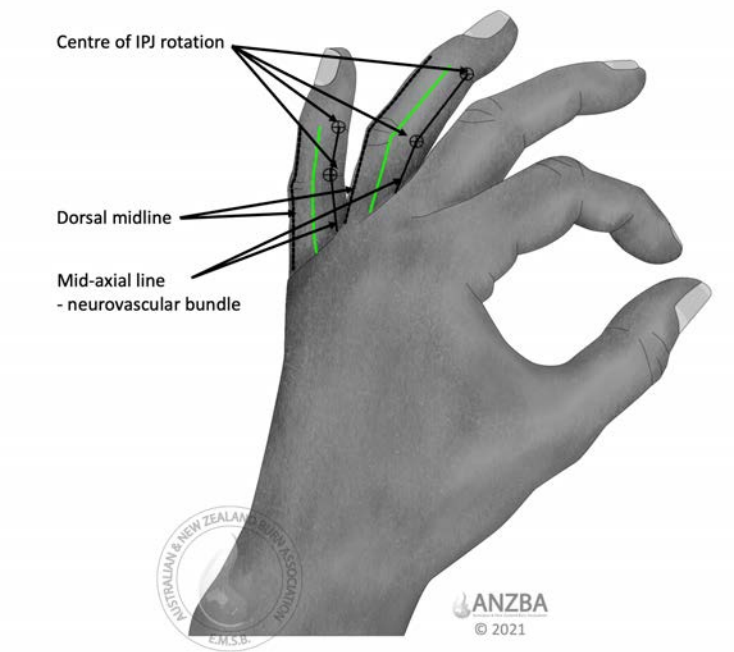
Note that the digital vessels to the thumb, unlike the fingers, does not run in the mid-axial line.

Dorsum of the Hand



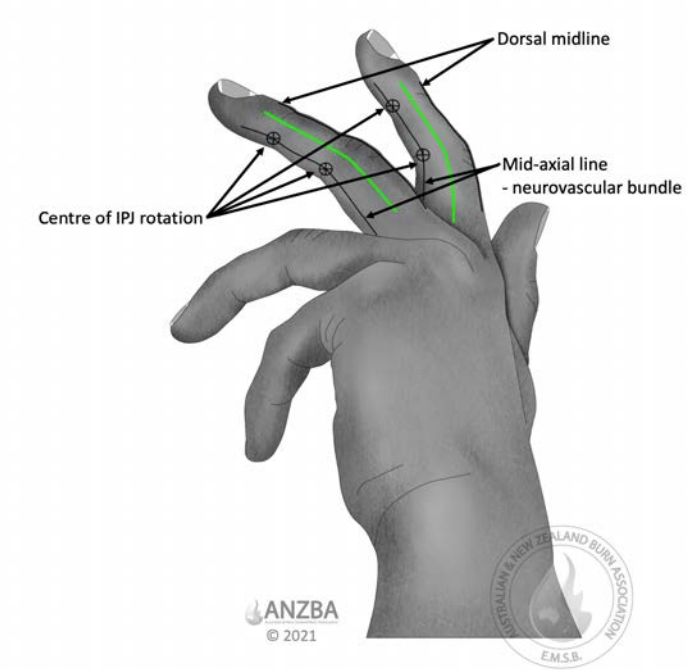
Escharotomy incisions on the dorsum of the hand run in the intermetacarpal space.

Note the escharotomy incisions on fingers is on the 'non-working border' of the digits (detail below). The escharotomy incision for the thumb is seen above in 'Radial Border of Hand / Thumb and Distal Forearm'.



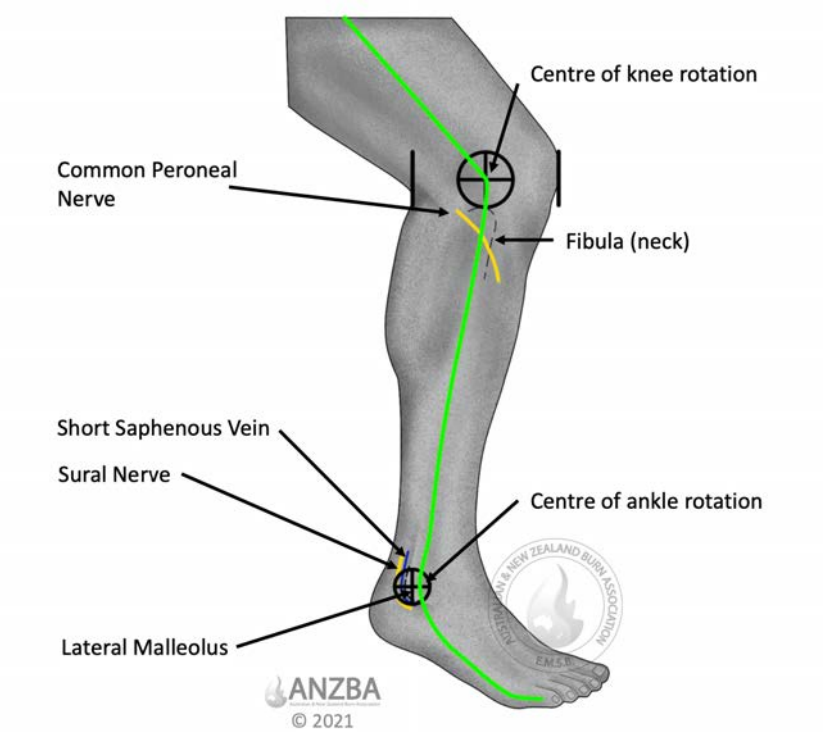
Fingers

Escharotomy incision for the little and ring fingers are placed on the 'non-working' border of the digits (radial) and are dorsal to the neurovascular bundles which run along the mid-axial line of the fingers (i.e. through the axis of rotation of the inter-phalangeal joints (IPJ). It is suggested they are place halfway between the 'top' / dorsal midline and the mid-axial line.



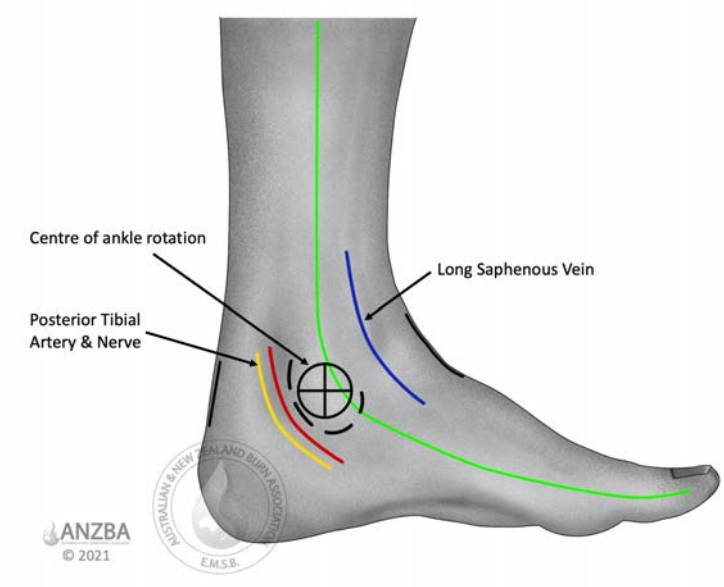
Index and Middle Fingers

Escharotomy incision for the index and middle fingers are placed on the 'non-working' border of the digits (ulnar) and are dorsal to the neurovascular bundles which run along the mid-axial line of the fingers (i.e. through the axis of rotation of the inter-phalangeal joints (IPJ). It is suggested they are place halfway between the 'top' / dorsal midline and the mid-axial line.



Lateral Lower Limb

Escharotomy incisions run along the mid-axial line, passing through the centre of rotation of the knee and ankle **EXCEPT** at the ankle where it passes anterior to the lateral malleolus to avoid exposing bone. At the level of the fibula beware of the common peroneal nerve as it passes around the neck of the fibula in a relatively superficial plane.



Medial Ankle

Escharotomy incisions run anterior to the mid-axial line to avoid exposing the bone. By passing anteriorly, the long saphenous vein is potentially at risk as it is superficial.

Selecting an Appropriate Dressing

Wound Care Product *What?*	Function *Why?*	Indications *When?*	Application *How?*	**Note / Precautions**
Silicone/foam • **Hydrophilic polyurethane foam + soft silicone layer +/- waterproof outer layer** **Also available with silver**	• Non-adherent • Conformable	• Superficial burns	• Cover with fixation/retention dressing	• **Do not use if any infection (unless using silver version)**
Hydrocolloid • **Hydrocolloid wafer**	• Aids autolysis of devitalised tissue • Provides moist wound environment • Absorbs exudate	• Superficial to mid dermal burns • Low to moderately exudating wounds	• Allow 2–5cm margin around wound. • Can remain intact 2–3 days • Wafers up to 5 days if no signs infection.	• **Do not use if any infection**
Impregnated Gauze • **Petroleum-impregnated gauze**	• Antiseptic dressing • Conformable	• Dermal thickness burns • Grafts & donor sites	• 2–3 layers for acute wounds to avoid adherence to the wound bed • Cover with secondary dressing • Change every 1–3 days	• **Soak off if adhered to wound bed**
Silver (eg Aquacel Ag™) • **Sodium carboxymethycellulose (CMC) & 1.2% ionic Ag in fibrous material** **Also Contreet H**	• Broad spectrum antimicrobial • Facilitates debridement • Absorbs exudate	• Mid to deep dermal thickness burns • Moderately exuding wound	• Allow 2–5 cm overlap to allow for shrinkage • Cover with secondary dressing • Review 7–10 days • Aim to leave primary dressing in place until healed	• **Exudate level indicates frequency of dressing change**
Silver (eg Acticoat™) • **Nanocrystalline Ag coated mesh with inner rayon layer.**	• Broad spectrum antimicrobial protection • Decreases exudate formation	• Dermal to full thickness burns • Grafts & donor sites • Infected wounds	• Wet with H_2O; drain and apply either side down • Moistened secondary dressing • Replace 3–4 days (Acticoat) or 7 days (Acticoat 7)	• **Temporary skin staining** • **Avoid if allergy to silver** • **Avoid hypothermia**
Silver (eg Flamazine™) • **Silver Sulphadiazine 1%**	• Reduces infection	• Infected wounds • Dermal to full thickness burns if only available option	• Apply generous amount to sterile handtowel to ease application • Apply to wound • Cover with secondary dressing	**Not recommended for most burns due to changes to wound bed appearance and frequency (daily) of required dressing changes**

- Wounds should be assessed regularly to ensure progress is being made as expected and product choice altered accordingly if required.
- Clinical photographs are a useful way to track and record individual patient progress. Consider discussing patient with your local burn service for guidance/ongoing follow up.

Referral & Transfer

SPAM

SIZE	PERSON	AREA	MECHANISM
>10 TBSA Adult	Pre-existing illness	Face / Hands / Feet Perineum / Major Joints	Chemical / Electrical
>5% TBSA Child	Pregnancy	Circumferential (*chest or Limb)*	Major Trauma
>5% TBSA Full Thickness	Extremes of age	Lungs (*Inhalation)*	Non Accidental Injury

*Note: Fulfilment of **any** of the above criteria is sufficient to warrant referral to a Burn Service.*

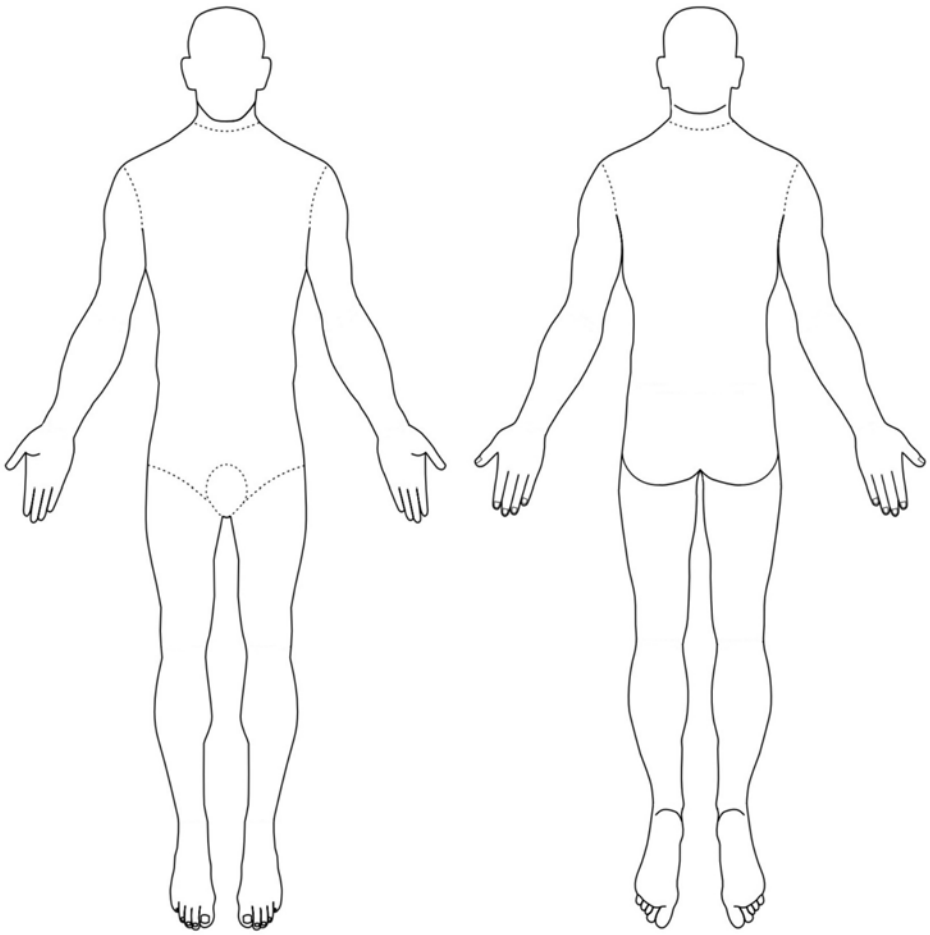
ISBAR

I - Identify	**Referring clinician** (you) – designation, location (± contact) **Receiver** – confirm person receiving information (designation, location) **Patient** information (name, hospital identification & DOB (age) *Not just patient, so if contact is lost, referring or receiving service can re-establish contact.*
S - Situation	State the immediate clinical situation and level of concern Summary of injuries / problems
B - Background	***Burn injury*** – 'E' in AMPLE - how / what / where & when did the injury occur ***Patient*** – 'AMPL' in AMPLE ALSO social issues/care and protection concerns
A - Assessment	Assessment / summary of ***Primary & Secondary Survey*** Assessment / summary of ***Burn*** – size & depth '***Vitals***' – HR, BP, RR, SaO_2, temperature & urine output
R - Recommendation	Decide best place for care – typically transfer to Burn Service Discuss transfer procedure - mode, timing, who is responsible (referrer or receiver) - escort, family members & other supports Treatment advice - resuscitation, wound care, escharotomy (? prior to transfer) Preparation for transfer - Circulatory… IV access & fluids - Urinary catheter - Pain… IV narcotics - G-I system… gastric drainage - Wound… wash & cover / tetanus *PREVENT HYPOTHERMIA*

EMSB TBSA Chart - Rule of Nines

Adult	9%	Head & Neck; Arm [shoulder - fingertip]
	18%	Anterior trunk; Posterior trunk; Leg [groin - toe]
	1%	Perineum
Child	9%	Arm [shoulder - fingertip]
(0 – 9 yrs)	18%	Head & Neck; Anterior trunk; Posterior trunk
	14%	Leg [groin – toe]

[For each year of life, take 1% from the head and add 0.5% to each of the legs
At 9 years, the 1% becomes the perineum]



T.B.S.A. BURNED

Head	_____
R) arm	_____
L) arm	_____
Ant trunk	_____
Post trunk	_____
R) Leg	_____
L) Leg	_____
Genitalia	_____
TOTAL	_____